

THE
WEST RIDING LUNATIC
ASYLUM

MEDICAL REPORTS.

EDITED BY

J. CRICHTON BROWNE, M.D., F.R.S.E.

VOL. IV.

LONDON:
SMITH, ELDER, & CO., 15 WATERLOO PLACE.
1874.

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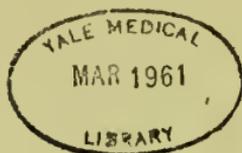
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'He that does not know that the human mind is organised, or that it is a spiritual organism, bounded or closed by a natural organism, in and according to which the mind produces its ideas, or thinks, must needs imagine that perceptions, thoughts, and ideas are only so many radiations and variations of light, entering by influx into the head, and presenting forms which a man sees and acknowledges as reasons and arguments: hut this is an idle imagination, for it is universally acknowledged that the human head is filled with brains, that the brains are organised substances, that the mind has its abode in them, and that its ideas are therein fixed and become permanent according to their reception and confirmation. You will ask, possibly, What is the nature of that organisation? I answer, It is an arrangement of the whole into series, as into fascicles or hundles.'

SWEDENBORG.



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P R E F A C E .

THE West Riding Lunatic Asylum Medical Reports, which were commenced, not without difficulty and discouragement, four years ago, are now securely established as an Annual Series. They have at least partially realised most of the purposes with which they were originated, and have met with a measure of approval ampler than was anticipated.

The Editor is under deep obligations to the contributors, who have made his task easy, and have supplied him with an amount of material greater than he has been able to utilise. Five papers, of much practical importance, have been, with sincere reluctance, excluded from the present volume.

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ON THE
PHYSIOLOGICAL IMPORT
OF
DR. FERRIER'S
EXPERIMENTAL INVESTIGATIONS INTO
THE FUNCTIONS OF THE BRAIN.

*An Address delivered at the Annual Medical Conversazione at the West Riding
Lunatic Asylum, Wakefield, November 25, 1873.*

BY WILLIAM B. CARPENTER, M.D., LL.D., F.R.S.

CORRESPONDING MEMBER OF THE INSTITUTE OF FRANCE.

IN complying with the wish strongly expressed to me by Dr. Crichton Browne, that I should lay before the large body of Medical men whom he annually assembles at his hospitable gatherings, my view of the scientific import of the results of Dr. Ferrier's very remarkable experiments, the earlier portion of which had been prosecuted at the Wakefield Asylum, I thought only of presenting to my audience what seemed to myself the Physiological bearing of such of those results as, from what I had heard and seen of them, I considered Dr. Ferrier to have established beyond reasonable question.

The time I can spare from Official duty has been so completely engrossed for several years past by other studies, that I could not presume to enter into such a critical examination of these results as would be required to satisfy Physiologists who have paid special attention to this department of inquiry. I can only say that none of the criticisms to which

they have been subjected appear to me to affect the positive fact (which can be vouched for by any number of competent witnesses), that Dr. Ferrier can *predict* with almost positive certainty the movements he will call forth by the localised stimulation of certain parts of the Cerebral convolutions; and that very dissimilar movements follow the application of the stimulus to points nearly adjacent. The definiteness of this localisation seems to me to negative the hypothesis that the movements proceed from the radiation of the electric currents, though these can be clearly proved to extend themselves over the neighbouring cerebral surface. And the fact that other experimenters have not obtained the positive results which Dr. Ferrier has over and over again publicly exhibited, merely shows, in my opinion, that they have not succeeded in obtaining the precise conditions which are essential to the success of the experiments.

As, for the reason already mentioned, I now scarcely venture to call myself a Physiologist, and as my Address was delivered without the slightest view to publication, I should not have consented to its appearance in these Reports, but for the earnest desire expressed by Dr. Crichton Browne to that effect.

At a time when we are hearing so much of our backwardness in Scientific research, as compared especially with our German *confrères*, it is gratifying to reflect that the greatest advances in the Physiology of the Nervous System which have been made during the last fifty years, have been chiefly the work of British investigators. It was Sir Charles Bell who first established the difference between the *Sensory* and the *Motor* nerves; it was by Dr. Marshall Hall that the *Reflex action of the Spinal Cord* was first distinctly demonstrated; Dr. Waller, contemporaneously with MM. Claude Bernard and Brown-Séquard (the latter of whom is half an Englishman), defined the *Vaso-motor* action of the Sympathetic system; and to these must now be added Dr. Ferrier, the remarkable results of whose Experiments upon the Brain it is my present object to discuss. In order that these results may be clearly understood, it is necessary first to

summarise what modern research had previously done to elucidate the elementary structure of the Nervous System, to determine the conditions of its action, and to define the relative functions of its different parts.

I need scarcely remind you of the now well-established distinction between the two components of the Nervous System, the fibrous or *tubular*, and the ganglionic or *cellular*. The central core of each nerve-fibre is a thread of *protoplasmic* substance, analogous to that of which the whole bodies of many of the lowest animals are composed. This central thread seems to be an intensified protoplasm of wonderful vitality and power, and to be the essential part of the fibre; the cylinder of fatty matter which surrounds it, and the membranous envelope outside, probably acting as insulators, like the gutta-percha or other insulating material round our telegraph wires. Each of the telegraph cords that cross our streets, holds about sixty wires; and every wire has its separate origin and termination. As with these wires, so with the nerve-fibres; each has its separate origin and termination, and must therefore be insulated, in order that it may be functionally independent of those with which it is bound up in the same nerve-trunk. In the nerve-trunks of the *Comatula* (feather-star), which I have carefully examined, I find no such insulation, there being no call for functional independence; for all the threads that proceed to the successive pairs of flexor muscles along each arm have the same function, namely, to produce the coiling-up of the arm, which is uncoiled by elastic ligaments as soon as the muscles are relaxed. In the ultimate distribution of the nerve-fibres of even the highest animals, whether to the skin or to the muscles, the central thread of each fibre escapes from its envelopes, and breaks up into finer fibrillæ, which inosculate with each other so as to form a network, very much resembling that formed by the protoplasmic pseudopodia that extend themselves from the bodies of Rhizopods. In the ganglionic centres the nerve-fibres come into continuity with *nerve-cells*, of which they may be considered as extensions. These cells appear to be the chief sources of power, standing to the fibres much in the same relation that the galvanic

battery stands to its conducting wires. } And in the retina of the eye, as also in the internal ear, we find the peripheral terminations of the fibres coming into relation with nerve-cells, in which it appears that the first changes take place which are concerned in the reception of sensory impressions. The grey or cellular Nerve-substance lies in the midst of an extremely close network of capillary Blood-vessels; whilst the supply of blood to the white or fibrous substance is far less copious.

Nerve-force closely resembles Electricity in its mode of transmission, and is obviously dependent, like galvanic electricity, upon Chemical change; but there are many points in which it differs from Electricity, particularly in its dependence upon the living condition of the Nerve-substance. That condition can be kept up only by an active Blood-circulation. Hence the Brain of Man, though not having more than one-fortieth of the weight of the entire body, receives about one-fifth of the whole mass of his blood; and by far the larger part of this fifth goes to the thin layer of grey matter which forms the cortical layer of the Cerebrum, and to the ganglionic centres at its base. The Blood serves two purposes. Its Nutritive materials enable the Nerve-substance to form itself, and thus to store up a vast amount of *potential energy*; while its Oxygen, by entering into new combinations with that nerve-substance, converts its potential into *actual* Nerve-force. In the ordinary state of the Nervous System, the battery (so to speak) may be considered as moderately charged, ready to send a Nerve-current whenever the circuit is completed; but if the supply of blood be excessive, the tension of the charge increases to such a degree that the discharge is effected spontaneously, as in the convulsions of Tetanus.

All Nervous activity is immediately and directly dependent upon a due supply of Oxygenated Blood; and this blood-supply is regulated by the Vaso-motor system of nerves. A complete interruption of the circulation through the Brain produces immediate and complete insensibility, sensibility returning as soon as the Blood is re-admitted; while a reduction in the Blood-supply appears to be the essential condition of Sleep. During our waking hours we are constantly using

up our *potential energy* faster than it can be re-supplied by nutrition; during sleep, on the other hand, the limited supply of Blood-plasma suffices for nutritive regeneration, whilst the reduction of Oxygen prevents conversion of the *potential* into *actual* energy. Sir A. Cooper experimented on an animal in full vigour, by tying two of the four great arteries by which blood is conveyed to the Brain, thus diminishing, but not breaking, the supply; he then compressed the other two, and the animal fell as if shot, with an entire suspension of consciousness, which returned immediately that the pressure was removed. Dr. Fleming, by compressing the two carotids, fell asleep for two minutes, and awoke believing he had slept two hours. Thus also patients in Hysteric coma, which probably depends upon a spasmodic constriction of the arteries of the Brain, will become suddenly unconscious while in the middle of a sentence, and, after remaining insensible for from five to twenty minutes, will suddenly come to themselves and complete the sentence. So, again, it is well known that a deficiency of Oxygen in the respired air enfeebles the Nervous power. The rarefaction of the air on the higher Alpine summits is a great drawback to the physical endurance of the mountaineer, who is obliged to stop very frequently to take breath.¹ And the continued breathing of a vitiated atmosphere dulls both the Intellect and the Moral Sense.

In tracing the gradual development of the Nervous System in the Animal series, we may begin with the very simple condition under which it presents itself in the Ascidian Mollusks. We find in one of these animals only a single *ganglionic centre*, having *afferent* nerves which proceed towards it from a fringe of sensitive filaments surrounding the entrance of the respiratory sac, and *motor* fibres issuing from it to the muscular walls of that sac. Any unsuitable particle which enters with the current drawn in by ciliary action, encountering the sensitive filaments, produces an impression which is transmitted by their afferent nerves to the

¹ Prof. Bert, of the Collège de France, has lately proved experimentally that the extremest *mal des montagnes* is at once relieved by the inspiration of air highly charged with oxygen. (*Comptes Rendus*, March 30, 1874.)

ganglionic centre; and a 'reflex action' is thus excited, which transmits an impulse through the motor nerves, and calls forth a sudden contraction of the muscular walls of the respiratory sac, whereby a gush of water is projected from its entrance, carrying the offensive particle to a distance; just as the act of coughing in Man, when excited by the passage of a crumb of bread or a drop of water 'the wrong way,' tends to drive it forth by an explosive blast of air.

Reflex actions of a much more complicated nature, and commonly characterised as 'instinctive,' are performed by Centipedes, Insects, &c.; in which we find a ganglion for every segment of the body, connected by fibrous cords with the ganglia of other segments, so as to form a chain, having at its anterior extremity the ganglia of Special Sense, by which (especially by the Optic ganglia) the independent actions of the segmental centres are harmonised and controlled. This chain of ganglia corresponds in function to the Spinal Cord of Vertebrate animals, with its upward prolongation into the cranium (*medulla oblongata*), which is the ganglionic centre of all the reflex movements of Respiration. Experiments on the lower animals, with observation of the phenomena of disease in Man, make it certain that, however the reflex actions of the Spinal Cord may seem to show a purpose (as when a decapitated frog tries to wipe off with one leg an irritant applied to the other), they may be performed *without sensation*. Hence such actions are distinguished as 'Excito-motor.' The higher forms of reflex action, however, require the guidance of sensation, and these are distinguished as 'Sensori-motor.' How purely *automatic*, even in Man, those movements may be, which, originally dependent on the Will, have come to be habitual, is shown by their continuance when the attention is entirely absorbed by an internal train of thought; the body of a philosopher, who is mentally working out some profound question, being carried along his accustomed track by the reflex action of his legs—each contact of the foot with the ground exciting the next movement,—so that he arrives at his destination without any knowledge of what brought him there.

In Vertebrate animals, the Cerebrum, superposed upon the

Axial Cord (under which term I include the Spinal Cord and the Sensory ganglia at its summit), may be regarded as the instrument of all those actions that deserve the title of *intelligent*; as involving the formation of 'ideas' and 'emotions,' and as being obviously directed to carry out a preconceived purpose. But animals that are simply governed by ideas and feelings are to be regarded as 'thinking automata;' and it is only when the action of these is controlled and regulated by the Will, as in the fully developed Human being, that the highest form of Intelligence can be properly said to exist. It appears to me an unmistakeable deduction from the facts of Comparative Anatomy, that the Cerebrum is a *superadded* organ, and not, as is commonly supposed, the fundamental part of the Brain. This consists of the succession of Ganglionic centres lying along the floor of the skull, which receive all the nerves of Sense, and from which the nerves of Motion take their departure; for it is of such a succession that the Brain of the Osseous Fishes is made up, their Optic ganglia being especially large, and what pass as their Cerebral lobes being probably homologous rather with the Corpora Striata. In addition to the Olfactive, Optic, Auditory, Gustatory, and Respiratory ganglia, the Human Brain includes two pairs of large ganglionic masses, embedded (as it were) in the Cerebral hemispheres, but really forming the summit of the Spinal Cord; into one of these, the *Thalami Optici*, the Sensory tract of the Cord may be traced; whilst into the other, the *Corpora Striata*, the Motor tract may be traced. Hence the two together may be regarded as the ganglionic centre of the nerves of *common sensation* and of the *motor* nerves which they excite to action; and as constituting, with the other ganglia, the *Sensorium commune*, by the instrumentality of which we become *conscious* of external impressions.

From this basal Ganglionic tract, a set of fibres radiate towards the cortical (ganglionic) layer of the Cerebral Hemispheres, which they thus connect with the Sensorium. One set of these fibres may be considered as passing upwards from the Sensory ganglia (especially the *Thalami Optici*), and as conveying to the Cerebral ganglia the effects of sensorial

changes; whilst the others may be considered as passing downwards from the Cerebral ganglia to the Motor centres, especially the *Corpora Striata* and *Corpora Quadrigemina*, through the instrumentality of which the Cerebral changes that minister to Intellectual and Emotional states express themselves in appropriate actions.

That this *Sensorium commune* is independent of the Cerebrum, is proved by the experiment of Flourens on a Pigeon, which has been frequently repeated since with the same result. The bird having been entirely deprived of its Cerebrum, it still turned to the light, raised its head at a noise, &c. I regard it also as the centre of the movements guided by sensations. I have seen the late Mr. J. S. Mill walking through Cheapside, at its most crowded time, with his mind completely engrossed in thinking out his System of Logic, which (he told me) was in great part mentally constructed during his walks between the India House and his residence at Kensington; and yet he never ran against persons or lamp-posts; for while the reflex action of the Spinal Cord maintained his movements, that of the Sensory ganglia directed and guided those movements. If the influence of Sensation passes from the Sensorium to the convoluted surface of the Cerebral Hemispheres, we have distinct perceptions or *ideas* of the objects which excite them; and there also lies the power of *Ideo-motor* reflexion. The Cerebrum furnishes the 'mechanism' of Thought and Feeling; but its reflex action is directed and controlled by the Will, in proportion as the Volitional power is developed.

It has been recently shown that the separate system of isolated ganglia and nerves known as the Sympathetic, has a special power of controlling the calibre of the Blood-vessels; and the nerves which thus regulate the supply of blood to the different organs are termed *vaso-motor*. This is a function of great importance. We see its exercise in the regulation of the blood-supply to Glands, whereby the quantity of their secretion is brought into accordance with the wants of the body, or is influenced by states of mind; the Lachrymal, Salivary, Gastric, and Mammary glands being thus specially affected. And it is probably through these *vaso-motor*

nerves, that the supply of blood to the various parts of the Nerve-centres themselves is regulated. This seems to serve as the key to what are termed *functional* diseases of the Nervous System, and especially to the various forms of what is known as Hysteria, in which there is deficient action of some parts and excessive action of others; these being subject to such sudden and extraordinary variations, that it is clear that no structural changes can be involved. Where the supply of blood to the Sensorial centres is in excess, a state of extreme nervous tension is produced; as we see in the excessive sensibility to impressions, whether physical or mental, that is characteristic of the Hysterical subject. And when this nervous tension reaches a certain point, it *discharges itself spontaneously* in Convulsions, just like an overcharged Leyden jar.—These well-known phenomena seem to me of great importance in the interpretation of Dr. Ferrier's experiments.

Returning to the Cerebrum, we find Comparative Anatomy and the history of Embryonic development to agree in showing that the Cerebrum of a Reptile or Bird is *not* the miniature of that of Man, but represents only its anterior lobes; that in the lower Mammalia the middle lobes sprout, as it were, from the back of the anterior; and that in the Quadrumana and in Man the posterior lobes sprout from the back of the middle. Thus, as it is the *anterior* lobe which is common to all creatures possessing a Cerebrum, while the *posterior* is peculiar to Man and his nearest allies, it is in the former, rather than in the latter, that (if there be any localisation of faculties) we should expect to find those which Man shares with the lower animals. But since the great *forward* development of the anterior lobes is as peculiar to Man and his nearest allies as the *backward* development of the posterior, we should on the same grounds expect that it is in some way instrumental to the higher Intelligence which characterises them.

The surface of the Cerebral Hemispheres in the Rabbit and other Rodents is smooth, or destitute of convolutions; and is formed by a thin layer of 'grey' or 'cortical' substance, composed of nerve-cells lying in the interstices of a very

close reticulation of capillary blood-vessels ; while the ' white ' or ' medullary ' substance of the interior is composed of nerve-fibres, which connect this cortical layer with the Sensorium beneath, and which also pass between the different parts of the cortical layer itself. In ascending the Mammalian series, however, we find the cortical layer folded into convolutions, by which its surface is largely increased, and brought into extended relation with that vascular envelope which supplies it with blood-vessels. The convolutions were formerly supposed to be destitute of regularity ; but the careful study of them by Leuret, Gratiolet, and others has shown that they are disposed on a regular plan, gradually increasing in complexity, which can be traced upwards through the *Quadruman*a to Man. It has been found, moreover, that the convolutions have a greater degree of complexity of arrangement, and differ more in the details of their disposition on the two sides, in the more intellectual races, than in the least advanced ; the *Cerebrum* of a Bush-woman, described by Mr. John Marshall, presenting an arrangement distinctly intermediate between that which characterises the *Cerebrum* of an educated stock, and that which is presented by the higher *Quadruman*a.

It was, until lately, the current doctrine of Physiology, that no stimulation of the *Cerebrum* would excite either sensation or motion ; but it has been recently found that the application of a weak galvanic current to its Cortical layer calls forth movements ;¹ and Dr. Ferrier has used with the best results the more intense but interrupted Faradic current of an induction-coil. The immediate effect of its application to the Cortical substance is to produce not only an afflux of blood, shown by the visible distension of the vessels, but an increased passage of blood through the capillaries, shown by its profuse flow from the great sinuses, which, in the quiescent state of the brain, had ceased to bleed ; and I am disposed to believe that it is the augmented activity of the re-action

¹ This was first ascertained by Hitzig in 1870 ; and the series of experiments which he subsequently prosecuted in conjunction with Fritsch, led them to conclusions which anticipated those of Dr. Ferrier to a greater extent than I was aware of when I delivered my Address.

between the Blood and the Nerve-substance, producing an excessive tension like that of an overcharged Leyden jar, rather than the direct stimulation of the nerve-substance itself, which causes the discharge of Nerve-force that produces movement,—the evidence of this being furnished by the *time* that is often required to call forth the action, as well as by the frequent continuance of action after the stimulation has ceased. This was especially noticed in Dr. Ferrier's first experiments, which were directed to the study of the phenomena of Epilepsy, and in which the application of the two electrodes to points of the Cerebral surface at some distance from each other, excited either partial or general convulsion, the most severe fits being induced when the electrodes were applied at the greatest distance.

'In all cases,' says Dr. Ferrier, 'whether the fits were partial or more general, the immediate antecedent was an excited hyperæmic condition of the cortical matter of the hemispheres. The irritation was entirely confined to the surface of the hemispheres; the electrodes being simply applied, without causing mechanical or deep-seated lesion.' And not only was there in every case a distinct interval between the application of the electrodes and the first convulsive movement; but there was occasionally 'a distinct interval of time *after the withdrawal of the stimulation*, before the condition of the grey matter had reached the pitch of tension requisite for an explosive discharge. This of itself is sufficient to show that the effects were not due to conducted currents or direct stimulation of the motor nerves of the muscles, but to an abnormal excitability or irritability of parts, whose function, it might be inferred, was to initiate those changes which would result in normal contraction of the muscles affected.'

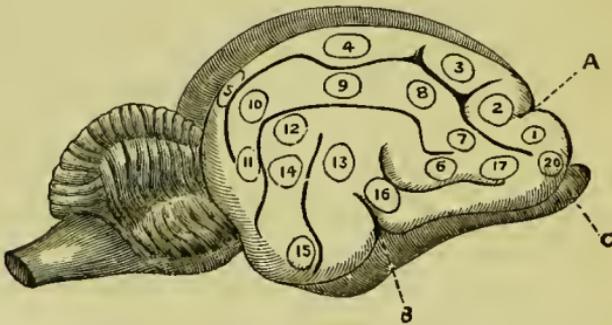
It seems to me that the above principles may be fairly extended to *all* the cases in which local faradisation produced special motor results; for although the *immediateness* of these results in the state of normal excitability might seem to indicate they proceeded from *direct* stimulation of the ganglionic nerve-substance itself, yet, when the excitability is depressed by exhaustion, a distinct interval is perceptible.

And that the 'expressive' movements excited by the localised faradisation really depend (like the epileptic convulsion) upon the hyperæmic state which it induces, seems to be further indicated by their frequent persistence after the discontinuance of the stimulation; a dog, for example, continuing to hold up his head and wag his tail, or to utter cries as of pain (though completely stupified by chloroform), after the withdrawal of the electrodes from the respective centres of these actions. For this could scarcely be the case if the stimulus acted *directly* on the nerve-substance; while it is quite consistent with the fact that the hyperæmic state does not immediately subside.

The general characteristic of the Movements called forth by the local stimulation of the cortical substance of the Cerebrum, is that they are such as involve the co-ordination of several distinct muscular actions; and resemble those which, in an animal in possession of its senses, we should regard as expressive of Ideas and Emotions.

Thus, in a Cat, the application of the electrodes at point 2 (Fig. 1) caused 'elevation of the shoulder and adduction of the limb, exactly as when a cat strikes a ball with its

Fig. 1.



Side View of Brain of Cat :—A, crucial sulcus dividing anterior convolutions; B, fissure of Sylvius; C, olfactory bulb.

paw;' at point 4, 'immediate corrugation of the left eyebrow, and drawing downwards and inwards of the left ear;' at point 5, 'the animal exhibits signs of pain, screams and kicks with both hind legs, especially the left, at the same time turning its head round and looking behind in an as-

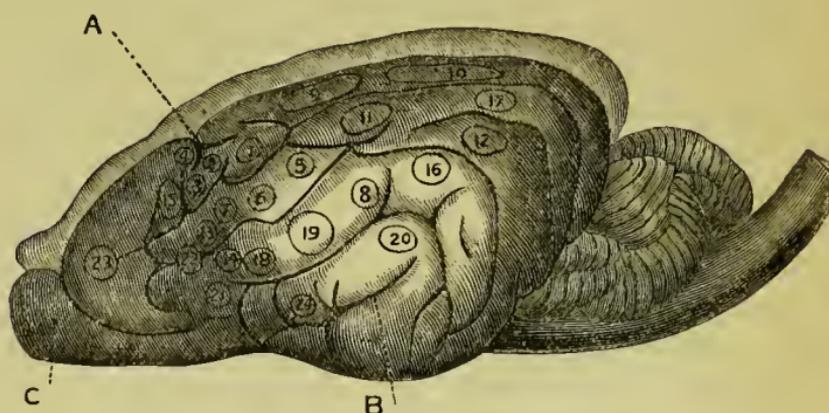
tonished manner;’ at point 6, ‘clutching movement of the left paw, with protrusion of the claws;’ at point 13, ‘twitching backwards of the left ear, and rotation of the head to the left and slightly upwards, as if the animal were listening;’ at point 17, ‘restlessness, opening of the mouth, and long-continued cries as if of rage or pain;’ at point 18 (on the under side of the hemisphere, not shown in the figure), ‘the animal suddenly starts up, throws back its head, opens its eyes widely, lashes its tail, pants, screams, and spits as if in furious rage;’ and at point 20, ‘sudden contraction of the muscles of the front of the chest and neck, and of the depressors of the lower jaw, with panting movement.’ Similar results were so constantly obtained, with variations obviously depending upon the degree of excitability and the strength of the stimulus, that the localisation of the centres of these and other actions was placed beyond doubt; the movements of the paws being centralised in the region between points 1, 2, and 6; those of the eyelids and face between 7 and 8; the lateral movements of the head and ear in the region of points 9 to 14; and the movements of the mouth, tongue, and jaws, with certain associated movements of the neck, being localised in the convolutions bordering on the fissure of Sylvius (B), which marks the division between the anterior and middle lobes of the Cerebrum—the centre for opening the mouth being in front of the under part of the fissure, while that which acts in closure of the jaws is more *in* the fissure.

A similar series of experiments on Dogs gave results that closely accorded with the foregoing; allowance being made for the somewhat different disposition of the convolutions, in accordance with the different habits of the animals; which showed itself in the higher development of the centres for the paw in cats, and for the tail in dogs.

Thus when the electrodes were applied at point 9 (Fig. 2), ‘the tail was moved from side to side, and ultimately became rigidly erect;’ within the circle 10, the application, ‘elicited only cries, as if of pain;’ at point 14 a continued application gave rise to the following remarkable series of actions:—‘It began with wagging of the tail and spasmodic twitching of

the left ear. After the cessation of the more violent spasms, the animal held up its head, opened its eyes wide with the most animated expression, and wagged its tail in a fawning manner. The change was so striking, that I and those about me at first thought that the animal had completely recovered from its stupor. But notwithstanding all attempts to call its attention by patting it and addressing it in soothing terms, it looked steadfastly in the distance with the same expression, and continued to wag its tail for a minute or two, after which it suddenly relapsed into its previous state of narcotic stupor.' The application of the electrodes to

Fig. 2.



Side View of Brain of Dog:—A, crucial sulcus; B, fissure of Sylvius; C, olfactory bulb.

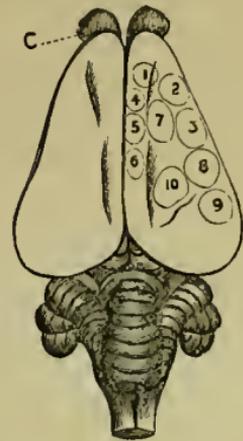
point 21 produced 'drawing back of the head and opening of the mouth, with a feeble attempt at a cry or growl (the animal very much exhausted). Repeated applications of the electrodes to this point and its neighbourhood caused whining and growling noises,' like those which a dog makes in its sleep, and which are supposed to indicate that it is dreaming.

Similar experiments having been made upon Rabbits, the results were again as accordant as it would be fair to expect; especially considering the difficulty in exactly localising the different centres, which arises from the absence of the landmarks afforded by the convolutions. It is curious that in this animal the centres of the mouth-movements seem to be the most highly developed; when these (2, 7, Fig. 3) are

faradised, 'there are munching movements of the upper lip, and grinding of the jaws, as if the animal were eating vigorously.'

Dr. Ferrier has since made a series of experiments on the Monkey; of which the details are as yet unpublished, but which seem to be yet more remarkable than the preceding, in the far greater variety of the movements called forth from different centres, and in their more distinctly expressive character. These results will also be of peculiar interest, on account of the close conformity which the simple arrangement of the convolutions in the Monkey bears to their more complex disposition in the Human Cerebrum. They correspond with those of the previous experiments in this important particular—that those centres of movement which may be regarded as giving expression to mental states that Man shares with animals beneath him, are all located in the *hinder part* of the *anterior lobes* and the *anterior portion* of the *middle lobes*,—the part of Man's Cerebrum which corresponds with the entire Cerebrum of the lower Mammalia. In the Cat and the Dog, which have the middle lobes fully developed, stimulation of their posterior portion produces no respondent movement. And not only is this the case in the Monkey also, but *the whole of the posterior lobe is similarly irresponsive*, as is also that *front portion* of the *anterior lobes*, which, in all the higher Mammalia, as in Man, has that forward as well as lateral development which markedly distinguishes it from the corresponding part of the Cerebrum of the Rabbit. Extirpation of the antero-frontal portion of the anterior lobes, from which the animal seems completely to recover corporally, induces a state of mental deficiency closely resembling dementia; the power of learning by experience, in particular, being abolished. Extirpation of the posterior lobes causes

Fig. 3.



Upper Surface of Brain of Rabbit:—A, cerebrum; B, cerebellum; C, olfactory bulb.

no loss of sensation or voluntary motion, but an apparent abolition of the instincts of self-preservation.

Before considering the further conclusions to be drawn from the results of Dr. Ferrier's experiments on the Cerebrum, it will be well to take into account those which he obtained from the application of the like stimulation to other Ganglionic centres forming part of the Brain. When either of the *Corpora Striata* was thus excited to activity, an immediate and rigid *pleurosthotonos* was excited in the opposite half of the body; the head being made to approximate the tail, the muscles of the face and neck being thrown into rigid tonic spasm, and the fore and hind limbs fixed and rigidly flexed. Apparently every muscle or group of muscles represented in the Convolutions, along with the lateral muscles of the body, were stimulated to contraction from the *corpus striatum*; the predominance of the flexors over the extensors, however, being very marked. Similar excitation of the *Thalami Optici*, on the other hand, gave no motor result whatever; from which it may be concluded that they have no direct connection with movement. That the irritation did not call forth cries or other signs of pain, might be supposed equally conclusive against the idea that these ganglionic centres are instrumentally connected with sensation; but when it is borne in mind, not only that the animal was under the influence of chloroform, but that the connection of the irritated *thalamus* with the Cerebral centres of the movements which express pain had been destroyed in order to expose this ganglion, the absence of any such expression seems adequately accounted for. Experiments on the *Corpora Quadrigemina* (or Optic Ganglia) were chiefly made on Rabbits, in which these centres are relatively very large and are easily exposed. The application of the electrodes to the anterior tubercles immediately calls forth a violent *opisthotonos*; so that, if the animal be not tied down, it executes a backward summersault which throws it off the table. The jaws are always violently clenched, and the pupils are dilated. These results do not militate against the idea of the connection of these ganglionic centres with the sense of Vision, which seems to be well established by

other evidence; but they show that they are also motor centres, especially for the extensor muscles. Stimulation of the posterior tubercles occasioned noises of various kinds.

Dr. Ferrier's experiments on the *Cerebellum* have led him to the unexpected conclusion that it contains the ganglionic centre of the motor nerves of the Eye; every kind of movement of the eyeballs—even rotation on their antero-posterior axes—being capable of excitation by stimulating some particular portion of the organ. The localisation of the centres of combined movements of the two eyeballs in particular lobules of the *Cerebellum* in the Rabbit, was extremely curious; and these results throw great light upon the obscurity which previously enveloped the precise function of the *Cerebellum*. That it was in some way concerned in the regulation and co-ordination of the Muscular movements, especially those concerned in the maintenance of the equilibrium of the body, has long been a general opinion among Physiologists, based in part on the results of experiments, and in part upon Pathological observation; the doctrine of the Phrenologists, who regarded it as the organ of the generative instinct, having been universally abandoned as untenable. But of the manner in which this power was exerted, nothing could be said to be precisely known. Now there can be no question as to the intimate relation between the guiding sensations we derive from Vision, and the co-ordination of our ordinary movements of Locomotion. In 'nystagmus,' which consists in a restless motion of the eyeballs from side to side, there is a difficulty in maintaining the equilibrium; and in 'locomotor ataxy,' in which disease of the posterior columns cuts off the *Cerebellum* from its normal relation with the Spinal cord, it is impossible for the patient to maintain his equilibrium with his eyes shut. So in the giddiness which most persons experience when they have rapidly turned round and round several times, it can scarcely be doubted that part at least of the result is occasioned by confusion of those Visual perceptions which would come through the oculo-motorial centres. That this regulation of the movements of the Eyes, and the harmonisation with them of the general movements of the body, constitute the entire function of the *Cerebellum*,

it would be premature yet to assert; but Dr. Ferrier's experiments seem clearly to establish the first of these, and strongly to indicate the second as essential parts of its action.

We now return to the inquiry as to the *import* of the experimental results previously detailed, in regard to the localisation of Cerebral action in the production of Movement, and its relation to Mental states.

In the *first* place, they unmistakably prove the correctness of the doctrine, that the Cerebrum, like the nerve-centres on which it is superposed, has a *reflex action* of its own; which manifests itself in the production of co-ordinated Movements, such as, in the normal condition of the animal, would be the expressions of Ideas and Emotions called forth by sensations. The Cortical ganglion is ordinarily excited to activity by the nerve-force transmitted upwards along the ascending fibres from the Sensorium; this calls forth respondent physical changes in its substance, which changes excite the states of consciousness that we designate as Ideas and Emotions; and respondent Movements are involuntarily called forth, which we regard as expressions of those states. The same movements are called forth (as in other instances) by stimulation applied to their motor centres; which are now proved to be definitely localised in the Cerebral convolutions.

But, *secondly*, it seems equally clear that these movements are called forth, not by the Mental states themselves, but by the Cerebral changes which are their *physical antecedents*. For we can scarcely believe that Ideas and Emotions can be called up by faradisation of the Cortical substance, in animals completely stupified by chloroform. And if we attribute any of those 'expressive' actions which are called forth by such localised stimulation, to the Mental states they would ordinarily represent,¹ we cannot refuse the like

¹ Dr. Ferrier was himself so much impressed in one case by the *intelligent* character of the succession of actions thus called forth, as to speak of it as 'evidently an acted dream.' But if *this* was, then *every other* must be regarded in the same light; and I fail to see in what the evidence of consciousness consists. It seems to me that it might just as well be said that the headless body of a Frog is animated by a directing Will, when one leg wipes off an irritant applied to the other.

character to the Epileptic convulsion called forth by the more general stimulation; a supposition at once disproved by the fact that in the typical forms of Epilepsy, convulsive movements, such as have now been traced to 'discharging lesions' of the Cerebrum,¹ take place *without any consciousness whatever*. Viewed in this aspect, Dr. Ferrier's results obviously afford additional support to the doctrine of 'Unconscious Cerebration;' by showing that important Cerebral modifications, of which only the *results* make themselves known, may take place outside the 'sphere of consciousness.'

In the *third* place, we seem able to draw from these experimental results a more definite *rationale* than we previously possessed, as to the automatic performance in Man of movements which originally proceeded from intentional direction. For it is clear that in Dogs, Cats, Rabbits, &c., the co-ordinated actions which result from localised stimulation of the Cerebral convolutions—expressing, by the Nervous mechanism proper to each species, the Mental states naturally called up by their Sensational experiences—are as truly the 'reflex actions of the Cerebrum,' as the simpler forms of movement are of the Axial Cord. Now the Nervous mechanism of Man *forms itself* in accordance with the modes in which it is habitually called into action; and thus, it may well be believed, any special modes of co-ordinated movement to which an individual has been trained, or has trained himself, come to be so completely the reflex actions of particular centres of his Cerebrum, that, if we could stimulate those centres by Electricity, respondent movements of the kind acquired by such special training would be the result. And since we now seem justified in asserting that such movements may be *executed* unconsciously, we may further regard it as at any rate conceivable that they may be *excited* unconsciously, even though such

¹ This view of the origin of those forms of Epilepsy which commence with convulsive spasm of the muscles ordinarily put in action voluntarily, as distinguished from those which primarily affect the muscles of Respiration whose centre of action is the Medulla Oblongata, is due to the clinical sagacity of Dr. Hughlings Jackson.

excitement comes through one of the organs of Special Sense.

The following statement, recently made to me by a gentleman of high intelligence, the Editor of an important Provincial Newspaper, would be almost incredible, if cases somewhat similar were not already familiar to us:—‘I was formerly,’ he said, ‘a Reporter in the House of Commons; and it several times happened to me that, having fallen asleep from sheer fatigue towards the end of a debate, I found, on awaking after a short interval of entire unconsciousness, that I had continued to note down correctly the speaker’s words. I believe,’ he added, ‘that this is not an uncommon experience among Parliamentary Reporters.’ The reading aloud with correct emphasis and intonation, or the performance of a piece of music, or (as in the case of Albert Smith) the recitation of a frequently-repeated composition, whilst the conscious mind is *entirely engrossed* in its own thoughts and feelings, may thus be accounted for without the supposition that the Mind is actively engaged in two different operations at the same moment; which seems to me tantamount to saying that there are two Egos in the same organism.

But, *fourthly*, these results entirely harmonise with the view I have long advocated, that the Cerebrum does not act immediately on the motor nerves, but that it plays downwards on the motor centres contained within the Axial Cord; from which, and not from the Cerebral convolutions, the motor nerves take their real departure. And the fact that all the muscles concerned in the ordinary movements of the body can be thrown into contraction by stimulation of these lower centres—the extensors through the *corpora quadrigemina*, while the flexors predominated when the *corpora striata* were stimulated—seems to show that the office of the Cerebrum is not immediately to evoke, but to co-ordinate and direct, the muscular contractions excited through these antagonistic primary centres; just as it controls the Respiratory movements whose centre is in the *medulla oblongata*.

In the *fifth* place, these experiments throw great light on the ‘crossed’ action of the several ganglionic centres con-

tained within the skull, which had previously been a matter of considerable obscurity; some phenomena of disease appearing to show that the motor centres of one side act on the nerves of the opposite side exclusively, whilst others seem to indicate that those of one side may affect the muscles on both sides. Anatomical investigation favoured the latter view, by showing that whilst some of the motor strands (*corpora pyramidalia*) which connect the Brain with the Spinal Cord, decussate, or cross to the opposite side, others pass continuously downwards without decussation. Now Dr. Ferrier found that the motor action of the *corpora striata* is strictly limited to the muscles of the opposite side of the body; being probably exerted solely through the decussating strands. On the other hand, the motor action of the *corpora quadrigemina* is not thus limited; the extensors of both sides being called into contraction by the application of the stimulus to either lateral half of the anterior pair; so that they would seem to act through both the decussating and the non-decussating strands. In his experiments on the *Cerebral hemispheres*, again, Dr. Ferrier found the motor action to be generally limited to the opposite side of the body; though in some movements, particularly those of the mouth, it was obvious that muscles of both sides were put in action. Now it is well known that extensive destruction of the substance of either hemisphere, if resulting from the *gradual* action of disease, may occur without any obvious loss of voluntary movement; though sudden injuries of a certain severity occasion paralysis of the opposite half of the body, which, however, is usually incomplete and of transitory duration. And I am inclined, with Dr. Ferrier, to accept the conclusion drawn by Dr. Broadbent from clinical observation, that the movements which are most independent on the two sides are those which are most completely paralysed by injury to one side of the Cerebrum; whilst those in which the co-operation of the muscles on both sides is required, may be sustained by the action of either Hemisphere. Not improbably the great transverse commissure (*corpus callosum*) here comes into action, enabling either Hemisphere singly to do the work—to a certain extent—of both; while there

seems some ground for the belief that the *left* hemisphere, which chiefly directs the movements of the *right* half of the body, is the 'driving' side. For in all save 'left-handed' persons, any movement which may be initiated by either limb is almost sure to be initiated by the right: thus, in beginning to walk, we almost invariably put the right foot foremost; and a person desired to hold up his hand, will as probably hold up his right hand.¹

But, *sixthly*, we have to inquire how far these experimental results justify the belief that there is any such localisation of strictly *Mental* states, as there is of the centres of the expression of those states in movement. And as to this it must be confessed that we are still very much in the dark, —the only fact that seems to afford any clue to the solution of the mystery, being the apparent coincidence between the motor centre of the lips and tongue in the lower animals, and that region in the Human Cerebrum of which disease is so often found to be associated with *Aphasia*. This association, however, seems by no means so constant as to establish a *causative* relation between the Physical and the Psychical state; and a careful examination of the phenomena of *Aphasia* would probably lead to the conclusion, that several distinct forms of disorder have been grouped under one designation. The *typical* *Aphasia* consists in the loss of the memory of words, or rather of the power of voluntarily recalling them; the patient understanding what is said to him, but not being able to reply verbally, because he is unable to call to mind the words which would express his thoughts. Such patients are exactly in the condition of the 'Biologized' subject, who, being assured that he cannot recollect his own name, finds himself absolutely unable to do so. But in other instances it would seem as if the defect were not so much in the want of the *memory* of words, as in the want of power to *express* them vocally; and this, not from paralysis

¹ In the well-known case of the murder of Mr. Blight by Patch, in which the sagacity of Sir Astley Cooper enabled him to infer, from an examination of the local circumstances, that the pistol must have been fired by a left-handed man, the prisoner, when called upon to plead and hold up his right hand, held up his left.

of the nerves of speech, but from an interruption to the action of the Will on the motor centres. And although there would seem strong ground for the belief that the memory of particular classes of ideas *may* be thus localised, yet it would be certainly premature to affirm that either the phenomena of disease, or the results of experiments, at present justify the belief that the region in question is the seat of the memory of words.

The analogy afforded by the specialisation of *downward* (motor) action, would lead us to anticipate that a like centralisation may exist for *upward* (sensory) action; and that particular parts of the Convulsions may be the special centres of the classes of Perceptual ideas that are automatically called up by sense-impressions; and anatomical investigation, particularly in the lower animals—in which such ideas may be supposed to prevail almost to the exclusion of the Intellectual ideas—may not improbably throw light on this relation. But in regard to those Mental processes which mainly consist in the selection, classification, and comparison of distinct Ideas, whether Perceptual or purely Intellectual, it still seems to me just as improbable as it formerly did,¹ that there can be special ‘organs’ for their performance, such as those named Comparison and Causality in the Phrenological system.—I consider, therefore, that the results of Dr. Ferrier’s experiments encourage the belief, that by the combination of Anatomical and Developmental study, of Experimental enquiry, and of Pathological observation, much light may be thrown on the Functions, not merely of the several Ganglionic centres which are aggregated in the Human Brain, but on those of the different parts of the great ‘Hemispheric ganglion’ formed by the convoluted layer of the Cerebrum.

¹ ‘British and Foreign Medical Review,’ October, 1846.

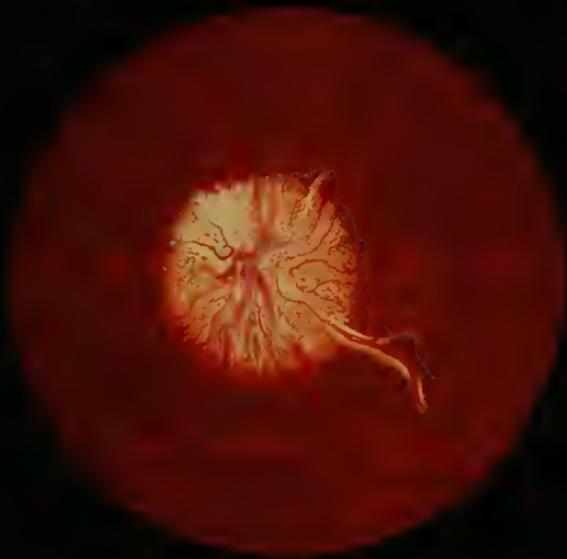
ON A
CASE OF RECOVERY FROM DOUBLE
OPTIC NEURITIS.

By J. HUGHLINGS JACKSON, M.D., F.R.C.P.,

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AND PARALYSED.

I HAVE so often written on the necessity for the routine use of the Ophthalmoscope in cases of Disease of the Nervous System, that I must now, in urging it again, write as briefly as possible. The case I have to relate, illustrated by two chromolithographs (Burgess), shows in a most striking manner the value of the ophthalmoscope to the physician.

The commonest ophthalmoscopical signs *which the physician sees* are those of optic neuritis; the neuritis is nearly always double. Except perhaps hemiplegia it is the most important of all the signs of intracranial disease. There is no more decisive evidence of the existence of an intracranial tumour (or other kind of adventitious product) than this easily discoverable pathological condition. Using common expressions, the discovery of optic neuritis enables us to decide that a patient's nervous symptoms depend on 'organic,' not on 'functional' disease ('coarse' or 'minute' changes). I do not say that the absence of optic neuritis negatives intracranial tumour (or other kinds of adventitious product). (See reference to remarks on minute changes in footnote p. 28.)





The ophthalmoscope is very easily used.

I have used the expression routine examination advisedly. I have insisted for many years, and I repeat, that we cannot tell whether a patient's optic discs are or are not in a pathological condition even so extreme as that illustrated by the chromolithograph No. 1, unless we actually look at them. There may be no defect of sight when the appearances of optic neuritis are extreme. The patient may make no complaint of his sight, and when asked about it may deny that there is anything the matter with it. But so far as I know I have convinced very few persons of the truth of my assertion, that sight is often unaffected in optic neuritis. It seems mere nonsense to some to assert that when there is severe optic neuritis the patient can read ^B *Brilliant* type.

Hence I continue to insist on the fact whenever a legitimate opportunity presents itself. I assert that we have frequently the *pathological condition*, optic neuritis, without the *symptom*, defect of sight. This is not a mere pathological curiosity.

Those who ignore this fact naturally do not in cerebral cases look at their patient's optic nerves until sight fails, *and thus they overlook the early and remediable stages of neuritis*. I believe that we should prevent amaurosis frequently if we always discovered the præ-amaurotic stage of optic neuritis and treated the affection energetically.

Those who wait until the patient's sight begins to fail, may err in taking as an early what is really a late stage of optic neuritis. So far as I know there is but one kind of optic neuritis from intracranial tumour (and other adventitious products). The ophthalmoscopical appearances vary extremely according to stage. Making conveniently, although arbitrarily, four stages, sight commonly fails in the third stage—a stage which is, I believe, by some called the 'swollen disc.'¹

¹ I may here refer to a lecture on 'Optic Neuritis from Intracranial Disease' which I published in the 'Medical Times and Gazette,' August 26, 1871. The following extract from that lecture refers to stages:—

'4. *The Stages of Optic Neuritis*.—I now tell you what you see in cases of optic neuritis at different stages. In the sense that there are abrupt differences, there are no stages; there are gradual changes from the beginning of the process through

The drawing (No. 1) shows that in speaking of optic neuritis with good sight one does not mean a mere slight change such as might be vaguely called 'congestion.' The more one uses the ophthalmoscope the less confidently one speaks of 'congestion,' and 'anæmia.' Sight may be quite good when the disc is very much swollen (and so much altered that there is really no true disc discoverable), when the arteries are scarcely traceable in the swollen *ci-devant* disc, when the veins, which are dark and partly concealed, give clear evidence of swelling by knuckling over the edge of the diseased patch; there may be scattered blotches of blood.

Those who do not look at the optic discs unless there be failure of sight will not only overlook the earlier stages of optic neuritis, they may overlook it altogether. For sight may not fail. I have seen not a few cases in which sight did not fail.¹ In the case I have to relate it did not. A common termination of optic neuritis is atrophy—what I call its fourth stage—but occasionally the swelling of stage 1 or 2 clears up, and, as in the case here illustrated, the disc resumes a normal or nearly a normal appearance. In this case the optic neuritis would never have been inferred; there was nothing whatever in the man's complaints or bearing to lead to a suspicion that his optic discs were abnormal. On the contrary, it would be said by most people that there 'could not be' anything the matter with them. Had I not used the

its ascent to a climax of acute change, and in its descent to the permanent change—atrophy. Nevertheless, although the changes are gradual, the appearances are strikingly different at different times, and most unlike at the two extremes—the height of acute change and permanent atrophy. We will make four stages. You will have gathered from what I have just said that this division into four is arbitrary. I used to make two stages only. It is, I think, convenient to make four, for learners, at all events. The following is an account of what is seen at different stages of a severe case. I use the expression 'severe' advisedly, as cases vary so much in degree and progress that I do not pretend to be able to describe 'typical' cases. Particularly observe that cases do not always run through these stages. There may be retrocession from either the first or the second stage, and *not* a progress to atrophy. I speak of what you may see by the indirect method of examination.'

¹ I have recorded a case showing this strikingly in the 'Medical Times and Gazette,' Dec. 7, 1872. A *résumé* of the chief points of it is given in the 'Roy. Lond. Ophth. Hosp. Rep.' Vol. 7, Part iv., Feb. 1873, p. 518, in Section 4, 'Recovery from Optic Neuritis.'

ophthalmoscope by routine the case would have been little more than one of paralysis of the third nerve. I should have ignored the *far more important symptom* (or, correctly speaking, pathological condition) optic neuritis.

I purposely give the case briefly, for without an autopsy it is of little value except as showing (1) that sight may be good when there is extreme neuritis, and (2) that the neuritis may disappear, the disc resuming what is practically a normal appearance. The patient did not recover from amaurosis; he never had any amaurosis to recover from. He took very large doses of iodide of potassium. My opinion is that this treatment prevented amaurosis, but as I always give large doses of iodide of potassium in such cases I cannot show more than a *post hoc*. Whenever I see optic neuritis I always give large doses of iodide of potassium.

A remarkably robust and healthy-looking Swedish seaman, 40 years of age, was admitted into the London Hospital on June 20, 1873. He had had a chancre many years ago, but apparently no secondary symptoms. He had had pains in the right side of his head for nine weeks, and after admission he had a little deafness of the right ear. One day, ten days before admission, he had some vomiting.

There was complete paralysis of the right third nerve which came on the day before admission. There was double optic neuritis. With the left eye he could read the smallest type, and denied that there was anything the matter with that eye; on the right side there was of course the defect of sight producible by palsy of accommodation. The first drawing of the left disc, was made by Burgess on June 30. The patient could then as always read No. 1 $\frac{1}{2}$ Snellen.

He went out apparently quite well on August 19. The palsy of the third nerve had disappeared rapidly under the administration of iodide of potassium; the neuritis had not disappeared. He went to sea.

He came to show himself on October 23. He looked quite well and felt well. The morbid changes in his discs were insignificant. Those on the left side are represented in the second drawing, which was taken by Burgess October 23. Seen for the first time, a good ophthalmoscopist would

hesitate to say that there had ever been any important acute change in that disc.

Such a case leads one to study carefully minute changes in the discs (as seen by direct examination), in order to make a retrospective diagnosis of optic neuritis.¹

It is probable that the case I have related was one of intracranial syphilis. But as there was no autopsy it is not worth while commenting on this aspect of the case. I have reported several cases of optic neuritis from syphilitic disease of the brain, as proved *post-mortem*. ('Medical Times and Gazette,' 1872-73-74.) I have recorded a marked case in the July number (1874) of the 'Journal of Mental Science.' I will now only say that *optic neuritis from syphilitic disease of the brain is not syphilitic optic neuritis*. Optic neuritis does not occur in such cases because syphilitic disease affects the optic nerves directly. There is a syphilitic tumour in the brain, and this causes optic neuritis, not in its character as a *syphilitic* lump, but in its character as a 'foreign body.' Any sort of mass in either the cerebrum or the cerebellum will cause optic neuritis.

I showed the original of the chromolithograph No. 1 at the meeting of the British Medical Association in August 1873. The following is cut from the museum catalogue. I extract it to show that I then believed that the neuritis would disappear leaving sight good.

'Ophthalmoscopic drawing by Burgess. The patient, a man, could read the smallest type (No. 1½ Snellen) on the day (June 30, 1873) the drawing was made. His sight is still (July 21st) good. It is believed that the abnormal change, will pass away, and that sight will remain good.—*Dr. Hughlings Jackson.*'

In looking up this entry I come across the catalogue notes of cases by my colleague, Dr. W. R. Gowers. As Dr. Gowers' opinion on any point in medical ophthalmoscopy is most valuable, I try to strengthen the position I have taken

¹ On this matter I have written in the 'Medical Times and Gazette,' Nov. 10, 1872; a reproduction of those remarks will be found in the 'Roy. Lond. Ophth. Hosp. Rep.' Vol. 7, Part iv., Feb. 1873, in Section 5, p. 520, 'Slight change in the optic discs in cases of cerebral disease.'

up by his testimony. I italicise those parts which bear on the points I have been urging. The cases Dr. Gowers relates have numerous other important bearings.

The following is cut out of the catalogue. (Drawings 176, 177, 178).

'Dr. Gowers on Optic Neuritis.

'1. Optic Neuritis, *with preservation of acuity of vision.* Left optic disc of a woman \ae t. 35 , suffering from headache, epileptiform convulsions, and paralysis of the right arm, due probably to syphilitic disease of brain. The position of the optic disc is occupied by a greyish red swelling, the redness being punctiform in the centre, striated on the peripheral portion. Vessels of nearly normal calibre, tortuous, and partly concealed on the swelling. The eye could read No. 1 Jäger. Field of vision of normal extent; blind spot of about three times the normal size. Both discs similar. *The neuritis cleared completely under antisyphilitic treatment, leaving no atrophy.*

'2. Slight optic neuritis, from a girl \ae t. 15 , suffering from epileptiform convulsions. Outlines of disc lost under a reddish swelling of moderate prominence, and of about twice the normal diameter of the disc. Redness punctiform in the centre. Vessels of normal size. Connective tissue about the vessels in the centre of the disc unduly conspicuous. *Could read the smallest test-type.*

'3. (a) Early stage of neuritis. Left optic disc of a girl, \ae t. 25 , suffering from right-sided convulsions, right hemiplegia and aphasia, due to a gliomatous tumour of the left hemisphere. A reddish swelling occupied the position of the disc, and concealed its outlines; the outer part was paler than the centre, and striated. Veins distended; arteries of normal size, partly concealed by the swelling. *The eye could read No. 1 Jäger. Both discs were similar.'*

Here Dr. Gowers gives the further course of the case. The girl became blind.

PATHOLOGICAL ILLUSTRATIONS OF BRAIN FUNCTION.

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THE object of this paper is to show some of the clinical bearings of the experimental researches on the Functions of the Brain, published in the last number of these Reports, by short commentaries on several cases extracted from the case-books and pathological record of the West Riding Lunatic Asylum. The cases themselves are reported almost verbatim, and being in the form of notes, not specially intended for publication, are to some extent deficient in detail. One special feature in them is the accuracy with which *post-mortem* appearances in the brain are given. There is nothing which gives greater cause for lament than the vagueness which characterises the great majority of reported cases of brain lesion. As a result of this, vast numbers of otherwise valuable cases are lost to those who wish to make use of them as data for arriving at the localisation of function from the side of pathology. It is much to be hoped that, in future, lesions affecting the hemispheres will be described exactly, according to some fixed system of nomenclature, as that of Turner or Ecker, and that general use may be made of such handy brain-charts as those edited by Tuke and Howden. It is only by accurate records of this nature that our knowledge of the localisation of function in

the human brain can be advanced. In the remarks on the following cases, attention has been chiefly directed to the relation between the symptoms and the pathological conditions of the brain, with especial reference to the harmony or otherwise of the recorded facts with the results of physiological experimentation on the brains of the lower animals. In this respect the negative as well as positive phenomena attendant on extensive brain lesion are equally deserving of notice.

CASE 1.—J. W.—Epileptic insanity. Lesion of the motor areas of the hemispheres.

Occupation.—Formerly a soldier. Admitted on December 30, 1873.

Facts observed by F. J.—Stated that he saw dogs and cats about him continually trying to tear the hedclothes or to seize hold of his own throat; every five or ten minutes he has epileptiform seizures, during which he grows very violent, requiring restraint.

Other facts communicated.—Tries to seize the poker or anything else that he may strike those in attendance upon him. Endeavoured to jump out of the chamber window. He has previously been in the asylum.

Æt. 50. Single. Religious persuasion—Protestant.

Second attack.—Duration one week. Cause unknown. Little is known at present of the patient. He was a soldier for twenty-one years, and is known to be a respectable and steady man. He was a patient here about ten years ago.

Mental and Nervous condition.—As far as can be ascertained he labours under no delusion but is decidedly demented. For certain events, such as his former residence here, his memory appears good enough, but for more recent events he professes not the slightest knowledge. He has a peculiar excited manner of speaking, and repeats very frequently that when last here Mr. Cleaton informed him his memory was entirely gone, which the patient considered a very bad business indeed; but the most peculiar feature in the case is the partial epileptic seizures which occur every five or ten minutes, without any loss of consciousness. The patient is suddenly seized with a convulsion in the left arm, the head is turned to the left as well as the eyes, and occasionally the muscular movements spread to the legs and right arm, but in a very slight degree indeed. After each seizure the patient's respiration is heavy, but there is not the slightest degree of unconsciousness, though he appears inclined to drop off to sleep. The patient is unable to stand, muscular power being entirely lost in left leg and decidedly diminished in left arm.

Physical condition.—Circulatory system: pulse 84; normal. Respiratory system: normal. Digestive system: tongue slightly coated, appetite fair. Is dirty and wet in his habits.

General appearance.—Height, five feet six inches; weight, 150 lbs.; hair, grey; eyes, brown; pupils, equal. Is in fair bodily condition, but has a few scratches and abrasions on left elbow, wrist, and hand, and a few trifling scratches on legs, and a small abrasion at base of sacrum.

Diagnosis.—Epileptic dementia.

Prognosis.—Unfavourable.

Progress of case.—January 2. The fits yesterday were much more severe, affecting the whole body. This morning it was observed, before a fit, that after a period of drowsiness and stupor the left hand and arm were stretched out, and the right hand and arm elevated above the head. A slight tremor was then noticed in the left arm, which was strongly flexed, and in both feet and legs. The legs were then drawn apart, the feet at the same time being twisted inwards and quivering violently. The tremors increased in severity, then the face, head, and eyes were affected, and strongly drawn to left side, and the mouth drawn to the left side and upwards; convulsion limited to the left side of face and arm. Loud puffing breathing beginning before cessation of fit, which lasted about forty seconds. Reflex action perfect in right leg but entirely suspended in left; whilst the right leg was abducted and quivering it was lifted up by the extensor muscles. Nitrite of amyl inhalation (gll. 20) for forty seconds produced drowsiness, beating movements of right hand, and dusky redness of face, neck and chest, accompanied with acceleration of pulse, which rose from 100 to 132.

At evening visit he seemed slightly improved and more conscious.

January 3.—Fits continue extremely numerous. During the night he had eighteen moderately severe, and twenty slight. Ordered Pot. Brom. ʒss. om. 4th hor.

January 4.—About ninety fits occurred during last night. Between 7 A.M. and 10 A.M. this morning he had six; from 10 A.M. until 1 P.M. no fits, but increased stupidity. Pupils decidedly contracted, complexion dusky. Pulse 100 and of fair strength. There is total loss of power on the left side, but a slight trace of reflex action in left leg. During inhalation of nitrite of amyl there is remarkable rousing whenever redness of face and acceleration of pulse and respiration occur; he awakes out of his drowsy stupid condition and becomes more lively and animated. During the first inhalation, sense of heat suggested to him his Indian experience, he thought he was in Calcutta and called for a punkah.

January 5.—Had eleven fits during the night, and six up to 10 A.M. Is still very dull and drowsy.

January 7.—His condition in all respects remains unchanged. The fits are exceedingly numerous and more severe than ever. Between the fits he is in a profound sleep, and yet he hears most of what is said near him and often makes some voluntary remark, such as asking 'when he is to be discharged.' He takes all his food.

January 8.—The fits continue of the same character but not so numerous; about thirty in twenty-four hours. He still lies in a stupid drowsy state, with eyes closed, as previously described. Pulse 105.

January 12.—Has had no fits for about sixty hours. Is more lively and talkative, and in about the same condition as when he came.

January 13.—Although always more or less rambling in his conversation, he was particularly deluded last night, speaking about dogs, &c., being in his room all day. He continues quite free from stupor and his general condition is improved. No fits.

January 17.—His condition remained the same for several days, but last night he appeared decidedly more feeble, and to-day he is altogether very much worse. His respirations are very hurried; his pulse 148, and sordes are beginning to form on the lips and teeth. He is quite conscious and can reply to questions, but if undisturbed lies in a drowsy state, frequently muttering in a very incoherent manner. Ordered ℥xii. of brandy in the 24 hours.

Evening visit.—He appears much worse. Pulse 160, and respirations rapid and laborious. He is rapidly sinking.

He died shortly after the preceding note was made.

Cause of death.—Epilepsy of unknown duration. 2. Pneumonia of a few days' duration.

Post-mortem examination.—Examination 27 hours after death. Weather mild. The body is very well nourished; there is a thick layer of fat over the abdomen. There are no external bruises nor marks of injury. There is a tattooed blue mark of a heart with a small cross rising out of its base on the front of the left forearm, and an anchor on the right forearm. Rigor mortis is present both in the upper and lower extremities. The left hand is fixed rigidly in a pronated and semi-flexed position. The left arm and leg are decidedly less in size than the right. Immediately below the elbow the left arm measures 9 inches in circumference, the right arm 10. The thighs, $4\frac{1}{2}$ inches above the upper end of the patellæ, measure, the left 14 inches, the right $14\frac{1}{2}$. The left calf measures $10\frac{1}{2}$ inches at its thickest part, the right $11\frac{1}{2}$ inches. There is some hypostatic congestion.

Head.—The skull is of average thickness and density and is fairly symmetrical. The dura mater is somewhat adherent and the sinuses contain only fluid blood. There is little or no thickening of the arachnoid, but there is a good deal of superficial wasting of the convolutions of the frontal and parietal lobes. The vessels at the base are perfectly normal. There is no visible hyperæmia and the pia mater strips with great ease. The grey matter is somewhat pale; it, as well as the white matter, is of fair consistence. There is no trace of softening, clot induration, or other organic change anywhere visible, although the most careful search is made. The ventricles are of average size and contain only a small quantity of fluid. The optic thalamus on the right side is smaller than its fellow on the left and its posterior end is attenuated. The whole brain weighs 47 ozs. Right hemisphere $20\frac{1}{4}$ ozs. Left hemisphere $20\frac{1}{2}$ ozs. Cerebellum $5\frac{3}{4}$ ozs. Pons Varolii ʒiv . grs. 39. Medulla oblongata ʒij . The medullary substance of the brain is perhaps a little firmer than it ought to be. No pathological change can be detected in the cerebellum, pons, or medulla.

Thorax.—No fluid in the pericardium. The heart weighs 12 ozs; left side empty. Right side contains fluid blood. The aortic valves are competent, but two of them contain hard bony spiculæ. There are several small atheromatous patches at the root of the aorta. There is considerable thickening of the muscular walls of the left ventricle. Right lung $30\frac{1}{2}$ ozs., completely adherent by old fibrous membrane. The upper lobe is completely solidified and is passing into a state of grey hepatitis. The lower lobe is congested and partially consolidated. Left lung, 18 ozs. free; slightly emphysematous at its anterior margin. On opening the abdomen an extravasation of dark-

coloured blood is found in the sheath and substance of the rectus muscle on both sides. There is no external mark corresponding with this extravasation. Liver 51 ozs., normal; spleen 5 ozs., normal; right kidney 5 ozs. capsule free; left kidney 5¼ ozs., capsule free; both quite healthy; bladder empty; bowels healthy. The spinal cord was examined 14 hours after the other organs had been examined. Nothing abnormal was found, except that on the posterior surface of the cord, and limited to about 4 inches of its lower extremity, there were found embedded in the arachnoid exceedingly minute cartilaginous and osseous plates. One or two of these plates were situated on the posterior roots of the nerves.

CASE 2.—W. B. Epileptic mania. Convulsions affecting left side. Extensive lesion of the right hemisphere.

Occupation, forgerman. Admitted on November 8, 1872.

Facts observed by R. B.—He has paroxysms of irritability and loss of temper. Gradual failure of the memory and intellect. Is careless and negligent in his habits, and performs his work and other actions without any subsequent recollection of what he has done. He is subject to epileptic fits, at which times the symptoms of mania are more predominant.

Other facts communicated.—His wife informs me that he has been very violent at times, and has committed acts of violence on her and his children. She also informs me that he has often threatened to commit suicide.

Æt. 57 years; married. Religious persuasion—none.

History from the relieving officer.—Patient has always been a dissipated character and has given much trouble at home. He is also an epileptic. Has been in the Bradford Union Workhouse since March 9, 1871, and during that time he has had many fits, the last occurring on the 3rd instant (November) when he had two. There is no record of any attempt at suicide, but he is said to have threatened it, and to be dangerous to others. There is no family history of fits, paralysis, or consumption.

State on admission.—Mental and Nervous condition. Patient's mind at present is clear and collected. He states that he was brought to Leamington from South Wales when about two years old and lived there for twenty years, after which he went to Scotland, and there began a drinking career which he kept up more or less till he was taken to the Bradford workhouse. He has been in the Bradford Infirmary with scurvy of the legs and has also had delirium tremens. He says he has only had two fits altogether, the first of these occurring two years ago, and the latter on Sunday last, November 3, or six days ago. Just before the fit he felt twitches in both arms and then he became unconscious, and when he came to himself again he felt bewildered and confused for some time. When about twelve years of age he received a blow on the back of his head with a poker, wielded by his sister, but though he says he was stunned he does not appear to have been unconscious, as he says he remembers his sister being corrected for her conduct. His eyesight is failing, and he has seen visions and suffered from pains in the head, but there have been no soundings in the ears. Circulatory system: heart normal. Respiratory system: normal. Digestive system: tongue slightly coated; bowels regular; appetite good. Genito-urinary system: normal.

General appearance.—Height 5 feet 5 inches; light brown hair; grey eyes, pupils equal and active, but crenated at the edges. In good bodily condition. No bruises or eruptions. There is the mark of an old buru below the lowest cartilage of the ribs. One vac. cic. on the left arm.

Progress of case.—November 15.—Going on very quietly. Makes himself useful in the wards. Takes his food well and sleeps well. No fits.

November 28.—Transferred to male 20 ward to work in the kitchen. No fits noticed since his admission.

January 25, 1873.—Quiet, but somewhat demented, as indicated by slowness of thought and speech.

April 9.—Several months since the patient had 1 fit during the day, the character of which could not be observed. In the last 24 hours he has had 2 very severe fits. Last night he was found upon his face in a fit almost suffocated. Ordered to be transferred to No. 4 ward and to sleep in the epileptic dormitory.

July 5.—Patient has occasional severe fits with long intervals. His mind is not yet much affected. To-day he showed a portion of tape-worm which he had passed. He was ordered treatment by extractum filicis liquidum and purgatives.

July 21.—The patient appeared in his usual condition until about 2 this morning when he had a very severe fit. He was removed to the epileptic dormitory, when the fits continued with extreme frequency. When seen at 10 A.M. to-day the fits occurred every 4 or 5 minutes, or even oftener, and had done so from 6 A.M.

The progress of two fits at that time was watched. The fit began apparently by drawing of head to the left side, tonic spasm of the left hand. Fully developed fit; clonic spasm of the left side of face, left arm, and left leg. Mouth, eyes, and hand drawn to the left side.

The body is bathed in perspiration, features livid, and mouth covered with foam. Respiration chiefly abdominal. Bronchi loaded. Pulse 120, compressible.

Another fit, 1st, hurried breathing; 2nd, twitching of eyebrows; 3rd, drawing of eyes and mouth to left, then other symptoms as before. The patient is quite comatose and insensible to all external impressions.

To have nitrite of amyl inhalations $\text{m}x.$ every ten minutes. Brandy and beef-tea injections every 3 hours.

Same date 3 P.M. No fits since nitrite of amyl was given. The patient has to a great degree recovered consciousness; hears, turns the head, and opens his eyes when spoken to.

6 P.M. Pulse 100, Respiration 56. Still no fits; continues conscious to a certain extent. Between 12 noon and 3 P.M. no nitrite amyl given. From 3 P.M. to 6 P.M., 5 minims every half-hour, to be continued till 10 P.M.

10 P.M. Patient much worse. No fits, but respiration has become embarrassed. He appears to be dying.

Died at 10.50 P.M.

Post-mortem examination.—Examination 46 hours after death. Weather exceedingly hot. The body is plump and very well nourished, there is a thick layer of fat on the abdomen. There are no bruises nor external marks.

Rigor mortis present in the legs but not in the arms. There is marked hypostatic congestion. There is great lividity of the features and neck.

Head.—The skull cap is thick and the hair coarse and bristly. The skull is rather thicker than usual generally, but is fairly symmetrical and of average density. Dura mater not adherent. Sinuses gorged with dark fluid blood. The whole brain weighs 38 ozs. There is slight thickening and milkiness of the arachnoid over the parietal lobes. There are a few atheromatous specks on the vessels at the base. In the right hemisphere, corresponding with the annectant and angular gyri and postero-parietal lobule, and round at the base to the uncinatè gyrus, there is a belt of depression and obvious softening and wasting of the cerebral substance. The pia mater, which is injected everywhere, strips freely except on the belt of depression, where it is adherent to the cineritious substance. On making sections of the brain, the convolutions corresponding with the belt of depression are found to be softened and of pulpy consistence. The white matter as well as the grey has a dirty brown colour. Right hemisphere 18 ozs. Left hemisphere 21½ ozs. Cerebellum 5¼ ozs. Pons varolii ¾ oz. Medulla oblongata ¼ oz.

The occipital lobe of the right hemisphere seems to be generally smaller than its fellow of the opposite side. Puncta sanguinea numerous. No trace of clot in the large ganglia.

Thorax.—No fluid in pericardium. Heart 15 ozs. Dark clots in the cavities. The aortic valves are competent. The lining membrane is stained of a deep red colour. The muscular walls are thickened and the cavities are dilated. The mitral valve is of unusually large size, and freely permits the passage of four fingers. Muscular substance pale and fatty. Right lung 22 ozs., not adherent, exceedingly congested, dark-coloured, and soft. Left lung 27 ozs., not adherent, engorged, and not crepitant at base.

Abdomen.—Liver 43 ozs., normal; spleen 6 ozs., soft and congested; right kidney 6 ozs., capsules strongly adherent; left kidney 6½ ozs., capsules strongly adherent; pyramids congested. Around the outer margins of the pyramids there are deep indentations and hollows. The cortical substance is very pale and shallow. Stomach 7½ ozs., normal.

CASE 3.—M. F.—Dementia. Cancerous tumour embedded in the right hemisphere.

Occupation, worker at a mill. Admitted on April 16, 1873.

Facts observed by R. F.—Wandering in his conversation; no knowledge of his nearest relatives or friends.

Other facts communicated.—Continually talking about persons and places at a distance, and imagining himself at other places. No control over the howels.

Æt. 40. Widowed. Religious persuasion—Independent.

History (from his sister).—This is his first attack. Last June a change was first noticed in him in the form of wandering in speech and general physical prostration. He has gradually been getting worse, and has latterly been unable to walk. This is probably due more to want of strength than to paralysis. Lately he has resided at home. He has lately become noisy

at nights and dirty in his habits. The origin of his illness is attributed to the sudden death in childbed of his wife about eighteen months ago. This loss he took much to heart. No history of insanity in the family. No drunkards in the family. His mother had an apoplectic fit, but it was not fatal. His father died at the age of 78. Family history good in other respects. The patient has not always been sober, though he has not been a drunkard. Ever since his illness he has had a ravenous appetite. He began to be very ill last June. The week before he left work he bled at the nose so much that it was thought he would die. After that he had sudden and violent pains in the temples and head accompanied by vomiting. The doctor attended him, but he did not get much better. He was then taken to the Leeds Infirmary, where he stayed three weeks and seemed to grow stronger. When he came from there it was noticed that he squinted, and that the pupil of the left eye was much larger than the other. The doctor who saw him again, said it was softening of the brain. His memory was nearly gone and he grew very weak, so much so that he could not walk or sit up. He took to his bed last November, and it was thought he would die almost every day until March, when he altered for the better. His sight went gradually. He called out the first time for a light at noon last Christmas, and has asked for a candle lighting during the day scores of times since. He was always wanting to eat, and never seemed satisfied, and slept a good deal of his time until lately, when he talked and shouted both day and night. When the attacks of pain came on he would be confined to bed for two or three days, unable to eat. About four months after the commencement of his illness the patient was taken to the Leeds Infirmary, where he remained for three weeks. On his return it was found that he squinted and saw double. After that his sight gradually got worse, and he gradually became unable to walk or to use his legs and arms; the pain in his head continued from time to time. There is no family history of cancer, and the patient never had any fits.

State of mind on admission.—He answers questions rationally, but his mind still refers constantly to the death of his wife. He thinks that he is in Gomersall (near Leeds). In conversation he always returns to the subject of his wife.

Bodily condition.—Circulatory system: pulse 92, small; heart sounds almost inaudible; extremities cold. Respiratory system: nothing abnormal audible anteriorly, but posteriorly there is to be heard at the right apex such harsh respiration that it may be described almost as cavernous; no crepitation here. Digestive system: tongue red and irritable at tip and edges, thickly coated; appetite very good, in fact almost ravenous. Genito-urinary system: he wetted his bed last night.

General appearance.—He is much emaciated. His knees are very stiff and seem fixedly flexed, as if he had been allowed to lie in bed for a considerable time. Hair dark, turning to grey. Eyes brown; left pupil larger than right; both are somewhat sluggish. He is nearly quite blind. One vac. cic. on the right arm. Height 5 feet 6½ inches; weight 104 lbs.

Progress of case.—April 18, 1873.—Not so well to-day, appetite being impaired. He is very feeble. Extremities (left) cold. Pulse 96. Eyes always jerked gradually to left side.

August 16.—Gets slowly worse; weaker and more debilitated. To-day a very large hæmatoma of left ear is observed. He is quiet and free from excitement.

History from the brother-in-law.—The patient was always quite well up till about twelve months before admission, when he began to complain of a great deal of pain in the forehead and head, especially on the right side.

For about three or four days after admission the patient complained of a good deal of pain in his head, and he generally eat with his head inclined to the right side.

According to the attendant of the ward where the patient was for the first fortnight of his asylum life he was paralysed on both sides to a certain extent, but the paralysis was most marked on the right side. He was quite crippled, and sat in a chair all day long and dozed. He had a voracious appetite, taking all given to him, and often asking for something more.

About a fortnight after admission the patient was brought down into ward 18; since then he has sat in a paralytic chair and appeared to be drowsy. This has been his chief characteristic. He could be readily roused, however, and when roused could speak rationally. He would generally take his food well, being fed; but latterly he would only take a little liquid. He gradually merged into a state of coma, in which he died. About two days before his death there were observed slight twitchings of the left side of the body. Both his arms as well as his legs seemed to be paralysed.

Death.—October 10, 1873. Assigned cause of death (as ascertained by post-mortem examination), tumour of the brain of uncertain duration.

Post-mortem examination.—Sectio 37 hours P.M. Weather mild. Body moderately well nourished. The conjunctivæ, face, hands, and knees are icteric. A few excoriations over the shins. Hypostatic congestion well marked. Rigor mortis complete in lower, but only slight in the upper extremities.

Head.—The skull cap is everywhere exceedingly thin. On its inner surface it has everywhere a rough granular appearance, and is specked with minute white dots. In the frontal region there are a great number of minute prominences terminating in sharp spiculæ or points, which cease almost abruptly at the coronal suture, a few only being found posterior to it on the parietal and occipital bones, most marked on the right side. Dura mater somewhat adherent along the line of the longitudinal fissure. The sinuses contain dark fluid blood. Beneath the dura mater the brain presents a flattened and compressed appearance. There is no trace of thickening of the arachnoid. The pia mater is thin and strips freely.

The whole brain weighs 52 ozs.; only a drachm or two of fluid escaped during the removal.

In the right hemisphere, occupying the parietal lobe, there is a tumour about the size of a small orange, which projects visibly from the brain mass. It occupies the postero-parietal lobule, the angular gyrus, and the upper extremity of the two first tiers of the temporo-sphenoidal gyri. It is of a dull pale purple colour, and is not demarcated from the cerebral substance into which it merges imperceptibly. The lower half of the tumour which is pale and more resembling the colour of the brain substance is slightly harder than the cerebral tissue, but the upper part is quite pulpy. No

wasting of the convolutions which are compressed together. On microscopical examination by Dr. Major, the tumour proved to be of a cancerous nature. The optic nerves and tracts were somewhat wasted.

Thorax.—About 1 fl. oz. of blood in the pericardium. The cavities of the heart contained dark clots and fluid blood, when emptied of which it weighed $7\frac{3}{4}$ ozs., and is fairly normal, the left ventricle being contracted. The right lung weighs $30\frac{1}{2}$ ozs. It is somewhat congested and œdematous. No tubercle. The left lung was firmly and universally adherent to the thoracic walls. The lower lobe is carnified. No tubercle.

Abdomen.—Liver weighs $41\frac{1}{2}$ ozs.; substance pale and soft. No stones in gall bladder, which is almost empty. Spleen weighs 6 ozs., normal. Right kidney weighs 4 ozs., left 4 ozs., normal.

CASE 4.—R. R.—Aphasia. Right Hemiplegia. Dementia. Lesion of both hemispheres, especially of the left.

Occupation, labourer. Admitted on December 5, 1873.

Facts observed by F. H.—A wild, vacant, and restless expression. Language unintelligible. Reasoning faculties disturbed and confused.

Other facts communicated.—Gets no sleep, wanders about at night, and is dangerous to those about him.

Æt. 63. Married. Religious persuasion—Church of England.

History.—Patient has been a very intemperate man; when drunk was always violent. Was twenty-one years in the army, and was much on foreign service. Three years ago he had an apoplectic fit, and since then has had two others; the right side has been partially paralysed ever since, and his language has been partially defective. After each apoplectic stroke his condition becomes worse. His mind has been seriously affected for the last fifteen weeks, but it has been weakened to some degree ever since the first attack. He has been occasionally violent, and has refused food. He is dirty in his habits.

Condition on admission.—*Mental condition.* Patient attempts to speak, and evidently wishes to communicate with those about him, but is unable to articulate anything beyond 'bah,' 'bow,' and occasionally other combinations of the letter 'b' with vowels. He appears to understand perfectly what is said to him.

Physical condition.—Circulatory system: heart's action irregular and feeble; no murmur; pulse 60, arteries corded. Respiratory system: no dulness, but respiration is very harsh at left apex, anteriorly. Digestive system: tongue clean, appetite fair, makes plenty of water, bowels not open since admission.

General appearance.—Height 5 feet 5 inches. Weight 114 lbs. His right arm is paralysed and rigidly flexed, and his right leg is very feeble. Hair light brown. Eyes blue, pupils equal and active. He is rather poorly nourished, but is free from bruises or eruptions.

Diagnosis.—Aphasia, with dementia.

Prognosis.—Unfavourable.

January 10.—Becomes feeble in mind and body, says nothing but 'hah.

February 5, 1874.—The patient died somewhat unexpectedly to-night.

No great change had occurred in his mental condition; he often attempted to speak, but never got beyond the monosyllables given above. During the day he sat helplessly in a chair, or was assisted to walk short distances in the airing grounds. At night he was occasionally slightly noisy, and always became so when visited.

A few days ago it was observed that the cold weather was affecting him severely. He was sent to bed and was more comfortable, but yesterday he was more feeble and died to-night.

Post-mortem examination.—Examination 40 hours after death; weather cold. The body is much emaciated but free from bruises or marks of injury, except a superficial scratch or abrasion on the right shin.

Head.—The skull is of average thickness and is quite symmetrical. The bones composing it have a yellowish tinge. The arachnoid is thickened, has a greyish appearance, and is buoyed up by an enormous quantity of subjacent serous fluid. The convolutions of the frontal lobe and of the upper and anterior portion of the parietal lobe in both hemispheres are wasted to an extreme degree. The intra-parietal fissure seems to form the posterior boundary of the wasting, which in front of it is very marked and behind it is scarcely perceptible. On the left side there is one special area of wasting and softening involving the third frontal, the external orbital, and the frontal end of the first temporo-sphenoidal gyri, the latter especially where it is recurved. In this area of softening the brain substance is broken down, pulpy, and has a dirty brown colour mottled with whitish spots. There is another extensive area of softening visible externally on that part of the brain which rests upon the cerebellum. It begins anteriorly at the middle of the under surface of the temporo-sphenoidal lobe, and extends backwards to the tip of the occipital, averaging about one inch in breadth, and involving the uncinate and internal temporal gyri. There the brain substance has a dirty greenish brown colour. There is no atheroma of the vessels. The whole brain weighs 1170 grammes. Eleven ozs. of fluid escaped during its removal. The grey matter is pale and the pia mater strips with the greatest facility. Cerebellum 143 grammes. The lateral ventricles are full of clear fluid.

Thorax.—There are 2 ozs. of straw-coloured fluid in the pericardium. The heart contains on both sides dark and decolorised clots; it weighs 225 grammes. The mitral valve is contracted to the size of a button-hole, admitting only the tip of one finger. The flaps entering into its constitution are enormously thickened. The aortic valves and others are normal. There is some thickening of the left side. There are no atheromatous patches nor vegetations. Right lung 888 grammes. Twenty-six ozs. of straw-coloured fluid in the pleura; no adhesions. The upper lobe is consolidated, noncrepitant and in a state of grey hepatitis. The lower lobe is crepitant but congested. Left lung 660 grammes. Twelve ozs. of fluid in pleural cavity; no adhesions. Lung emphysematous at its margin and generally congested.

Abdomen.—Liver 1,250 grammes. Very firm and hard. Spleen 67 grammes. Small, hard, and shrivelled. Right kidney 160 grammes. Left kidney 177 grammes. Capsules do not strip freely. The renal substance is exceedingly hard and the cortical substance is shallow.

CASE 5.—A. B.—Melancholia. Lesion of both hemispheres, but chiefly of the right.

Occupation.—Housewife. Admitted June 28, 1873.

Facts observed by C. J.—Her morose, sullen aspect, and general behaviour. Loss of memory, great depression, and inability to answer questions.

Other facts communicated.—She has on three occasions attempted suicide. Has also attempted to throw a child into the fire.

Æt. 44. Married. Religious persuasion—Wesleyan.

History.—This is the patient's first attack, and it began nine weeks ago by restlessness, especially at night, rambling conversation, and refusal of food. She became very low-spirited, and was influenced by the idea that her soul was lost because she had been bad to all. She had not altogether lost affection for her relatives, but has become very irritable, and on one occasion was about to throw one of her children into the fire. She has also struck her other children repeatedly in a manner previously unusual to her. She has on three occasions attempted suicide, twice by strangulation, and the other time by concealing a razor 'with intent,' &c. The patient has had two 'strokes,' the first of which is said to have occurred 10 years ago. She lost power over her left arm, and the faculty of speech. Both suddenly returned after a short period, but the duration of disability is not known. The second 'stroke' is said to have occurred three years ago, and was attended with similar phenomena. No cause assigned. The patient has had six children, three of whom are dead. The last was born five years ago. No miscarriages. Some years ago she had spinal disease (so called), and has always been in rather weak health. No history of rheumatism or of other serious illness, nor of intemperance.

Additional facts of patient's history elicited from her husband.—Twenty years ago she began to suffer from 'gurdy' (fits); these were characterised by sudden attacks of unconsciousness. She was not in the habit of falling to the ground, but would stagger to a chair; there was no cry and no actual convulsion, but rigidity of the arms and flexion of the fingers; this passing off was followed by stupor lasting from half an hour to two hours. The actual 'strokes' occurred as stated in the history first given. In the first there was loss of consciousness for twenty-four hours, in the second for thirty hours. The phenomena of both were the same, viz., coma (sudden) with loss of power affecting the left arm. It is not known whether there was any convulsion of this or other parts, nor is it remembered whether the face was drawn to either side. In both instances, however, after coming to herself her speech was found to be much impaired, being reduced to a lisping as it is described—but this gradually passed off. With regard to the paralysis, which was confined to the left arm, this also gradually became less, until it manifested itself only in inability to perform certain delicate movements, such as sewing, knitting, &c. Sensibility of the limb does not seem to have been affected, but the point is uncertain. Three years ago she began to complain of severe pain in the head, mostly towards the back, which, while present, used to cause her to hold her head, cry out, and exclaim that 'she must go

crazed from pain.' After this she was subject to sudden lapses of memory, for instance she would address her husband, commence a sentence, and then suddenly stop, saying she had forgotten what she had meant to say.

Condition on admission.—Mental. Her statements are somewhat rambling. She is extremely dejected and miserable, her mind constantly dwelling on her supposed sins and misfortunes. She constantly exclaims that she has no home, and no clothes, and that she will never be able to pay the debts incurred for her food and clothing here.

Bodily.—There is some degree of subclavicular flattening on both sides of the chest, but no abnormal sounds are audible. Heart normal. Pulse 80, small. Tongue slightly dry and glazed. She does not take food well. Hair dark brown, turning to grey. Eyes brown. Pupils equal, but small. Conjunctivæ pearly. Features regular. Cheekbones prominent. Rather tall of stature.

Genito-urinary system. Urine Normal.

Diagnosis.—Melancholia merging into dementia.

Prognosis.—Doubtful.

July 9, 1873.—Patient is in a very feeble state, but on the whole is taking food better than on admission.

August 8, 1873.—Still very demented and troublesome. She is very thin and her face has the pale sallow complexion suggestive of *organic brain disease*.

October 9, 1873.—Very feeble; frequently refuses food.

December 1, 1873.—Has had an attack of diarrhœa. She is now in a very feeble state, but the diarrhœa is better. No tympanites, but the abdomen is tender. Breathing harsh and prolonged, at the left apex no dulness, great emaciation. Pulse 100, temperature normal.

December 8, 1873.—During the last week the diarrhœa has returned, and the patient has been very obstinate in refusing nourishment. She has taken a mixture of bismuth and opium. Yesterday, though much reduced, she appeared on the whole improving, the diarrhœa having ceased, and had taken more nourishment than for some time previous. Last night, however, she became much worse. There has been no diarrhœa, but she is now in a state of collapse, pulse imperceptible. She has taken only a very little wine to-day, and is almost unable to swallow.

Died December 8, 1873.

Cause of Death.—Dysentery of about ten days duration with chronic atrophy of the brain of uncertain duration.

Additional note.—The paralytic symptoms, if any, were so slight as to escape detection, nor did she manifest any subsequently, her mental condition, however, prevented her engaging in such work as sewing, in which it might have been detected. Mentally patient continued as on admission, not much demented apparently, but all her thoughts occupied with the delusions that her soul was lost, that she could never pay for her food, &c., and on this account refusing it to the last.

Post-mortem examination.—Fifty-eight hours after death. Body much emaciated but free from bruises or other marks of injury. The surface of the abdomen is green from commencing decomposition. Rigor mortis

present to a slight extent in the upper and more so in the lower extremities. Hypostatic congestion very slight.

Thorax.—Pericardium normal. Heart $7\frac{1}{2}$ ozs. Cavities contained de-colourised clots and dark blood. Valves competent. No atheroma. Substance pale. Lungs. Right, $18\frac{1}{4}$ ozs. Slightly adherent at apex and in front. Substance healthy. Left, $26\frac{3}{4}$ ozs. Lower lobe in a state of red hepatisation. Upper lobe crepitant. Liver, $44\frac{1}{2}$ ozs. Substance soft and pale. Spleen, 3 ozs. Firm in consistence, but pale in colour. Kidneys, right $3\frac{1}{4}$ ozs. Capsule here and there adherent. Substance pale but otherwise normal. Uterus and ovaries normal.

There is great congestion of the mesentery and meso-colon, the veins being distended with dark blood. The small intestines are free from ulceration or congestion, but in the whole of the large intestine are minute ulcers commencing just above the caput carcum, a few in the ascending colon, much more numerous in the transverse and descending colon and rectum with punched out margins. There is also much congestion of the mucous membrane in the latter regions.

Head.—Skull. Not symmetrical but more capacious on the left side. The sinuses contained dark clots and fluid blood. There is no thickening of the arachnoid. Whole brain weighs $45\frac{1}{2}$ ozs. Pia mater thin and strips freely.

Left hemisphere weighs $20\frac{3}{4}$ oz. In it there is considerable wasting localised in the upper part of the parietal lobe and involving chiefly the upper part of the ascending parietal convolutions and the postero-parietal lobule, a little also in the first annectent. The other gyri are plump and well nourished.

Right hemisphere weighs $18\frac{1}{4}$ ozs. In it there is wasting of the convolutions in the same position as in the other hemisphere and extending also a little forwards into the first tier of frontals. There is also a very remarkable belt of wasting and softening extending from the tip of the occipital lobe posteriorly to the tip of the temporo-sphenoidal lobe anteriorly. This belt corresponds with the horizontal limb of the Fissure of Sylvius and the extension of that backwards. It involves the upper and part of the middle tier of temporo-sphenoidal gyri, the lower tier of frontals, the lower extremities of ascending frontal and parietal gyri, the angular gyrus, the second and third annectents, and the superior tier of occipitals. It is of irregular breadth, being about $1\frac{1}{2}$ inches in its widest part and in some places is hollowed out to the depth of $\frac{3}{4}$ inch below the level of the adjacent gyri. The wasted hollows are covered or lined by the arachnoid and pia mater, which in this situation is thickened and congested. The hollows present a dirty pinkish brown appearance. The large vessels at the base present a few spots of atheromatous deposit. In and around the hollows before described the brain substance is soft and pulpy and the softening is most marked in the grey matter.

There is no trace of old or recent clot. Cerebellum $5\frac{3}{4}$ ozs. Pons varolii $3\frac{1}{4}$ ozs. Medulla oblongata $\frac{1}{4}$ oz., apparently normal.

In Cases 1 and 2 we have two examples, in which mental aberration was associated with epileptic convulsions, a com-

bination pointing to some morbid condition of the brain, either induced by the epileptic attacks, or more probably the common cause both of the mental and convulsive phenomena. Post-mortem examination revealed in Case 2 very manifest lesion of the hemispheres, while in case 1 the appearances were not so striking, but yet sufficient to indicate some morbid process going on in the cortical centres with total absence of any abnormal condition of the other parts of the encephalon. In both, however, there were, apart from central causes, possible peripheral sources of the epileptic state, viz. in case 1, the occurrence of cartilaginous and osseous nodules in the membranes of the lower part of the spinal cord, and in case 2 the existence of tape-worm. It is well known that ento- or epi-peripheral irritation of such a kind may give rise to epileptic convulsions, but an analysis of the symptoms gives no special reason for supposing that in these two cases the conditions mentioned were concerned in the production of that condition of the nerve centres which manifested itself in the convulsive phenomena or the cerebral symptoms. The chief point of interest is to determine from what centres the discharge proceeded, whether from the lower as the medulla, or from the cerebral hemispheres themselves. It is more usually supposed that the medulla oblongata is the primary seat of the convulsions of epilepsy, and that the cerebral symptoms are secondary to changes in the cranial circulation conditioned by the same affection of the medulla. This theory fails to account for numerous facts in connection with the mode of origin and sequence of the phenomena of an epileptic attack, and especially in those cases of partial seizures, which, without manifesting all the symptoms of what are usually considered necessary to constitute a genuine case of epilepsy, are clearly of the same nature, and frequently merge into a general seizure, with all the usual convulsive phenomena and abolition of consciousness. On the other hand, these peculiarities (and it is these that are most deserving of attention) are capable of being fully explained when regarded as dependent on some morbid condition of the cerebral hemispheres in the first instance. In my former paper in these

Reports I have related experiments on the lower animals, which show that convulsive attacks with all the phenomena of idiopathic epilepsy can be caused by irritation of the surface of the hemispheres, and that the order of occurrence of the convulsive spasms may in great measure be determined according to the point from which the irritation primarily proceeds.

Dr. Bartholow (*American Journal of the Medical Sciences*, April, 1874) has likewise produced the same effects in a human being, by electrical irritation similarly applied to the cerebral hemispheres. In my paper above referred to, and in a more recent one on the same subject (*see Abstract Localisation of Function in the Brain. Proceedings of the Royal Society*, No. 184, 1874), following in the footsteps of Fritsch and Hitzig, I endeavoured to demonstrate the localisation in the cortex of the hemispheres of areas or centres of origination of simple and combined muscular movements, and the convulsive phenomena resulting from irritation of the hemispheres were accounted for by serial or simultaneous discharge of these differentiated centres.

Some objections have been urged by Dupuy, Carville, and others against the theory of localisation of motor centres in the hemispheres. They are disposed to think that the motor phenomena I have described are rather the result of irritation of ganglionic centres, or motor tracts.

It is contended that the electric currents employed by me for irritation are conducted from the surface of the brain to the basal ganglia, and that thus the movements are called forth. It is urged that if the cortical centres are motor, they ought to behave in the same way as the lower motor centres under the influence of anæsthetics such as chloroform and ether; and it is argued that the cortex of the brain cannot contain motor centres, on the ground that in the state of complete narcosis no results are obtained on irritation of the hemispheres, while the lower ganglia and motor nerves are still capable of excitation by the same stimulus.

In reference to these objections it is no doubt true that a certain amount of extra polar conduction of the electric

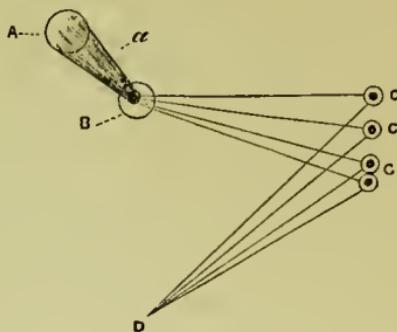
current is observable in the brain as in all tissues, but it is apparently forgotten that a certain intensity of the current is required before the function of the centres is excited. Were the results simply due to conduction, it would be expected that motor phenomena would be manifested by application of the electrodes to any part of the surface of the hemispheres. This, however, is not the case, for there are certain parts of the brain, stimulation of which gives rise to no external phenomena whatever. Thus, in the monkey, the occipital lobes, the antero-frontal regions are apparently non-excitabile. Nor is it the case that the negative results are due simply to distance of these parts from the basal ganglia, for stimulation of the Island of Reil in the monkey, which lies in much closer relation to the corpus striatum than any other part of the hemispheres, gives no results on stimulation, while the convolutions overlying this invariably and at the same time give definite motor results. Were the movements simply due to conduction, surely they ought to be called forth much more readily in this case than in any other.

One great fact of the first importance is that the results are uniform and are predictable. This of itself proves that there is no haphazard conduction. And, in addition, the stimulation of regions in close local relation to each other is followed by uniform results of a very different character. The conduction, if such is the case, must follow always the same definite line, and even this explanation involves the supposition of localised paths having differentiated connections with underlying ganglia. As regards the action of anæsthetics, I have myself pointed out that the encephalic centres are differently affected by those as well as by other conditions, *e.g.* the state of the circulation. In the condition of anæsthesia the hemispheres lose their excitability before the corpora striata, and the corpora striata before the corpora quadrigemina. These facts prove that the excitability of the centres varies, but are very far from countenancing the theory that, because the cortical centres are not affected in the same way as the lower centres of motion, they are therefore not motor in any sense. It must be

remembered that in regard to the nervous centres there is a progressive evolution from the simple reflex up to the highest psychological manifestations, and that a complex system of integrations and differentiations can be traced through the spinal and basal ganglia up to the cortical centres, the highest of all. It is quite intelligible that the conditions of reflex and sensori-motor or automatic excitability may differ greatly from those which are the basis of psychological manifestations.

The very fact that in the state of complete narcosis the hemispheres are not excitable, while the lower ganglia continue so, seems completely to overthrow the view that the results are due to conduction. For unless it is proved that the anæsthetic annihilates the conductivity of the brain tissue to electrical currents (and this is nowhere suggested), the absence of manifestation indicates that centres in the hemispheres have lost a function which they had formerly possessed. And it is only necessary to mention the fact that destruction of the centres whose excitation causes certain movements, results in paralysis of these very movements, and none others, while destruction of those centres which are not motor causes no paralysis of motion whatever. These facts I have succeeded in demonstrating in the case of monkeys. The mode of explaining away the phenomena adopted by Dupuy, Carville, &c., seems altogether incompatible with these facts. Owing however, to the use of terms such as 'motor centres,' which are susceptible of a different signification, some misconception has arisen as to the views which I hold as to the motor functions of the hemispheres. By the term motor centre as applied to a particular part of the cortex of the brain, I have endeavoured to signify the fact that this part is in direct communication with the motor tracts and their ganglia, and that its function is to excite co-ordinated muscular action of a definite kind of the nature which we call voluntary. What the multiplicity of connections may be which the part of the brain has with the basal ganglia and the spinal ganglia of the nerves supplying the muscles which it sets into combined action, is a matter which can hardly be revealed by mere

physiological experimentation. But that it produces its effect through and by these is the fact which I have endeavoured to explain. A simple diagram will serve to explain more clearly the meaning I attach to the term cortical motor centres.



Let A represent the cortical centre for say the action of closure of the fist; B the corpus striatum, c, c, c, the spinal nuclei in connection with the corpus striatum and the motor nerves of the muscles concerned in the act; and D the resultant act. The functional activity of A is to cause the action D through the combining centres situated between it and the muscles set in action. A is therefore motor in function, and is usually exercised in what we term an act of will. Stimulation of the individual motor nerves or spinal nuclei, could not be made artificially so as to produce the combined act. Stimulation of the corpus striatum usually produces the combined action not of the differentiated centre A alone, but of more or all the centres which exist in the hemispheres. Generally I have found that all are set in action. Dr. Burdon Sanderson in an interesting paper recently communicated to the Royal Society has demonstrated the fact that irritation of the medullary fibres corresponding to *a* between A and B causes the same action as results from the stimulation or functional activity of A itself. This is quite in accordance with the view I have taken, though, owing to my use of the term motor centre, Dr. Sanderson had been led to suppose that I had regarded the *motor nerves* of the muscles as being carried continuously up to the cortex and there combined and co-ordinated. The electric stimulus applied to *a* will cause a

stimulation essentially the same as that caused by the functional activity of A, and produce the effect D, but after removal of the grey matter corresponding to A, voluntary motion is lost as far as D is concerned. D, however, may still be effected through the influence of the sound hemisphere provided it be an act which is usually associated with that of the opposite side of the body. The basal and spinal ganglia in this case are set in action on both sides, owing either to their physiological or anatomical association with each other.

I see no objection to the use of the term motor as applied to the cortical centres when it is understood in the sense above described.

That these centres or areas have a motor signification is further demonstrated by the results of irritating or 'discharging lesions' of the surface. This view was acutely reasoned out by Dr. Hughlings Jackson from the facts of clinical research. Physiological and pathological experiments completely harmonise. I have already called attention to the great similarity between the sequence of the convulsive spasms in the artificially induced epilepsy by irritation of the surface of the brain, and the results of Hughlings Jackson's clinical observations. Dr. Bartholow's case furnishes an interesting physiological experiment of the same kind on the human brain. Cases 1 and 2 are illustrations of the same facts. In both the spasms were limited (almost entirely) to the left side, commencing in Case 1 with the left arm, succeeded by turning of the head and eyes to the left, retraction of the left angle of the mouth, and by convulsive action of the left leg. Rarely the right side became affected. In Case 2 the sequence was much the same. One great difference, however, between the two cases is that in Case 1 there was no loss of consciousness, while in Case 2 this always occurred in the fit. To explain these phenomena.

In the cortex of the brain, and related to each other in a constant and definite order, are individual centres for each separate muscular action involved in the epileptic convulsion, and the theory is that the convulsions are due to the discharge of these centres in a tolerably uniform manner, much depending on the primary source of the irritation. The motor

centres of the limbs are situated in the convolutions bounding the Fissure of Rolando. The centres for the leg are situated in the postero-parietal lobule, and upper part of the ascending parietal convolution. The hand and arm centres are localised in the ascending parietal and upper divisions of the ascending frontal; centres for different actions being distinctly differentiated. The posterior divisions of the superior and middle frontal convolutions contain an area stimulation of which causes the head and eyes to be directed to the opposite side, and the pupils to dilate. The facial muscles and the muscles of articulation have their centres in the ascending frontal in regions corresponding to the posterior extremities of the middle and inferior convolutions respectively. A more detailed description of the exact localisation and extent of these centres will be found in my 'Memoir on the Localisation of the Functions of the Brain,' presented to the Royal Society (*vide* Abstract). In the artificially induced epileptic convulsions by electrical irritation of the brain in the lower animals when the irritation starts primarily from any one particular centre, it is the first to be thrown into action, and then the others are discharged, usually in a certain order. The order most commonly observed is that the centres seem discharged from before backwards, beginning with the head and eyes, which are most anterior, and ending with those of the leg, which are situated furthest back. This is the case apparently from whatever portion of the hemisphere the irritation proceeds, whether starting from a motor centre, or from the more posteriorly-situated sensory areas. Epileptic convulsions can be produced with quite as great readiness by application of the irritation to the sensory areas as to the motor centres themselves. In some cases it would seem as if convulsions of a more general nature can be so excited. And it would appear as if in such cases the loss of consciousness occurs more early in the train of symptoms. And there is strong ground for the assumption that by affections of the sensory centres alone those forms of epilepsy are to be accounted for which are ushered in by sensorial illusions followed by loss of consciousness without motor phenomena. These forms may, however, pass into motor epilepsy, and it

seems as if the irritation is transferred from the sensory to the motor centres, just as there is a transference of nerve energy from a receptive to a motor centre in the phenomena of reflex action. It is doubtful whether consciousness becomes lost when the motor centres of the brain are alone implicated. An epileptic spasm may be confined to one particular group of muscles when the irritation is restricted to its cerebral centre alone. This may continue for some time without loss of consciousness, and usually it is not until the same morbid condition has affected several motor centres, and caused general convulsions that consciousness becomes lost. This last factor may be entirely absent as in Case 1. It is probable that the morbid condition of the brain, which, affecting the motor centres, causes convulsions, causes loss of consciousness by implication of the sensory regions. Consciousness largely depends on the continuous receptivity of the sensory regions to impressions of an ento- or epi-peripheral nature, and a sudden perversion of the sensory regions may interrupt the seriality and lead to loss of consciousness just as a temporary hemiplegia may result from excessive action of the motor centres. Probably the situation of the morbid changes in the brain in Cases 1 and 2 may in some measure account for the differences observable in regard to the affection of consciousness. In Case 1 the parietal regions were principally affected—convulsions motor in function; while in Case 2 the lesion affected the angular gyrus, the first annectent and the uncinatè gyri, all centres of sensation.

The loss of consciousness was one of the first symptoms in Case 2, while in Case 1 it did not occur.

In both these cases it is assumed that the existing lesions gave rise to some degree of irritation of the brain, while gradually progressing to destructive action. Destructive lesions affecting the cortex of the brain manifest themselves externally, more especially when they affect motor areas. Those movements which are distinct and independent of each other on both sides of the body, such as the various complex and individual movements of the hand, are specially affected; those which are antagonised are weakened to such an extent that lateral distortion results, as in the facial muscles,

owing to the influence of the sound hemisphere. In this way is to be explained the lateral deviation of the head and eyes in cases of hemiplegia. Thus in left hemiplegia the head and eyes are directed towards the right side. The centre for this action, situated in the convolutions of the frontal region, being interfered with by lesion of the right hemisphere, the action of the same centre in the sound left hemisphere causes the lateral deviation of the head and eyes to the right owing to the loss of counterbalancing action normally exerted by the other side. Movements which are normally associated with those of the opposite side are less affected, while lesions of the mouth centres which exert bilateral action do not cause any very characteristic affection of motion. It is different when the lesions involve the sensory regions of the brain. In such cases the lesions may undergo gradual and extensive development without causing any obvious loss of sensory perception. In Case 2 a destructive lesion existed in what corresponds in monkeys to the seat of visual perception (*viz.* the angular gyrus).

In the monkey I found that loss of sight in the opposite eye resulted from destruction of this region, but the blindness was only of temporary duration. The explanation seems to be that in regard to sensory perception a compensation may be effected by the sound hemisphere, the lower centres being intact. It is important not to forget the fact of the brain being a double organ. To cause complete loss of function it is necessary (except in the case of some movements) to have destruction of the corresponding parts in both hemispheres. This so rarely occurs that many have concluded from the absence of special symptoms, notwithstanding the existence of extensive lesions of one side, that brain function is not capable of localisation in definite regions. The duality of the brain and the possibility of compensatory action is not taken into account. While, however, a destructive lesion may not give rise to obvious symptoms, an irritating lesion on one side may cause very marked sensory illusions and hallucinations. Case 2 had at one time seen visions. This in all probability coincided with a condition of irritation affecting the substrata of visual perception and memory.

Among other symptoms in this case it was noted that the eyesight was failing. Owing to the absence of information as to the condition of the retina and optic nerve, it would be difficult to ascribe this to a central cause, for it may have been the result of optic neuritis dependent on the brain lesion. In regard to mental aberration as the result of brain lesions, it is easy to account in a general manner for its occurrence, but as to special manifestations, psychological difficulties have to be encountered which our present knowledge of the brain hardly enables us to meet successfully. It is to be noted that in the above cases, as well as in some of the others, lung affections existed, to which death may have been due. Whether the brain and lung affection stood in causal relation to each other it would be difficult to say, but Brown-Séguard, and more recently Nothnagel, have pointed out that brain lesions frequently induce abnormal conditions of the lung. This is a point in pathology deserving of careful observation.

Case 3 is an example of a class of cases more frequently met with in the wards of a general hospital than in a lunatic asylum. It presents several well-marked symptoms of cerebral tumour, with few which would indicate the seat of the tumour beyond the fact that it was in the right hemisphere. The intense headache, the vomiting, and blindness resulting from optic neuritis and subsequent atrophy of the optic nerves, all pointed to the diagnosis of tumour of the brain. The position of the tumour and the mode of growth are sufficient to account for the absence of convulsions such as one would have expected had the tumour commenced on the surface of the hemisphere. It is also probable that the tumour, growing slowly, caused rather displacement than actual destruction of the centres whose region it invaded. From its causing gradual increase in the intracranial pressure it would cause considerable impairment of the brain functions in general, and to this must be attributed the drowsiness and sluggishness of intellect, as well as the diminution of the voluntary control over the muscular movements. The affection of the postero-parietal lobule would have given reason for supposing that the muscular weakness should have

shown itself specially in the left-leg. This however seems not to have been the case, for the attendant stated that the paresis was most marked on the right side. It is questionable whether this observation is altogether accurate. While there were no very evident symptoms indicating the exact localisation of the tumour in the hemisphere, there are some points of considerable interest which may aid diagnosis in similar cases. Squinting was observed and frequently a jerking of the eyes to the left side. Irritation of the angular gyrus causes in monkeys a movement of the eyes to the opposite side. Whether a similar condition existed here can only be surmised, but it is at least probable, and the dilatation of the left pupil may have been due to the same cerebral condition. The pain was referred to the right side of the head. The seat of the pain, however, does not always coincide with the position of the tumour. In reference to this symptom of cerebral disease much may be learnt by percussion of the skull. On several occasions by smart percussion I have been enabled to determine approximately the seat of the disease, and sometimes brought out local pain though not formerly complained of.

Case 4 is another addition to the now numerous recorded cases of aphasia, coinciding with destructive lesion in the left hemisphere. It is not, however, a simple and uncomplicated case of aphasia, for in addition to the usual phenomena of aphasia, we have a condition of dementia, much more marked than can be accounted for by mere loss of the faculty of speech. This condition of dementia coincides with very extensive atrophy of the region of the brain supplied by the anterior cerebral artery, and on this it evidently chiefly depended. Much has been written on the subject of aphasia, and many differences of opinion still continue to exist among those who have directed their attention to it. It is not my intention to enter into any lengthened examination of the theories which have been advanced, nor to controvert the opinions of those who deny all localisation of brain function, including a 'speech centre.' The researches of Broca and the numerous confirmations of his observations which have been put on record, taken with

the results of my experiments on monkeys and the lower animals, seem to me to establish the fact of a localisation of the faculty of speech and to explain at least the broad features of the pathology of aphasia. I have shown that the region which governs the movements concerned in articulation is that which is the seat of lesion in aphasia. The region is symmetrically situated in both hemispheres, each one possessing the power of originating co-ordinated movements of the lips and tongue in a bilateral manner. 'Broca's convolution' is usually described as being the posterior third of the inferior frontal convolution of the left hemisphere. It would be more in accordance with the facts of experiments on monkeys, as well as with the cases which have come under my own observation, to localise the speech centre in the *operculum* (*Klappdeckel*) which is included between the ascending and horizontal limbs of the Fissure of Sylvius and which immediately overlaps the Island of Reil. While 'Broca's convolution' is placed in the left hemisphere, the centre for bilateral co-ordination of the speech muscles exists, as I have said, in both hemispheres. To this bilateral action of each centre is to be ascribed the peculiarity of loss of the faculty of speech without paralysis of the articulating muscles. For as regards the mere muscular action, one centre is capable of carrying it on when the other has been disorganised. This contrasts in a significant manner with the coincident hemiplegic paralysis of the arm and occasionally of the leg which frequently exists along with the aphasia. The centres for the movements of the arm and hand are in close contiguity to the centre of articulation, and hence the lesion which causes aphasia usually involves these also. More rarely the leg is affected owing to the fact that being at a greater distance the centres are less often invaded by the softening. These results are to be found when the cortex of the brain alone is implicated, the ganglia being intact, and furnish conclusive proof of the localisation in the brain cortex of the centres for voluntary motion. These centres, however, have another signification in so far as they form the motor substrata of mind. Besides being centres for the accomplishment of acts of volition,

they form the organic centres of the memory of accomplished acts. The centres for articulation besides their function of setting in action the complex and delicate movements involved in articulate speech, have the power of permanently recording the results of their functional activity. Words represent complex movements of articulation guided by impressions of sound. The memory of words must have an organic basis in that part of the brain which is the centre involved in their execution, for the memory of words is nothing else than the memory of the articulating processes which have been effected under the guidance of auditory sensations. The ideas of which words are the arbitrary symbols have no organic relation to that part of the brain where words are remembered except by associating fibres. The ideas in physiological language have their organic seat in those parts of the brain specially related to the nerves of common and special sensation, for all ideas are ultimately reducible to impressions of sense as their basis. The records of these in the brain cells furnish the material of all thought, and by their various combinations and associations constitute the foundation of all complex conceptions. The utterance of a word, with an appreciation of its meaning, involves the physiological activity not merely of the speech centre, but also of those parts of the brain related to the various senses concerned in the perception of the qualities of the object signified. Thus the mention of the word 'orange' indicates not only the activity of the speech centre, but also, of the sensory centres of colour, smell, taste, form, &c., all of which are concerned in our concept of the object. This is a concept of the simplest form, but the analysis of more complex ideas must necessarily be the same in principle.

In aphasia, consequent, as it usually is, on disease of the left hemisphere, the *memory* of words is not lost, nor is the person incapable of appreciating the meaning of words uttered in his hearing. What is lost in aphasia is, as Hughlings Jackson so clearly discriminates, merely the power of *voluntarily* using words to express ideas. As both sides of the brain are symmetrical and work conjointly, the memory of words may remain in the right hemisphere after

the occurrence of lesion in the left. The explanation of the loss of the faculty of speech is to be attributed to the unequal preponderance of the left hemisphere in the inclination of voluntary actions. The left hemisphere, like the right side of the body, is the leading or driving side, so that lesion of the left side is like the loss of the right hand. It requires long education to enable the person to accomplish with his left hand all the delicate manipulations of which the right hand was capable. The leading action of the left hemisphere may, however, be merely an accident of education or heredity, and there is no reason why articulate speech should not be the function of the right side. That such may be the case, there are pathological grounds for believing, viz. in those where aphasia has resulted from disease of the right hemisphere, and in those rare cases where lesion of the left speech centre has not caused the usual results.

In the case before us, as in most other cases of aphasia, there is retention of the faculty of intelligent comprehension of what is said, while there is total loss of speech. This is explicable on the grounds already given, viz. the integrity of the sensory tracts, the seat of the sensory memory or organic basis of ideation. There is reason to believe that speech may be regained by the education of the right hemisphere. But, in perhaps the great majority of the cases of complete recovery, the lesion of the left hemisphere has not been of such a nature as to incapacitate the part from ever again regaining its former function. Of this a very instructive example was brought under my notice in the West London Hospital by my colleague, Dr. Wiltshire. The case was that of a woman who had had an attack of aphasia with some degree of right hemiplegia, due probably to embolism. Recovery took place, and the faculty of speech was restored, but, curiously, at each menstrual period accompanied by profuse menorrhagia, the aphasic condition returned and lasted some days. On post-mortem examination I found softening almost entirely confined to the region overlapping the Island of Reil, and also involving the Island itself in the left hemisphere. The explanation of the recurrent aphasia is to be

found in the fact that in an already impoverished brain, with considerable lesion of the speech centre, the anæmic condition of the system particularly manifested itself in the temporary annihilation of the function of the most enfeebled part. In many cases of total destruction of the speech centre of the left side, a condition of more or less dementia becomes developed. Of this our asylums furnish many instances. The right side never, or only in a very imperfect degree, acquires the function formerly exercised by the left. Probably in addition to the lesion of Broca's convolution, there may in such cases be more extensive affection of the brain, and this seems to be the explanation in a great measure of the degree of dementia which was manifested in Case 4.

Although words are not absolutely essential to the reasoning process such as is manifested by the lower animals, yet the higher flights of reasoning, like the higher mathematics, require the use of symbols for the purpose of getting rid of the burden of details. Of still greater importance to the race, the use of symbols enables one generation to profit by the experience of the past, and advance beyond it. A person who has lost the faculty of speech is reduced to reasoning of a more simple process, for thought means internal speech, so that as compared with his former self he is in a condition of dementia. The dementia in the case before us coincides not merely with lesion of Broca's convolution, but also with very considerable atrophy of the frontal regions. Lesion of the frontal convolutions is of itself sufficient to account for a state of dementia. Experimentally I have found that destruction of the frontal regions of the brain of the monkey induces a condition resembling dementia.

In reference to lesions situated in this region, it is of importance to remember the fact of the duality of the brain. Conclusions are hastily drawn against the theory of localisation from unilateral lesions in this situation. Cases are on record, such as the celebrated American 'crowbar case,' where extensive lesion on one side in this situation caused no very obvious symptoms of loss of intellectual power. With extensive destruction on both sides, however, we

should always expect marked diminution of the intellectual powers, without loss of voluntary motion or sensation. The posterior parts of the brain were also affected in Case 4, but the symptoms do not exhibit any character which can be definitely associated with the abnormal condition of these regions.

Case 5 presents a complexity of symptoms not altogether easy of explanation by the lesions of the brain discovered on post-mortem examination. The seat of the lesions accounts for the degree and kind of paralysis observed, while the mental condition, that of melancholia, is more difficult of exact explanation, and probably may have been dependent as much on general mal-nutrition of the brain as on localised affection of the hemispheres. The mode in which the disease began to manifest itself seems to indicate that the lesions which were found to exist, were the result of apoplectic extravasations which had before death undergone absorption, but which had caused limited destruction of the brain tissue. The more marked symptoms were at first comatose attacks accompanied by a slight aphasia, or at least affection of articulation, and left hemiplegia affecting specially the left arm. The degree of paralysis of the left arm at the time of death seems not to have been of a very marked character, but the state of the patient's mind was such as to make it difficult to ascertain its exact amount, and the movements specially affected. The patient was said to have had spinal disease, but the symptoms on which this diagnosis was based are not detailed, and it is left uncertain whether a degree of loss of power over the legs is indicated. The lesions of the brain are of a very extensive nature. In the first place the upper part of the ascending parietal convolution and postero-parietal lobule seem to have undergone wasting on both sides. From the situation of these lesions one would have expected some degree of affection of the movements of the lower extremities, because these parts in monkeys are concerned in certain voluntary movements of the legs. That the lesions affected the cortical substance to such an extent as to cause loss of function appears not to have been the case; possibly what was taken as spinal dis-

ease may have been some degree of loss of power over the lower extremities.

The most marked pathological appearance was the deep belt of softening which ran in almost a horizontal line from the posterior to the anterior extremity of the right hemisphere. It involved the superior tier of the occipital lobe, the second and third annectents, part of the angular gyrus, the lower extremities of the ascending parietal and ascending frontal convolutions, the upper and part of the middle temporo-sphenoidal convolutions, and extended into the inferior frontal convolution. From the affection of the lower extremities of the ascending parietal and ascending frontal it would have been expected that loss of power over the left hand and interference with articulation should be manifested, as this region contains the mouth centres, and (at the lower part of the ascending parietal) some of the hand centres. The ascending parietal in nearly its whole extent in monkeys is concerned with movements of the fist. As the ascending parietal was not affected in its whole extent in this case the degree of paralysis which was likely to exist persistently should be very slight. This is quite in accordance with the symptoms recorded. At the commencement of the establishment of the lesion, the functional disturbance would naturally be greater, and to this is to be attributed the more complete paralysis of the left hand which at one time existed. Some degree of aphasia is recorded, but which speedily disappeared; and it was perhaps more a defect in articulation than loss of speech. Had lesion, to such an extent as existed in the right hemisphere, been found in the same part of the left hemisphere, aphasia of a typical form should have been expected. This is another instance of disease of the right speech centre not causing aphasia, which bears out the usually recognised pathology of this affection.

Besides the motor regions, the sensory regions of the brain are involved to a considerable extent. Nearly the whole of the superior temporo-sphenoidal convolution is invaded by the softening. This convolution in the monkey is, according to the results of my experiments, the seat of auditory perception. The effects of irritation of this convolution

are such as to warrant the conclusion that impressions of sound are excited, and destruction of the same confirms this opinion. It is, however, difficult to ascertain with any great degree of certainty the loss of auditory perception; and in regard to the sensory regions of the brain, owing to the compensatory action of the hemispheres, permanent affection of sensory perception is not usually the result of unilateral lesion. No diminution of the sense of hearing is recorded in this instance, but in this there is nothing remarkable. The upper part of the middle temporo-sphenoidal convolution is also involved. The function of this region is as yet obscure, nor is any light thrown on it by this case. In regard to the occipital lobes much has yet to be learnt as to their specific function. Irritation of these lobes by the induced current gives rise to no outward manifestations, and the only effect I observed after extensive destruction of these lobes on both sides in a monkey, was a remarkable state of depression with refusal of food such as one sees in cases of melancholia; sensation and voluntary motion being unaffected. The facts are such as to indicate a relation to the systemic sensations and the emotions founded on them. This would agree with the results obtained by Schroeder van der Kolk, who states that in melancholia with depression he has found the posterior parts of the brain diseased. In the case before us melancholia was a prominent symptom, and considerable softening of the occipital lobe on the right side was found *post mortem*. Whether these two facts stand in causal relation to each other can only be conjectured, but they would at least bear out Van der Kolk's statements. Much may have been due to the general weak bodily health and mal-nutrition of the brain. An anæmic condition of the brain with visceral troubles may of themselves have been sufficient to account for the depressed state manifested by this patient.

In the foregoing remarks I have confined my attention principally to tracing the relation between the symptoms during life and the lesions of the hemispheres which were revealed after death. It will, I think, be seen that they afford numerous confirmations of the conclusions which physiological experiment seemed to warrant. And though much yet requires

to be done, especially in reference to the psychical function of the brain, there is every reason to believe that the union of physiological experimentation with pathological observation will ultimately succeed in unravelling even this obscure subject, and establishing mental physiology and pathology on a more tangible basis.

THE
URINOLOGY OF GENERAL PARALYSIS.

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THE condition of the renal secretion has always been looked upon by physicians as capable of affording valuable information regarding the general condition of the system, and, in many cases, the knowledge obtained from a careful examination of the urine has been turned to good account in the diagnosis and treatment of disease. The indications derived from such an examination are not indeed, in all cases, so exact as might be desirable, but this is due, in some measure at least, to the imperfect state of our knowledge regarding the real origin and mode of formation of some constituents of the urine, and to defects or difficulties in our present methods of analysis. There is little doubt, that, as our knowledge of physiological action increases, and our methods of analysis become more perfect and easy of application, we shall be able to meet and overcome many existing difficulties, and to determine, with some approach to accuracy, the real value of the various elements of the urine, in their pathological as well as physiological relations.

When we consider that the kidneys are the principal channels for the elimination of the superfluous and effete products resulting from the operation of the various organic processes going on in the system, it is not surprising that alterations or perversions of these processes should be accompanied with corresponding changes in the character and

composition of the urine. Accordingly we find, in many cases, changes of a sufficiently definite and palpable nature to be easily detected and sufficiently well understood to be connected, to some extent, with their proper pathological cause. Such an instance may be found in the state of the urine in pneumonia, where the chlorides are greatly diminished in quantity or even entirely absent. Here we have a well-ascertained morbid condition giving rise to alterations in the secretion sufficiently appreciable to our present methods of examination, and a rational explanation of the change is to be found in the abstraction of chlorides from the blood, by the exudation into the air-cells of the lungs. Examples of a similar nature might be multiplied, and I have no doubt that even the slightest deviations from the standard of healthy nutrition, in any part of the system, are similarly registered in the condition of the urine, though we are not able, in many cases, to detect the nature of the changes ; or, if detected, to connect them with any known morbid process.

It is, perhaps, too much to say, that we shall ever be able, from a mere examination of the urine, to pronounce upon the real nature of the morbid action in any individual case. Such a result is hardly to be expected, considering the extreme complexity of the destructive processes going on in the various tissues, and the consequent liability of the urinary secretion to vary even under conditions of healthy nutrition. Nevertheless, such considerations as above indicated, lead us to hope that a more extensive and careful investigation of the characters of the urine, under different conditions of the system both healthy and morbid, may be attended with results of some importance, and may even tend to the explanation of morbid states which are as yet little known or ill understood. It was not, therefore, without some hope of being able to elucidate the pathology of general paralysis of the insane, that I undertook an investigation of the condition of the urine in that disease, the results of which I purpose to give in this paper.

On enquiring into what had been already done in this subject, I found that the constitution of the urine in the

insane had been made the subject of investigation by several observers, amongst others, by the late Dr. Sutherland and Dr. Beale, who together examined the urine of a number of patients labouring under different forms of mental disease, determining the relative amounts of water, solid matter, organic matter, saline matter, and phosphates in each case. The results are given by Dr. Sutherland in a paper in the *Medico-Chirurgical Transactions* for 1855, and the conclusions he arrives at are the following:—

1. A plus quantity of phosphates exists in the urine in the paroxysms of acute mania.

2. A minus quantity exists in the stage of exhaustion in mania, in acute dementia, and in the third stage of general paralysis of the insane.

3. The plus and minus quantities of phosphates in the urine correspond with the quantitative analysis of the brain, and of the blood; for a plus quantity of phosphorus is found in the brain, and a slight excess of albumen in the blood of maniacal patients; and a minus quantity of phosphorus and albumen are found in the brains of idiots, and a minus quantity of albumen in the blood of paralysis of the insane.

4. The plus quantity of phosphates in the urine of cases of acute mania, denotes the expenditure of nervous force, and is not a proof of acute inflammation in this disease.

The cases of general paralysis examined appear to have been too few to justify conclusions of any value regarding that form of mental disease; and in regard to some of the cases given, there is reason to doubt whether they were really cases of general paralysis. For instance, in one case recorded by Dr. Beale in his work on the urine, the attack is said to have lasted two months, and to have ended in complete recovery, a fact which renders the diagnosis, to say the least, questionable. Besides, the investigations of both the observers mentioned were unsatisfactory, inasmuch as they determined only the relative amounts of the different constituents in a certain quantity of urine, without regard to the actual amount passed in a given time. It is clear, however, that, in order to appreciate the value of the several constituents of the urine, in relation to the healthy or ab-

normal nutrition of the various tissues, we must know the absolute amount of each passed in a given time. Of this we can form no appropriate estimate from knowing merely the percentages; for in one case, with a high percentage of solids, and a small quantity of urine passed, the absolute amount of solids may be no greater than in another, where the percentage of solids is low and the quantity of urine voided large; and yet, if we trust to percentages, we should set down the amount excreted as much greater in the one case than in the other. We must therefore collect the whole of the urine passed in the twenty-four hours, and calculate the absolute amounts of the several constituents from an analysis of a given quantity of the mixed secretion. This has been done in patients labouring under various forms of mental disease by Mr. Adam Addison, whose paper on this subject appeared in the *Medico-Chirurgical Review* for April, 1865. In this paper Mr. Addison gives the results of an analysis of the urine in forty-eight different cases, determining in each case the absolute amount of urea, chloride of sodium, phosphoric and sulphuric acids, excreted during several successive periods of twenty-four hours. His cases include seven of general paralysis of the insane, and his conclusions regarding them I shall give in his own words.

‘In states of excitement the quantities of chloride of sodium, urica, phosphoric and sulphuric acids, are less than in the quiescent state. In the demented cases the quantities are about normal—some slightly above, and some below the mean. In two cases the excretion, according to weight, was also very near the mean healthy standard, and in one case the phosphoric acid was above it, without reference to weight. In the last stage of general paralysis it is impossible to obtain the twenty-four hours’ urine, but I have no reason to suppose that the result would differ much.’

In stating his results Mr. Addison compares his cases with the average normal standard as given by Dr. Parkes in his work on the urine. It appeared to me, however, that a fairer standard of comparison might be found in the urine of even a small number of healthy men, under conditions of hygiene and diet similar to those existing in the case of the general paralytics to be examined. Accordingly, before

commencing the examination of patients, I got six healthy attendants to subject themselves to experimentation. These men were put on a fixed diet of a fairly nitrogenous character. Their urine was then collected for three successive periods of twenty-four hours, and the absolute quantities of urea, chloride of sodium, phosphoric and sulphuric acids, were determined each day by careful volumetric analysis. The results of this examination are shown in the accompanying tables. Table I. gives the daily average of three days' excretion in each case, with the mean of the six cases, without reference to weight. In Table II. I have given the average amount excreted daily for every kilogramme of body weight. The urea, chloride of sodium, phosphoric and sulphuric acids, are given in grammes; the quantity of urine in cubic centimetres.

TABLE I.

Showing the average daily amounts excreted by six healthy men irrespective of weight:—

Men	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1016	1625	33·658	6·334	3·270	1·851
2	1021	1610	40·875	8·730	3·135	2·205
3	1016	1880	37·140	9·576	3·600	2·365
4	1018	2135	39·114	11·993	4·085	2·923
5	1022	1485	40·254	8·316	3·970	2·415
6	1025	1215	40·428	6·025	3·610	2·294
Mean	1019	1658	38·578	8·495	3·611	2·342

TABLE II.

Showing the average daily amount excreted per kilogramme of weight in six healthy men:—

Men	Weight in kils.	Average daily excretion per kil. of weight				
		Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	77·11	21·073	·4365	·0821	·0447	·0240
2	80·73	19·922	·5063	·1081	·0388	·0273
3	64·00	29·361	·5834	·1496	·0562	·0369
4	80·73	26·446	·4845	·1480	·0506	·0362
5	78·00	19·042	·5760	·1066	·0508	·0309
6	63·50	19·134	·6366	·0948	·0568	·0361
Mean	74·01	22·496	·5242	·1149	·0496	·0319

It should be stated that the healthy men, furnishing the materials for the above tables, were, during the continuance of the examination, employed in light ward work, or in airing ground exercise with the patients—duties which involve only a small amount of muscular exertion. As regards exercise, therefore, they may be considered almost on a par with the patients afterwards examined.

The results given in these tables differ somewhat from those obtained by other observers in the case of healthy men. I have therefore recorded in Table III. the amounts of urea, chloride of sodium, phosphoric and sulphuric acids, as given by Dr. Parkes. This table is taken in part from Mr. Addison's paper 'On the Urine of the Insane.' The results are, however, reduced to grammes in order to facilitate comparison with Tables I. and II.

TABLE III.

Showing Dr. Parkes's results, reduced to grammes:—

	Urea	Na Cl	P ₂ O ₅	SO ₃
Minimum	18·535	—	1·601	1·124
Mean	33·208	11·471	3·162	2·016
Maximum	44·614	—	5·184	2·666
Excreted per kil. of weight	·5081	—	·0486	·0305

On comparing this Table with I. and II., it will be seen that the results I have obtained are higher in the case of all the constituents except the chloride of sodium, the mean daily amount of which is 3·976 grammes less than that given by Dr. Parkes. The quantity of phosphoric and sulphuric acids is slightly greater. The most marked increase, however, is in the urea, the average of which is greater by 5·37 grammes in my cases than the mean given by Dr. Parkes. This result is no doubt due, in a great measure, to the character of the diet; and, as the diet was the same for all the patients examined as for the healthy cases, I still consider that the results given in Tables I. and II. form a fairer standard of comparison than those in Table III.

Taking these results then, as a means of comparison, I

shall now proceed to give the details of an analysis of the urine in several cases of general paralysis under various conditions. Afterwards I shall sum up the results and compare them with the results obtained in the case of the healthy men, and with those of other observers in the same field.

The method of analysis employed in all the cases was the same as in the healthy men examined. The chlorides, estimated as chloride of sodium, were determined by the nitrate of mercury process. The urea was found by Liebig's process, with a graduated solution of nitrate of mercury. The phosphoric acid was determined by Sutton's process, with a graduated solution of nitrate of uranium, and the sulphuric acid by precipitation with chloride of barium. The solutions used were those prepared by Sutton and Co. of Norwich. The results in the case of the urea, chlorides, phosphoric and sulphuric acids, are expressed in grammes. The quantity of urine passed is given in cubic centimetres.

During the period of examination the patients were placed under the care of special attendants, whose duty it was to collect the urine; and I have every reason to think that the quantities given are as near an approach to the truth as could be attained even in the case of healthy men.

The cases selected for examination, in the first instance, were those in as early a stage of the disease as could be found in the asylum. Six well-marked cases were taken, and, after being put on the same diet as in the case of the healthy men previously examined, their urine was collected for three successive periods of twenty-four hours, and the quantities of urea, chlorides, phosphoric and sulphuric acids, determined in each case. The same patients were then ordered extract of Calabar bean in $\frac{1}{4}$ -grain doses three times a day, and, after a sufficient interval, the urine was again collected for three successive days and tested as before. In each of the cases, as detailed below, the results without medicinal treatment are given in the first table, those under Calabar bean in the second.

CASE 1.—W. II., æt. 35; admitted June 30, 1873. Duration previous to admission, six months; has exalted delusions relating chiefly to personal strength, and manifests a general feeling of well-being; bodily condition good; weight 73·48 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1016	2340	53·820	10·440	2·948	3·159
2	1017	2050	53·300	10·325	2·706	3·485
3	1017	2150	55·900	6·450	3·268	3·719
Average	1017	2180	54·340	9·072	2·974	3·454
Av. pr. kil. of wgt.	29·667	·7396	·1133	·0407	·047	

The urine was clear and natural in colour each day. A film of uric acid formed on the surface after standing a few hours.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1016	1700	56·100	3·400	1·870	2·635
2	1013	1750	45·500	4·750	2·100	2·275
3	1014	2020	52·500	4·616	2·626	3·235
Average	1014	1823	51·366	4·255	2·198	2·714
Av. pr. kil. of wgt.	24·6733	·6986	·0579	·4297	·0369	

Urine clear and of pale colour; no deposit or film of uric acid on standing.

CASE 2.—II. K., æt. 42; admitted May 15, 1873. Duration on admission about a year; has well-marked optimistic delusions, such as that he is possessed of immense sums of money and is on intimate terms with several noblemen; well-marked physical symptoms; bodily condition fair; weight 70 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1027	1000	41·000	12·000	1·400	2·600
2	1022	2075	66·400	8·300	3·423	3·320
3	1019	1600	52·800	11·200	2·240	3·520
Average	1023	1558	53·400	10·500	2·354	3·146
Av. pr. kil. of wgt.	22·257	·763	·1500	·0336	·0449	

In this case the first day's urine was somewhat high-coloured and gave a light flocculent deposit suspending some oxalate of lime; second day's clear, with no deposit; third day's also clear, but on standing a short time deposited uric acid.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1015	1250	37·650	3·025	1·875	2·825
2	1022	1050	34·650	4·700	1·995	2·363
3	1021	1000	35·000	4·500	2·000	2·000
Average	1019	1100	35·766	4·075	1·956	2·392
Av. pr. kil. of wgt.	15·714	·5109	·0582	·0279	·0342	

Urine clear on each of the three days, remaining so after standing twenty-four hours, a film of uric acid forming on the surface.

CASE 3.—P. R., æt. 50; admitted January 6, 1873. A change had been observed in him for five years previous to admission, but he was able to follow his occupation till within a few weeks. At the time of these observations, he showed the most characteristic symptoms of general paralysis, both mental and physical. He was fairly nourished, and weighed 69 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1021	500	18·900	2·320	·972	·918
2	1020	1650	57·750	7·825	2·805	3·300
3	1017	1070	32·100	2·715	1·284	1·819
Average	1019	1073	36·250	4·286	1·687	2·012
Av. pr. kil. of wgt.	15·550	·5253	·0621	·0244	·0292	

After standing a short time the urine became opalescent, and yielded a light deposit containing oxalate of lime.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1 and 2	1015	1950	54·600	5·900	3·315	3·315
3	1019	1100	39·000	3·750	2·310	2·365
Average	1017	1016	31·200	3·220	1·875	1·893
Av. pr. kil. of wgt.	14·724	·4521	·0466	·0271	·0274	

On this occasion the urine was turbid and alkaline when passed, and deposited phosphates in abundance on standing.

CASE 4.—G. O., æt. 34; admitted October 13, 1873. For two years previous to admission, his mind had become gradually enfeebled. Is, however, unconscious of any mental defect and repudiates the idea of being ill. Physical symptoms well marked. Is fairly nourished, and weighs 63·5 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₂
1	1014	1070	26·750	4·420	1·070	1·712
2	1018	1850	55·500	6·475	2·775	2·960
3	1019	1250	43·750	3·750	2·000	2·250
Average	1017	1390	41·833	4·882	1·948	2·307
Av. pr. kil. of wgt.		21·890	·6588	·0769	·0306	·0373

The urine of the first day gave no deposit; that of the second day deposited uric acid on standing a few hours; on the third day a few crystals of oxalate of lime were found.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₂
1	1023	1300	51·250	3·900	2·210	2·600
2	1021	1400	46·350	4·100	2·240	2·800
3	1024	1020	38·446	3·550	1·938	2·193
Average	1023	1240	45·348	3·850	2·129	2·531
Av. pr. kil. of wgt.		19·527	·7141	·0606	·0335	·0398

A few crystals of oxalate of lime were found in the deposit of the first day's urine, which was very slight. On the second and third days there was an abundant deposit of uric acid.

CASE 5. C. W., aged forty-three, admitted November 4, 1872. Had been in another asylum since August 1871, and at that time had well-marked symptoms of general paralysis. Is subject to considerable excitement at times, and during the time of observation was excited for some part of each day. Has a systolic murmur at the base. Bodily condition fair; weight 70 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₂
1	1016	1300	42·900	8·450	1·950	1·950
2	1018	1650	49·300	8·250	2·310	2·475
3	1018	1650	51·150	12·375	2·475	3·465
Average	1016	1533	47·783	9·692	2·245	2·630
Av. pr. kil. of wgt.		21·900	·6826	·1384	·0321	·0375

In this case the urine was very high-coloured, and gave an abundant deposit of amorphous and triplo phosphate. There was also a slight trace of albumen.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1016	1500	42·550	6·000	2·100	2·700
2	1016	2170	65·868	5·340	2·821	3·797
3	1015	1740	45·140	6·485	3·480	3·045
Average	1016	1803	51·152	5·942	2·800	3·180
Av. pr. kil. of wgt.	25·561	·7307	·0849	·040	·0454	

Urine high-coloured, slightly albuminous, and giving a considerable deposit in which were found some fatty casts and uric acid crystals. The bulk of the deposits consisted of urates.

CASE 6.—J. T., æt. 36; admitted October 3, 1873. Duration previous to admission not known; was tried for pig-stealing in August 1873, but acquitted on the ground of insanity. Physical symptoms of general paralysis well marked; extravagant ideas not prominent, but denies any deterioration in mental or bodily health; bodily condition fair; weight 62·6 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1031	750	34·500	6·375	1·050	2·287
2	1020	2040	61·200	12·240	3·672	3·264
3	1078	1325	46·475	8·612	2·782	2·252
Average	1023	1372	45·544	9·075	2·501	2·601
Av. pr. kil. of wgt.	21·754	·5677	·1449	·0399	·0415	

On the first day there was an abundant reddish brown deposit, consisting chiefly of urates. On the second and third days deposit was less, and contained some oxalate of lime.

Under Calabar Bean.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1017	2200	64·900	6·400	3·960	3·300
2	1019	1350	35·640	4·700	2·160	2·295
3	1016	1150	29·750	5·350	1·955	1·782
Average	1017	1533	43·763	5·483	2·692	2·459
Av. pr. kil. of wgt.	24·488	·6290	·0875	0·430	·0392	

Urine pale and clear, depositing a slight amount of uric acid on standing.

This was one of those hitherto rare cases of general paralysis in which partial recovery takes place, and I had an opportunity of again examining the urine after restoration to comparative health. At the time of this examination the symptoms had been arrested for many weeks, the patient showing no mental peculiarity whatever, and being regularly employed at his trade as a mason preparatory to his discharge from the asylum. The only remaining symptom was a slight tremulousness of the muscles of the face and a certain thickness of articulation. He was in good bodily condition, and weighed 64·5 kils. I give below the results of the examination of the urine first without medicine, and then under the influence of Calabar bean. Dietetic and other conditions were the same as in the other cases.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1017	1515	43·690	5·623	2·548	2·215
2	1016	1675	40·095	4·690	2·278	2·177
3	1017	1425	39·900	5·653	1·853	1·995
4	1015	2500	48·750	10·000	3·000	3·250
Average	1016	1449	43·109	6·491	2·420	2·592
Av. pr. kil. of wgt.		22·310	·6683	·1006	·0375	·0401

The urine was only faintly acid and on standing gave a pretty abundant deposit of phosphates.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1015	1600	41·600	5·600	2·624	2·560
2	1019	1050	38·850	4·725	2·058	2·100
3	1019	1510	52·850	6·040	2·869	2·869
4	1015	1700	39·100	5·400	2·788	2·720
Average	1017	1315	43·100	5·441	2·584	2·562
Av. pr. kil. of wgt.		19·000	·6682	·0843	·0400	·0396

It will be seen on comparing the two sets of results in this case that there is no material difference between those obtained during the progress of the disease and those obtained during its partial arrest. The quantity of urea is in both cases considerably above the normal; the sulphuric acid is very near the mean of health; while the chlorides and phosphoric acid are considerably below.

The following six cases were treated in the same way as the six already given, only in them the dose of Calabar bean was increased to $\frac{1}{3}$ grain three times a day.

CASE 7.—R. B., æt. 45; admitted Sept. 4, 1873. Duration of disease previous to admission five months. Has had well-marked exaltation of ideas, but does not show this much at present; physical symptoms well pronounced; bodily condition fair; weight 65·7 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1007	1450	17·400	3·190	1·160	1·015
2	1008	2250	33·750	9·000	2·475	1·800
3	1010	1425	28·500	6·412	2·137	1·567
Average	1008	1373	26·550	6·201	1·924	1·461
Av. pr. kil. of wgt.		20·875	·4036	·0942	·0292	·0222

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1090	2200	44·000	4·400	1·980	2·620
2	1008	1350	20·250	3·700	1·215	1·215
3	1007	2010	22·814	3·015	1·809	1·728
Average	1008	1853	29·021	3·705	1·668	1·854
Av. pr. kil. of wgt.		28·173	·4412	·0563	·0253	·0280

On both occasions the urine was very faintly acid, soon became opalescent on standing, but gave no bulky deposit.

CASE 8.—H. V., æt. 48; admitted April 14, 1873. Duration previous to admission, six months; has very well-marked delusions of an exalted character, and well-pronounced physical symptoms; bodily condition, good; weight 66·7 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1025	800	32·000	5·200	1·840	1·720
2	1020	1150	34·500	12·750	2·139	1·725
3	1023	1100	34·100	13·200	3·080	2·200
Average	1023	1017	33·530	10·350	2·019	1·882
Av. pr. kil. of wgt.		15·251	·5028	·1550	·0302	·0232

The first day's urine gave an abundant yellowish-white deposit of urates and uric acid, that of the second day a less abundant deposit of the same nature, and on the third day there was only a light mucous cloud with some crystals of uric acid.

Under Calabar Bean						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1017	1170	35·100	5·380	1·987	2·165
2	1024	970	32·980	5·860	2·328	2·134
3	1025	710	31·593	3·840	1·917	1·846
Average	1022	950	33·224	5·026	2·077	2·048
Av. pr. kil. of wgt.		14·247	·4982	·0753	·0311	·0307

Urine at first clear, giving a very slight deposit of uric acid on standing.

CASE 9.—D. H., æt. 46; admitted November 8, 1872. Duration of disease previous to admission, five weeks; is very slow in manner, somewhat demented and has a general feeling of self-satisfaction; physical symptoms well marked; weight 68·7 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1015	1950	36·500	6·875	2·340	1·950
2	1016	2170	40·450	6·340	2·821	2·647
3	1023	1750	50·400	5·500	2·800	2·887
Average	1018	1956	42·450	6·238	2·653	2·489
Av. pr. kil. of wgt.		28·48	·6179	·0908	·0386	·0362

The reaction was only faintly acid, and the urine soon became opalescent, but gave no proper deposit.

Under Calabar Bean.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1013	1250	25·000	4·875	1·875	1·875
2	1021	1310	41·965	3·965	2·620	2·358
3	1013	1400	36·400	4·800	1·960	1·890
Average	1015	1320	34·455	4·546	2·151	2·041
Av. pr. kil. of wgt.		19·214	·5015	·0661	·0313	·0297

CASE 10.—R. H., æt. 47; admitted September 21, 1872. Duration previous to admission, about two years. On admission had well-marked symptoms of general paralysis both mental and physical. Is now considerably demented but has a general feeling of well-being; is in fair bodily condition, and weighs 62·6 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1031	320	11·200	1·440	·320	·572
2	1024	1240	48·360	4·960	1·736	1·984
3	1023	500	20·500	4·750	·750	·900
Average	1026	686	26·686	3·716	·935	1·132
Av. pr. kil. of wgt.	10969	·4262	·0593	·0147	·0180	

On the first two days the reaction was neutral, the urine high-coloured, and yielding an abundant deposit of a light yellow colour consisting chiefly of urates, but containing also triple phosphatè. The third day's urine was acid and gave no distinct deposit, but became opalescent after standing a short time.

Under Calabar Bean.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1015	640	14·080	1·625	·576	·640
2	1025	540	21·600	1·350	·972	1·404
3	1028	260	9·520	·560	·320	·496
Average	1023	480	15·066	1·158	·623	·846
Av. pr. kil. of wgt.	7·658	·2406	·0185	·0100	·0135	

The urine of the first two days was clear at first, but on standing gave a deposit of uric acid, that of the third day deposited urates in abundance.

Case 11.—I. S., aged forty-six; admitted February 4, 1873. Duration before admission, about eight months; has well-marked symptoms both mental and physical; is fat and well nourished, and weighs 73 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1012	2400	36·000	4·800	2·400	2·400
2	1018	1500	45·000	6·500	2·250	2·625
3	1018	2000	50·000	6·400	3·400	2·900
Average	1016	1966	43·667	5·900	2·683	2·642
Av. pr. kil. of wgt.	26·850	·5981	·0808	·0367	·0362	

The reaction was only faintly acid, there was no proper deposit but a slight opalescence.

Under Calabar Bean.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1018	1000	28·000	3·500	1·700	1·840
2	1023	800	29·600	3·450	2·000	1·680
3	1027	650	24·360	2·570	1·551	1·865
Average	1023	817	27·320	3·173	1·750	1·795
Av. pr. kil. of wgt.	11·1918		·3742	·0434	·0239	·0245

The urine of the first two days was clear, but gave a considerable deposit of uric acid on standing; that of the third day was turbid, and deposited urates in abundance. Reaction acid.

CASE 12.—H. H., æt. 32; admitted February 21, 1872. Previous duration of disease, two months; shows great self-satisfaction; considerably demented; is in good bodily condition and weighs 63·5 kils.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1015	1630	37·490	3·260	2·445	1·875
2	1010	2300	34·500	4·140	2·300	1·725
3	1011	1650	33·000	3·475	1·980	1·897
Average	1012	1860	34·996	3·625	2·242	1·832
Av. pr. kil. of wgt.	29·291		·5511	·0578	·0353	·0288

Urine pale and clear; no deposit.

Under Calabar Bean.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1013	1130	29·380	3·256	1·356	1·356
2	1016	1000	26·000	2·560	1·300	1·550
3	1021	540	17·280	2·350	·994	·945
Average	1017	890	24·220	2·720	1·216	1·284
Av. pr. kil. of wgt.	14·015		·3814	·0428	·0191	·0202

The following three cases were first examined under conditions similar to those already given. They were then put on an ounce and a half of pure alcohol daily, and after an interval of one day, their urine was again examined.

CASE 13.—G. S. æt. 41; admitted November 11, 1873. Previous duration of disease, about thirteen months. Is full of delusions of an exalted character and has all the physical symptoms of general paralysis; is in fair bodily condition, and weighs 77 kilogrammes.

Without Alcohol						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1018	2180	56·680	6·540	3·488	3·597
2	1017	1700	39·850	5·100	2·040	2·465
3	1011	1200	31·200	4·500	1·488	1·560
Average	1015	1693	42·576	5·380	2·338	2·541
Av. pr. kil. of wgt.	21·987		·5529	·0698	·0304	·0330

In this case there was a considerable deposit of uric acid after the urine had stood for 24 hours.

With Alcohol.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1018	1330	34·245	4·660	2·128	2·128
2	1017	1530	38·750	5·295	2·448	2·907
3	1019	1770	47·790	4·325	3·186	3·805
Average	1017	1476	40·260	4·760	2·587	2·946
Av. pr. kil. of wgt.	19·168		·5227	·0618	·0336	·0382

In its general characters the urine did not differ materially from that of the three days without alcohol. The amount of uric acid deposited was apparently greater.

CASE 14.—W. G., æt. 41; admitted Oct. 28, 1873. Previous duration not known; symptoms of general paralysis at first not apparent, now exceedingly well marked; is in good bodily condition, and weighs 66 kilogrammes.

Without Alcohol						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1020	2850	59·850	5·700	4·560	4·902
2	1013	2640	50·060	7·820	2·904	2·956
3	1016	1040	29·650	3·280	1·560	2·496
Average	1016	2160	46·520	5·600	3·008	3·452
Av. pr. kil. of wgt.	32·727		·7050	·0850	·0455	·0523

The urine was clear, faintly acid, deposited phosphates when heated, and became opalescent on standing.

With Alcohol						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₂
1	1017	1000	26·500	3·500	1·720	1·640
2	1022	1260	46·010	3·780	2·671	2·898
3	1027	1340	48·240	3·216	3·082	3·149
Average	1022	1200	40·250	3·498	2·491	2·562
Av. pr. kil. of wgt.		18·181	·6098	·0530	·0377	·0388

Characters the same as without alcohol. There was a slight deposit of uric acid on the second day.

CASE 15.—H. E., æt. 45; admitted June 30, 1873. Previous duration not known. Was at first despondent, is now buoyant and cheerful and full of the most exalted and extravagant delusions; in moderate bodily condition, and weighs 66·5 kils.

Without Alcohol.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₂
1	1012	1850	46·625	4·300	1·332	2·180
2	1012	1150	22·850	2·874	1·245	1·317
Average	1012	1500	34·737	3·087	1·288	1·748
Av. pr. kil. of wgt.		22·554	·5238	·0464	·0193	·0262

Urine pale and slightly opalescent, depositing phosphates on the application of heat. On the second day there was a considerable deposit of uric acid.

With Alcohol.						
Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₂
1	1023	550	20·440	2·320	1·430	1·210
2	1011	1150	28·750	3·500	1·380	1·426
3	1015	500	15·500	2·000	·750	1·100
Average	1016	733	21·563	2·606	1·186	1·245
Av. pr. kil. of wgt.		11·022	·3242	·0392	·0179	·0187

On the first day the urine was turbid from urates, but tolerably clear on the following days. Large quantities of uric acid were deposited each day.

In the cases that follow, except 16 and 17, the disease was in a much more advanced stage than in those hitherto given. In the last four it was impossible to collect the whole of the urine passed in the twenty-four hours, on account of the faulty habits of the patients. As much as possible, however, was collected at different periods of the day, and an analysis made of the mixed urine thus obtained. The figures given in these cases represent the quantities of the various ingredients contained in 1,000 cubic centimetres of the secretion. The results, as will afterwards be seen, do not appear to differ materially from those obtained in the less advanced cases; but, as it was impossible to get the patients to take the entire quantity of food and drink prescribed in the other cases, the comparison is scarcely a fair one.

CASE 16.—W. C., æt. 25; admitted Sept. 27, 1872; had well-marked exaltation of ideas; is now considerably demented, and in rather poor bodily condition; weight 53 kilogrammes.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1014	2120	38·160	4·240	1·908	2·120
2	1018	1740	52·250	4·610	3·480	2·697
3	1017	1800	46·650	3·520	3·240	2·340
Average	1016	1886	45·686	4·123	2·876	2·385
Av. pr. kil. of wgt.		35·584	·862	·0778	·0542	·0450

Urine at first slightly opalescent; film of uric acid forming on the surface after standing a few hours.

CASE 17.—E. G., æt. 42; admitted Oct. 30, 1873. Previous duration one year; has well-marked symptoms, mental and physical; is in good bodily condition, and weighs 70 kilogrammes.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1018	1550	45·000	6·975	2·170	2·170
2	1027	1250	42·650	3·500	1·875	3·312
3	1023	1450	43·375	4·750	2·050	3·652
Average	1023	1416	43·675	5·075	2·032	2·711
Av. pr. kil. of wgt.		20·288	·6237	·0725	·0290	·0387

CASE 18.—P. F., æt. 51; admitted March 4, 1872. Previous duration, about two years. Has a well-marked history of general paralysis. Is at present quiet, demented, helpless, and dirty in his habits; in fair bodily condition.

Days	Sp. gr.	Quantity	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1022		41·000	2·500	1·700	1·400
2 and 3	1025		40·000	1·500	1·300	3·600

The urine of the first day gave only a slight deposit, suspending a few crystals of uric acid. That of the second and third days gave a very abundant deposit of almost pure uric acid in all varieties of forms. In both cases the supernatant fluid remained clear.

CASE 19.—H. S., æt. 41; admitted March 20, 1871. Previous duration, a year. History in the asylum shows a well-marked case of general paralysis, with little excitement. Is at present in good bodily condition, much demented, and dirty in his habits.

Days	Sp. gr.	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1018	31·500	3·000	1·500	1·400
2 and 3	1014	20·450	1·560	1·450	1·750

In this case also there was a considerable deposit of uric acid.

CASE 20.—C. T., æt. 33; admitted May 23, 1870. Has a history of exalted delusions and excitement; is now quiet and demented.

Days	Sp. gr.	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1013	19·000	2·106	2·500	·850
2	1026	44·000	3·000	3·560	2·300
3	1032	60·500	2·850	2·850	2·700

The urine was alkaline and tender, and on standing deposited an abundant yellowish white sediment of urates, triple phosphate, and amorphous phosphates.

CASE 21.—J. C., æt. 23; admitted May 27, 1871. Has a history of considerable excitement, with exaltation of ideas; is now much demented and helpless, but in fair bodily condition.

Days	Sp. gr.	Urea	Na Cl	P ₂ O ₅	SO ₃
1	1012	17·500	2·500	1·100	1·350
2 and 3	1018	25·650	3·450	1·300	2·600

The first day's urine was alkaline, and deposited amorphous and triple phosphates. On the other days the secretion was acid, and yielded a considerable deposit of uric acid.

I come now to the last division of the task which I have proposed to myself in this paper, namely, to institute a comparison between the results obtained in the cases of general paralysis examined, and those found in healthy men, and to enquire how far the difference, where such exists, may be explained by a reference to known or supposed changes in the conditions of nutrition. In the first place, I shall give a summary of the results obtained without medicinal treatment of any kind, and compare them with our assumed healthy standard. I shall then contrast the results under the influence of Calabar bean with those obtained in the same cases without it; and, lastly, I shall refer shortly to the cases in which alcohol was administered.

Cases of general paralysis under no medicinal treatment.—In most of the cases examined, the general character and appearance of the urine did not differ materially from those observed in health. In some, however, the composition appeared to be less stable, the urea having a tendency to undergo decomposition at an earlier period than in health. In only one case was there any trace of albumen found. The *reaction* was in general acid, though only faintly so in most instances; in some, it was neutral, and in a few decidedly alkaline. The alkaline reaction appeared chiefly in the more advanced stages of the disease. The *deposits* most frequently observed were uric acid, urates, and phosphates, crystalline and amorphous. In one or two cases small quantities of oxalate of lime were found on several occasions, but this was by no means common. By far the most frequent sediments were uric acid compounds, and this notwithstanding the fact that the reaction was in most of the cases but feebly acid.

The *specific gravity* varied within very wide limits—the minimum recorded being 1007, and the maximum 1032. The latter occurred in an advanced stage of the disease, where there seems in general to be a tendency to concentration of the urine. This fact is due, no doubt, in some measure, to the circumstance that patients in this stage of the disease are unable to make their wants known, and though they devour greedily all that is given them in the way of food,

may not always be supplied with the necessary quantity of fluids. Taking all the cases examined, the average specific gravity was not much different from that of the healthy cases taken as a standard of comparison, being in both a fraction over 1019. In six cases, the total amount of solids, as found by careful evaporation and weighing, averaged 69·24 grammes, as compared with an average of 70·845 grammes in six healthy cases similarly treated. Here, as in the case of the specific gravity, the range was much wider in the paralytics than in healthy men, being in the former from 40·4 grammes to 96·8, and in the latter from 59·8 grammes to 87·9. The amount of solids, as calculated from the specific gravity by Dr. Christison's formula, corresponded pretty closely with that found by experiment, the greatest discrepancy being where the specific gravity was low.

With regard to the *quantity* of urine passed, in the seventeen cases in which the whole amount was collected, the average daily quantity was 1,565 cubic centimetres. The average excretion in health, as given by Dr. Parkes, who has collected the averages of various observers, is set down at about 52½ fluid ounces, or nearly 1,500 cubic centimetres. It will thus be seen that the average quantity excreted by seventeen general paralytics is slightly in excess of that generally given as the mean average of health. In the six healthy men whom I examined, however, the average daily quantity was 1,658 cubic centimetres, or nearly 100 cubic centimetres more than the average of seventeen general paralytics. On the other hand, when estimated with reference to body-weight, the relation is reversed, the average weight of the general paralytics being considerably less than that of the healthy men. In the healthy cases, the average quantity excreted in twenty-four hours for every kilogramme of weight was 22·496 cubic centimetres; while in the general paralytics it amounted to as much as 23·31 cubic centimetres. Judging from these figures, therefore, it would appear that the quantity of urine excreted is not materially different from the average of health.

The quantity of *urea*, as in the case of the solids generally, was found to be exceedingly variable in different cases. In

some it fell as low as 11 grammes for one day, while in others the excretion amounted to over 60 grammes in the twenty-four hours.

Taking the average of the three days on which the urine was examined, the lowest daily amount was 26·5 grammes, the highest 54·34. The range in the healthy men, similarly estimated, was from 33·65 to 40·875 grammes, the mean being, as before stated, 38·578 grammes. Of the seventeen general paralytics examined, in five the average of three days' excretion was below the mean of health; in the remaining twelve the quantity was considerably higher. The mean of all the cases was 41·19 grammes, representing an increase of 2·5 grammes over the healthy mean. In the more advanced stages of the disease, where it was impossible to estimate the entire quantity eliminated, there still appeared indications of an increase in the amount of urea. In one case it rose as high as 60 grammes in 1,000 cubic centimetres; and though no approximate estimate could be formed of the quantity of urine passed, yet each result seems to indicate a probable increase in the absolute quantity. It may be mentioned that this high percentage of urea corresponded with a specific gravity of 10·32, the highest recorded in these observations. When estimated in relation to a definite weight of body, the increase in the urea becomes still more apparent. The average daily excretion per kilogramme of weight in seventeen general paralytics was ·6061 gramme, as compared with ·5242 gramme in six healthy cases, and ·5081 the normal of health as given in Table III. In some cases, no doubt, the quantity excreted is less than the normal average of healthy men. It may be, however, that in these cases the amount excreted during health was habitually smaller than the average; but, as the patients do not recover, it is impossible to compare the results with the *individual* healthy standard, and this point must remain undetermined. In the case of partial recovery, where the urine was examined both during the progress of the disease and after its partial arrest, we have seen that the results of the two analyses are almost identical. Here, however, though the symptoms are at present in abeyance, there is

little doubt that the same morbid condition of nutrition exists as when the symptoms were more observable. Hence we are not warranted in drawing any conclusion from the results obtained. If, however, we find that, in a large number of cases of disease, the average amount of any urinary constituent is habitually higher than the average in healthy cases examined under the same hygienic and dietetic conditions, I think we may fairly conclude that the tendency to increased elimination is in some way connected with the presence of the disease in question. On the whole, therefore, the balance of evidence seems to be in favour of an increase in the quantity of urea.

In trying to arrive at a rational explanation of the increased elimination of urea, we are met at the outset by the difficult question of the mode of origin and formation of this substance. The solution of this question has engaged the attention of some of the ablest physiologists of the present day, and much has been done of late years towards the advance of our knowledge on this subject. It cannot be said, however, that we are yet in a position to give a satisfactory explanation of the processes involved in its formation, or to discover the various conditions influencing its discharge from the system. It is quite clear from its composition that its formation involves the destruction of some nitrogenous compound, but it has long been matter of discussion how or in what part of the system this destruction takes place. An opinion long held sway that it was generated at the expense of the muscular tissue. Liebig, with whom this idea originated, believed that the nitrogenous elements of the food could not become disintegrated, so as to give rise to urea, without first passing through the stage of organised tissue. In his work on 'Animal Chemistry,' this great chemist says, 'the flesh and blood consumed as food yield their carbon for the support of the respiratory process, whilst the nitrogen appears as uric acid, ammonia, or urea. But previously to these changes the dead flesh and blood become converted into living flesh and blood, and it is, strictly speaking, the carbon of the compounds formed in the metamorphosis of living tissues, that serves for the produc-

tion of animal heat.' In another part of the same work, he says, 'there can be no greater contradiction with regard to the nutritive process than to suppose that the nitrogen of the food can pass into the urine as urea, without having previously become part of an organised tissue.' It would appear, therefore, that this distinguished chemist held that urea could not result from the nitrogenous ingesta until they had become part and parcel of an organised tissue, and that it is the wear and tear of tissue that causes the disintegration of the nitrogenous compound, giving rise, on the one hand, to the carbon compounds used up in the production of heat and force; and, on the other, to urea as a residue to be eliminated from the system. There seems, however, to be good grounds for the belief that only a small portion of the urea originates in this way. Within the last few years experiments have been made by Parkes, Fick, Wislicenus, Ed. Smith, and others, which show that muscular exercise is not attended with any marked increase in the quantity of urea eliminated, but, on the other hand, that the ingestion of an increased quantity of albuminous food exercises a very notable influence. In a series of experiments conducted by Professor Parkes on two healthy soldiers, he found that on a non-nitrogenous diet, and at rest, the urea fell, in one case, from 35 grammes (which was the daily amount eliminated under ordinary diet) to 16·7, and, in the other, from 26 to 15 grammes. Under the same diet, with the addition of severe exercise, the increased amount of urea was very small, being, in one case, 1·589 gramme in two days, and, in the other, only 0·223 gramme in the same period. After their return to ordinary diet and occupation, these men excreted for the first four days a quantity of nitrogen considerably in excess of the average amount. This excess was referred by Dr. Parkes not to increased muscle waste during the period of severe exercise, but to an excess in the nitrogenous food consumed during the time immediately following the exercise. From these and similar experiments by other observers, it would appear that the effect of muscular exercise in causing waste of albuminous tissue, and consequent elimination of urica, is much less than had been supposed, and, on the other

hand, that the amount of urea excreted is directly proportioned to the quantity of nitrogenous matter ingested. It is, therefore, highly probable that the nitrogenous constituents of the food, without becoming an integral part of the blood or tissues, may and constantly do undergo disintegration, giving rise, on the one hand, to hydrocarbonaceous compounds which are either directly applied to the production of force and heat, or are stored up, in the form of fat, for the future wants of the system; and, on the other, to urea, which must be regarded as the inutilisable portion of the nitrogenous principles. This is not, however, to be regarded as the only source of urea. It is a well-known fact that even after prolonged abstinence, urea is found in the urine, and the same is the case on a strictly non-nitrogenous diet. In such cases, the urea is no doubt derived from the disintegration of the albuminous tissues, which, under these circumstances, may become subservient to the wants of the system in the same way as the nitrogenous elements of the food. Again, the muscles, and all highly organised animal structures, are, as a consequence of their vitality, liable to a constant process of disintegration and repair. If the system is in a healthy condition, these two actions counterbalance each other, and the amount of nitrogen eliminated remains unchanged. If, however, the nutritive activity is in any way impaired, the quantity of nitrogen given off may preponderate over the amount assimilated, and hence we have wasting of the tissues, and an increase in the amount of urea excreted.

It will thus be seen that the chief points to be taken into consideration, in seeking for an explanation of increased or diminished elimination of urea in any case, are the amount of nitrogenous material ingested with the food, and the relation of the reparative process to that of disintegration and decay. The amount of nitrogen eliminated by the bowels may also have an important bearing on the quantity of urinary nitrogen. I have not been able to determine whether this consideration accounts for the small amount of urea obtained in a few of the cases of general paralysis examined, but I think it probable that such may be the case.

As we have before seen, the mean average amount of urea in all the cases examined was considerably in excess of the normal. Now this increase can hardly be accounted for by the quantity of nitrogen ingested, as the diet was the same in the general paralytics as in the healthy men, and each patient was induced to consume as nearly as possible the same quantity. The fact seems to point rather to an increased disintegration of the protein compounds of the organised tissues, the nutritive activity being so far impaired that adequate repair does not take place. Nor is this inconsistent with the conditions existing in general paralysis. In many cases we observe a gradual and progressive emaciation, notwithstanding the fact that large quantities of food are greedily consumed. In other cases, no doubt there is a tendency to corpulence at certain stages of the malady, and in these cases the patients are generally quiet, contented, and fatuous. In those where emaciation is observed, on the other hand, we find muscular restlessness and excitement prevailing to a greater or less extent. These two classes of cases, though at first sight very unlike, will be found on closer examination to present many similar characters. In both, it may be presumed there is a defective power of nutrition and an inability to build up the more complex nitrogenous tissues. Hence that part of the nitrogenous principles of the food, that should otherwise go to the renovation of the muscles and other highly organised tissues, undergoes a retrograde metamorphosis, and appears in the renal secretion in the form of urea. On the other hand, the hydro-carbonaceous compounds, resulting from the disintegration of the food, would seem, in the one case, to be at once expended in the production of force and heat, while in the other class of cases they are stored up in the form of fat.

The quantity of *uric acid* excreted was not made the subject of direct experiment, on account of the want of a volumetric test. As we have before seen, however, this acid and its compounds formed a very frequent deposit even in those cases where the urine was not of more than average acidity. There is, therefore, some probability that the quantity would have been found increased.

The quantity of *chlorides* was considerably less than the average of health. In all cases, however, the quantity of chloride of sodium is subject to great variation, and we can easily see why it should be diminished in cases of general paralysis, when we consider that by far the larger proportion of it is derived from the food directly, and that patients in this condition are by no means fastidious as to the amount of condiments taken with their food. The average quantity of chloride excreted daily, as given by Dr. Parkes, is 11·471 grammes. In the six healthy men examined, I found the mean average of three days' excretion to be 8·495 grammes, the minimum being 6·025 and the maximum 11·993.

With regard to the other two constituents that were made the subject of analysis, namely, the *phosphoric acid* (P_2O_5), and *sulphuric acid* (SO_3), the latter did not differ much from the average of health, the quantity being in most cases, however, slightly increased. The phosphoric acid was uniformly diminished, and this was perhaps the most notable aberration from the healthy standard observed in the course of these investigations. It was certainly by far the most uniformly present. The mean average of phosphoric acid excreted daily in health is, according to Dr. Parkes, 3·162 grammes, that of sulphuric acid 2·016, the phosphoric acid being about a third more than the sulphuric. In many cases of general paralysis this relation was reversed, and in the majority of the cases the sulphuric acid was slightly greater. This result may have been due in some measure to the slight increase in the quantity of sulphuric acid itself, but much more to a diminution in the phosphoric acid. The daily average of phosphoric acid for seventeen cases was 2·218 grammes, that of sulphuric in the same cases 2·381; in other words, the quantity of phosphoric acid is nearly a third less than in health, while that of sulphuric remains unaltered or is even slightly higher. Subjoined is a table showing the average quantities of the various constituents examined in six healthy cases and in seventeen general paralytics, first without reference to weight, and then as excreted according to a definite body-weight.

TABLE IV.

Showing the Average Excretions in Six Healthy Cases and Seventeen General Paralytics.

	Daily Average		One kilogramme of weight excretes	
	Health	General Paralysis	Health	General Paralysis
Quantity	1658 c. c.	1565 c. c.	22.496 c. c.	23.310 c. c.
Urea	38.578 grms.	41.190 grms.	.5242 grms.	.6061 grms.
Na Cl	8.495 "	6.300 "	.1149 "	.0934 "
P ₂ O ₅	3.611 "	2.218 "	.0496 "	.0334 "
SO ₃	2.342 "	2.381 "	.0319 "	.0354 "

Under Calabar bean.—This remedy has been of late years recommended by Dr. Crichton Browne in the treatment of general paralysis, and has been extensively administered at the West Riding Asylum with some good results. In the twelve cases in which the urine was examined under its influence, the average results show that there is a general tendency to diminished excretion not only of the urea but of all the other constituents which were estimated. This was not an invariable consequence of the administration of the Calabar bean. In eight only out of the twelve cases was the average quantity of urea excreted less than the average of health. In the remaining four there was a slight increase. On the whole, however, there appears to be sufficient indication that, under the influence of this remedy, the tendency to increased excretion of urea is held in check to some extent. A glance at Table V. will show that, taking the average of the twelve cases, the difference in the results with and without Calabar bean is sufficiently marked.

TABLE V.

Showing the Average Quantities Excreted in Twelve Cases with and without Calabar Bean.

	Daily Average		One kilogramme of weight excretes	
	Without Calabar Bean	With Calabar Bean	Without Calabar Bean	With Calabar Bean
Quantity	1497 c. c.	1235 c. c.	22.061 c. c.	18.266 c. c.
Urea	40.585 grms.	34.325 grms.	.5864 grms.	.5202 grms.
Na Cl	6.961 "	4.013 "	.1027 "	.0582 "
P ₂ O ₅	2.180 "	1.928 "	.0321 "	.0285 "
SO ₃	2.299 "	2.086 "	.0339 "	.0308 "

Under the influence of alcohol.—The position of alcohol as an alimentary substance has been long subject of discussion, and there is still great diversity of opinion as to whether it is entitled to be ranked with articles of food possessing real nutritive value. It is contended by some that it passes out of the system in an unconsumed state, and therefore cannot be considered as possessed of any alimentary value. Such is the view taken by MM. Lallemand, Duroy, and others abroad, and by Dr. Ed. Smith in this country. On the other hand, it is believed by some that the principal part of the alcohol ingested undergoes consumption in the body. Liebig's view, based on chemical considerations, was that it is consumed by oxidation, like other non-nitrogenous alimentary principles, and this seems to be the opinion entertained by Drs. Dupré, Thudichum, Anstie, and others who have experimented on the subject. On the whole, as the evidence stands at present, there appears to be a balance of opinion in favour of the view, that the amount of alcohol eliminated as such in the breath and urine is only a small proportion of the entire quantity ingested, and that the chief portion undergoes consumption in the body. Apart, however, from the question of its alimentary value, alcohol exerts a notable influence on the various functions of the body. It is generally stated that it diminishes tissue metamorphosis, and therefore we should expect a diminution in the quantity of urea, as indicating the amount of tissue destruction. There is, however, considerable discrepancy in the results of the experiments of different observers on this point. The general opinion is in favour of a diminution of the urea. It will be seen, on referring to the three cases whose urine I examined under the influence of alcohol, that the results are in favour of this view. The average quantity of urea excreted daily without alcohol was 43·27 grammes, while that under the influence of alcohol amounted only to 34·243 grammes. Each of the other constituents examined are also slightly diminished, though the difference is not so marked as in the case of urea.

The results of my observations may be summed up in the following propositions:—

1. The quantity of urea varies above and below the average of health, being in the majority of cases considerably increased. Probably also the uric acid is increased.
2. The quantities of chlorides and phosphoric acid are notably diminished; that of sulphuric acid remains about normal.
3. The specific gravity varies within wider limits than in health, but the mean does not differ materially.
4. The absolute quantity of urine passed is slightly below the average of the healthy cases examined, but, estimated according to weight of body, the amount excreted by seventeen general paralytics was slightly in excess of that excreted by six healthy men.
5. Under the influence of Calabar bean there is a considerable diminution in the quantity of all the solid constituents, especially the urea.
6. The results obtained in the three cases treated with alcohol, are in favour of the view that both the quantity of urine and the amounts of solid constituents are diminished under the influence of that substance.

CEREBRAL ANÆMIA.

By J. MILNER FOTHERGILL, M.D. Edin., M.R.C.P. Lond.

ANÆMIA of an organ can be defined as a decrease in the actual amount of arterial blood passing through that organ in a given space of time. This is permitted by the power possessed by the blood-vessels of dilating and contracting, and so altering their calibre and permitting of a greater or less blood-flow through them. The nervous arrangements by which this alteration of calibre is accomplished are the vaso-motor nerves, and, according to Schiff, another network of nerves running over the arterial walls which possess an inhibitory action. The vaso-motor nerves produce contraction of the circular muscular coat of the arterioles; and the inhibitory nerves counteracting the action of the vaso-motor nerves produce dilatation of the vessels and enlargement of their calibre. By these means the vascular supply of a part is regulated according to its wants. Thus, when digestion is going on, the arterioles of the stomach become dilated, and the mucous lining becomes highly coloured and vascular—resuming its previous paleness when the act of digestion is completed. So it is with the brain—when functionally active, it is highly vascular; during sleep, it is pale and bloodless. That heightened condition of mental activity which is produced when the mind is bent to study energetically and determinedly, and of which we are acutely conscious, is coincident with a certain condition of increased vascularity, evoked by the action of the will upon the nerves of the cerebral blood-vessels through the cerebral cells. This is in harmony with the physiological axiom that the

functional activity of an organ is in direct proportion to its blood supply; and that a condition of vascular turgescence, amounting almost to erethism, is indispensable to the full working of every organ. When we remember these physiological facts, it becomes at once obvious that a condition of anæmia of an organ entails an impaired functional activity, and an enfeeblement of its working power; and that anæmia of the brain will exercise a profound influence over the working of that organ, not only as regards its physical relationships, but also as regards those psychical actions with which it is so intimately associated. We might *à priori* expect to find mental torpidity and depression, muscular lethargy, and an impairment of the organic processes of life, as the consequences of cerebral anæmia. Before this article is completed, we shall have seen that such are actually the outcomes of this condition. The mental states which are thus originated are by far the most important of the modifications so induced, and throw into the shade by comparison the physical sequels: though these latter are not themselves unimportant. The casual relationship of cerebral anæmia to some most important matters affecting our social life, will become clearly apparent as we proceed.

The first point to be considered in regard to cerebral anæmia is the possibility of its occurrence; for until this is demonstrated it is futile to proceed to the consideration of its consequences. The circulation within the encephalon is very active, and the vascular supply is unusually profuse, so as to admit of great functional power on the part of the intra-cranial contents. But the encephalon is enclosed in an unyielding bony case, the skull; how then, it may be asked, can it become more vascular at one time than another? Kellie and Dieckenhoff first announced that the skull was an inverted bell-jar, and that there could be no change in the actual bulk of its contents—a view espoused by Abercrombie, Hammernik, Reid, and Hughes Bennett. The experiments upon which this view was based showed that after fatal hæmorrhage there is still a quantity of blood retained within the skull, and that the brain is not so bloodless as the other parts of the body. Blumenbach first de-

monstrated that the vascularity of the brain is not always the same, and experiments to prove this have been performed by Sir George Burrows, Donders, Kussmaul, and Tenner, Arthur Durham, Hammond, and others—experiments which show conclusively that changes can take place, and do take place, in the vascularity of the encephalon; the brain becoming turgid during excitement, and in sleep, a period of functional quiescence, becoming pale and exsanguine.

These alterations in the circulation within the cranium are permitted by the presence of the cerebro-spinal fluid. When the brain becomes more vascular the amount of that fluid is lessened; when the turgescence passes away, the bulk of that fluid is increased. ‘As the cerebro-spinal fluid can readily find its way from the sub-arachnoid spaces of the cranial into those of the spinal cavity, and as it is no less readily absorbed than reproduced, it evidently serves as an equaliser of the amount of pressure within the cranial cavity; permitting the distension or contraction of the vessels to take place, within certain limits, without any considerable change in the degree of compression to which the nervous matter is subjected.’ (Carpenter, ‘Human Phys.’ § 567.) An interesting experiment is related by Hilton (‘Rest and Pain,’ p. 27), by which this is well illustrated. After an elaborate preparation in the dead subject, he found that an increase in the amount of blood within the cranium produced an outflow of the cerebro-spinal fluid, and that when the cerebro-spinal fluid was pressed back again into the cranium the blood was displaced and exuded. That the cerebro-spinal fluid permits of varying amounts of vascularity in the contents of the skull is now generally acknowledged; and that its ready effusion and absorption permit of quick and sudden vascular changes is also recognised.

But there is something more than a general back and forward play betwixt the intra-cranial vascularity and the cerebro-spinal fluid. The researches of Robin and His have demonstrated that there are in the substance of the brain channels along which the blood-vessels run; and their observations are corroborated by Lockhart Clarke and Batty Tuke. These channels are termed the ‘perivascular spaces,’

within which lie the blood-vessel and its accompanying lymphatic vessel surrounded by a clear fluid, containing nuclei, globules, and sometimes corpuscles. When the brain is quiescent, the blood-vessel lies quite free, surrounded by fluid ; when the brain is functionally active, the vessel dilates and the fluid is absorbed, so that the brain becomes highly vascular without compression of the nervous structures ; when the activity is over, the vessel contracts and the space betwixt it and the margins of the perivascular space is again filled with fluid. This action has not yet been actually seen ; but there can exist little doubt as to the truth of what is written here. The channels exist in the first place, while blood-vessels dilate and contract according to the functional activity of an organ ; and some such arrangement must necessarily exist in a bone-bounded space. Logical induction would indicate perivascular spaces if they had not already been recognized and delineated.¹ Obersteiner has detected lymph corpuscles in these perivascular spaces.

By this beautiful arrangement of one large reservoir with numerous channels perforating the brain substance, along which run the blood-vessels, it becomes possible for the brain to enlarge or become turgid with blood when in action, and to settle down into a condition of vascular quiescence during rest, within its bony case and without disturbance of its own proper structure. The fluctuations in the actual bulk of blood within the cranium are accompanied by corresponding fluctuations in the amount of cerebro-spinal fluid ; ‘ while in all instances in which the bulk of the brain has undergone an increase, whether from the production of additional nervous tissue or from undue turgescence of the vessels, there is either a diminution or a total absence of this fluid.’ (Carpenter, § 567.) In atrophy of the brain we find the *hydrocephalus ex vacuo* of Niemeyer. Having determined the primary question of the possibility of cerebral anæmia, we can now proceed to the further consideration of the actual state of the brain itself and its blood-vessels, in this condition. When anæmia comes on, whether induced by sleep or by

¹ See a woodcut in the writer's paper in the last vol. of these Reports.

compression of the carotids it matters not, the brain sinks down and becomes pale in colour. The vessels become less and less distinct as their blood-current is diminished; especially is this the case in the arterial vessels, but the veins on the surface also become smaller and less distended. There is a general diminution of the vascular area of the brain, and a quiet, placid blood-flow. Such is the actual condition of matters in cerebral anæmia; and a return to the normal state is accompanied by a blush on the surface of the brain, a filling of the vessels with blood; the smaller vessels which had become invisible to the eye are again recognizable, and the veins which had contracted to an abnormally small calibre resume their ordinary diameter and capacity. These observations, made by means of pieces of watch glass luted into the skulls of animals (rabbits and dogs), are corroborated by the revelations of the ophthalmoscope as to the normal condition of sleep and as to distinct states of cerebral anæmia during wakefulness.

The anatomical appearances in cerebral anæmia according to Niemeyer are as follows:—‘The substance of the brain is discoloured; the grey substance appears paler and more resembles the white. The latter is very milky and shining. On section, few if any blood points are seen in the cut surface. The vessels of the cerebral membranes are empty and collapsed. We do not always find a considerable amount of fluid in the sub-arachnoid space. Kussmaul and Tenner could not prove, on examination, any increase in the cerebro-spinal fluid, which, on theoretical-grounds, they had expected to find.’ There is some discrepancy here betwixt Niemeyer and Kussmaul and Tenner, who themselves say, ‘Lastly the experiments according to the method of Donders did not afford any explanation of the increase and diminution of the quantity of serum in the brain and membranes in cerebral anæmia or hyperæmia. There is certainly every justification theoretically for the assumption that the quantity of serum will increase and decrease inversely with the quantity of blood.’ The experiment of Hilton, before alluded to, distinctly bears out this inference, and demonstrates the reality of the relations, in amount, betwixt

the cerebro-spinal fluid and the blood within the cranium. The pathological changes when atrophy or brain-wasting follows the condition of cerebral anæmia will be given further on, when the progress of the disease is considered.

Cerebral anæmia is induced by different and various means, which may be given forthwith; leaving the diagnosis, to be discussed after the exact nature of the change, according to causation, has been detailed. The conditions under which it is found are as follows:—

A. General anæmia. B. Spanæmia. C. Unfilled vessels. D. Disease of the heart. E. Pressure on the Cerebral Vessels. F. Embolism. G. Venous Stasis. H. Apoplexy. I. Gouty spasm of the Cerebral Vessels. J. Organic Disease involving the Cerebral Vessels. K. Vaso-motor disturbances of the Cerebral Vessels. L. Artificial Production by Medicinal Agents.

A. *General Anæmia.*—This affects the brain by decreasing the amount of arterial blood flowing through it; and in so vascular an organ any diminution in the general bulk of blood is readily felt. Haller has estimated the amount of blood going to the brain as one fifth of the whole bulk of the blood. Any reduction then in the general amount of blood must soon be apparent in the encephalic circulation. Consequently we find cerebral anæmia associated with constant drains upon the system, as piles, menorrhagia, or lactation, rapidly growing morbid growths, &c.; and with imperfect nutrition, as imperfect supply of food, dyspepsia, &c.

B. *Spanæmia.*—This is also a cause of cerebral anæmia, and arises in various ways, as for instance in malarial poisoning; reabsorption of fæcal matters; lead poisoning; syphilis, bile products in excess, lithiasis, or in any form of blood-poisoning which hinders the formation of blood corpuscles; the excessive use of acids or alkalies; the prolonged administration of mercury, copper, &c. Acute fevers also often lead to the breaking down of the red blood corpuscles and the production of a spanæmic condition, which,

in its turn, exereises a deleterious action upon the different tissues including the highly vascular brain.

C. *Unfilled Vessels.*—In this condition of the vessels we find a very efficient cause of cerebral anæmia in its truest sense. When the blood-vessels are but insufficiently filled by the bulk of the blood, the head, placed at the highest point of the organism in the ordinary postures of sitting and standing, is deprived of its blood supply for the blood falls away from it, by the force of gravity, into the more dependent parts. In this form of cerebral anæmia is the effect of posture most marked. The symptoms are all aggravated on standing up, and relieved when the recumbent posture is again assumed. Handfield Jones quotes a most instructive case from Abererombie illustrating well the effect of posture. The case was that of a gentleman of thirty, who was greatly reduced by a complaint of the stomach. ‘As the debility advanced he became very deaf, and this symptom varied in the following instructive manner. He was very deaf while sitting erect or standing, but when he lay horizontally with his head quite low, he could hear very well. If when standing he stooped forwards, so as to produce flushing of the face, his hearing was perfect; and upon raising himself into the erect posture, he continued to hear distinctly as long as the flushing continued; as this went off, the deafness returned.’ In cerebral anæmia of this form the effect of change of posture is very marked, and in exsanguine invalids swooning, vomiting, and other evidences of a failing circulation within the encephalon are induced by merely raising the head from the pillow, to say nothing of graver changes of posture. Under conditions of less profound anæmia, persons more readily faint when standing than when in the recumbent posture. This condition of cerebral anæmia is not always the result of general anæmia, from which it is intentionally separated, but may be occasioned by an excess of blood in other organs. Thus Niemeyer describes it as occurring from the use of Jounod’s boot; and we are all practically familiar with its occurrence after tapping of the abdomen or the emptying of a long distended bladder, where dangerous syncope is apt to come on

if the recumbent posture be not carefully maintained. Indeed it is sometimes necessary under such circumstances to maintain artificially a certain pressure upon the abdominal veins, which would otherwise become so full of blood as to cause syncope, sometimes so grave as to endanger life. This is the more necessary to be kept in remembrance as 'the intestinal vessels are so capacious that when they are fully dilated they can hold all the blood in the body' (Brunton on Shock and Syncope. 'Practitioner,' 1873).¹ A condition of general venous fulness is also found in adynamic conditions of the heart, with or without actual valvular disease. Here as in the above-mentioned condition of fulness of the abdominal veins, the effect of posture is distinct. It is usual to ascribe the syncope which follows the assumption of the erect posture in these cases to the inability of the left ventricle to lift the increased weight of the column of blood now thrown upon it, and there is no doubt that that is an important factor; though the force of gravity upon the general blood column in the veins must not be overlooked, and is a not insignificant element. The blood gravitating into the abdominal veins is not passed over by the right ventricle to the left, and a temporary arterial anæmia follows. Probably the syncope is due sometimes to the first factor, and at other times to the second.

D. *Disease of the Heart.*—This is often a direct cause of cerebral anæmia, as we have just seen. Fainting, more or less complete, and varying degrees of impaired functional derangement of the brain, are the consequences of cardiac inability. In the case of an old lady sudden attacks of cerebral anæmia of an acute character came on whenever she over-exerted herself, or was emotionally perturbed. There was marked aortic stenosis. Rest and cardiac tonics always and quickly relieved her. In another case of congenital malformation of the heart, the functional activity of the brain rested almost upon position. The recumbent posture was indispensable to the committing to memory of pieces of poetry, and even to the recalling of them, when the

¹ This was found by Goltz in Virchow's 'Archiv,' xxvi. p. 11, and xxix. p. 394. This dilatibility of vessels is described by Schiff in 'La Nazione,' Aug. 9, 1872.

youth was desired to repeat them. Dr. Langdon Down tells me of a case where a wealthy old lady was very much depressed by the idea that she had no clothes to wear. He found the heart much dilated and acting irregularly. The administration of digitalis restored the heart's rhythm and improved its contractions, and in a few days the delusions had fled. The relation of cerebral symptoms to disease of the heart is much insisted upon by Stokes, and the term he uses for acute attacks of cerebral anæmia is that of 'pseudo-apoplexy.' He relates a most striking case of a man with aortic disease and a weakened heart who was able to ward off the coming attack by hanging his head downwards. Mitral stenosis and fatty degeneration of the heart are commonly associated with swooning, vertigo, and the other consequences of an imperfectly sustained intra-cranial circulation.

These acute attacks of cerebral anæmia occurring during heart disease have rather curiously been regarded by some writers as of a genuinely apoplectic character; and venous fulness by presenting a *vis à fronte*—by impeding the arterial flow, has been credited with the production of true apoplexy from arterial rupture. This is obviously improbable on the face of it; for venous fulness implies and necessitates the condition of arterial emptiness, except in cases of general increase of the bulk of the blood, as in hydræmia, and the attacks are really 'pseudo-apoplectic.' The attack may be fatal, but it is not apoplexy when arising under the circumstances of heart failure; it is not an attack of apoplexy proper, but of acute cerebral anæmia, or lack of arterial blood in the brain.

E. *Pressure on the Arteries supplying the Brain.*—The effect of pressure upon the carotids in producing cerebral anæmia of various degrees of intensity according to the amount of pressure is not only known scientifically, but has found a practical illustration in the *modus operandi* of the garrotter, by which complete unconsciousness is quickly secured. Kussmaul and Tenner describe a long series of operations upon the ascending blood-vessels in the neck with their results. In all of them there were evidences of cerebral

anæmia, in some form or other, as swooning, convulsions, paralysis, &c.

F. *Embolism*.—The washing of a portion of a blood clot into a cerebral vessel is an effective measure for cutting off the blood supply along the distribution of that blood-vessel. Sudden paralysis is thus not uncommonly induced. The commonest form is that of right hemiplegia with aphasia induced by a clot in the left middle cerebral artery. The anatomical explanation of the frequency of this affection is that the left middle cerebral artery lies in the straightest line from the heart, and so the clot is borne by the blood current into that particular locality. Local softening of the brain is the usual outcome of embolism.

G. *Venous Stasis*.—This is not an uncommon cause of arterial anæmia of the brain. ‘It is not merely the presence of blood in the vessels of the brain, but the supply of oxygenated arterial blood that is indispensable for the normal functions of the organ: it is evident that, even where the absolute amount of blood in the brain is not diminished, but where its circulation and distribution are changed so that only a small amount of blood enters through the arteries, and but little escapes through the veins, the same symptoms must arise as in true anæmia.’ (Niemeyer.)

H. *Apoplexy and other Forms of Pressure*.—Niemeyer is also inclined to regard the symptoms of pressure in apoplexy, tumours, extravasations, effusions, and other diseases which encroach upon the intra-cranial space, as due to the consequent anæmia of the compressed nerve mass, rather than to any pressure upon the nervous tissue itself interfering with or arresting its function. He further states that Traube, Leyden, and others, have adopted his conclusions. The weight of evidence is certainly in their favour.

I. *Gouty Spasm of Cerebral Vessels*.—In all cases of the retention of effete products in excess, and especially the products of tissue metamorphosis, the vaso-motor centre is affected, and spasm of the muscular walls of the peripheral arterioles results. This general condition is evidenced by the hard sustained pulse, the characteristic sphygmographic tracing, and the large bulk of pale urine of low specific

gravity, indicating a high blood pressure on the glomeruli. At first the blood supply to the brain is well maintained by the high arterial tension and the slight resistance offered by the thin muscular walls of the encephalic vessels, and such persons often possess high mental powers and capacity for sustained exertion, while at the same time they are hasty and irascible. The powerful hypertrophied left ventricle, and the accentuation of the second sound produced by the forcible closure of the aortic valves under high arterial tension, tell of arterial fulness. 'Persons who suffer from active hypertrophy of the heart with enlargement of the carotids, and in whom more blood flows to the brain, are for the most part excitable, and come easily into ebullition' (Schroeder v. der Kolk). But as the disease progresses, and the arteries become atheromatous, and the heart walls are structurally undermined by fatty degeneration, the blood supply to the brain becomes less abundant, and ultimately even insufficient at the best; while it is liable to fits of passing diminution, owing to arteriole spasm, and then conditions of marked anæmia are induced. Irascibility is the characteristic *par excellence* of a brain fed with blood laden with gout-poison, and hastiness is habitual; but as the condition of heart failure becomes slowly developed and superimposed upon the gouty condition, the characteristics of cerebral anæmia are blended with those of lithiasis. The psychical results of this complex condition will command our attention further on.

J. *Organic Disease involving the Blood-vessels.*—In certain forms of cerebral disease, the blood-vessels become involved, their walls are thickened and their lumen diminished, and consequently the blood current through them is distinctly reduced in volume. In his 'Morisonian Lectures' this year Dr. Batty Tuke, in speaking of cerebral lesions in syphilitic lunatics, says: 'You will notice that the arteries are more or less thickened as to their muscular coat, and more especially as to their outer fibrous coat, the latter surrounding the vessels in concentric rings for a very considerable extent; it appears also to be cemented, as it were, by a viscid gummatous material in which amyloid bodies are occa-

sionally seen. The effect of this thickening is to occlude completely the vessels in many instances, and in all to materially modify their calibre.' Dr. Herbert Major, of the West Riding Asylum, also informs me that in numerous instances he has had occasion to notice the implication of the cerebral blood-vessels in general disease of the brain, and the diminution of their calibre thereby. Whatever may be the symptoms of the original condition which instituted the disease of the blood-vessels, the consequences of cerebral anæmia must ultimately be united to them, and the indications of the primitive disease be tinted with the results of an insufficiency of arterial blood.

K. *Vaso-motor Disturbances of the Cerebral Vessels.*—This division is separated from that of gouty spasm by design, as it would have been confusing to have included the effects of irritants upon the vaso-motor centre with vaso-motor conditions of disturbance arising through the sympathetic nerve. By keeping the two entirely distinct, one source of confusion is avoided.

We are all familiar with the relations of the sympathetic nerve and the vaso-motor nerves of the different arteries. Emotional conditions manifest themselves through this complex mechanism, as in the blush of shame and the pallor of fear. These emotional states, acting through the sympathetic and the vaso-motor nerves, alter the calibre of the peripheral arterioles, and on this the change of colour of the face depends. We also know that section of the sympathetic in the neck produces dilatation of the blood-vessels of the head; and that irritation of the upper cut surface produces contraction of the vessels and pallor. The branches of the common carotid are in close relationship with the cervical portion of the sympathetic, and the internal carotid is intimately associated with the ascending branches of the upper cervical ganglion. These nerve fibrils may be traced along it into the vessels of the pia mater and the orbit. On the other hand the vertebral arteries are supplied with vaso-motor fibrils from the inferior cervical ganglion.

The relations of the carotid and vertebral arteries to the

different portions of the brain are not by any means unimportant, especially since the localization of function in the brain has been established by the brilliant observations of Dr. Ferrier. The empirical guesses of Gall and Spurzheim, and the more accurate pathological observations of Schroeder van der Kolk, are now having direction and precision given to them by the close clinical studies of Hughlings Jackson, and the direct experiments of Fritz and Hitzig, and still more of our countryman Ferrier. There is no longer a standing ground remaining for those who might still feel inclined to hold the ancient views as to the brain being a general nerve mass divisible into cerebrum, cerebellum, pons, medulla, &c. The cerebrum is being gradually mapped out until the distinction in function betwixt Broca's convolution and those which lie behind the fissure of Rolando is as palpable as the difference in function betwixt the medulla oblongata and the corpus striatum. The relations of the arteries of the brain to their regional distribution are now invested with an interest they have not hitherto possessed.

The internal carotid will be seen to give off the posterior communicating branch of the circle of Willis, after that the anterior choroid dipping into the interior of the cerebral mass, and then dividing into the anterior and middle cerebral arteries, from the latter of which pass numerous small branches to the corpus striatum. The anterior portion of the brain and the eye, the middle portions of the brain with the pia mater over them, are supplied by the branches of the internal carotid. On the other hand the vertebral arteries supply the cerebellum partly before they unite, partly after,—the pons and medulla being supplied by the basilar artery, and so most positively secured as to their blood supply by the presence of the circle of Willis—and then the two branches of the basilar artery, the posterior cerebral, after taking up the communicating branches, pass onward and supply the posterior portions of the brain. To put it broadly the carotid supplies the anterior and middle portions of the cerebrum, the vertebral the posterior portion, while the parts at the base and interior of the brain are fed by branches from the circle of Willis and the basilar

artery, and are so secured as far as may be against the risk of being cut off from their supply of arterial blood.

From the relations of the sympathetic and the ascending blood-vessels in the neck it follows that the vaso-motor nerves of the blood-vessels of the anterior and middle portions of the cerebrum are derived from the upper cervical ganglion, while the blood-vessels of the posterior portion of the brain are derived from the inferior cervical ganglion, which is in close communication with the abdominal portion of the sympathetic. This point is of some importance, as we shall see when we come to the different effects produced by disturbance in the blood supply of the different portions of the brain—albeit there is no forgetfulness of the free inosculations of the blood-vessels, and doubtless of their vaso-motor fibrils in the encephalic mass.

It is very obvious from the foregoing observations that the brain is in close relationship with different emotions by their effect upon the sympathetic and through it again with the cerebral blood-vessels. The blood-vessels of the brain are in the most direct line from the heart; and from this fact, from their size and the rapidity of their blood flow, the functional activity of the brain is closely bound up with the force of the heart's action and the conditions of the circulation. Depressing emotions affecting the emotional centres and the action of the heart lead to diminished functional activity of the brain, while exhilarating emotions exalt the action of the brain. If the depressing emotion be pronounced, it is easy to see how it may be perpetuated, and the first shock be continued by a sustained condition of cerebral anæmia from an enfeebled circulation.

Let us take *shock* for example. Whether shock be physical or psychical, it exercises a profound impression upon the vascular system. According to Mr. Savory, 'Shock is the paralyzing influence of a sudden and violent injury to nerves, over the activity of the heart.' This is acute surgical shock; but a more chronic and less marked condition of shock possesses the same action in a modified form. The symptoms of shock are pallor and coldness of the skin, weak pulse, oppressed and sighing respiration, dilated pupils, and sickness

(Brunton, *op. cit.*). There is obviously here a profound impression made upon the brain through the sympathetic, and the shock to the circulation is felt very markedly in the vascular encephalon. Any severe and unpleasant surprise is usually accompanied or immediately followed by diminution in the mental activity, a partial imperfect paralysis, a loss of self-possession. It may be that the shock falls upon the brain itself in the first place, or rather upon the cortical cells, and thence is transmitted to the organic nervous system; instead of upon the sympathetic first, and then secondarily upon the brain. And this at once leads us to the relation of the cerebral cells to the intra-cranial blood supply on the one hand, and to the manifestations of cerebral activity on the other.

It must be distinctly understood that no attempt is here made to establish a theory that varying conditions of mind, of exaltation and depression, either in the sane or the insane, are simply and solely dependent upon the amount of arterial blood passing through the cerebral mass. We must admit that the cerebral cells possess the power of regulating their blood-supply according to their functional activity, as much, or perhaps more than other tissues; still we must equally admit that the alterations in the blood supply affect their functional activity, and a full supply of blood evokes functional activity in the cerebral cells,¹ while a scanty supply of blood is followed by impaired brain action. Bucknill and Tuke in speaking of the relation of vascular and cellular changes express themselves very distinctly on this subject, and to the following effect:—‘In most instances of insanity arising from physical causes it is probable that the pathological condition of the cerebral cells is subsequent to, if not dependent upon, the pathological condition of the cerebral capillaries. It is unnecessary to go through the roll-call of the physical causes of mental disease: suffice it to say that

¹ ‘It is a known fact, that deformed, hunchbacked individuals, in whom the blood flows more quickly and strongly towards the brain, are remarkable for vivacity of spirit. Persons with long necks are mostly quieter and slower, those with short necks more lively, more restless and impetuous; but in this again there are exceptions.’ Schroeder van der Kolk. These views were also held by Bichat.

injuries to the head, fever, suppressed discharges, alcohol, and other noxious agents can only influence the cerebral cells through the medium of the capillaries. With regard to many other physical causes of mental disease, it is scarcely possible to doubt that a pathological condition of the cerebral vessels is not only an essential condition of the disease, but that it is also one which takes place in order of time antecedent to any pathological condition of the cerebral cells.' The whole consideration of the relations of the brain to its blood-supply by these writers is most excellent. In introducing the subject of cerebral anæmia they state, 'The efficient cause of numerous cases of insanity is actual loss of blood, or a deficiency of its nutritive powers, occasioned by insufficiency of food, or by impediments to the conversion of food into healthy blood; or by the numerous anti-hygienic influences which limit the quantity or weaken the nutritive quality of the blood.'

Griesinger says: 'Chronic constitutional diseases very frequently cause the development of insanity. Of these we may place in the first series all states of weakness and anæmia resulting from great loss of blood (in childbirth for example), from continued hunger and misery, from self-inflicted fasting (religious asceticism of former times), after too prolonged nursing; finally, in consequence of the most various general and local maladies which impair digestion, blood-formation, and nutrition. Anæmia likewise plays a very important part in the production of a number of other neuroses; and we see that even within the limits of physiology, a bodily condition in which the nutrition is lowered renders more easy a state of irritation in the functions of the nervous system, more or less disturbance of sleep,' &c.

After these references to the writings of alienist physicians, the importance of an adequate supply of healthy blood to the proper action of the cerebral cells becomes sufficiently apparent. For if those advanced conditions known as insanity can be so produced, minor conditions not entailing mental aberration will undoubtedly often be so occasioned, and the action of the cerebral cells be crippled by an insufficient supply of arterial blood in other individuals than the

inmates of asylums. 'The state of the blood in the minute vessels is the circumstance lying nearest to that ultimate molecular change resulting in functional activity, with which until recently our powers of observation have been able to make us to some extent acquainted.' (Bucknill and Tuke.) Whether then in shock or other depressing conditions the disturbance in the nutrition of the cerebral cells is the first step, or whether there is first produced an alteration in the cerebral capillaries, and secondarily an impaired blood supply to the cells in the cortical substance, it is quite certain that there is an impairment of the blood supply; and that this is causally related to the enfeebled and diminished functional activity of the brain. In conditions of mental depression, in acute dementia, and in melancholia, the circulation is distinctly affected, as we shall see hereafter.

But while we can thus see how depressing emotions or vascular conditions can affect the functional activity of the brain, and understand quite clearly the influence of extrinsic causes in producing a diminished circulation within the encephalon, and especially the cerebral hemispheres, there is still left another factor that must not be omitted, and that is the diathesis of the individual. The reason why troubles exercise but a transient effect upon some, while the impression made upon others is permanent, and not rarely ineffaceable, lies largely in some original diathetic condition. There are those who are habitually sorrowful and depressed, often with small hearts and languid circulations, in whom lesser causes of depression act with greater effect, and in whom the recuperative power against such depressant action is but imperfect and inert. Others again of the sanguine and arthritic diatheses soon react against depressing forces: they are hopeful, buoyant, and to some extent irrepressible; they recover from conditions of depression comparatively quickly, and with strong hearts and a well-maintained circulation they press onward, strong and self-reliant.

Much too lies in the past history of the individual—that past which exercises so decided an influence over all of us. There may be, and often are, in that past, long stretches of

physical ill health, or psychical disturbance; long mental strains over what may appear to others trivial matters, but invested with much importance by the individual himself; an erroneous or mischievous education; disappointed aims and hope deferred; or the actual presence of slow, exhausting, debilitating disease, or psychical overstrain; and then, when the immediate exciting cause comes, the system yields before it, powerless to resist or withstand it. Under such circumstances those extrinsic disturbances which evoked from Pinel the invariable question, 'Have you suffered vexation, grief, or reverse of fortune?'—a question, Griesinger says, as often answered in the affirmative in his experience as in Pinel's—produce a profound impression; like that of an acute disease upon a person in broken health, or a sudden demand on a man already beggared and bankrupt.

L. *Artificial Production by Medicinal Agents.*—A condition of cerebral anæmia with its consequences, psychical and physical, is not uncommonly produced by the administration of remedial agents, especially by the large doses of bromide of potassium now in vogue. The symptoms, as given by Nothnagel, are loss of memory, confusion and torpidity of thought, a tardiness in comprehension and answer, as if the nerve messages travelled but slowly, and depression. Several cases are related in the *British Medical Journal* for 1869-70, illustrating this; in one case the use of 'malaprop' words was the distinguishing feature, and a diminution of the dose was at once followed by the disappearance of this pathognomonic symptom.

Conditions of cerebral anæmia are now deliberately induced by the use of agents which depress and slow the heart's action, as for instance the Calabar bean, in states of cerebral hypervascularity; and for this purpose Dr. Crichton Browne has used the physostigma to control the wild outbreaks of general paralysis; and Dr. Forbes, of Shoreditch Infirmary, has employed it in the treatment of acute maniacal conditions with benefit and satisfaction. The idea is not new, and the same end was sought of old by free bleedings or the administration of huge doses of tartar emetic. By these

means not only the mental excitement, but the violent muscular movements are controlled, and that, too, evidently by diminishing that blood supply on which the activity of the cerebral cells rests.

Diagnosis of Cerebral Anæmia.—For the elucidation of this important matter, the subject may conveniently be divided into two sections: (1) acute cerebral anæmia; (2) chronic cerebral anæmia. In this latter division it will be necessary to consider the condition both in the sane and insane, as the more pronounced conditions are only to be met with, on a sufficiently large scale to make the observations trustworthy, in the wards of our public asylums.

(1.) *Acute Cerebral Anæmia.*—In its most pronounced form this condition forms the pathological basis of the syncope, or fainting fit, with which we are all so familiar, where the sudden failure of the heart's action leads to arrest of the cerebral function from cessation of the flow of arterial blood—a condition very commonly produced by surprise or other emotional exciting cause, in women chiefly. We recognise the same condition in the loss of consciousness and muscular power which follow ligature of the carotids, or compression of the brain by an apoplectic clot, or the sudden shutting off of the arterial current by embolism. The symptoms are loss of consciousness, loss of muscular power, pallor of the face, dilatation of the pupils, and a sighing respiration. Where the anæmia is less instantaneous, we have convulsions. The diagnosis of acute cerebral anæmia is usually not particularly difficult. There is also a condition of what may be termed sub-acute cerebral anæmia, a condition lasting over a few days at the termination of an acute disease, ere the convalescence is inaugurated and established. 'Abercrombie describes how children may lie, for a day or two, in a state of stupor closely resembling coma from organic disease—insensible, with dilated pupils, eyes open and insensible, the face pale and the pulse feeble, and yet recover under the use of wine and nourishment. This state is induced by causes of gradual exhaustion, going on for a considerable time. It may occur in adults, though less frequently than in children.

The features of the condition will sometimes assume the following aspect, when occurring after sharp hæmorrhages. There is a blanched condition, with headache, flashes of light before the eyes, or noises in the ears, (of which the gravest perhaps is the ringing of bells); confusion of thought and loss of memory, with great muscular prostration. That is to say, to the condition of brain failure from arrest in the arterial flow are added certain positive symptoms which are allied, in character and causation, to the hallucinations or delusions commonly found in the graver forms of the chronic condition in asylum inmates.

Another condition of subacute cerebral anæmia corresponds with the frontier line which lies betwixt the actual typhoid condition of acute disease and the establishment of convalescence. The delirium and coma are gone, and the intelligence is coming back, fitfully and irregularly, like the bursting of sunlight through the rifts in dark clouds on the breaking of a rain storm; the returning apprehension being marked by a capricious irritability and petulance, ever hailed as a good indication by experienced nurses. The little trivialities of the *ménage* of the sick room are of paramount importance to the shortened mental vision; the outer world is a distant unknown land lying without the present sphere of the intelligence; and the exhausted brain cells feebly supplied with blood, itself only commencing to regain its normal purity, act but slowly and sluggishly, and only tediously recover the broken strands of the interrupted consciousness. Usually the returning intelligence bridges over the numerous solutions of continuity without much delay, but at other times a condition of mental imbecility, of enfeebled intellectual power, obtains for some considerable time.

(2.) *Chronic Cerebral Anæmia*.—This condition may be either a continuation of the acute condition, or it may come on gradually without any preliminary stage which is entitled to be called acute. Here there is a blunting of the mental perceptions, a loss of tone in the action of the brain centres; an enfeeblement of the general intelligence, and a languor in the muscular movements. At times, however, there is a species of irritable and adynamic activity in the anæmic

brain, which manifests itself in an uneasiness and restlessness not altogether dissimilar to the movements of a caged animal. Nor is the idea of a psychological resemblance so very strained. In each there is a recognition of the being controlled by external forces; in the animal by the bars of its cage, in the other case by some vague, undefined, external restraining force. But in time this condition either melts away in returning mental vigour, or is merged into a condition of mental torpidity and diminished muscular movement. This irritable condition varies with the severity of the anæmia: it is profound in some melancholics; it is less pronounced, but still distinct, in many of those slighter conditions of cerebral anæmia which are compatible with an unrestrained existence.

In addition to this general condition, there are numerous objective signs and subjective symptoms of high diagnostic value, which may be profitably considered and appraised *seriatim*.

The condition of the pupil is perhaps one of the most generally constant indications; and dilatation of the pupil, speaking now very generally and broadly, is one of the commonest accompaniments of cerebral anæmia. It is not the place here to consider at length the very difficult and complex question of dilatation of the pupil, with those varied causation and curious relationships, which constitute it one of the most vexed questions of medical symptomatology; it is enough to say that commonly the condition of cerebral anæmia is accompanied by dilated pupils, and as improvement proceeds, the pupils diminish in size.

Pallor of the face is rarely wanting in cerebral anæmia. The face possesses a large and extensive vascular supply, and pallor of it indicates a distinct modification of the condition of the circulation, whether it takes its origin in a physical condition, as in hæmorrhage, or in a psychological state, as in fear. It is pronounced in fainting, in ligature of the common carotid, and in other conditions of impaired arterial blood supply. Pallor of the face indicates lack of arterial blood, while duskiness and lividity are the indices of venous fulness. When a deep impresson on the sympathetic involves the

innervation of the heart, and impairs the ventricular contractions, pallor of the face and forehead follows as surely as cerebral anæmia; they are alike the outcomes of a condition which stands to each in a causal relationship, and consequently they are usually found together.

Pallor of the eye is also a diagnostic sign of value. Dr. Handfield Jones says, 'the state of the eye is, perhaps, a more sure guide than that of the skin. A red, injected, eager eye can scarcely imply anything else than a hyperæmic brain; and a pale, dead white languid eye almost certainly indicates the reverse.'

Ophthalmoscopic Signs are very valuable, and the condition of the eye tells of the vascular condition of the encephalon, the blood supply of each being derived from a common source. In conditions of cerebral anæmia there is in the eyeball a state of retinal anæmia. The optic discs are pale, but it is not the brilliant pallor of atrophy—the tint of the choroid is dull, and there is a want of that distinctness and sharpness in the picture which is such a marked feature of atrophy. Another distinguishing condition between this anæmic state of the disc and atrophy is to be found in the uniform greyish-white appearance of the disc, and the fact that one disc is in exactly the same condition as the other, and also that no partial anæmia of one optic disc can ever be seen. So that, should there be vascularity of a portion of the optic disc, and whiteness of the remainder, we may be pretty certain that we have not to do with anæmia. Such is the description given by Dr. Aldridge in his article on 'The Condition of the Eye in Acute Dementia.' The same observer kindly examined for me some of the melancholics in the West Riding Asylum, who were being examined by me in order to illustrate several points in this paper. In these he found that 'the arteries were small, and the veins of normal size, and that there was an absence of choroidal glow.' The ophthalmoscope lights up the pathology of cerebral conditions as regards the vascular system very distinctly, and furnishes most valuable diagnostic indications.

The Expression of the Facial Muscles is not without value in this cerebral condition. There is a mingled look of sad-

ness and general impairment of expression. The only positive expression is that of gloom. In some cases it is rather the shadow of sadness falling across expressionless, immobile features; at other times the leading impression, mental pain, is stamped upon the countenance, and we can say

This man's face, like a tragic leaf,
Foretells the nature of a tragic volume.

Indeed, the muscular system generally is affected, and the movements are languid, feeble, and constrained. The impairment of cerebral activity is seen in the loss of tone of the muscles, and the disinclination towards movement generally. In some cases the inner disquietude reveals itself in general physical restlessness; in others fixed painful ideas are accompanied by sustained action of the corrugators of the eyebrows, and the depressors of the angles of the mouth; these combined states of allied activity contrasting strongly with the general mental and muscular lethargy.

The General State of the Circulation is a matter of the utmost importance in the consideration of cerebral anæmia, not only from a pathological, but from a diagnostic point of view. In order to be able to speak with a fair claim to be heard on this matter, extensive observations as to the condition of the circulation in various forms of insanity have been made by the writer in the wards of the West Riding Asylum. In a paper in the last volume of Reports, the relations of the vascular system to conditions of encephalic hyperæmia were considered.¹ It was found that in hyperæmia there was a firm pulse, high arterial tension, a strong heart with loud sounds, especially the aortic second, and a well-maintained blood supply in the brain. Such a condition of the vascular system is associated with stages and states of mental exaltation, and activity of the cerebral cells. But a totally different state of matters is found along with conditions of

¹ 'The Heart-Sounds in General Paralysis.' Much point was given to these observations by the paper of Dr. Wilkie Burman on 'Heart Diseases and Insanity.' He gave the cases where insanity is associated with disease of the heart, with cardiac inability, and our observations mutually corroborate each other. Vol. III. 1873.

cerebral anæmia and mental depression. There is just that opposite condition which the foregoing observations had led one *à priori* to expect. If a condition of vascular turgescence is essential to functional activity, and this is universally admitted, a condition of anæmia will be equally allied with functional lethargy; and to this rule there is no exception in the case of the cortical substance of the brain. But it was obviously insufficient to assume such a state of matters, and the synthetic workings of the moral consciousness alone were quite inadequate to the task of inducing a well-founded personal conviction, to say nothing of the necessity for direct evidence, in order to satisfy the minds of others: so an extensive series of observations were made on two several occasions, each extending over several days, and including the examination of several scores of patients, most of those who were seen on the first occasion being re-examined on the second visit, many of them being further examined by Dr. Aldridge with the ophthalmoscope, while Dr. Lauder Brunton took pulse tracings from them with the sphygmograph.

The condition of the vascular system was distinct. The heart's impulse was slight; the first sound was generally thin, short, and valvular, more distinct towards the apex, and very low over the base; the muscular factor of the first sound was much impaired; the second sound was thin and slight, contrasting very markedly with the second sound in mania and general paralysis. There were in some, uncompensated valvular lesions, dilatation of the ventricles, or fatty degeneration of the muscular walls; but these were mere allied conditions, and in no way essential; in the great bulk of the cases they were entirely wanting. There was no change to be detected by percussion—indeed the change in absolute bulk did not proceed to the extent that rendered it recognisable by percussion, that is in the hands of a sober observer, though perhaps it might have been discernible enough by the enthusiastic devotees of physical examination, the inspired expounders of the minutæ of physical signs. The essential points were an adynamic condition with impaired functional power. The arteries were small, diminished

in volume, and the pulse was feeble and easily compressible. The following tracing is that of a typical case.



Nor is this condition of unfilled arteries necessarily accompanied by evidences of the opposite condition of general venous fulness. There seems to be a general diminution of the bulk of blood of the entire area of the vascular system, the curtailment of the area being very distinct in advanced or marked cases. The condition conveyed strongly the impression of an atrophy of the vascular system, if such a phrase be permissible; the encephalic circulation being markedly involved, both in the diminished calibre of the blood-vessels and in the enfeebled blood current. The surface generally was cold, and the temperature low. Though no especial observations were made on this point, it is well known that the temperature of melancholics is pretty constant, and slightly below the normal average. If this latter were not the case, and the surface and extremities were kept normally warm by a free blood flow through them, the general internal temperature would soon be so reduced that it would no longer be compatible with life. The small loss of heat by the cool surface is the condition absolutely necessary to the maintenance of a proper internal temperature.

Cold Hands.—In these conditions the extremities are seriously implicated, and both the hands and feet are cold, indeed sometimes chillingly cold, and often blue from venous stasis. Especially is this the case where there is absence of exercise, and much muscular lethargy. The hands attract attention at once—they are cold from lack of arterial blood and loss of heat, and blue from venous congestion. The general low temperature and defective nutrition affect the extremities very distinctly. The patient is generally thin and wasted from defective nutrition and exercise; the skin is dry,

and often withered or wrinkled, and loses its freshness. The altered skin, the wasted muscles, the lack-lustre eye,¹ and the blue hands, form together a picture, which is characteristic and expressive.

Drowsiness is one of the most marked symptoms of cerebral anæmia, and is commonly combined with sleeplessness; of the connection of these two apparently contradictory conditions clinical observation leaves no doubt. Anæmic out-patients of an hospital ordinarily complain of inability to discharge their duties from drowsiness, while at the same time they cannot sleep at night. Hammond, in his recent work on *Nervous Diseases* (art. 'Cerebral Anæmia'), takes up this question and delivers himself with much decision. Admitting that cerebral anæmia and sleep are indissolubly connected, he argues that the falling away of the blood by gravity from the head in the upright and sitting postures, in which the drowsiness becomes so marked, is the cause of the inclination to sleep, or rather to sleepiness; while the filling of the encephalic vessels with blood when the head is laid on the pillow, and the horizontal position assumed, is the consequence of the blood gravitating into the head, no longer at the top of the organism. There is no doubt much truth in what he states, and the clinical facts warrant the hypothesis, which, however, savours a little too strongly of *à priori* reasoning. The dizziness of this condition of unfilled cerebral vessels is usually relieved by the assumption of the recumbent posture, as is swooning, which is merely a more extreme and acute condition, by a similar position. The effect of position over the intellectual power is well shown in the cases quoted by Sir George Burrows, and in the case of the cyanotic youth mentioned before.

Headache is a very common subjective symptom of cerebral anæmia. The pain is dull, persistent, and unvarying, and the sensation is not uncommonly as if the skull was opening, or the upper half of the calvarium was being lifted off. Chronic headache in conditions of cerebral anæmia is usually, or at least often, vertical; while frontal headache is

¹ The lack of lustre is probably due to an anæmic condition of the irides, which sparkle in the excitement of cerebral hyperæmia, or functional activity.

rather associated with passing conditions of exhaustion from sustained intellectual labour. The *why* of this will be seen presently. Vertical headache is most distinctly associated with anæmic conditions of asthenic gouty states, and is often of much diagnostic value, often, also, pointing very clearly the direction which our therapeutic measures must take if we wish them to be successful. At other times headache, often vertical, sometimes frontal, is found along with those anæmic states where the patient complains chiefly of 'low spirits,' simple states of mental depression and unhappiness, from a defective blood supply to the encephalon.

Vomiting.—This is commonly induced in the more pronounced conditions of cerebral anæmia, when the sitting posture is suddenly assumed, even in bed. It would appear to be due to a sudden anæmia of the roots of the vagus, and a consequent contraction of the stomach, as it is free from nausea, and accomplished without an effort, *i.e.* action of the abdominal muscles.

Palpitation.—This is common in severe hæmorrhage, and is rarely absent in the course of well-marked cerebral anæmia. It is not induced by any obstruction offered to the circulation and consequent effort to overcome it—the ordinary exciting cause of palpitation; but is due to the anæmia of the vagus roots, and consequent comparative loss of power in the vagus, which inhibits or controls the heart's contraction. There is a distinct back and forward play betwixt the heart and the brain by means of the function of the vagus. When the brain, including the vagus roots, is well flooded with blood, the vagus is thrown into action, and the heart's contractions are retarded: when there is less blood in the brain, the vagus does not act strongly, and quicker, sharper contractions of the ventricles drive a freer blood supply to the brain. In these conditions of chronic cerebral anæmia arising from emotional conditions, this compensating action through the vagus seems lost, partially or wholly; and palpitation is only found in the less pronounced cases, and is, so far, not of bad prognostic import.

Sighing Respiration is also a phenomenon of diagnostic value. It is commonly found in conditions of cerebral

anæmia, and is classed by Griesinger among the physical indications of melancholia. The sigh is indicative of unhappiness negative as well as positive. It is a very curious objective indication of an emotional condition. Whether it is a mere vague movement undeserving of our consideration, or whether it is a reflex action of some utility, a more probable view, it is not easy to be positive. The sucking action of a long inspiration affects the veins; and by so acting upon the encephalic veins induces a brisker movement of the blood in them, and quicker arterial circulation, thus the sigh may be actually useful, and not without practical value in affording relief.

Constipation is the well-known concomitant of disturbance within the encephalon. All forms of cerebral disease may be accompanied by constipation, and constipation is usually associated with impaired functional activity of the brain. The effect of loaded bowels over the brain is notorious, and the enlivening effects of a brisk cathartic in cerebral torpidity are familiar to us all. This is well seen in heart disease. A load in the bowels is no rare cause of a melancholic condition, and its removal is at once followed by cheerful brain activity. Epilepsy is sometimes distinctly associated with intestinal accumulations. But it is quite possible that constipation may stand to cerebral anæmia in a causal as well as a consequential relationship. Ludwig and Dogiel found that the touch of a finger along the intestines was at once followed by an accelerated circulation within the cranium.

Torpidity of the bowels combined with a load in their interior may so affect the cerebral vessels, and through them the cortical cells, by means of the sympathetic, as to induce reflex spasm and a condition of cerebral anæmia. This much appears certain, that there is such a relationship betwixt inaction of the intestines and disturbances within the encephalon that the one usually induces the other, and that whichever comes first in the order of time, the other commonly follows, and so each condition aggravates the other; a point of some practical importance in directing the treatment. The constipation may not uncommonly be the result

of the cerebral torpidity, the implication of the involuntary muscle in the general muscular lethargy arising from the cerebral condition.

General Muscular Condition.—The diminished activity of the cerebral cells from the impairment in their blood supply is manifested in the condition of the muscular system generally. In slighter and less pronounced cases there is languor, lassitude, and disinclination towards as well as inability for exertion. The individual is listless, unenergetic, and easily exhausted. This is the condition of matters in the ordinary cases of our out-patient rooms. In more marked cases, as in acute dements and melancholics with stupor of asylum wards, there is almost muscular immobility. The same position is maintained for hours, and the disinclination to change the posture is pronounced. The only muscles thrown into action are those expressive of depression and sadness, and in confirmed cases these are almost fixed in tonic spasm. The whole muscular system tells of the inactivity of the cerebral centres, of their impaired functional activity. Nor is the evidence thus furnished invalidated by the restlessness which obtains in the early stages of grave cases, preceding or alternating with the general inactivity; just as we shall see mental irritability preceding and alternating with the psychical torpidity. The restlessness and irritability are sometimes in so far good that they indicate that activity has not yet been abolished and obliterated, but such is not generally the case. 'It is a physiological fact that the excitability of a nerve is increased a short time before it is entirely lost, and that the greatly increased excitability of a nerve is not a sign of increased normal nutrition, but, on the contrary, of its diminution. It is true we do not know why this is, but the knowledge that it is so renders it less strange that in gradually developing anæmia of the brain symptoms of irritation should, as a rule, precede those of paralysis, and that when the anæmia does not attain a high grade, only the symptoms of irritation should be seen' (Niemeyer). There is a striking analogy betwixt the condition of impaired muscular movement and imperfect mental activity. Nor can this cause any surprise when we remember that each takes its

origin in the cells of the cortical substance, and that alterations in the working of these cells will alike affect both these outcomes of their activity. At the same time that we recognise that these cells are the originators of thought and muscular movement, we must not overlook the fact that they are also the recipients of sensations. Consequently we may look for disordered, impaired, perverted, or at times exalted sensations as the concomitants of the altered condition of action and of thought.

Modifications of Sensation assume at times the character of abolition, more or less complete, of sensation—the anæsthesiæ being local or general; at other times of disorder, now perversion, now exaltation, or even of something actually added. In the latter division we find the origin of many of the delusions and even hallucinations which do not prevail merely among the insane, but haunt many whose intellectual integrity enables them to recognize the true nature of the case. The cutaneous anæsthesiæ, too, are also found amidst the presumably sane. Local anæsthesiæ are common among hysterical patients; while general cutaneous anæsthesiæ obtain in the condition known as religious ecstasy. There is also a form of anæsthesia found usually among melancholics, which assumes the type of an arrested or interrupted sensation. ‘I see, I hear, I feel,’ say they, ‘but the object does not reach me. I cannot receive the sensation: it seems to me as if there was a wall between me and the external world.’

More commonly there is merely an impaired conducting power, so that the sensation travels but slowly along the nerve tracks, and there is an unwonted delay in conception and answer: a condition often seen in the lighter stages of the typhoid condition, and also in the cerebral anæmia artificially produced by bromide of potassium. There are also perversions of the special senses, as of sight, smell, taste, and hearing, recognized by the sane as hallucinations, but not so recognized by the insane, who adopt these perversions and weave them into the web of their ordinary thought. That these disordered sensations are more commonly found among the insane, and are also more pronounced than in the

sane, amounts to nothing further than that in diseased intellects erroneous impressions founded on modifications of sensations are more probable and more stable than in those who, being sane, are able to recognize the true explanation and so check the morbid feelings, and prevent their assuming a dominancy over their mental processes.

‘Nothing would be more erroneous,’ says Griesinger, ‘than to consider a man to be mentally diseased because he had hallucinations.’ Physicians themselves have had them, as for instance Van Helmont, Andral, Leuret, and Schroeder van der Kolk, who had hallucinations and phantasms when he had a load in his colon. Visions, or hallucinations, are common among religious persons, as for instance Luther, Spinoza, and Swedenborg. Goethe, Sir Walter Scott, Pascal, and Tasso suffered from these disorders affecting the imagination. An excellent instance of how hallucinations may arise from disease in the organs of special sense is furnished by Esquirol in the case of a blind maniacal Jewess in Salpêtrière, who saw the strangest things. After death the optic nerves were found atrophied along their whole length. Optical hallucinations are notorious among the victims of delirium tremens. They occur as visions of banquets to famishing men—as living fountains to those perishing of thirst. Anæmic women in our out-patients’ rooms complain much of black specks before their eyes. I had a patient, a boy, anæmic when convalescent from typhoid fever, who commonly asserted that he had just found a sovereign in the street, but could never produce it; nevertheless it was a recurring hallucination which disappeared as he grew stronger. Hypochondriasis is commonly enough founded on some disordered or abnormal sensations.

The modifications of sensation produced by the effect of an impaired supply of arterial blood to the cerebral cells, their functional activity thus being affected, are in harmony with the other outcomes of this anæmic condition.

Psychical Symptoms.—The mental outcomes of cerebral anæmia are, as we have hinted before, the most important of its consequences; though the physical sequels, as we have seen, are not always trivial. As the physical condition is a

combination of lethargy and torpor with a certain amount of adynamic irritation, so is the psychical state one of mental inactivity with intervals of irritability, which in the advanced cases passes on to fatuity. Midway between these two conditions lie the modified sensations, impairment with perversion, which we have just considered. All go to illustrate the enfeeblement of the working of the cerebral cells, and each is but the complement of the others. The effects of cerebral anæmia upon the mental processes of the brain have attracted the attention of most acute observers. Thus Niemeyer gives ‘attacks of convulsions and loss of consciousness as the symptoms of complete and sudden arterial anæmia; general symptoms of irritation and depression as those of incomplete and gradually developing anæmia;’ while Griesinger writes, ‘Anæmia of the pia mater and cortical substance, which is sometimes met with in acute and chronic conditions, as remarkable pallor of the cranial contents, merits, under certain circumstances (deduced from clinical observations of the case), great consideration. It may be a co-symptom of general anæmia. The great influence which this state of the blood exercises, especially in females, and, when acute in its origin, upon the development of the most varied nervous and psychical anomalies, is well known; and it is also admitted that all sorts of symptoms of irritation and torpidity of the cerebral organs may originate from this source alone. . . . When the anæmic state is developed slowly, and its course chronic, it appears principally to give rise to states of intellectual weakness and dementia.’ Loss of mental power, of sustained thought, is the first step; and along with this defective functional activity of the brain co-exists irritation, a general painful state, which when marked is denominated melancholia, ‘a vague feeling of oppression, anxiety, dejection, and gloom.’ There is as well as mere intellectual enfeeblement a general apprehensiveness, a true panphobia, which is popularly denominated ‘low spirits;’ which exists in every shade, from the fits of passing depression to which all are subject up to the most confirmed melancholia. The normal tone of the mind is subverted, and feebleness and dread usurp its place. There is a condition

of psychical pain, the homologue of physical pain, which Romberg says 'is the prayer of a nerve for healthy blood,' which is ever looming, or even 'pressing prominently into the foreground of the consciousness.' Dark presentiments and gloomy thoughts first suggest themselves, and then are pondered over until they become persistent fixed ideas.

This condition of depression and panphobia in anæmic conditions of the brain contrasts instructively with the hilarity, joyousness, and freedom from care which characterise conditions of hyperæmia, especially when artificially induced, as by alcohol. The tone of the thought is most distinctly influenced by varying conditions of vascularity of the cortical substance of the brain. Of this we are all conscious in the varying shades of thought which prevail according as we are full of bounding energy with a good circulation, or are exhausted or depressed with a feebly acting heart and compressible arteries.

'To be weak is to be miserable,' is a maxim upon which Dr. Maudsley is ever insisting; and it is especially true of mental conditions, where weakness is so closely associated with apprehension. Long ere these changes are sufficiently pronounced to constitute insanity or to warrant seclusion in an asylum, there exists this brooding condition, the *stadium melancholicum*; and in ordinary practice we commonly meet with well-marked instances of these depressed conditions of mind, which are the outcome of a defective supply of arterial blood to the nervous centres. This *stadium melancholicum* constitutes a condition which undoubtedly often runs on into insanity, but there is every reason to suppose that recovery is possible without an actually insane condition being induced; as much as that the congestion which is frequently the forerunner, or rather the preliminary, stage of inflammation, may be recovered from without having necessarily to proceed on to actual inflammation. But in either case slight determining causes may produce very distinct results, and determine the progress in the direction of disease. In this mental condition trivial external causes excite grave commotion, and shake rudely the mental equilibrium already trembling: it is a mixed condition of debility

and instability which renders comparatively unimportant exciting causes often of grave and momentous import as regards their effects. There is something more too than mere instability. The whole consciousness seems to concentrate itself upon a train of painful thought, and what would produce in healthy individuals but mere fits of passing depression is now crystallised into permanent wretchedness. Not only so, but this condition of panphobia commonly takes the place of the will, and crowds the mental horizon with dread and weird figures, the mingled products of vague fear and disordered sensation.

What doth ensue,
But moody and dull melancholy,
(Kinsman to grim and comfortless despair;)
And at her heels a huge infectious troop
Of pale distemperatures and foes to life?

An incubus settles down upon the mental processes which the enervated will and impaired brain power are incapable of dislodging.

This condition of mind which has just been described, a condition of wretchedness not rarely intensely acute, is frequently accompanied by a confused sense of being controlled by some extrinsic force, or of being under the influence of some outside power. When such a mental condition exists in the ignorant and superstitious, it often assumes the concrete shape of a belief in being bewitched, or in being overlooked by the evil eye: subjective conditions of mind deserving of deeper consideration than they usually receive, viz. a contemptuous sneer. In the more educated it may take the form of a pursuit by the officers of justice, entailing the creation of some imaginary crime in order to account for their pursuit; or it may assume the form of an imaginary or magnified sin which calls out the terrible powers of omnipotence in order to secure its due punishment. Such mental conditions in those banned by the Church, where an outraged conscience and a well-founded remorse prepared the ground for the reception of the crop of ideas sown by spiritual forces and evolved by the tremendous power of the Church, especially where co-existent depressing physical con-

ditions cut down the supply of arterial blood to the brain of the excommunicated individual, have doubtless often shaped themselves into forms of Satanic possession, and converted disordered sensations into the consequences of supernatural interference. When indeed this sensation of being under some external force is combined with perverted or exalted sensations, a distinct direction is given to the morbid thought, and the general sense of misery gathers together into some distinct concrete delusion. When these morbid sensations are internal, they may suggest the idea of an animal or a devil being in the intestines; when cutaneous, of being flogged with scorpions wielded by demons. Or hyperæsthesiæ of the special senses may occasion delusions and voices suggesting the most blasphemous ideas or shrieking out the horrors of the inevitable future; or perverted smell, originating the impression of putrefaction or corporeal decay, may be ever present to the imagination of the wretched individual. Under less tragic circumstances disordered sensations may stubbornly resist the action of the will, and sensible and intelligent people have been known to entertain the idea of poison in their food from disorders in the gustatory nerve. At other times the full intelligence of wakefulness can subordinate and control these morbid impressions, but they assert themselves and become dominant in the condition of dreaming. The waking thoughts may be modified pain, but the dreams are saturated with intense misery. This aggravation of the prominent thought in dreaming and on awakening is well known to alienist observers. To some it appears due to 'the unwonted force and activity which attend all the operations of the mind at this period' (waking), but it is equally possible that the explanation lies in the fact that these conditions are near the more pronouncedly anæmic condition of sleep. The first view, however, is rendered somewhat tenable by the fact that during those conditions of irritability and excitement, which occur at intervals during this condition of depression, the ruling ideas still retain their possession, and the periods are not those of partial relief but of aggravated wretchedness. The sense of misery is ever paramount, and

these intervals are only exacerbations of the ordinary condition, and the predominant impressions are merely more vivid or more intensified.

The psychological outcomes of cerebral anæmia are not by any means confined to the inmates of asylums, or to those who could fitly be consigned to these institutions, even for remedial purposes; they meet us at every turn in common life. By study of the more marked cases, we can educate ourselves to note more readily this diathesis of gloom, of doubt, and of apprehension, when we are brought into contact with it in our every-day walk. We see it in the over-anxious business man, who finds that his apprehensions and his powers of sustained thought are moving in opposite directions, or growing in inverse proportion to each other; in the poorly fed mother, who strives to suckle her child, to give to it the nutrition she can so ill spare from her own scanty stores, for it is the only sustenance she can procure for it; we see it in the acutely painful condition which follows a debauch, and is denominated by the Scotch 'the horrors;' or, again, we note it in the mental break-down which so commonly occurs in women when suddenly deserted by their lovers, where physical disappointment is linked with psychical chagrin. The mental condition is closely intertwined with the bodily state, and the greater the physical exhaustion the more pronounced the mental symptoms. The mental sufferings of those whose health is thoroughly broken often far exceed their physical sufferings. How many unfortunate beings are there, especially poor women, who are regarded as passionate, quarrelsome, and fractious, or as not exercising due control over themselves, who really deserve our warmest sympathy. Their brain, ill-fed, exhausted, and irritable, is unable to control the impulses which are all the more easily generated under these circumstances, and self-control is almost if not wholly unattainable. Their condition is very piteous; and remonstrance and expostulation are not effective curative agents.

We are but too apt to give but a superficial attention to conditions of mental misery that are not at once obviously

apparent, or with which we do not individually sympathise, and to pass from imperfect analysis to *à priori* synthesis. Thus we recommend them as did Mrs. Chick to her sister-in-law, Mrs. Dombey, to make an effort, even when in *articulo mortis*, or intimate to them that they should not give way to their feelings. This is about as rational as to tell a suffering individual to forget the pains of neuralgia, or to advise him by an effort of will to subdue or blot out the agonies of cancer. The wretched sufferer would only too gladly do so, if this were simply possible. The grievous load, however, is not to be dislodged by any power which they can bring to bear upon it; and it is cruel mockery to assert that they can emancipate themselves, if they but would, from their terrible thralldom. The simple fact is they cannot, not they will not.

The psychical outcomes of anæmic brains are intimately bound up with several conditions common in our social life; so common and yet so piteous that they deserve consideration. The first condition is a form of the intense wretchedness just described, which seeks oblivion. It is that terrible craving after alcoholic stimulants which obtains so largely among women. It is not the drinking which comes of sociality, for it is stealthy and secret, nor yet the taking of the dram which enables a man to get more out of himself: it is the employment of alcohol as a moral anæsthetic. The weight of gloom and misery which presses so heavily impels the sufferer with an irresistible power to the means of relief. The action of alcohol is to increase the force and rapidity of the heart's contractions, and also to dilate the blood-vessels of the brain. This is just what is wanted to relieve the condition under which the unfortunate woman is actually suffering; and the intensity of her misery is the measure of the force which impels her conduct. No wonder then that when once the sensation of relief afforded by alcohol is experienced, it becomes a tempting demon, and that it is resorted to again and again; as the gloomy thoughts, becoming fixed by habit, and the depression which normally follows alcoholic indulgence, added to that already existing, drive, with resistless might,

the miserable and much to be pitied woman to her baneful solace.

It is futile under such circumstances to talk of moral depravity and to preach of good resolutions; the passion which is now the impelling force is only comparable with the sensations of hunger in a famished brute. Relief from the existing misery is the sole, or at any rate the ruling desire; and it sweeps before it, like a resistless flood, the wreck of principles, resolutions, self-respect, and even regard for the opinion of others. It is worse than useless to attempt to approach this condition from the side of an appeal to the higher centres of morality, religion, or obvious duty; every attempt at thought, at realisation of the actual state of matters, only adds blank despair or poignant self-reproach to the existing weight of misery, and gives a fateful impetus to what is already a resistless whirlpool or a consuming fire. Actual medical treatment of the physical condition alone affords the slightest prospect of success in these unhappy cases. In two well-marked instances which came under my own notice, both women of acute susceptibilities, prepossessing appearance, and endowed with a large share of mental attractiveness, indeed, much superior to the women of their class, both mothers of families and affectionate wives—the filling of the brain with blood by appropriate measures did away with the craving for stimulants, by abolishing for the time the physical condition on which the craving rested; and it was only when the action of the remedies wore off that the terrible propensity returned, to the consequences of which they both ere long succumbed.¹

A clear distinction must be made betwixt those cases where women merely like the effects of alcoholic stimulation, a condition compatible with intervals of sobriety when the stimulant is unattainable, and the condition described above, where sobriety is distinctly intolerable, and the relief of intoxication is eagerly sought after and procured at any cost.

Under other circumstances the psychical sequels of cerebral anæmia assume another aspect altogether, viz., religious

¹ The line of treatment was abandoned in consequence of my leaving the neighbourhood, and no substitute for it adopted.

melancholia. This form is associated with spinsterhood, the form just described rather with a married existence. Much too depends upon diathesis, education, and the surroundings. The doubtfulness, the loss of self-reliance, the vague apprehensiveness of evil, the general dread, which we have seen are the consequences of a defective arterial blood supply to the brain, here assume a religious or theological aspect. This melancholia has sometimes been regarded by the Church as evidence of religious feeling and of spiritual life. The periods of excitement, of exacerbation, became periods of ecstatic devotion. The hallucinations became visions; and the unfortunate melancholic was ultimately perhaps canonised as a saint, and held up to others, not as a beacon to deter, but as an example to be followed.

And so it is still. The loss of volitional power, which is so marked in melancholia, and the domination of the leading ideas, carefully fostered, render these unhappy creatures helpless; the limitation of the will is enervating and paralysing. The apprehensions of this condition become fearful and unpardonable offences; the morbid thoughts, the outcomes of the functional activity of cerebral cells ill-fed and starved, assume the form of omnipotent decrees; and abnormal sensations are the pains of hell. At first sight it seems monstrous that such should be the actual explanation of much theological fanaticism, yet too often such is the case. Doubtless severe religious self-introspection, a gloomy creed, and a monotonous existence have often led to insanity. The rule and practice of frequent fasting, of nights spent wholly or in part in prayer, rivet upon the deluded devotees the fetters first thrown around them by a morbid self-feeling, sometimes itself the offspring of unsatisfied physiological impulses. The brain, ill-fed, half-starved, and unrested by sleep, is at once irritable and exhausted, susceptible and impotent. One species of power alone remains, and that is an intense form of obstinacy as to their delusions and their habitual trains of thought.

If a volume of portraits of the saints were submitted to Mr. Darwin, that gentleman would experience no difficulty in recognising in the features of some of them the charac-

teristics of the depressing emotions, which may be recognised by an ordinary observer of physiognomy in the action of the corrugators of the eyebrows and the depressor muscles of the angles of the mouth. On many of them there would be found stamped the deep depression of religious humility blended with spiritual pride, which first moulds the plastic features, and then leaves to sleeplessness and incomplete starvation the task of successfully hardening them as it were into stone, and of graving on them more deeply and distinctly the characteristic lineaments of morbid depression and cerebral anæmia.

The effects of an imperfect blood supply to the cerebral cells are strikingly illustrated by the peculiar mental changes which are undergone by the sufferer from advanced heart disease. These go far to establish the conclusion that the vascular changes are not always the result of primitive changes in the brain cells affecting the blood supply as a secondary action, but that modifications in the vascular supply to the pia mater and the grey matter of the convolutions do exert a consequential action upon the functional working of the cerebral cells, and the thoughts evolved therefrom. When organic disease of the heart has not been accompanied or followed by compensating muscular hypertrophy, or when that compensation has itself become worn out, we find that the effect so produced upon the circulation exercises a distinct action upon the mental operations of the individual. The resolute, determined, even-tempered individual becomes vacillating, capricious, and irritable. Self-reliance and confidence give way to distrust and suspicion. Sustained thought is no longer possible, and periods of mental irritability alternate with intervals of depression. Weak obstinacy and irresolute perverseness are blended with well-marked suspiciousness of the motives of others. This has been admirably illustrated by George Eliot in her delineation of the last days of Peter Featherstone, in 'Middlemarch,' in his suspiciousness, duplicity, and vacillation betwixt his two wills. The original character of mind is there in every case, but it is modified by the effects of disease in the vascular system. The impaired and often intermit-

tent blood flow to the cerebral centres affects their working and exercises an influence over the products of their activity. The normal flow of ideas becomes first tinted with depression and apprehension, and then ultimately deeply dyed with suspicion. In some cases the thought is saturated with distrust, and the natural affection for friends and relatives is metamorphosed into aversion or even absolute hatred. Melancholy, suspicion, moroseness, and sullenness have been described as the special characteristics of those cases of insanity which are complicated with disease of the heart. And such mental attitudes are what we might expect from individuals under restraint, when a different cast of behaviour and rôle of conduct would obtain and characterise their actions if free to manifest their feelings and to make others conscious of their power in their suspicions and caprices. All those who have had to come much in professional contact with sufferers from advanced heart disease know the extreme difficulty often experienced in maintaining pleasant relations with them, or rather in avoiding open rupture, in consequence of their varying moods, the accessions of distrust, manifested in the form of loss of confidence in their medical adviser, and the general irritability which often strains to the utmost point of tension the relations betwixt the patient and his physician.

More marked still are the mental peculiarities in those numerous cases where disease of the heart is associated with disease of the kidneys, and where the symptoms of defective blood supply to the brain are combined and blended with the irascibility which is almost pathognomonic of the presence of lithates in the blood. Then the brain is not only sparingly supplied with blood, but the nutritive fluid itself is poisoned with the products of tissue metamorphosis. The passionate outbursts of the gouty in their paroxysms resemble rather the nerve storms of the imperfectly controlled child than the outbreaks of anger or indignation in the ordinary adult. There is a gusty violence about them which sometimes attains the terrific force of a tornado. These outbursts occur, however, while the heart is still fairly sound and the bodily

powers are as yet unimpaired, so that an element of vigour is present and in action. Here there is not an impaired blood supply due to any failure in the circulation, but to spasm of the cerebral arterioles excited by the gout poison. Nevertheless, here there are still the characteristic and essential elements of that depression and apprehension which are pathognomic of cerebral anæmia. The illustrious Sydenham, in writing of the mental changes to which he was subject during attacks of gout, expresses himself to the following effect: 'The body is not the only sufferer, and the dependent condition of the patient is not his worst misfortune. The mind suffers with the body, and which suffers most it is hard to say. So much do the mind and reason lose energy, as energy is lost by the body, so susceptible and vacillating is the temper, such a trouble is the patient to others as well as to himself, that a fit of gout is a fit of bad temper. To fear, to anxiety, and to other passions the gouty patient is the continual victim, whilst as the disease departs the mind regains its tranquillity . . . Melancholy so-called is pre-eminently the inseparable companion of gout.' Such is the testimony of one of the most competent of observers. When then upon this condition are engrafted the psychological consequences of heart failure—an almost inevitable pathological outcome in the course of time—the mental condition becomes such as to be a deep misfortune to the individual and all around him. To those brought ordinarily in contact with him he becomes simply terrible, his friends approach him almost with fear, which in those dependent upon him becomes something more pronounced, as the invalid asserts the consciousness of power with an almost malignant enjoyment. This picture is neither exaggerated nor even highly coloured, and this combination of irascibility and apprehension, irritability and distrust, is at the bottom of much of the ferocity of temper and selfish cruelty, born of suspicion, which has characterised the latter days of despots and tyrants, and notably of Henry VIII.

Different as these various conditions are, they present

family characteristics which proclaim their close relationship. It is rather the externals which mask the resemblance than any essential loss of likeness in the cases themselves. In all there exists that sense of anxiety and apprehension which falls into grooves or channels already traced out by the pre-existing temperament. In one the irritable brain craves the solace afforded by stimulants; in another the depression takes the form of anxiety as to the future, the soul's place in eternity; nor are these forms in which the distemper clothes itself interchangeable in the same individual, they depend upon radical differences in the mental constitution of each. But as to the effects of gout poison or of bile upon the character of the individual, or the effects of a failing circulation, they present less marked points of difference; and a similar physical condition, making due allowances for different diatheses and temperaments, produces like results, or at least results presenting such a family likeness as clearly to indicate their common origin. And if this origin is a physical condition, springing from a modification of the circulation through the pia mater and grey matter of the brain, we must extend to each of these sufferers an enlightened charity, and not attribute to malicious perverseness, natural depravity, or rebellious wilfulness, conditions for which the sufferers are little if at all more responsible than they would be for a halt in their gait after paralysis, or for suffering from palpitation or dyspnœa on exertion when labouring under valvular disease of the heart, or for the tint of their skin when jaundiced from biliary disturbance.

Even in acute cardiac affections, these effects upon the mind are sometimes produced, and Fliut ('Diseases of the Heart,' 1872) describes two cases of acute pericarditis where the prevailing delusion of the delirium was the having committed a crime, and all the efforts were directed towards an escape from the imaginary emissaries of justice.

Local Cerebral Anæmia.—At times, however, there exists an anæmic condition of one part of the cerebrum without the rest of it being implicated. Hence we have marked depres-

sion or 'low spirits' without the perceptive or intellectual faculties being impaired. At first sight this may appear impossible, in consequence of the inosculation of the terminal branches of the three cerebral arteries. But when we remember the localised necrosis or brain softening which commonly follows an embolon in the cerebral vessels, we cannot avoid the conclusion that the circulation of the brain is divided into areas but little or imperfectly connected with each other. Our ideas, associated with the circle of Willis, have learned unconsciously to group themselves into an impression that the circulation in the cerebrum is as general as, until recently, the functions of the cerebrum were held to be. The lines of both are more distinctly marked out than we have hitherto been in the habit of thinking. Anæmia of one or other of these vascular areas may exist without the rest being affected; and one of the most curious points that strikes us in the consideration of the relations of perverted emotions to cerebral anæmia is this, that there may exist a distinctly morbid condition of the emotions, as depression, general apprehensiveness, melancholia, indeed, without the intellectual powers being implicated. The acuteness of perception is unimpaired, and the condition of sadness does not exert any influence over the intelligence; it may give a tone to the conclusions arrived at, but it does not interfere with the power of observation nor the perception of the facts. Indeed, the person may be quite conscious of this emotional perversion. Thus Rousseau writes: 'I feel bitterly my wrong conduct and the baseness of my suspicions; but if anything can excuse me, it is my mournful state, my loneliness.' The intellect here was perfectly conscious that there was a morbid condition of the emotions which it was evidently unable to control. It will appear at first sight to most readers improbable, and to some perhaps even absurd, that such psychical conditions should find an anatomical explanation. Yet such is the case!

We have seen before that the anterior and middle portions of the brain are fed by the branches of the internal

carotid, and from these arise the arteries of the corpora striata, while the posterior lobes of the cerebrum are supplied by the vertebrals after their temporary coalescence into the basilar artery. We further saw that the vaso-motor nerve supply of these vessels was also differently derived. The internal carotid is supplied by the ascending branches of the superior cervical ganglion, which receives the ascending branches of the thoracic ganglia, including the fibres of the splanchnics which ascend from the abdomen. Now it is admitted that for sensations to pass from one point to another a nerve track is indispensable. By this communication, in all probability, we have the emotional portion of the brain affected by conditions in the abdominal viscera—and there are a plentiful supply of cases to illustrate this connection. I say, ‘in all probability,’ because it might be suggested that the nervous communication ran along the vagus, or even along the communications existing betwixt the sympathetic and the spinal nerves, and up the cord. There is, as yet at least, no evidence of any sort to substantiate these views, while the view adopted possesses not only an intrinsic probability, but even some actual positive evidence. Cyon and Aladoff have shown that the vaso-motor nerves of the liver actually run down the vertebral arteries, though the lower cervical ganglia, after which they form the annulus of Vieussens and then pass on to the first dorsal ganglion, and thence through the gangliated cord of the sympathetic, the splanchnics, to the cœliac ganglion and along the hepatic vessels of the liver.

The accompanying wood-cut demonstrates this; and is kindly lent me by Dr. Lauder Brunton, in whose lectures on Diabetes in the *British Medical Journal* for January, 1874, it first appeared.

Here we see that there is a direct anatomical association betwixt the nerves of the liver and those of the vertebral arteries; and from this we can comprehend the condition of melancholia existing in derangement of the liver, without the intellect being affected; for if a nerve track has been traced in one direction, the co-existence of another track passing in the opposite direction is not very conjectural, and

will doubtless be found when equally carefully sought after. The portion of the brain which is supplied by the vertebral arteries, and consequently affected by vaso-motor disturbance of the nerve supply of these vessels, is according to Schroeder van der Kolk and Laycock the portion in which are located the emotions. We are familiar with emotional disturbance or melancholia in other abdominal affections than those of

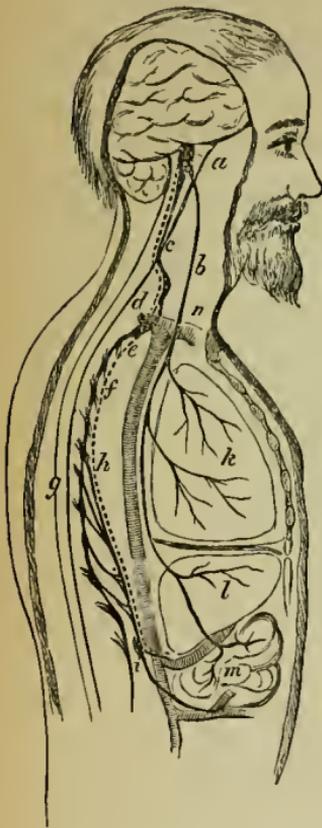


Diagram showing the course of the vaso-motor nerves of the liver, according to Cyon and Aladoff. These nerves are indicated by the dotted line which accompanies them. *a.* Vaso-motor centre; *b.* Trunk of vagus; *c.* Passage of the hepatic vaso-motor nerves from the cord along the vertebral artery; *d.* Fibres going on each side of the subclavian artery, and forming the annulus of Vieussens; *e.* First dorsal ganglion; *f.* Gangliated cord of the sympathetic; *g.* The spinal cord; *h.* Splanchnic nerves; *i.* Celiac ganglion, from which vaso-motor fibres pass to the hepatic and intestinal vessels; *k.* The lungs, to which fibres of the vagus are seen to be distributed; *l.* The liver; *m.* The intestine; *n.* The arch of the aorta.

the liver merely; it co-exists with faecal accumulation in the colon, with Addison's disease, with uterine displacement, and with peripheral irritation in the reproductive system. It appears highly probable that there are other than hepatic fibrils in the vertebral vaso-motor nerves, and that it is by such mechanism that this emotional disturbance is brought about.

We can also comprehend how disturbance of the emo-

tional area of the brain is not connected with or accompanied by any corresponding change in the intellectual processes, nor yet with motorial impairment. For this synthesis can fairly be carried beyond the point reached by Van der Kolk and Laycock. When the anæmic condition is more general, and the intellectual centres as well as the emotional are involved, as in acute dementia, we find that there are motorial anomalies. Here again will anatomy, supplemented by physiological investigation, come to the rescue, and throw a ray of light over the darkness and obscurity which have hitherto surrounded this subject. The blood supply of the corpora striata and of the motor centres of the convolutions, in front of the fissure of Rolando, are alike drawn from the distribution of the internal carotid. A condition, then, involving the anterior and middle cerebral lobes will include the motor centres. When then we have the intellectual centres involved, we find motorial disturbances; when the emotional centres alone are affected, there is no loss of motorial power. A person may be melancholic without impairment of either intellectual or motor power; but in the dement the loss of muscular energy is as marked as is the paucity of ideas, or mental inactivity. Possibly the motor and ideational centres may lie over one circulatory area, while depression and perverted systemic sensations may occupy another vascular distribution of their own. At least such association is found in the combination of motor and intellectual disturbance in the general paralytic, where the anterior and middle lobes are chiefly implicated; while emotional disturbance associated with abnormal systemic sensations, are commonly found together in the melancholic, where, according to Van der Kolk at least, the morbid changes are found in the posterior lobes.

The very divisions of melancholia seem to bear out such an hypothesis. Thus we find Hypochondriacal M., where we have the depressing thoughts centred upon some organ, or area; Delusional M., associated with lost, perverted, or exalted systemic sensations; Religious M., so certainly related with the reproductive organs; while in Atonic M. mind and motor power are alike nearly annihilated, the con-

sciousness of time and place is lost, even the sensations connected with bodily necessities are blotted out in oblivion, and there is a psychical void combined with muscular paresis.

Prognosis.—The prognosis of simple cerebral anæmia is usually good. If there be no well-defined organic disease standing in a causal relationship to it, most cases will improve, especially under appropriate treatment and therapeutic measures based on a true conception of the pathology and the real state of matters. Probably those cases where the darkness and gloom are alone dissipated by alcohol are the least promising of the forms unconnected with obvious organic disease; and the hopelessness of those cases where women drink systematically is generally recognised. Of the other forms, of course the condition is more serious where the affection has become so pronounced as to require seclusion; but even then, if the depression be vague and general, the prognosis is far from unfavourable. If, however, these vague and shapeless impressions assume a concrete form, and gather together into a fixed idea, the prognosis is worse, and if to these are added hallucinations, the prospect becomes very dark. One point in prognosis must never be overlooked, and that is, that in all mental disease there is a mental and a physical factor, and that the prognosis is graver when the physical disease leads to mental disease than when the reverse is the case. If, for instance, the delusions of being acted upon by some external agency be founded upon disordered or perverted, but still actual sensations, the prognosis is bad, and decidedly much worse than when they arise in some abnormal functional action of the cerebral cells. Where the condition of cerebral anæmia depends upon disease of the blood-vessels or organic changes in the heart, the prognosis is as bad as is that of the physical condition on which it depends.

Progress of the Malady.—This may be either towards recovery, or the direction may be downwards. When the latter occurs the physical changes are those of brain wasting. There is actual atrophy of the brain substance, especially of the cortical layer, the convolutions are shrunken,

the sulci are open channels filled with fluid, and the brain has a water-logged, sodden appearance. There is the *hydrocephalus ex vacuo* of Niemeyer, 'which develops as a necessary result of diminution of the size of the brain'—the unavoidable outcome of the decrease in bulk of the other contents of the encephalon. The mental symptoms undergo corresponding changes. At first there is melancholia, that is, depression, with perversion of thought, but after a certain point is reached, a condition of dementia is entered upon, which involves the extinguishing of thought. In other cases there is acute dementia, settling down into chronic dementia, a confirmed hopeless condition. In either case there is first mental disturbance, which gradually merges into annihilation of mind. We are now confining ourselves to those cases which commence in vaso-motor disturbance or in a derauged action of the cerebral cells, not those dependent upon visible organic disease. The physical condition keeps pace with the mental state. At first there is loss of muscular energy, the gait is languid or unsteady; as the disease progresses there is marked disinclination to all movement, and the tendency is to assume one position which is persistently retained. The failing brain power shows itself in impaired motion, heralding the abolition of motorial energy and power, and in mental perversion or deficiency, the precursors of psychical torpidity and intellectual decay. Not uncommonly the last change of all is induced by exhaustion or starvation; at other times by intercurrent disease, especially pneumonia, so frequently found in chronic conditions of debility.

In the production of the more marked or advanced conditions the importance of sleeplessness must never be overlooked. During sleep, we have every reason to believe, the actual nutrition of the brain is conducted; the more highly vascular condition of waking being associated with the functional activity of the brain. The slower current of the sleeping state favours the tissue nutrition: and consequently a condition of sleeplessness is antagonistic to or incompatible with brain nutrition and repair. When there is cerebral anæmia, and consequently an impaired condition of the brain

cells, sleeplessness interferes with the recovery of their lost power, and tends to perpetuate and aggravate the condition of matters; the weakened organs cannot recover their normal integrity, indeed, can only further degenerate under such circumstances. The unrested brain is less equal than before to the proper performance of its function; the thought is more confused and bewildered, the mental images are blurred in outline and less distinct, and thought is painful and only called out by effort; there is indeed a condition of mental lethargy and suffering induced which enables one to estimate the destructive effects of the want of rest. In anæmic conditions of the cerebrum such sleep as is obtained is often unrefreshing, as it is either broken by disturbing dreams or neutralised by the aggravation of the mental distress which attends the awakening of these sufferers. There is a vicious circle established, and the disturbance in one part of it adds to and aggravates the other portions, the morbid changes act and react, and a steadily downward progress is first inaugurated and then maintained.

One point there is in this advance towards utter vacuity which is not without interest, and that is the effect of habitual thought. Abolition of mind and of motor power are alike the finale of dementia following melancholia and of the mental annihilation of general paralysis. But even in the lowest depths are traceable the distinguishing characteristics of the ruling ideas. The stamp of unhappiness is impressed as distinctly upon the facial lineaments of the one, is as apparent and unmistakable, as are the fitting gleams of joyous satisfaction which light up fitfully the fatuous features of the other.

Treatment.—It is obvious enough from the foregoing considerations that the treatment of cerebral anæmia is a matter calling for the exercise of thought in each particular case. This is the more necessary in that the plan of treatment pursued exercises a decided influence over the progress of the case, that is, in those cases which are not associated with irremediable organic changes; in these last cases unfortunately the relief that can be afforded is but slight,

and the progress of the case is steadily downwards. In the other and much larger class of cases the prognosis is generally good; at the same time, that prognosis must often turn upon the aid treatment can afford to the natural recuperative powers. It is abundantly evident from what has gone before that the treatment requisite to each form must be widely different, according to the varied causal indications, and that a careful consideration of every case must precede the conclusion as to what therapeutic plan should be pursued. An outline of the leading lines of treatment only can be given here—a species of skeleton map merely, the filling up of which must entirely rest upon the exigencies of individual cases and be directed by particular needs.

The two main lines of treatment are the psychical and the physical.

The psychical treatment is important in every case. The mental characteristics of cerebral anæmia, of an insufficient blood supply to the cerebral cells, are essentially presentiment and gloom; and it behoves the medical adviser to bear this in mind and to provide the unhappy patient with cheerful and exhilarating surroundings, so as to divert as far as possible the current of habitual gloomy thought. Frequently it will be found necessary to indicate a new residence in order to enable the patient to break the thread of the mournful thought suggested, or fostered by, the familiar association with the immediate surroundings, and to give substantial aid to the will in its attempt to regain its authority and power. Distraction is desirable; and pleasant mirthful company and scenes of varied attractions are often eminently useful. Travel in those whose circumstances will permit of it may be profitably resorted to. Physically too the glow produced by walks in the fresh air, and the natural exhilaration induced by bright days and the keen pleasure of being bathed in sunlight, are of much utility. The consciousness of mental energy, the play of fancy and the flow of thought produced in all of us by bright inspiring weather, indicate how beneficial and curative indeed must such action be upon the unhappy and the melan-

cholic. Equally important is the matter of the disposition of those with whom the patient is chiefly brought into contact. Some people seem as if by design created for the induction of sadness and the suppression of joy; such persons are not desirable associates for those who are suffering from the gloom induced by cerebral anæmia. Others again diffuse joyousness around them, their very presence suggests mirth, and the ring of their voice is a gloom-dispelling music. Persons of this disposition are natural curative agents to the mournful and the sad.

The physical treatment of cerebral anæmia includes or even largely depends upon the administration of remedial agents of diverse character and producing widely different effects. In order to render the matter clearer, and to place the complex business in a sort of lucid arrangement, the whole subject may be again divided into those sections which were found convenient for the purpose of rendering the pathology more distinctly intelligible.

In general anæmia hæmatics are indicated, and of these iron stands pre-eminently first. It is not, however, a matter of indifference which form of iron is selected. Frequently the milder forms, such as the ammonio-citrate and the potassio-tartrate, are digested and assimilated when the more powerful preparations, as the tincture of the muriate or the dried sulphate cannot be so utilised. At other times the administration of the tincture of the muriate is rendered much more effective by its being given in acetate of ammonia. Properly too all hæmatics should be given shortly after a meal, and so be incorporated with the food; indeed, we must regard iron, when given in the doses we use for its hæmatic action, as distinct from the tonic effects of larger doses, and, under these circumstances, as an addition to the ordinary food rather than as a medicine. Arsenic may be combined with iron profitably in the treatment of general anæmia. The two can be administered together in an excellent form, viz., that of a pill, which admits of the co-administration as a vehicle of some cathartic, commonly indicated in these cases; indeed, we are all familiar with the fact that slight purgation often assists materially in bringing the system

under the effects of chalybeate remedies. The following formula will very frequently be found useful.

℞ Acid. Arseine, gr. ʒ
 Ferri Sulph. Ex Sic. gr. vi.
 Pulv. Pip. Nepal, gr. xii.
 Pil. Al. et Myrrh.

At other times it may be more desirable to combine the chalybeate with vegetable tonics, as quinine, strychnine, or calumba. When dyspepsia lies at the root of the anæmia, bismuth and calumba may often usefully precede the administration of ferruginous tonics. All sources of anæmia in the form of draining discharges must be arrested and controlled. Especially is this matter to be attended to in the case of females, and menorrhagia and leucorrhœa are each to be subjected to their appropriate treatment, and cured if possible.

Where there is spanæmia, hæmatics must be combined with more specific measures, as quinine in malarial anæmia, iodide of potassium in lead-poisoning, potash in lithiasis, and cathartics in fæcal anæmia. The cases given by Dr. Allbutt, in his Paper in the last volume of these Reports, brilliantly illustrate the effects of specific treatment over the melancholia of syphilitic anæmia, and the uselessness of the ordinary tonics in specially produced conditions. After weeks of treatment by tonics and sedatives without relief, specific treatment was adopted in the case of a gentleman who had had syphilis, and who had become quite melancholic. ‘As he passed under the influence of mercury and iodide of potassium his morbid fancies were dispelled, and his mental conceptions underwent a transformation which surprised even himself.’ The effect of all so-called blood poisons is to break down the blood, or to arrest the formation of blood corpuscles, and consequently in spanæmic conditions hæmatics are comparatively useless and inoperative, unless combined with the proper specific in each morbid condition.

Where there is a defective blood pressure, due to unfilled vessels, the treatment of general anæmia may be profitably assisted by the administration of some agent which will tighten down the two muscular ends of the arterial

system upon the contained blood column—by acting simultaneously upon the heart and the peripheral arterioles—and so raise the vascular tension. Such agents we possess in digitalis, caffeine, theine, quinine, all of which tend to fill the arteries and pump the blood out of the veins. Strychnine and belladonna have a similar general action, while they dilate the blood-vessels of the cerebro-spinal system; belladonna selecting the meso-cephalic vessels, while the action of strychnine falls rather on the spinal blood-vessels. Position is at times a matter of consequence. Vomiting and vertigo come on when the sitting posture is assumed even in bed, and the recumbent posture becomes a necessity. The raising of the head brings into play the effects of gravity upon the blood, and the vessels of the head are emptied as the blood falls away into the dependent parts; and the loss of the elasticity of the arteries, usually and normally found as the consequence of the blood pressure upon their elastic walls, no longer efficiently counterbalances the force of gravity.

In venous stasis within the skull, no matter how originated, two indications present themselves, viz., to remove the *vis a fronte* by relieving the venous congestion, and to increase the arterial flow. It is not irrational or inconsistent under these circumstances to combine venesection with the administration of stimulants. Such was the plan of Carmichael as given by Stokes. Agents which fill the arteries with blood are specially indicated here. When the venous stasis is due to some mechanical obstruction to the venous flow the treatment must be specially adapted to the individual case.

In gouty spasm of the cerebral vessels a mixture of potash and colchicum in infusion of buchu is indicated, and with these may be combined alkaline laxatives, as Pullna or Friederichshall water, or seidlitz powders. Here the removal of the materies morbi, the effete products of tissue metamorphosis, or of the hystolysis of peptones never converted into tissue, from the blood is the direct means of doing away with the arteriole spasm, which is the consequence of their presence in excess. Even in atonic or sup-

pressed gout such treatment may advantageously precede the administration of chalybeates and alkalies combined. This latter combination is best achieved by the addition of the bicarbonate of potash to the potassio-tartrate or ammonia-citrate of iron in a bitter infusion, or in buchu.

In organic disease of the vessels of the encephalon no line of treatment offers a satisfactory prospect. When cerebral anæmia is the result of remedial agents their suspension will soon restore matters to a healthy condition.

The treatment of those vaso-motor disturbances which constitute cerebral anæmia, whether arising in some alteration in the cerebral cells themselves, or taking their origin in some other causal agent and affecting the cortical cells secondarily and consequentially, first demands our intelligent and attentive consideration. It may be induced, as we have seen, by some emotional shock falling upon a susceptible organisation, or one already depressed by numerous and repeated blows. The depression of the heart's action and the contraction of the cerebral vessels indicate the administration of agents which affect these conditions contemporaneously. Such agents we possess in alcohol and belladonna. Belladonna, Dr. Crichton Browne tells me, is only useful in the early stages of emotional melancholia, where it is eminently beneficial. More extensive trial of it may establish the fact that it requires to be pushed until its physiological actions are induced ere its beneficial action becomes properly apparent, just as has been found to be the case with the calabar bean, its antagonist and antidote. Such trial of it seems *à priori* indicated in the treatment of what is denominated the *stadium melancholicum* which so commonly precedes actual insanity.

Alcohol is a powerful therapeutic agent in such conditions, and Hammond advocates its use very strongly. That alcohol possesses the power to disperse gloomy thoughts and to expel for the time mournful presentiments we all know well. We have also seen how mischievously such power may be utilised in the cases of those unfortunate creatures who abandon themselves to alcoholic intoxication, and who do unquestionably find in it temporary if dearly purchased

relief. We may also form some estimate of how alcohol often acts most beneficially in holding in abeyance the incursion of a condition of gloom or despair which would otherwise settle down upon the mind, and usurp a persistent sway. Amidst the sins of alcohol let its utility here not be forgotten; it has often given a temporary gleam of hope which has prevented the intellect from abandoning itself to utter despair; and the flash of hopefulness thus induced has often been the herald and pioneer of the steady growing light of returning trustful energy. In the treatment of cerebral anæmia the proper use of alcohol, and its administration in connection with nutritive food and chalybeates, is not only unobjectionable but even powerfully beneficial.

M. de Metz, at Mattray, found the addition of wine to nutritive food often absolutely requisite in order to enable boys of a sluggish and lymphatic disposition to maintain their good resolutions. But after all, shrewd as was the mind which observed how the mental operations could be acted upon by physical agents, and adroit the consequent action, it was only the inversion of the old plan of putting stubborn resolute children upon a diet of bread and water until they came to see the errors of their ways.

Another narcotic agent is found most serviceable in those forms of cerebral anæmia which assume the shape of melancholia with sleeplessness, namely, opium, in small but continuous doses. It is not only that opium conduces to sleep, the restorative effects of which upon these cases are most beneficial, but it exercises a decided influence, when so administered, upon the ultimate peripheral vessels. Pott found this out in the treatment of senile gangrene; and there seems to be no room for doubt but that the steady continuous action of oft-repeated small doses of opium has a decided effect upon the terminal vessels, dilating them, and so conducing to a more perfect and sufficient blood supply with more perfect consequent tissue nutrition in cases of chronic bloodlessness; whether cutaneous in chronic ulceration, more general in senile gangrene, or specially located as in the pia mater and cortical substance of the brain in melancholia.

There is something in opium which renders it the hypnotic *par excellence* in this condition, and which separates it from that class of agents which conduce to sleep by their direct depressant action upon the heart and blood-vessels (see 'The Depressants of the Circulation,' *Brit. Med. Jour.*, January 3, 10, and 17, 1874), by directly inducing cerebral anæmia, and which are here directly and distinctly contra-indicated. Such agents aggravate the pre-existing condition, increasing the tendency to anæmia and depression, the conditions which we are so anxious to remove and not to foster. These agents, as hydrate of chloral and bromide of potassium, retard recovery in the convalescent, and tend to transfer the condition from that of temporary melancholia into that more advanced and permanent condition, chronic dementia. The conclusion is independently arrived at but equally strongly entertained by Dr. Crichton Browne and Dr. Hammond. Hammond also lays stress upon another point to be attended to in the therapeutics of cerebral anæmia, and that is the evil of too suddenly or too freely filling the cerebral vessels by the combined use of hæmatics and tonics. This point is also insisted upon by Dr. Handfield Jones, who expresses himself thus: 'Nothing is more common than to find anæmic patients complaining of headache from the administration of the necessary tonics, because their nerve centres have been brought into such a state of hyperæsthesia by the impaired nutrition that they can hardly tolerate anything of a stimulant nature. A little excess, therefore, even of spanæmic blood, may cause distress to a feeble brain, which, after it has acquired a more healthy tone, will bear and be benefited by a larger amount of much better blood. The case is similar to that of the starved man, whose very preservation depends upon his being fed most sparingly for some time.' In many cases bromide of potassium may be combined beneficially with iron; and this is the class of cases where such union is indicated.

Finally, attention must always be paid to the bowels in the anæmic conditions of the brain with consequent depression. Schroeder van der Kolk was radically cured of a disturbed mental condition by the evacuation of a mass of scybalæ.

Ludwig and Dogiel proved by their experiments the relation of the brain circulation with the intestines; and the quicker brain circulation induced by increased action of the intestines is one lamp among the many lights which illumine the path of rational therapeutics, and indicate the way to the relief and cure of conditions of cerebral anæmia.

ON THE
THERAPEUTIC VALUE OF COLD TO
THE HEAD.

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THE object of this Paper is to bring under the notice of those who may be interested in the subject some experiments which have recently been performed with a view to ascertain, if possible, the actual effect produced upon the animal economy by the application of cold to the head.

I am indebted to Dr. Crichton Browne for the suggestion that this subject would well repay investigation, and also for many valuable hints which have greatly assisted me in my researches.

The very short period only, during which I have been able to devote my attention to this important subject, has prevented my treating it in an exhaustive manner at present; but I trust the few facts I have ascertained, and the comments I have made upon them, will not only prove interesting but will add a little to our somewhat scanty knowledge of the therapeutical action of cold as applied to the shaven and unshaven scalp, its *methodus medendi*, if, indeed, in some cases it possesses any curative action whatever, often perhaps being little understood.

As the cold water cure is literally as 'old as the hills,' it of course formed part of that empiric art which held universal sway for so long a period, and which is only gradually being swept away as we find ourselves armed more and more strongly in that honesty which our increased know-

ledge and means of scientific investigation give to us. Although held in high repute for centuries before, we find little mention of it from the tenth until the seventeenth century, when it seems to have again come into favour on the Continent, and was then styled *the new method*. That Celsus strongly recommended cold affusions in affections of the head, whilst Hippocrates had frequently prescribed cold as a remedial agent, we all know: and it seems extremely probable that Clephantus, who practised as a physician about 300 B.C., was as well acquainted with cold as we know that he was with hot affusions, which he is said to have recommended in intermittents.

I have been as yet unable fully to corroborate, by additional experiments, some of the results which I now publish, but as I have taken every care to avoid error, I do not doubt that further investigation will prove the accuracy of most of them.

In estimating the effect of cold on the blood supply to the head, I have taken advantage of a most ingenious instrument invented by Ludwig,¹ and known by the name of 'Ludwig's Strom-uhr.' This instrument enables one to estimate by volume the amount of fluid which passes through the vessel to which it is applied in a given time, and as it becomes more generally used, will, I am sure, be of the greatest practical value in all investigations like the present. For the knowledge of this instrument I am indebted to Dr. Lauder Brunton, who has very kindly, for the purposes of this Paper, placed his strom-uhr at my disposal.

I think it may be fairly stated that where the ice-cap is ordered to be applied, it is with the belief that it will have some direct action on the tissues within the cranium such action being manifested either by a decrease in their temperature, a lessening of their blood supply, or by a sedative action on the nervous centres. That any such effect can be produced directly through the scalp and skull-cap, my experiments seem to negative; but that some such effect may result from reflex action, at least temporarily, is extremely

¹ See Ludwig's 'Arbeiten aus der physiologischen Anstalt zu Leipzig. Zweiter Jahrgang,' 1867.

probable.¹ That cold is a powerful agent for good or evil cannot be denied; we know that its prolonged local application will result in the death of the part to which it is applied; and that when animals are long exposed to its influence, destruction of life will ensue, this exposure soon causing great torpor, a tendency to sleep becoming more and more irresistible, this being followed by a kind of apoplectic condition, gradual increasing asphyxia, and death.¹

In *post-mortem* examinations which have been made on those who have succumbed to intense cold, congestion of the brain, lungs, and other organs has generally, if not invariably, been found. Quelmalz,² on examining a body after death from cold, found the cerebral vessels engorged with blood; Rosen³ also observed great congestion of the vessels of the brain; Dr. Kellie⁴ found in two cases examined by him a bloodless state of the vessels of the scalp, but engorgement of the sinuses of the brain and effusion into the ventricles. That the knowledge of these facts should make former writers cautious in recommending even for a short period the application of intense cold is not surprising; and thus we find them expressing various opinions as to the length of time that cold to the head should be continued.

One writer on this subject, whilst recommending the brief application of cold in cases of congestion of the encephalon, especially warns us that a more prolonged use of it may produce the very state of things that a shorter application of it would be efficacious in removing.⁵ That cold applications to the head might have some reflex action in diminishing the flow of blood to adjacent parts is referred to by a former writer, who says, 'Cold applied to the scalp will be found to act as a sedative—not only on the vessels of the integument, but (by a sympathy which is well known to exist between the larger and smaller vessels) upon the carotids also, and the flow of blood will be de-

¹ Pereira's 'Materia Medica,' vol. i., Art. Cold.

² Quelmalz, *Prog. quo Figoris acrioris in corpore effectus expedit, &c.*, in 'Halleri Disp. Med.,' vol. vi., 1758.

³ Rosen, *Anat.* p. 142.

⁴ Kellie, 'Trans. Med. Chirurgical Society of Edinburgh,' vol. i, p. 84.

⁵ 'Dictionary of Pract. Medicine Art. Cold. J. Copland, M.D.

creased.’¹ Liston also refers to this sympathetic action, for when writing of cold applications, he says that ‘they act by constricting the superficial vessels, with which those more deeply seated sympathise to a certain extent.’² The extent, duration, and constancy of this *reflex* action of cold applications, as applied to the head, forms the subject of Section II. of this Paper. The direct action that cold may have in lowering the temperature of the intra-cranial tissues is considered in Section I. In Section III. I give the results of two experiments with Ludwig’s strom-uhr, showing the effects of the application of cold to the head on the flow of blood through the carotid artery.

SECTION I.—*Has the application of cold to the scalp any direct action in abstracting heat from the intra-cranial tissues?*

The following experiments were undertaken for the purpose of investigating this question:—

EXPERIMENT I.—The body of a man, æt. about 30, was twenty-four hours after death prepared in the following manner. The left common carotid was carefully dissected out, and divided at a convenient distance from its bifurcation into external and internal divisions, the external being tied close to its origin; the left vertebral artery was now found and divided; the corresponding arteries on the opposite side were treated in a similar manner, the accompanying veins of each being carefully dissected out and severed.

A large tub was now filled with hot water, which was carefully kept at the same temperature by an attendant appointed for the purpose. To the bottom of the tub was fixed a forcing pump, the discharge pipe of which, by means of a bifurcated brass nozzle, was connected with the common carotid and vertebral arteries of the left side. A small hole was now made in the left parietal bone near the vertex, and the bulb of a delicate thermometer introduced in a slanting direction underneath the dura mater. All being ready, the water, which was kept at the temperature of

¹ ‘Cyclopædia of Pract. Med.’ Edited by John Forbes, M.D., and others. Vol. i. p. 136.

² ‘Elements of Surgery,’ p. 20, 2nd edit., 1840. Liston.

110° F., was steadily pumped into the left vertebral and carotid; after some manipulation and the exercise of a little patience, the water was forced through, some of it returning by way of the veins and some through the medium of the circle of Willis, by the right vertebral and carotid. The temperature in twenty minutes had risen, as indicated by the thermometer in the brain, to 75° F., but it would now rise no higher, and after waiting ten minutes, during which it remained steadily at the same point, bladders containing a mixture of ice and salt were applied all over the cranium, care being taken to protect the orifice through which the thermometer projected. As long as the water in the tub was kept at the temperature of 110° F., the temperature of the brain remained at 75° F., but on lowering the temperature of the water, the thermometer in the head quickly fell a corresponding amount. Having been applied for fifty minutes the bladders were removed, having produced no effect whatever. The experiment might have been longer continued, only the tissues gradually by endosmosis became distended with fluid, making it more and more difficult to use the pump, and threatening to vitiate the experiment. The temperature of the room was 52° F. The difference between 110° F. and 75° F.—that of the brain and the water in the tub—must be taken as the loss of heat in the passage of the water through about 12 ft. of gutta-percha tubing. This length, however, was greatly shortened in the following experiment, and the difference in temperature from this cause much diminished.

EXPERIMENT II.—The body of a man, æt. about 50, was thirty-six hours after death prepared in the following manner. The subclavian was tied on the right side immediately external to the vertebral artery, and the same was done on the left side, the two external carotids being also tied. The thorax was then opened, and an incision made in the superior vena cava. The ascending portion of the arch of the aorta was now severed, and the nozzle of the discharge pipe of the pump firmly tied into it. The descending portion of the arch was ligatured. The body was placed in a hot water bath and raised to the temperature of 98° F. A small hole was

made in the frontal bone with a centrebit, and the bulb of the thermometer pushed downwards and backwards until it would go no further. All being ready the pumping was commenced, the water in the tub being 110° F., the thermometer in the brain registering 98° F., the temperature the body had previously been raised to.

The temperature inside the skull soon rose to 100·1° F., and having remained at this for ten minutes, a mixture of ice and salt was applied all over the cranium in bladders. After twenty minutes there was no diminution in the temperature, the thermometer in the brain still standing at 100·1° F. During the next five minutes, an atheromatous spot at the root of the left carotid began to show signs of giving way, and means were taken to prevent it; during the time that our attention was directed to this, the temperature was seen to be falling, as shown by the thermometer in the skull, and when our arrangements were completed it registered only 97° F.; however, on taking the temperature of the water in the tub, this was at once explained, as the temperature had fallen 3° F., or exactly the difference registered by the thermometer in the brain. The cold had therefore been applied thirty minutes without effect.

The temperature of the water was now raised to 108° F., and the bulb of the thermometer moved up from the floor of the cranium so as to occupy a more central position. It then registered 68° F., and after twenty-three minutes showed not the slightest alteration. The temperature of the room was 56° F. The bulb of the thermometer was found in the first instance to have rested upon the floor of the skull near the left crus cerebri, but slightly posteriorly to it.

Observations.—Of course the conditions under which these experiments were made widely differ from those existing when cold is applied to the head in the living subject, but they resemble them as nearly as it is possible to make them. The fact that is determined by them seems to be that as long as a fluid of a certain temperature is passing through the vessels of the intra-cranial tissues, the application of even the most intense cold to the outside of the scalp has no effect in lowering their temperature.

This result is not altogether surprising; for when we consider the character of the tissues through which the cold has to penetrate, and which in the living body are of course permeated by a constantly renewed supply of warm blood, the difficulty is to understand how it is possible that any loss of temperature of the deep tissues whatever could be expected to take place by the direct abstraction of some of their heat from them under such circumstances. One could almost expect that the external tissues might be completely frozen without the internal being in the slightest degree affected. But we must bear in mind that the temperature of the scalp can never, except momentarily, be lowered to freezing point without running serious risk of subsequent inflammation of its tissues. The amount of cold then which can be brought to bear through the scalp outside it, on the bony skull-cap beneath, is at the most extremely limited.

The following further experiments were performed on the living animal, every care being taken to keep the animal well under the influence of chloroform during the whole operation.

EXPERIMENT III.—A dog weighing 26 lbs. was placed under the influence of chloroform, and a small opening was made in the skull-cap, sufficient to allow the very small bulb of a thermometer, especially made for the purpose, to be introduced well into the longitudinal fissure dividing the two hemispheres of the cerebrum; this was accomplished with only a very small amount of hæmorrhage. The thermometer soon registered 98° F. When it had continued the same for ten minutes, small gutta-percha bags¹ containing a mixture of ice and salt were applied all over the surface of the shaven scalp. In ten minutes after the application of the cold, the temperature was 97·8° F., in seventeen minutes after it still registered the same; but now unfortunately an overdose of chloroform was given, and the ice-cap and thermometer had to be removed whilst artificial respiration was

¹ The gutta-percha tissue is very convenient for this purpose, and when cut into the shape desired, the edges can be readily fastened, having been first moistened with chloroform.

performed, but as this proved unsuccessful, the experiment was thus early brought to an end.

EXPERIMENT IV.—This experiment was performed in a similar manner to the last, on a dog weighing 15 lbs. In this case every care was taken not to give an overdose of chloroform, and the application of cold was continued for 55 minutes. The temperature before the application of the ice and salt mixture was 98·2° F., after 10 minutes 98·1° F., in 15 minutes 98·0 F. After this it did not fall, but at the end of the experiment had risen to 98·1° F. The temperature in the rectum was 98·2° F. the whole time. The following table will show at a glance the results obtained.

TABLE I.

Time of application of cold	Temperature in skull	Temperature in rectum	Temperature of laboratory
Minutes 0	° F. 98·2	° F. 98·2	° F. 60
" 10	98·1	98·2	"
" 15	98·0	98·2	"
" 25	98·0	98·2	"
" 35	98·0	98·2	"
" 45	98·1	98·2	"
" 55	98·1	98·2	"

Observations.—It will at once be seen that in both these experiments the results agree, and seem to corroborate those ascertained by the experiments on the dead body. It is true that in both cases the temperature was in a few minutes affected to the extent of ·2° F., but that this was caused by the direct abstraction of heat from the internal tissues, is negatived by the fact that decrease in temperature was not progressive, as it would have been in that case; but on the contrary the temperature in the second experiment rose again after a certain time. I think then we are justified in concluding that the temporary reduction of temperature in these two experiments was not due to the mechanical abstraction of heat by the cold applied, but rather to some reflex effect on the vessels, or a general lowering of the blood temperature.

SECTION II.—*Has the application of cold to the scalp any indirect or reflex action in lowering the temperature of the intra-cranial tissues?*

That cold has no direct effect when applied to the scalp in abstracting heat from the intra-cranial tissues, has been indicated by the results of the experiments recorded in the preceding section. I shall now proceed to inquire whether indirectly it may have such action, by irritating the peripheries of the sensory nerves to which it is applied; and thus producing reflexly through the vaso-motor centre such increased inhibitory action on the vessels as to materially diminish the vascularity of the parts in nearest relation with them, and consequently reduce their temperature.

That cold when applied to the skin may produce very considerable effects can hardly be doubted by anyone. Reflex arrest of the heart's action, even, may occur from the application of intense cold to the skin;¹ and the respiratory function may also be stopped by the sudden application of cold to the lips and nostrils.² That cold applied to a given part produces reflexly contraction of the vessels of a part distant from it can be proved by experiment, and that it does this by acting as an irritant to the afferent nerves can be clearly shown. To prove that this is so, let me briefly state the effect of excitation of afferent nerves on the vaso-motor centre.

Dr. Burdon Sanderson, in the 'Handbook for the Physiological Laboratory,'³ most thoroughly enters into this question, and gives many experiments by which the separate facts may be proved. It would serve no purpose to repeat them here, so I shall simply state their results.

It has been determined that the vaso-motor centre, although constantly in activity, can be so stimulated by impressions conducted to it through the afferent nerves as to

¹ Bruce Jones and Dickenson, 'Journal de la Physiol. de l'Homme,' i., 1858, p. 72.

² Brown-Sequard, 'Arch. of Scientific and Pract. Med.,' Jan. 1873.

³ 'Handbook for the Physiological Laboratory,' Brunton, Foster, Klein and Sanderson. London, J. and A. Churchill, 1873.

increase its function, thus—by curarizing a frog or rabbit, dissecting out a sensory nerve, such as the peroneal, dividing it and irritating its central end, the same reflex effect, namely, general contraction of the arterioles, can be produced as results from excitation of the vaso-motor centre in the medulla oblongata; although, it seems, in perhaps a slightly less degree. It can also be shown that by the excitation of the peripheral end of a cut vaso-motor nerve the same effect is produced on the parts supplied by it as is produced by the direct excitation of the vaso-motor centre in the medulla; but excitation of its central end produces no change whatever. It is also a most important fact that by exciting the central end of a cut sciatic nerve in one extremity of a frog, the effect produced on the opposite extremity is the same as if its nerve had been the one subjected to excitation. We see then that by the excitation of the peripheries of some sensory nerves, we produce reflexly an excitation of the vaso-motor centre, which not only shows its effects in the part to which the exciting agent is applied, but in parts remote from it, although evidently in near sympathetic relation with it. That cold, as the agent of excitation, produces this effect has been shown by Brown-Séguard,¹ who found that by plunging one hand in cold water the temperature of the other hand was considerably lowered; and that this was not caused by a general lowering of blood temperature was proved by the fact that the temperature in the mouth remained unaltered. I have repeated this experiment on the inferior extremities of a healthy adult man, and my results seem to corroborate the above fact, only perhaps the effects produced were rather less in degree.

EXPERIMENT V.—A healthy adult man, one hour after dinner, was placed in an arm-chair, with one foot in a basin containing a mixture of ice and salt, and although he complained of a certain amount of pain from the intense cold, he very pluckily allowed the experiment to be continued for nearly two hours, only occasionally removing his foot for a moment or two, when the aching was excessive. His pulse,

¹ Brown-Séguard, 'Experimental Researches applied to Physiology and Pathology.'

respirations, and temperature were observed every ten minutes. The following table shows at a glance the results which were obtained.

TABLE II.

Time	Pulse	Respiration	Temperature			Length of application
			Mouth	Ankle	Room	
P.M.			° F.	° F.	° F.	Minutes
3.40	86	18	98.4	95.4	66	0
3.50	86	"	98.4	95.0	"	10
4.0	84	"	98.4	93.6	"	20
4.10	84	"	98.4	94.0	"	30
4.20	84	"	98.4	94.4	"	40
4.30	84	"	98.4	94.4	"	50
4.40	84	"	98.4	94.6	"	60
4.50	82	"	98.3	94.6	"	70
5.0	82	"	98.3	95.0	"	80
5.10	82	"	98.3	95.2	"	90
5.20	80	"	98.4	95.4	"	100
5.30	80	"	98.4	95.4	"	110

The temperature of the ankle was taken in the depression behind and below the internal malleolus, the bulb being covered with a pad of lint and a woollen sock.

Observations.—This experiment, which was repeated with practically the same result, seems to indicate that the application of intense cold to one extremity produces a rapid fall in temperature in the other, followed by an irregular but gradual return to the normal standard, and in some cases to a little above it. That this reflex action does not extend to the head and neck is clearly shown by the temperature in the mouth remaining unaltered. We might thus fairly conclude that whereas the one inferior extremity is in near nervous relation with the other, the head and neck certainly are not so connected with the limbs.

Wishing to ascertain if similar results could be obtained by a like application of cold to one side of the head only, the following experiment was performed.

EXPERIMENT VI.—An adult man was, two hours after breakfast, asked to lie on a bed, and was covered over with a sheet and counterpane. The bulb of a thermometer was

placed between the cheek and upper gum of one side, and another was similarly placed on the other side; the indices of both projecting from the mouth so as to be easily read off when required. Another thermometer was placed in the right axilla. None of them were removed during the whole experiment, which lasted one hour and twenty minutes. The cold was applied to the whole of the right side of the head, including the ear, so as to affect as many branches of the afferent nerves on this side as possible; gutta-percha bags containing a mixture of ice and salt were placed in the necessary position, and retained there during the whole time; but doubtless a fair crop of hair had some effect in modifying the action of the intense cold on the tissues to which it was applied, thus preserving them from serious injury. The following table exhibits the effects produced on the pulse, respirations, and temperature.

TABLE III.

Time	Pulse	Respiration	Temperature			Time of application	Temperature in room
			Mouth same side	Mouth opposite side	Axilla		
A.M.			° F.	° F.	° F.	Minutes	° F.
10.45	80	16	98·8	98·8	98·2	0	67
10.50	80	"	98·6	98·8	98·2	5	"
10.55	80	"	98·4	98·7	98·4	10	"
11.0	80	"	98·2	98·3	98·4	15	"
11.5	78	"	98·0	98·4	98·4	20	"
11.10	78	"	97·8	98·3	98·4	25	"
11.15	78	"	97·8	98·3	98·4	30	"
11.20	75	"	97·7	98·3	98·3	35	"
11.25	75	"	97·6	98·2	98·2	40	"
11.30	75	"	97·6	98·1	98·2	45	"
11.35	78	"	97·5	98·0	98·1	50	"
11.40	78	"	97·4	98·0	98·0	55	"
11.45	78	"	97·4	98·0	98·0	60	"
11.55	78	"	97·4	98·0	98·0	70	"
12.0	78	"	97·4	98·0	98·0	75	"

The experiment was not longer continued, as the man complained of feeling pain about the head, and it was a considerable time before the part to which the cold had been applied recovered its sense of feeling, and the reaction was very marked.

Observations.—In this experiment we have the advantage of comparing the temperature of the parts in direct relation with those to which the cold was applied, with other parts in symmetrical relation to them; and it will be seen that although the temperature of the former was more decidedly affected, there was a distinct gradual decline of the temperature of the latter, which, however, did not coincide with the other. It will also be seen that the temperature in the axilla, after rising 2° F., gradually declined until it coincided with that in the left side of the mouth—the opposite side to which the cold was applied. We thus seem to have three effects produced in this experiment:—First, a considerable and comparatively sudden, and then gradual decline in temperature on the side to which the cold was applied; secondly, a gradual but not so considerable decline on the opposite side; and thirdly, a rise of temperature at first, succeeded by a gradual decline, in the axilla. It would have been interesting to have continued this experiment, as the temperature during the last twenty minutes had remained stationary in all three situations, and might possibly have shown a tendency to rise again, as will be seen to have been the case in some other experiments.

To ascertain the effect of the application of cold to the whole scalp on the temperature of the body generally, the following experiments were made.

EXPERIMENT VII.—Jane M., *æt.* 38, suffering from acute mania. The temperature in the axilla was found to be $100\cdot6^{\circ}$ F., the respirations were 22 per minute and the pulse 98. Gutta-percha bags containing a mixture of ice and salt were applied to the whole surface of the unshaven scalp. In twenty minutes the result was—temperature $100\cdot4^{\circ}$ F., respirations 22, pulse 90. In forty minutes the temperature was $100\cdot5^{\circ}$ F., respirations 22, and the pulse 90. It was now thought advisable to remove the ice-bags. The patient was in a state of delirious excitement the whole time, and seemed in no way relieved by the application.

EXPERIMENT VIII.—W. B., *æt.* 40, a man in fair bodily health. His temperature in the axilla was $98\cdot4^{\circ}$ F., his respirations 18, and the pulse 82 per minute. A sphygmo-

graphic tracing was taken, and found to be in every respect normal. A mixture of ice and salt was applied in bags to the unshaven scalp for forty minutes; after which the temperature in the axilla registered 98·2 F. The respirations were 18 per minute and the pulse 100. A sphygmographic tracing was again taken, and found to closely resemble that obtained before the cold was applied. This was the only case in which the pulse was accelerated, and I cannot help thinking that such a result must have been accidental.

In the following experiment bags of the same capacity as those used in the two last experiments were filled with ice and salt, and applied to the leg of an adult man around and below the knee joint.

EXPERIMENT IX.—A. O., æt. 31, a man in a state of good bodily health. His temperature in the axilla registered 98·4° F., his respirations were 16, and his pulse 82 per minute. The ice-bags were applied around and below the knee joint for forty minutes; after which the temperature in the axilla was 98·5° F., the respirations were 16, and the pulse 80 per minute. The sphygmographic tracings taken from the radial pulse before and after the application of cold showed no alteration in character. This man complained of having the toothache very badly before submitting to the cold application, but on its termination he declared that it had quite gone, and seemed much astonished at what he thought to be a novel cure for toothache!

EXPERIMENT X.—G. N., æt. 28, a man in a fair state of bodily health. His temperature in the mouth registered 98·5° F., his respirations were 16, and his pulse 75 per minute. Bags containing a mixture of ice and salt were applied over the abdomen for thirty minutes, when the temperature in the mouth registered 98·4°, the respirations were 16, and the pulse 70 per minute.

Observations.—These four comparatively simple experiments seem to indicate that the application of intense cold to the scalp, or to any other region of the body, has little effect in lowering the general temperature when applied for such periods as twenty or forty minutes, which is probably quite as long as is judicious under any circumstances. The

same fact is, of course, demonstrated to a certain extent by the experiments of Brown-Séguard before referred to.

EXPERIMENT XI.—One man in a former experiment having shown himself somewhat tolerant of the application of intense cold to the scalp, I determined to thoroughly envelope his head in a gutta-percha bag containing a mixture of ice and salt for as long a time as possible, and his temperature in the mouth and axilla was taken every ten minutes for one hour and fifty minutes, with the following result.

TABLE IV.

Time	Pulse	Respiration	Temperature			Time of application
			Mouth	Axilla	Room	
P.M.			° F.	° F.	° F.	Minutes
3.45	84	16	98.4	98.2	66	0
3.55	84	"	98.2	98.2	"	10
4.5	82	"	98.2	98.2	"	20
4.15	82	"	98.1	98.2	"	30
4.25	82	"	98.1	97.8	"	40
4.35	82	"	98.1	97.8	"	50
4.45	78	"	98.2	98.1	"	60
4.55	80	"	98.1	98.1	"	70
5.5	78	"	98.1	98.4	"	80
5.15	78	"	98.2	98.6	"	90
5.25	78	"	98.3	98.4	"	100
5.35	78	"	98.3	98.2	"	110

Observations.—It will be seen that in this experiment the temperature in the mouth and axilla gradually fell for the first hour, the former $\cdot 3^{\circ}$ F., and the latter $\cdot 4^{\circ}$ F. In the second hour, however, it gradually rose again, until at the end of the experiment the temperature in the axilla was the same as at the commencement, and in the mouth only $\cdot 1^{\circ}$ F. less.

The same kind of experiment was performed on dogs, rabbits, and pigeons, the results of which I give in the table following. In each case cold was applied in the form of a mixture of ice and salt for twenty minutes, the hair, fur, or feathers, as the case might be, being as much as possible removed from the scalp. The temperature was taken in the anus.

TABLE V.

Experiment	Animal	Temperature		
		Before application	10 minutes after	20 minutes after
		° F.	° F.	° F.
XII.	Rabbit	100·0	99·9	99·9
XIII.	"	99·9	99·9	99·8
XIV.	"	100·0	99·9	99·9
XV.	Pigeon	107·7	107·7	107·7
XVI.	"	108·0	108·0	108·0
XVII.	"	108·2	108·2	108·1
XVIII.	Dog	99·3	99·3	99·3
XIX.	"	99·1	99·0	99·0
XX.	"	99·1	99·0	99·0

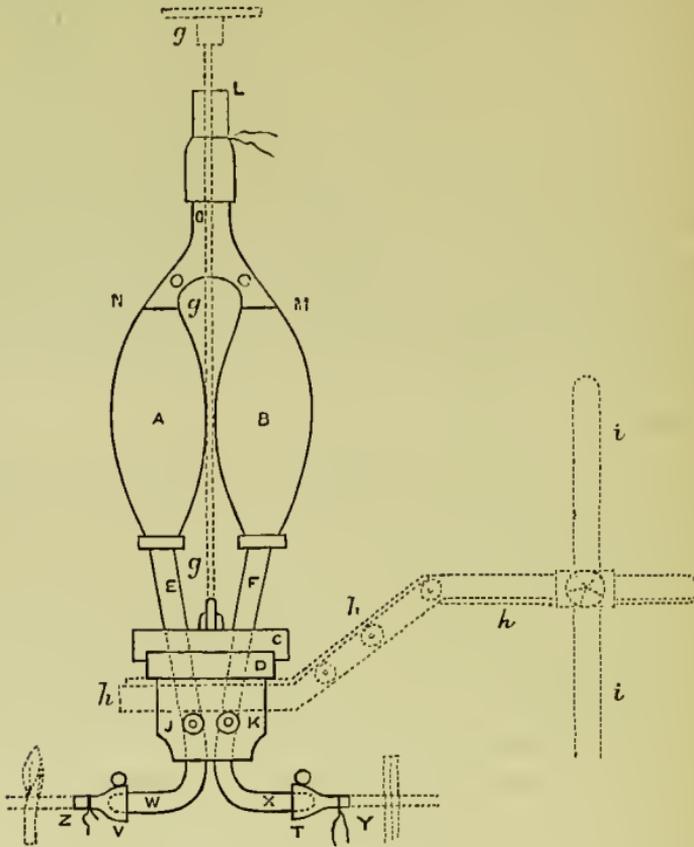
Observations.—In the rabbits and dogs the temperature is thus seen to have been very slightly affected, and perhaps this may be owing to the fact that the very vascular ears of these animals were involved in the cold application. But in the case of the pigeons, where the area over which the cold can be applied is very small, no diminution in temperature took place.

SECTION III.—*Has the application of cold to the head any appreciable action in lessening the blood supply of the intra-cranial tissues?*

Had the experiments in the preceding sections shown that a great reduction of intra-cranial temperature resulted from the application of cold to the scalp, it would scarcely have been necessary to have inquired what effect this might have had upon the blood supply to the encephalon; but seeing that so far the experiments have proved how little the temperature is affected, I have taken advantage of Ludwig's Strom-uhr for the further investigation of the subject, and although I have not had the opportunity of performing the elaborate experiments narrated by Dr. J. Dogiel¹ as having been made by him with this instrument, I am able to give the results of two experiments I have satisfactorily completed very recently.

¹ Dr. J. Dogiel, 'Die Ausmessung der strömenden Blutvolumina,' &c. Berichte über die Verhandlungen. Leipzig, bei S. Hitzel, 1868.

The accompanying wood-cut from a drawing which I have made of this ingeniously contrived instrument—without which I fear the most elaborate description would fail to convey a correct idea of its form and mode of application—will, I hope, enable those who are not acquainted with it to understand its construction. The strom-uhr is best applied to an animal paralysed with curara, and in some further experiments which I hope to make with it on the actions of



certain drugs on the animal economy, the administration of this agent will be absolutely necessary; but as the present inquiry only relates to the application of an external remedy, I have performed these experiments upon animals under the influence of chloroform, without I think having in any degree vitiated their results.

Description of Instrument.—Ludwig's Strom-uhr consists of two glass bulbs, communicating with one another, and

with both orifices of the cut vessel to which this instrument is applied, in such a manner that whilst the *central* bulb (A) is filling with blood from the *central* orifice of the cut artery (z), the *distal* bulb (B) is discharging its contents into the *distal* orifice of the cut artery (y). The instrument is so contrived that by means of two metal discs, the upper one of which (c) revolves upon the lower one (d), the two bulbs can be made to exchange their relative positions. Each disc has two orifices, which when the instrument is in its proper position exactly correspond to one another. The two glass bulbs (A and B) are connected by means of two metal tubes (E and F) with the two orifices in the upper disc (c), and by means of which and the two orifices in the lower disc (d) they communicate with the two tubes (w and x), which fit into the canulas (v and t) which are fastened into the orifices of the cut artery (y and z). The disc (c) can be so turned that the *central* bulb (A), when quite full, may immediately change places and become the *distal* bulb (B); whilst the *distal* bulb (B) (which has been emptying whilst the central one was filling) changes its place in a like manner and becomes the *central* or filling bulb (A). So that during the experiment we have the glass bulbs bearing this relation to one another:—That whilst the one is *filling* (during which time from its position in relation to the heart it is called the *central* bulb), the other is emptying (and from its position is called the *distal* bulb). Directly the *distal* bulb is empty it is turned round by the operator and becomes the *central* bulb, and *vice versa*.

As I have before stated, the two orifices in the lower disc (d) correspond with those in the upper disc (c), in such a manner that during the process of turning, the circulation through the instrument is momentarily stopped until the *distal* and empty, and the *central* and filled bulbs exactly occupy one another's previous positions, at which time the circulation through them is immediately restored.

The instrument is turned by means of an inverted (σ) shaped brass tube which fits on to two pegs on the upper disc (c), and is indicated in the wood-cut by the dotted lines (g g g). During the experiment the strom-uhr is held in position by

the brass clip (*h h h*) which slides up and down on the rod (*i i*) which is fixed at the head of Czermak's support. This support, however, cannot well be represented in a wood-cut.

To prepare the instrument for use.—Some fresh blood must be obtained from some animal of the same species as the one to be experimented upon (a small dog or large rabbit will be found the most convenient). The blood must then be defibrinated and filtered through linen, and by means of the opening (*κ*), to which is attached a small piece of gutta-percha tubing, the filtered blood must be introduced into the distal bulb (*B*) until it reaches the mark at the top of it (*μ*); the tubing must then be tied. A glass pipette will be found the best means of introducing the defibrinated blood. Now through the opening (*λ*), to which is also attached a small piece of tubing, defibrinated blood must be introduced until it reaches the bottom of the central bulb (*A*), which is at the top of the brass tube (*E*); the gutta-percha tubing must then be tied.

The next step is to introduce through the gutta-percha tubing (*ι*) at the top of the instrument some olive oil (the finest that can be obtained, and which has first been well shaken up with several quantities of warm water for the purpose of removing its slimy impurities), which is best done by means of a very fine pipette, great care being taken that no air bubbles are introduced with it. Sufficient oil must be used to fill the bulb (*A*), the glass tube connecting the bulbs (*A* and *B*), and its perpendicular extension (*ο ο ο*). The gutta-percha tubing must now be tied and the instrument should be placed in a bath of the temperature of the blood of the animal to be operated upon until all is ready for its application. But before this is done, in fact directly after the defibrinated blood has been introduced, the disc (*c*) should be turned *half* round, so as to close the orifices in the disc (*D*) leading into the tubes (*w* and *x*), or else all the defibrinated blood will run out by them.

The Experiment.—The animal to be operated upon should be placed on Czermak's support, and its head fixed in the proper position by the holder. The artery to be operated

upon must be dissected out, cleared for about half an inch, and divided, bull-dog forceps having previously been put upon the artery above and below the seat of division. The cut orifices should now be measured with calipers, and canulas of the proper size selected, which should then be introduced into the cut orifices and tied there. All being ready the strom-uhr must be fixed by means of its tubes (w and x) in the canulas (v and τ), and held in position by the brass holder (h h h). The bulbs must now be turned another half round, so that the bulb containing the defibrinated blood is the *distal*, and the bulb containing the oil is the *central* bulb. The bull-dogs should be removed, and if the conditions requisite for success have been duly observed, the oil in bulb (A) will be forced round into bulb (B), driving its contents into the artery (γ). The moment the fresh blood rising up in (A) reaches the line (N), by which time the oil in (B) will have descended to just above the commencement of tube (F) at the bottom of bulb (B), the instrument must be rapidly turned and the time the bulb took to fill noted. The capacity of the bulbs being known, and the number of turns requisite in a given time noted, it will be easy to calculate the quantity of blood passing through the instrument in that given time, and thus by means of this strom-uhr it will be possible to discover in a practical manner the effect that certain internal and external remedies may have in increasing or decreasing the volume of the blood flowing through the artery to which it is applied. To ensure success in this experiment great care is requisite in every step of it, a very little thing being sufficient to suddenly bring it to a termination. The chief points to be observed are as follows:—

1. The blood must be absolutely fresh and thoroughly defibrinated.
2. The instrument must be perfectly clean.
3. The oil must be deprived of every impurity by being well shaken with different quantities of warm water.
4. The room must not be cold, or coagulation will take place.
5. The orifices of the cut vessel must not be stretched by the canulas, but the canulas must exactly fit into them.

6. The neck of the animal must not be too much stretched, but should lie in an easy position.

Even if all the above conditions be observed, the experiment may yet be interrupted if the greatest care is not taken to turn the instrument at the proper time; for if the least drop of oil should get into the distal tube by its not being turned in time, the results of the experiment will be vitiated. Also, should coagulation take place in the bulbs, the experiment must be at once stopped, and an extra stromuhr (which should have been prepared at the same time as the other) inserted as quickly as possible. To prevent coagulation Dr. Dogiel recommends a glass casing containing hot water, which can be applied around the bulbs, but this is not absolutely necessary, especially with bulbs of small dimensions, through which the blood current is pretty rapid, and when the experiment is performed in a well-warmed room. The room should be kept at an even temperature throughout. A sponge dipped into hot water, and rapidly run over the outer surface of the bulbs between the periods of turning, I have found useful in preventing this tendency to coagulation.

For a further account of this instrument, and the many difficulties besetting its successful application, as well as for an exhaustive statement of the method by which the time taken for the filling of the bulbs is registered with great exactness on a revolving drum, by means of a spring lever, I refer my readers to the Paper by Dr. J. Dogiel before referred to.

EXPERIMENT XXI.—A rabbit weighing 1,960 grammes was put under chloroform and placed in proper position on Czermak's support. An incision was made in the side of the neck and the common carotid artery was dissected out and carefully cleared from the surrounding tissues. Two bulldog forceps were placed upon it about half an inch apart, and it was divided midway between them. The orifices were accurately measured with calipers, and suitable canulas selected, so that they might as nearly as possible correspond with the calibre of the artery. A canula having been secured in

each orifice by means of a fine silk ligature, the strom-uhr, which had been previously prepared, and had since been kept in water of the temperature of 100° F., was now fixed in its proper position, and the experiment was commenced by the removal of the forceps on either side of the artery.

The number of times that the bulbs filled and emptied was accurately noted by an assistant, whilst the bulbs were carefully watched by the operator, that their positions might be reversed at the exact moment when the blood in the central one reached the mark surrounding the upper end of it. Five minutes was the time during which each observation lasted, and the average of four observations was accepted as the correct result. A mixture of ice and salt was then applied to the shaven scalp for thirty minutes, and an observation was made during each five minutes, the average result of four observations being, as before, taken as the correct one.

TABLE VI.

No. of observations	Temperature in anus	Blood stream in minims per second	Average blood stream in minims per second	Temperature of room	Observations
	° F.			° F.	
1	101.2	6.75	} 6.775	68	Rabbit weighed 1,960 grammes.
2	101.1	6.9		"	
3	101.3	6.4		"	
4	101.3	7.05		"	
5	101.2	6.6	} 6.750	"	Cold applied 10 min. " 15 " " 25 " " 30 "
6	101.2	6.9		"	
7	101.0	6.3		"	
8	101.1	7.2		"	

EXPERIMENT XXII.—This experiment was performed on a rabbit weighing 1,630 grammes. It was conducted in exactly the same manner as the former one, and its results were corroborative.

TABLE VII.

No. of observations	Temperature in anus	Blood stream in minims per second	Average blood stream in minims per second	Temperature of room	Observations
1	° F. 100·2	6·0	} 5·925	° F. 70	Rabbit weighed 1,630 grammes.
2	100·1	5·85		"	
3	100·1	5·7		"	
4	100·1	6·15		"	
5	100·2	6·3	} 5·926	"	Cold applied 10 min. " 15 " " 25 " " 30 "
6	100·1	5·7		"	
7	100·2	5·5		"	
8	100·0	6·15		"	

Observations.—By a reference to the above tables it will be found that the average result of the four observations before and after the application of cold to the scalp is almost identical, and therefore indicates that no appreciable difference in the quantity of the blood stream through the common carotid artery, in such animals as those operated upon, is demonstrable by the strom-uhr as resulting from the application of intense cold to the head for thirty minutes.

Similar experiments can be easily repeated, and it will perhaps be as well to curarize the animal instead of subjecting it to the influence of chloroform; but I cannot help thinking that one drug would have quite as much influence as the other in vitiating the results of the experiments, if any such vitiation really takes place. It is, however, true that the quantity of curare necessary can be more accurately estimated than can the quantity of chloroform or chloral.

Having come to the end of my experiments, I shall now proceed to summarise their results, which are as follows:—

1. Experiments I. and II. directly indicate that so long as a current of warm fluid is passing through the intracranial vessels, the application of intense cold to the external surface of the scalp has no effect in abstracting heat from the intra-cranial tissues of the dead body to which the cold is applied.

2. Reasoning by analogy, and taking into due con-

sideration the results of Experiments III. and IV., it seems almost certain that the same holds good with regard to the application of cold to the living body.

3. That the application of cold to one part of the body produces a diminution of temperature reflexly in other parts of the body in symmetrical relation with it, as has been demonstrated by Brown-Séguard, is further shown by Experiment V.

4. This principle holds good when the scalp is the part to which the cold is applied, and the intra-cranial tissues those expected to be acted upon reflexly, as indicated by Experiments III. and IV., in the latter of which the intra-cranial temperature fell $\cdot 2^{\circ}$ F. So small an effect, however, must be considered unimportant, when we take into consideration the intense cold necessary to produce it.

5. That the effect produced on the body generally is no more than a scarcely appreciable diminution of temperature, is indicated by Experiments V. to X. on the human body, and by Experiments XII. to XX. on the bodies of animals. In one case only, Experiment VIII., is the decrease as much as $\cdot 4^{\circ}$ F., in all the others being not more than from $\cdot 1^{\circ}$ F. to $\cdot 2^{\circ}$ F., and in all those experiments that were sufficiently prolonged, after the application of cold had been continued for a certain time, the temperature remained stationary for about ten minutes, and then showed a decided tendency to rise again.

6. That the application of cold to other parts of the body than the scalp has some effect—though less in degree—on the general temperature of the body, is shown by Experiments V., IX., and X.

7. That although the frequency of the heart's action was in most of the experiments decreased to the extent of from four to six beats in the minute, the strength of each beat, as indicated by the pulse, was slightly increased.

The therapeutic action of cold, when applied to the scalp, may be shortly stated, then, as follows:—It causes a slight lowering of the temperature of the intra-cranial tissues by reflex action; a slight diminution of the temperature of the body generally, by the direct action that cold has in

lowering the temperature of the stream of blood passing through the capillaries in direct contact with it; and a slight decrease in the frequency of the heart's action. All these effects, however, are so insignificant in degree and temporary in duration, that taking into consideration the violence of the remedy adopted, one cannot help thinking that a greater effect in the same direction may be more easily produced by other and less violent means, and the patient be saved the pain and discomfort of having his head shaved and afterwards enveloped in a freezing mixture as long as one may dare to continue its application.

Dr. Crichton Browne informs me that of the large number of cases in which he has seen the ice-cap applied, not only in the acute maniacal attacks of the insane, but in numerous other primary or secondary affections of the encephalon, he can scarcely recall to mind one in which he could confidently state that a beneficial result had been obtained. Other physicians to whom I have spoken on this subject tell me that the result of their own observations agree with those expressed by Dr. Crichton Browne, as certainly do my own.

Seeing the very slight effects that even the most intense cold has produced in the foregoing experiments, we cannot wonder that some observers should long ago have begun to doubt seriously the efficacy of this remedy in those affections of the encephalon for which it has been prescribed.

It cannot be denied that one effect of the application of intense cold to the scalp must be to prevent a considerable portion of the normal blood supply of this very vascular area from following its proper course. It must therefore be accommodated somewhere else; and although the usual reply to this objection is that the vessels of the abdomen can contain almost any amount of blood without harm resulting, it cannot, I think, be deemed quite satisfactory. One is compelled to acknowledge that any such considerable obstruction to the flow of blood through the terminal branches of the external carotid must result in some increase in the blood pressure in the terminal branches

of the internal carotid, and which in some cases might be most prejudicial. I think the possibility of some such action should not altogether be lost sight of.

It should always be borne in mind, when applying remedies whose therapeutical action mainly depends on the effect produced by the irritation they cause to the peripheries of the nerves over the region of whose distribution they are applied, that the reactionary effect will be in inverse ratio with the primary effect resulting from the irritation of the nerve peripheries affected by the application.

That cold has some sedative action cannot I think be denied; most of us can I daresay testify to the relief experienced on applying cold to the forehead in cases of severe headache, but how this effect is produced does not as yet seem capable of satisfactory explanation.

That the vascular system is but little concerned in this action is indicated by the foregoing experiments, and I think we must go no further than purely nerve elements to discover the cause of this sedative action.

It may be that the decreased temperature and consequent lessened nerve sensibility produced by the cold upon the extremities of the sensory nerves, are shared in somewhat by the centres within the cranium from which they spring; and thus it may be that that portion of the sensorium to which the irritation on the nerve peripheries in the stomach or elsewhere—causing headache—is conveyed, shares in the diminished sensibility of those centres, and is therefore less capable of receiving impressions as vividly as before; the sensation of pain felt by the patient being consequently much lessened. When we consider the proximity of the central tracts of the fifth, and the pneumo-gastric branch of the eighth pair's of nerves, it seems probable that cold applied to those peripheries of the fifth distributed over the forehead should produce such an effect in parts in close proximity to the origin of the pneumo-gastric as to be capable of somewhat modifying the impression in the course of its transmission to the sensorium from the peripheries of that nerve irritated by a disordered stomach. The fact that a draught of cold spring water will often produce a sensation of pain in

the forehead, passing off, however, as soon as its temperature is raised by the heat of the stomach, would seem to strengthen the grounds for belief that this explanation may be the correct one.

A curious fact in connection with these experiments was the immunity from serious consequences following the application of such intense cold as about 0° F. to different parts of the living body for periods of time so considerable.

In only one case was the reaction sufficiently marked to give any cause for anxiety, and in that fortunately no harm resulted.

ON INHIBITION, PERIPHERAL AND CENTRAL.

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CASUALTY PHYSICIAN AND LECTURER ON MATERIA MEDICA AND THERAPEUTICS

AT ST. BARTHOLOMEW'S HOSPITAL.

THE external indications of nervous activity vary according to the nervous centres which are in action, but they may be included under three heads:—action of voluntary muscles, action of involuntary muscles, and secretion. Thus we recognise motor activity in the strong grip of the hand, the quick and decided motion of the limbs, and the firm compression of the mouth. Sensations and emotions may also manifest themselves through the medium of involuntary muscles, and do indeed evidence themselves most distinctly in the half-voluntary, half-involuntary muscles of expression, but they have in addition a most extraordinary action on the involuntary muscles and secretions, so that the pale face and cold sweat of excessive pain, the bounding pulse, flushed cheek, and bright eye of pleasurable excitement, and the lack-lustre eye and flowing tears of sorrow are no less evidences of these sensations and emotions than the contractions and quiverings of the facial muscles. So long as thought is unconnected with emotion it has little if any influence on the involuntary muscles and secretions, but manifests itself either by words and symbols or by deeds. It is only by external manifestations that we can ascertain the existence of either thoughts or emotions. Visions as wonderful as those of Milton, and schemes as daring as those of

Cromwell, *may* have passed through the brain of many a rustic, but so long as they led neither to expression nor action it was impossible for his fellows to give him credit for them, and thus he remained inglorious because he was mute. Many a man receives news either good or bad without the slightest change of expression, and it is hard to say whether he feels any emotion or not. But experience has shown everyone that seeming impassiveness may cloak the most intense feelings, and thus we are accustomed to say in such a case that the man either does not feel or he possesses great self-control. In this very expression we recognise the existence of another form of nervous activity, the external manifestation of which consists, not in any positive action, but in the very opposite, viz., in the absence of the movements by which the reaction of the organism to any stimulus is generally indicated.

This restraining or inhibitory action of the nervous system is quite as important as any of the others it possesses. Indeed it is this rather than any other power which distinguishes the man from the boy, and its cultivation is one of the chief ends of education. A slight temptation will lead a child to gratify its appetite without regard to consequences, a slight injury will make it cry bitterly, and a slight provocation will make it rush with blind fury against its tormentor. A man, on the other hand, will often deny himself every gratification for years together in order to ensure a comfortable provision for his old age; like an Indian at the stake, he may preserve an unmoved countenance while suffering the severest tortures; like St. Lawrence on the gridiron, he may even joke at the vain efforts of his persecutors; like a man of the world, he may meet his greatest enemy with a smiling countenance while his heart burns with rage; or, like a true Christian, he may subdue the angry feelings themselves and learn even to love his enemies.

The first lesson that a child gets at school is one of inhibition; it is taught to sit still and restrain the movements which external impressions acting on its excitable nervous system prompt it to make. The whole education is, or ought to be a continuation and expansion of this lesson, teaching

the boy to restrain his appetite, curb his temper, and do his duty, however disagreeable it may be. The highest praise is given by the wise man to the attainment of this power; for Solomon says that he that ruleth his spirit is better than he that taketh a city, while he illustrates the direful consequences of its absence by the simile, 'He that hath no rule over his own spirit is like a city that is broken down and without walls.'¹

But it is not in the restraint of passions and emotions alone that inhibition is observed; it occurs throughout all the body, and it would almost seem that every motor centre has a corresponding inhibitory one. The simpler the centres the easier is it to study their actions, and we shall therefore consider inhibition in isolated nerve centres before proceeding to such complex structures as the spinal cord and brain.

The first instance of inhibition, that is, of nervous action producing restraint instead of excitement, was discovered by Edward Weber.² He observed that when the vagus nerve is irritated the pulsations of the heart, to which the nerve is distributed, instead of becoming quicker become slower, or cease altogether. The heart contains within itself numerous ganglia, which keep up its rhythmical contractions even for some time after it has been removed from the body. The terminal branches of the vagus nerve in the heart are connected in some way with these ganglia, and whenever it is irritated the ganglia cease to act on the muscular substance, and the heart stands completely still in a relaxed condition. The branches of the vagus which have this action resemble motor nerves in their conveying an irritation applied to them towards the periphery and not towards the centre, and also in their origin, for although they run in the vagus they are really derived from the spinal accessory nerve, and only join the vagus near its origin.³ The other fibres of the spinal accessory go to muscles, and when they are excited they set the muscles in action, but those going to the heart do not end in the muscular fibres but in the ganglia, and they

¹ Proverbs, xvi. 32, and xxv. 28.

² Wagner's 'Handwörterbuch' Bdl. iii., 2, p. 31.

³ Waller and Schiff. Schiff's 'Lehrb. d. Phys.' i. 420, and Heidenhain, 'Studien des Physiolog. Anstalts zu Breslau,' 1865. p. 109.

produce rest instead of motion, relaxation instead of contraction. Other parts of the circulatory system have a nervous arrangement somewhat like that of the heart, only that in them the contraction is more tonic and not so distinctly rhythmical as in it. Thus the blood-vessels of the penis are kept in a state of moderate contraction by the stimulus which the vaso-motor nerves supply to their muscular walls. This stimulus is derived in part at least from ganglia lying close to the vessels, and to these ganglia proceed certain nerves, the *nervi erigentes*, which arise from the sacral plexus.¹ When the nerves are irritated the ganglia cease to stimulate the vascular walls, and these consequently relax and yield to the current of blood which pours into and distends them, so that the organ becomes swollen and turgid, and erection takes place.² After the irritation of the nerves has ceased, the ganglia reassert their power over the vessels and cause them to contract, so that the blood is again driven out and the organ becomes flaccid. A similar arrangement occurs in the submaxillary gland. There are small ganglionic structures in the gland close to the vessels,³ and a branch of the chorda tympani, which passes to them, acts as an inhibitory nerve in much the same way as the *nervi erigentes*.

This will be rendered more intelligible by a glance at the accompanying figures. In Fig. 1, A is the artery, *m*

FIG. 1. the ganglion lying close to it, which causes it to contract, and *n* is a nerve fibre connecting the two together. So long as they are not interfered with, *m* acts constantly upon A, and keeps it moderately contracted; but, whenever the inhibitory nerve *i* is excited, it arrests the action of *m*, and A dilates.

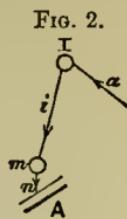
The inhibitory action of the vagus, of the *nervi erigentes*, and of the chorda tympani may be excited reflexly by stimulation of a sensory nerve, as well as by the application of an irritant directly to the trunk of the nerves themselves. An irritating vapour held before the nose of a rabbit will often

¹ Lovén, 'Arbeiten aus d. Physiolog. Anstalt zu Leipzig,' 1867, p. 19.

² Eckhard, 'Wien. Sitz. Ber. xlv. (Abth. 2)' 1862, p. 342.

³ Lovén, Op. cit. p. 19.

stop the heart instantaneously; friction of the skin near the genitals of a dog will, under certain circumstances, cause erection of the penis;¹ and a little vinegar placed on the tongue will dilate the vessels of the submaxillary gland. In each case the stimulus is conveyed by the sensory nerve to an inhibitory centre in the medulla or spinal cord, and this being called into action arrests the activity of the motor ganglia. This is represented diagrammatically in Fig. 2, where *a* is an afferent nerve corresponding to the sensory nerves of the nose, genitals or mouth in the above-mentioned examples, *i*, an inhibitory centre in the medulla or cord. The other letters are the same as in Fig. 1.



In the case both of the penis and submaxillary gland, the ganglia, which seem to act as vaso-motor centres, are placed close to the vessels, and the inhibitory nerves are efferent nerves, conveying impressions from the centre to the periphery, just like the vagus. In the case of many vessels, however, the vaso-motor centres do not lie close to them, but are placed at a distance from them in the medulla oblongata (as in Fig. 3, *m*), and the inhibitory nerves pass to it. Thus the vaso-motor centre for the intestinal vessels lies in the medulla, and stimuli are conveyed down to them from it through the splanchnic nerves. These may be represented by *n* in Fig. 3.



When the splanchnics are cut, and the communication severed between the medulla and the intestinal vessels, they become dilated, and the pressure of the blood in the arterial system generally is much diminished. But the action of the vaso-motor centre for these vessels may be arrested, and a similar dilatation produced by irritating a nerve which proceeds from the heart to the medulla. It has received the name of depressor, from its power of depressing or lowering the blood pressure, and its obvious use is to lessen the resistance the heart has to overcome, whenever this may become necessary, either from too great pressure in the arteries or weakness or over-distension of the heart itself.

At the same time that it dilates the arteries, it also slows

¹ Goltz, Pfüger's 'Archiv.' viii. p. 460.

the cardiac pulsations by acting reflexly through the vagus, and this double action on the heart and vessels of course lowers the blood pressure much more rapidly than either would singly. This nerve may be regarded as belonging to one or other of two classes, for it may be considered as an inhibitory nerve of the same sort as the vagus or *nervi erigentes* (*i*, Figs. 1 and 2), only passing upwards instead of downwards (*i*, Fig. 3), because the vaso-motor ganglion is situated in the medulla instead of being near the vessels; or it may be considered to be an afferent nerve corresponding to the nasal or buccal nerves in two of the foregoing examples, conveying an impression towards an inhibitory centre in the medulla (*a*, Fig. 2). On the first view we must make the improbable assumption that inhibitory centres for the vaso-motor system of the intestinal vessels exist in the heart, whereas on the latter we consider that both the vaso-inhibitory and vaso-motor centres are situated in the medulla, and are connected by short, instead of by long communicating fibres, as in Fig. 4. My friend, Professor Rutherford, thinks that this subject is more easily understood if we regard the motor ganglia of the heart, and, I may add, the motor ganglia of the vessels in the penis and submaxillary gland, as little bits of the general vaso-motor centre which have been removed from the medulla to the periphery, by which process the communicating branches (*i*, Fig. 4) between the vaso-inhibitory and vaso-motor ganglia have become elongated, so as to form long nerves like the vagi and *nervi erigentes* (*i*, Fig. 2). It appears to me to be still simpler to regard the ganglia, both motor and inhibitory, as if they had been originally apart, and situated, as they are in some of the lower animals, near the organs they have to regulate, their connection with these organs being effected



by short filaments, and their connection with each other by long ones. When massed together so as to form the medulla, cord, and other nervous centres, the ganglia are further removed from the organs over which they preside, and the filaments passing from them to these organs necessarily become elongated, while those connecting them with each

other become very short, as in *i*, Fig. 4. Some exceptional ganglia, like those of the heart, of the penis, and of other organs, still retain their long connecting filaments (vagi,¹ nervi erigentes, &c.), as in *i*, Fig. 2. The ganglia of the intestine connected with the cerebro-spinal system by the splanchnics afford another example of the same sort as the heart, and so do the ganglia of the uterus,² and probably the bladder also, although the nerves connecting them with the spinal cord are not so large and distinct as the vagi or splanchnics. The splanchnics are compound nerves, and contain both afferent and efferent fibres. The latter are the inhibitory nerves of the intestines, and when irritated arrest their peristaltic movements, though the manner in which they do this is still under discussion. Some authors consider that they arrest the action of the motor ganglia in the intestine in the same way that the vagi arrest the action of the motor ganglia in the heart, while others think that they do so by causing contraction of the intestinal vessels, and thus preventing the blood which serves as their normal stimulus from coming in contact with them. In the case of the heart this difficulty does not arise, for its action can be immediately arrested by irritation of the vagus in the frog, although there are no blood-vessels, not even capillaries, in the heart of this animal.³ It is therefore evident that the inhibitory action of the vagus is exerted immediately on the cardiac ganglia, and not through the medium of the blood-vessels in the frog; and we have no reason to suppose that it is otherwise in the higher animals, although vessels are to be found in their hearts. Reflex paralysis from excessive inhibition in the spinal cord will be hereafter discussed more particularly, but it may be mentioned here that it has been attributed by Brown-Sequard to spasmodic contraction of

¹ The vagus is regarded by Goltz as an *intercentral* inhibitory nerve, 'Centralblatt d. Med. Wiss.' 1864, p. 690.

² Presence of ganglia in uterus shown by Dr. Lee, 'Phil. Trans.' 1841. Their action, independently of the central nervous system, shown by the uterus contracting when its blood supply is arrested, and their regulation by the nerve centres by the uterine motions which follow irritation of the medulla, Oser and Schlesinger Stricker's 'Med. Jahrb.' 1872, part i.

³ Hyrtl, Sitz. Ber. d. Wien. Acad., 1858, xxxiii., p. 572.

the vessels in the cord.¹ The occurrence of inhibition in the heart without the intervention of blood-vessels, however, renders it by no means improbable that it takes place in the cord also by direct action of inhibitory nerves on motor cells, without any change necessarily occurring in the vessels.

The sphincters ani and vesicæ usually remain in a state of tonic contraction in uninjured animals. In dogs whose spinal cord has been divided in the dorsal region, the sphincter ani remains contracted, but instead of the contraction being constant and steady, as before, it becomes rhythmical, and if the finger be introduced into the anus, the sphincter is felt to contract and relax alternately about twenty-five times per minute.² In this rhythmical contraction the sphincter somewhat resembles the heart, and just as the cardiac contractions can be reflexly arrested by irritation of the nose, so the contractions of the sphincter may be stopped by irritating the foot. So long as the irritant is applied the movements cease, but when it is removed they recommence and become stronger than before.³

The fact that the contraction of the sphincter ani becomes rhythmical after division of the spinal cord, while it was previously steady and constant, is of great interest, for Goltz attributes the rhythmical contraction to the irritation produced by the finger in the anus, and instances this as an example of a constant irritation producing rhythmical contractions. Now another example of clonic or intermittent spasms following a constant irritation was noticed by Nothnagel in the frog,⁴ and ascribed by him to paralysis of inhibitory centres. Nothnagel's experiments will be described afterwards, when we come to speak of inhibitory centres in the spinal cord, and it is unnecessary to enter upon them here, but I mention them now as they throw considerable light upon the curious phenomenon observed by Goltz, and enable us to ascribe the rhythmical nature of the movements with some probability to the absence of some inhibitory or regulating force, which usually was transmitted downwards

¹ Quoted by Handfield Jones, 'Functional Nervous Disorders,' 1873, p. 13.

² Goltz, Pflüger's 'Archiv.' viii. loc. cit.

³ Goltz, Op. cit.

⁴ Nothnagel 'Centralblatt d. Med. Wiss.,' 1869, p. 11.

from some of the higher centres along the cord so long as it was intact, but was cut off by its division.

After division of the spinal cord in dogs, touching or pressing the foreskin and tickling the anus, or touching it with a wet sponge, cause the sphincter vesica to relax and a stream of urine to be expelled. At first it flows in a steady stream, and afterwards in jets from the action of the bulbo-cavernous muscles. The contraction of these muscles is at once arrested by pressing on the foot of the animal.¹

One of the most important instances of reflex action is afforded by another well-known experiment of Goltz, which is known as his tapping experiment or Klopversuch.² This experiment shows that similar results to those which follow irritation of the depressor nerve may be obtained by irritating the intestines themselves. It is of especial interest, both as affording an explanation of the phenomena of shock and aiding our comprehension of the extraordinary changes which any intestinal affection produces in the vascular system generally, and of the remarkable influence it exerts over the cerebral circulation and the mental functions which depend so much on a proper supply of blood to the brain cells. The excessive faintness which usually accompanies nausea, and the feelings of giddiness which often affect persons who suffer from dyspepsia, together with a weak circulation, are also more easily understood if we assume that the same changes in the circulation may be produced by an irritant acting on the mucous surface of the intestinal canal as by one applied to its serous coat.

Goltz's experiment consists in tapping sharply on the abdomen of a frog several times. If the heart and vessels have been previously exposed, so as to render the changes in them visible, it is found that after a tap or two the heart stands still, and then after a short pause begins to pulsate again. But about the same time that the heart stops, the abdominal vessels, and especially the abdominal *veins*,³ dilate widely, and form a large reservoir in which nearly all the

¹ Goltz, Op. cit.

² Goltz, 'Centralblatt d. Med. Wiss.,' 1863, p. 17; and 'Virch. Archiv,' xxvi., p. 11.

³ Goltz, 'Centralblatt. d. Med. Wiss.,' 1863, p. 593.

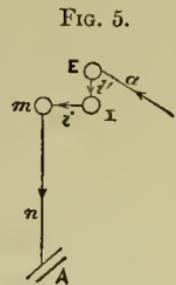
blood in the body collects, so that if the animal be suspended vertically no blood can reach the heart, which therefore remains empty. Although it continues to beat it sends no blood into the arteries, and the circulation ceases unless the animal be laid horizontally, so as to allow the blood to trickle from the abdominal veins into the right auricle.

In this experiment irritation of the mesenteric nerves by tapping on the intestine has a double action. It produces inhibition firstly of the heart, and secondly of the vasomotor centre for the intestinal vessels, and especially of the intestinal veins. These two actions, however, are independent of each other, for a few gentle taps will arrest the heart without affecting the vessels, and when they have once become dilated they remain in that condition after the heart has resumed its work. The depressor has likewise a double action on the heart and vessels, but it is more closely connected with the heart and the mesenteric nerves with vessels. In both instances then we see that the nerves act, not only on the organs with which they are closely connected, but also on other organs with which their connection is not so apparent.

The diminution in blood pressure which occurs after irritation of either the depressor or mesenteric nerves has a tendency to deprive the brain of its proper supply of blood, and thus to produce syncope. A blow on the stomach not unfrequently causes fainting, or even death from shock. We must distinguish between the two conditions, for although the state of fainting at first sight more nearly resembles death than does that of shock, it is much more transient and is in reality much less dangerous. Although one cannot say that fainting is due to an affection of the depressor nerve, and shock to an affection of the mesenteric nerves, yet these two conditions closely resemble in their comparative duration the results of irritating the depressor and abdominal nerves artificially.

Fainting often occurs from witnessing some horrible sight, or from the great emotional excitement produced by the sudden reception of very sad or very joyful news. Its production in this manner is also a reflex action, though of a

more complicated kind than when it happens after a blow in the epigastrium. In the latter case an impression is transmitted up the mesenteric nerves to the medulla, represented diagrammatically in Fig. 4. In the former the optic or acoustic nerves transmit an impression to the emotional centres in some part of the brain, and thence a stimulus proceeds to the medulla, there exercising a similar action to the one which proceeded from the stomach. This is diagrammatically represented in Fig. 5. where E represents an emotional centre, and A a nerve of special sense. The inhibitory action of emotions on vaso-motor centres is very evident in the blush of modesty or shame, and on other centres in the relaxation of the sphincters of the bladder and anus from fear, or the arrest of labour pains by the sudden entrance of a stranger into the room where a parturient woman is lying. A good instance of their action on secretion is afforded by the Indian ordeal of making persons accused of theft chew rice and then spit it out. The innocent reduce it in their mouth to a soft pulp, but the secretion of saliva is entirely arrested in the guilty by the fear of detection, and they spit it out as a dry powder.¹ The effect of intense grief in arresting the secretion of tears is another example of the same kind. This emotion probably exerts an injurious action on the health, and produces emaciation, debility, and sometimes death, in great measure through its action on the intestinal canal, lessening the secretion of the digestive juices and thus diminishing the actual nutriment absorbed by the organism. In consequence of this slow starvation the sufferer pines away from day to day, till reduced to a mere shadow, without any symptom of actual disease.



We have now considered reflex inhibition affecting secretion and involuntary muscles in the case of the heart, the vessels of the penis, intestine, and face, as well as the sphincters of the bladder and anus. We have seen that it may be produced by an impression made on the cerebro-

¹ Carpenter's 'Physiology,' 7th ed., p. 811.

spinal nerves of a part of the body having apparently almost nothing to do with the organ affected, as in stoppage of the heart by irritation of the nose; by an impression made on the organ itself, as in the case of the heart and intestinal vessels; on one closely connected, as in the same instance; or by an impression on the nerves of special sense acting through the emotional centres.

We now pass to inhibition of voluntary muscles, the simplest example of which is probably afforded by tickling. A gentle titillation of the palms of the hands, soles of the feet, or skin under the armpits, excites in most persons an almost irresistible impulse to withdraw the irritated part out of reach of the irritant. This impulse is of a reflex nature, depending entirely on the spinal cord, and has been noticed, as in J. Hunter's famous case, after the cord had been divided high up, and the patient was no more conscious of the movements of his feet than the spectators were, although the motions were more violent than normal. Such movements can be much restrained by the will, and many persons can stop them altogether when they try to do so, though if tickled unawares they will almost always start at first before they have brought their will into play. Others again cannot restrain their movements, however much they try, the reflex power of the cord being greater than any inhibitory power they can bring to bear upon it. But whenever the titillation is performed more forcibly, so as to convert it into scratching rather than tickling, the great tendency to reflex movement suddenly ceases, and little or no effort is required to prevent those movements which were previously irresistible. Nothing can be easier than to verify this upon the soles of one's own feet, but it is much easier to make sure of the fact than to explain it satisfactorily. It appears to me to be in all probability due to there being two sets of ganglia in the cord itself, one motor and one inhibitory. The motor is more readily excited than the inhibitory, and causes violent movements, which the inhibitory centres of the brain cannot restrain without the greatest difficulty, though they are readily controlled by the inhibitory ganglia in the spinal cord. A slight titillation excites the motor, but not the inhibitory spinal ganglia, a stronger

pressure stimulates the inhibitory centres also, and thus arrests the movements without any action being required on the part of the inhibitory centres in the brain. We may try to explain this either by supposing that there are two distinct sets of nerves proceeding from the skin to the cord, one of them having the power to excite inhibitory, and the other to excite motor centres. Farther, we must suppose that these sets of fibres are endued with different degrees of excitability, the motorial ones being stimulated by a slight touch, but the inhibitory ones only by a stronger impression. This is represented in Fig. 6, where *s* is the skin, *a* the fibres proceeding from it to the motor ganglion *m*, and *e* those going to the inhibitory ganglion *I*; *i* is the fibre by which *I* arrests the action of *m*, and *i'* that by which the brain exerts

FIG. 6.

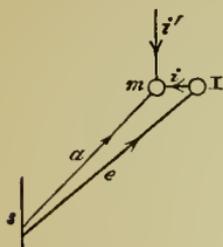
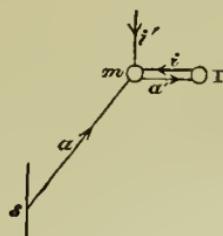


FIG. 7.



a similar action. The different fibres by which *m* acts on the muscles have not been introduced into the diagram.

This hypothesis, however, is a very clumsy one, and we explain the facts quite as well by supposing that there is only one set of afferent nerves (*a*, Fig. 7) from the skin to the cord, which transmit a slight impression only to the motor ganglia *m*, but convey a stronger one along *a'* to the inhibitory ganglia *I* also, which then react through *i* upon the motor ones. This latter supposition renders intelligible the fact that it is only when something is drawn quickly and lightly across the skin, so as to make a slight and transient impression on the ends of many sensory nerves, that tickling is felt. If the pressure on the skin is heavier, or if the motion over it is slow, the effect is quite different, and this is just what we might expect if a short and slight impression

travels only to the motor ganglia, and a stronger or more lasting one goes to the inhibitory beyond them.

It may appear to some, however, that I am here speculating on the mode in which the inhibitory centres in the cord are affected by stimuli, without having any good reason for believing in the very existence of these centres. I think however that Nothnagel has demonstrated this pretty clearly.¹ It is well known that if the head of a frog be cut off, or its brain removed so that the spinal cord is the only great nerve centre remaining, the animal will draw up its leg with clockwork-like regularity whenever the foot is pinched or an irritant such as vinegar or other dilute acid is applied to it. The time which elapses between the application of the irritant and the withdrawal of the foot varies according to the strength of the irritant. But if the strength of this remains the same the time will hardly vary more than a second or two at each application, and this will continue for hours together, so that to study the effect of any agent whatever upon the reflex function of the spinal cord is the easiest thing possible. Now Nothnagel found that when the sciatic nerve of one side, *e.g.*, the *left*, is irritated by a faradic current, no reflex action can be obtained in the other, or *right* leg, by any irritant whatever applied to the *right* foot. This arrest of reflex action endures so long as the current continues, but as soon as it ceases the reflex irritability reappears. It is extremely difficult to explain this phenomenon except by attributing it to excitation of certain inhibitory centres by the irritation of the sciatic nerve, and as the brain and spinal cord had been removed it is hard to see where those centres could be except in the cord itself.

The first thought that occurs to one is that the arrest of reflex action was only apparent, and was really dependent on tetanic spasm of some of the muscles, which kept the leg fixed in one position, and prevented it from moving. This was not the case; all the muscles were completely relaxed and the leg hung perfectly limp. There was no more evidence of tetanic spasm than there is in the heart when its pulsations have

¹ Nothnagel, 'Centralblatt f. d. Med. Wiss.,' 1869, p. 211.

been stopped by irritation of the vagus. In both cases the action seems to have been one of pure inhibition, arresting the production of motor energy.

The only muscular contractions observed took place when the faradic current was applied and removed.

At the moment when it was applied to the *left* sciatic a momentary jerk occurred in the *right* leg, which then remained motionless and insensible as already described as long as the current passed. After the current ceased several clonic contractions often occurred in the right leg. But this condition only continued while the cord was in its normal state. The results are very different when the sciatic is irritated twenty-four hours after the removal of the brain, and sufficient time has elapsed for certain alterations to take place in the cord. Clonic contractions then occur in the right leg whenever the faradic current is applied to the left sciatic, and continue all the time that it is passing. This instance of a *constant* irritation producing *clonic* convulsions is of great interest as giving us some insight into the cause of intermittent spasm generally. Nothnagel attributes it to paralysis of the inhibitory centres in the cord, which he thinks lose their vitality sooner than the motor centres.

Before going any farther, it may be well to attempt to give some sort of explanation of the manner in which rhythmical actions may be supposed to arise from a constant stimulus, and how inhibition may affect them. For illustrations of this we shall return to the heart, although rhythmical action is perhaps universal in the body, and constant contraction is only apparent. Indeed, Herbert Spencer extends the prevalence of rhythm much more widely still, for in his 'First Principles' he says, 'Rhythm is a necessary characteristic of all motion. Given the co-existence everywhere of antagonist forces, a postulate which as we have seen is necessitated by the form of our experience, and rhythm is an inevitable corollary from the persistence of force.'¹

The usual supposition regarding the occurrence of inter-

¹ Herbert Spencer's 'First Principles,' 2nd ed., p. 271.

mittent or rhythmical movements, arising from a constant stimulus, is that the nervous action in motor ganglia is prevented from transmitting itself to the muscles by some resistance opposed to it, so that it must gradually increase and reach a certain intensity before it can break through this opposition and set the muscle in action, just as when air is blown through a tube which dips into water, it does not pass in a steady stream, but in a succession of bubbles. The air is constantly pressing onwards, but it is not at first able to overcome the resistance of the water, and it gradually accumulates at the mouth of the tube until its tension is sufficient to overcome the resistance of the water, and a bubble passes off. The tension is thus reduced, and a certain time is necessary before it again rises sufficiently to cause a second bubble.

In somewhat the same way we suppose that certain changes, probably of a chemical nature, are going on constantly in a motor centre, *m*, Fig. 4, which tend to cause contraction of the muscle *A*, but there is a resistance to their transmission through the nervous path *n* to the muscle. The change is going on and generating motor power constantly under the influence of a stimulus supplied to the motor centre either by something in the blood, as in the case of the respiratory centre, or by an irritation transmitted to it along a sensory nerve, as in the spinal cord. But it is only at intervals that the motor power thus produced becomes sufficiently great to break through the resistance which opposes its action on the muscle, and causes the latter to contract, so that between every explosion of this sort a period of rest is necessary. The quickness with which explosion follows explosion may be increased either by accelerating the production of motor energy in the nerve centre or diminishing the resistance to its expression. On the other hand, the intervals may be increased by diminishing the production of motor energy, or augmenting this resistance. The simplest example of this sort of action is afforded by the heart, which normally makes a sudden contraction, then relaxes, and, after a certain pause, contracts again. Its action may be rendered slow or even be arrested

altogether by irritating the vagus, and its pulsations may be greatly increased by irritating the cervical sympathetic.

We suppose the cardiac pulsations to be due to the rhythmical motor impulse supplied to the muscular fibres by ganglia in the walls of the heart itself, and we may explain this action of the vagus by supposing it either to increase the resistance to the passage of this impulse from the ganglia to the muscle, or to retard the production of motor force in the ganglia. If it simply increased the resistance, the motor impulse would accumulate, and we should expect that when the heart did beat its pulsations would be very strong. This is not the case, however, for Ludwig and Coats have shown that after irritation of the vagus the heart-beats are weaker instead of being stronger than before.¹ We are thus led to adopt the second alternative, and suppose that the vagus lessens the production of motor force. On this supposition, the results of Ludwig and Coats are just what we should expect, and we find it further corroborated by other facts. In almost all cases nerves are sooner exhausted than muscles, and when we find a mixture of muscles and nerves ceasing to act, we are generally right in laying the blame on the nerves. Now the motor ganglia of the heart cannot go on indefinitely, and they may be exhausted just like other structures. It is natural that if they are working hard they should be soon exhausted, if they are working slowly they should last long. It is found by experiment that if the vagus is stimulated so as to make the heart work slowly it will continue to beat for a long time, but if galvanism be applied in such a way as greatly to accelerate its pulsations it quickly becomes exhausted and stops.² This stoppage we may fairly attribute to exhaustion of the motor centres, and the long vitality displayed when the vagus is irritated to its diminishing the changes in them and consequently preventing their force from being expended.

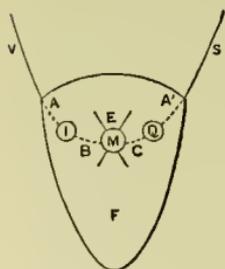
It appears, however, that the vagus does not act upon the motor centres directly, but only through the medium of

¹ Coats, Ludwig's 'Arbeiten,' 1869, p. 200.

² Ludwig's 'Physiologie,' vol. ii. p. 91.

certain inhibitory ganglia,¹ which are the real causes of the diminished production of motor energy. These ganglionic centres are usually set in action by the vagus, but they may be excited even when the vagus has been paralysed by certain poisons, and thus it is that we know of their existence apart from that nerve. All phenomena connected with the cardiac movements are studied most easily in the heart of the frog, which is much less complicated than that of the higher animals, though its nervous arrangement seems to be essentially similar. The frog's heart may be stopped either by galvanising the vagus or the venous sinus, between the vena cava and the right auricle. As the vagus enters the heart at this point, it might be thought that the effect of galvanising the sinus was due to irritation of the ends of the vagus in it, but this is not the case. If the frog be poisoned by a minute dose of nicotia the vagus is paralysed, and galvanising it no longer has any effect on the heart. Notwithstanding this, the pulsations cease at once if the venous sinus is galvanised, and this we attribute to excitement of the inhibitory ganglia by the current, although the vagus through which they usually receive their stimulus is completely paralysed.² Other arguments might be adduced in support of this view, but I have given these in another place, and it is unnecessary to enter upon them here. The

FIG. 8.



accompanying diagram may render more intelligible the ideas regarding the nervous system of the heart which I have been trying to convey. The muscular walls of the heart itself are indicated by the letter F, the motor ganglia by *m*, and the nervous filaments which connect these by E. The letter v denotes the vagus, and i the inhibitory centre through which it acts on M. A is the termination of the vagus in the heart, and is the part supposed to be paralysed by nicotia while i still remains active. It will be seen that on the other side of M there is another centre Q corresponding to i.

¹ Schmiedeberg, Ludwig's 'Arbeiten,' 1870, p. 45.

² Schmiedeberg, Op. cit. p. 42; and Truhart, 'Nicotinwirkung. Inaug. Diss. Dorpat,' 1869, p. 50.

This indicates a supposed quickening centre, which increases the production of motor energy in M instead of diminishing it as I does. It is connected with the sympathetic nerve s . When this nerve is irritated it quickens the pulsations of the heart. The reason for supposing that it acts on the motor centres M indirectly through other ganglia, and not directly, is that when it is irritated the pulsations of the heart do not become quicker till after a short time has elapsed, and they remain quick after the irritation has ceased. This seems to indicate the presence of some apparatus between the sympathetic nerves and the motor ganglia, more especially as the pulsations are quickened immediately when the current is sent through the heart itself instead of through the sympathetic nerves, and they again become slow immediately after the current has been discontinued.¹

There is no other part of the body in which inhibition has been so thoroughly studied as in the heart, and the diagram given above, though quite hypothetical, may help us to understand the mechanism by which it is effected. Although we cannot say that the arrangements for inhibition are the same elsewhere as in the heart, it is by no means improbable that they are similar, and we may perhaps profitably apply this diagram to the explanation of inhibition in the nervous centres. In the heart we have single contractions following each other at considerable intervals, but if we suppose the resistance to the action of the motor centres to be lessened, so that instead of having to accumulate long, and to rise to a considerable intensity before being able to act on the muscular fibres, the motor energy could be transmitted to them in small shocks, quickly succeeding one another, there would not be time for the fibres to relax before another contraction was induced, and thus they would remain steadily contracted. This is exactly the condition we find in voluntary muscles. However constant their contraction may seem to be, it is found on examination to consist of a great number of small contractions following one another so rapidly that they can only be detected by the use of delicate

¹ Schmiedeberg. *Op. cit.* p. 47.

instruments. But if the muscles, or motor centres, or both, are wearied by exertion, the contractions become less rapid and more extensive, so that anyone may see them if he will only perform the simple experiment of holding a few pounds' weight at arm's length for five minutes. When his arm begins to shake he may steady it by an effort of the will, which being translated into physiological language probably means that he transmits from his cerebrum a certain impulse which increases the production of energy in those cells which directly preside over the motions of the muscles. Very soon however this stimulus loses its effect, and the trembling goes on as before. It has already been mentioned that the sphincter ani executes rhythmical movements in the dog after the spinal cord has been cut although it remained steadily contracted before the operation was performed. This seems somewhat analogous to the trembling just mentioned, though it is hard to say exactly to what cause it should be attributed.

There are also certain inhibitory centres which are situated within the cranium itself. These have been localised in the frog by Setschenow,¹ who found that irritation of the optic lobes, which correspond to the corpora quadrigemina of the higher animals, greatly diminishes reflex movements.

An attempt to discover and localise similar inhibitory centres in the higher animals was made by Simonoff, and he states that he actually did succeed in diminishing reflex action by irritating the brain of dogs by an electrical current.² I have repeated his experiments on kittens in the West Riding Asylum, where my friend Dr. Crichton Browne supplied me with all the necessary apparatus, and for his kindness on this and many other occasions I take this opportunity of thanking him most heartily. Unfortunately I have not been able to confirm Simonoff's observations, although I have irritated the brain both by constant and induced currents, and have varied the irritation applied to the foot by using sometimes an induced current,

¹ Setschenow, 'Physiologische Studien über die Hemmungsmechanismen,' c. Berlin, 1863.

² Simonoff, Du Bois Reymond and Reichert's 'Archiv,' 1866, p. 545.

and sometimes pinching as an irritant. The number of experiments I have performed, however, is small, and a more extended series may yet enable me to produce, like him, an inhibitory action by stimulating the brain.

McKendrick¹ has endeavoured to ascertain whether reflex action is diminished by the application of a faradic current to the cerebral convolutions, and has occasionally witnessed a slight diminution, but more generally has obtained no positive result. He has found however that the convulsions which occur in pigeons immediately after decapitation may be arrested by the application of a faradic current to the upper part of the spinal cord. When movements of the wings are excited by the application of a gentle galvanic stimulus to their nervous centre in the cord, the motions are at once arrested by a faradic current to a part of the cord nearer the medulla. These results may be ascribed to the presence of inhibitory centres in the cord itself, but McKendrick considers them to be rather the results of irritation of nervous fibres passing down in the cord from cerebral inhibitory centres than of excitation of the inhibitory centres in the cord itself. Although the experimental data regarding inhibitory centres in the brain is still defective, yet sufficient evidence has, I think, been adduced to warrant us to believe in their existence, and to accept provisionally Simonoff's conclusions. According to him the most decided inhibition is obtained by stimulating, not the corpora quadrigemina as might have been expected, but the frontal lobes, those very parts of the cerebrum which my friend Professor Ferrier has found to have no motor action whatever. It seems therefore quite possible that it is in this part of the brain that the inhibitory centres are situated which enable a man to keep his hand or foot motionless while some one is tickling it, but it would be rash indeed for anyone to assert this positively without more evidence than can at present be brought to bear on this point.

Several months ago my friend Dr. Crichton Browne observed a case which seems to show not only that inhibitory

¹ McKendrick, 'Edin. Med. Journal,' Feb. 1874.

centres exist in the brain, but that these may be morbidly excited by cerebral disease. He has headed it in his notebook, 'Inhibitory action of the brain,' and with his permission I extract it verbatim.

'February 3, 1874.—M. M. W., who is now labouring under erysipelalous encephalitis in Ward No. 2 ; I noticed yesterday that while there was distinct evidence of irritation of the surface of the cerebrum (incessant pronation and supination of the hands) there was an abolition of reflex action of the cord. Tickling the soles of the feet and pricking the toes produced no movement whatever. This is a most interesting observation. It seems to prove that nerve currents set in motion by irritation of the brain or of some of its convolutions transmitted down the cord may inhibit reflex action.

'Two days ago subsequently to this entry the man became completely unconscious, and reflex action as determined by pricking and pinching returned. After death the brain was found disorganised by erysipelalous changes.'

In this case excessive inhibition seems to have been produced by excitement of the cerebral centres through inflammation and to have ceased when these centres became disorganised.

Another good example of inhibition is afforded by the effect of stimulation of various nerves upon respiration.

The respiratory movements, though performed by muscles having striated fibres, are regulated by a nervous centre chiefly situated in the medulla oblongata, and acting independently of the will, though it may be influenced to some degree both by it and by the emotions. It is not quite certain whether it is a reflex centre kept in action by constant stimuli applied to it through the medium of sensory nerves, or whether its stimulus consists in chemical changes taking place in itself and depending on the condition of the blood with which it is supplied. Nor has it yet been certainly determined whether it is a single or double organ, though it is usually assumed to be double, *i.e.*, to consist of an inspiratory and an expiratory centre. The inspiratory one is at all events the more important in ordinary breathing, and so long as respi-

ration is tranquil there is hardly any active muscular exertion at all in expiration, the air being expelled from the lungs by the return of the ribs and abdominal viscera to the positions from which they had been removed by inspiration. But if the breathing is impeded, expiration becomes more active, and may become very marked during emotional excitement, as in the acts of sighing or laughing.

Respiration may be completely arrested by irritation of certain nerves, and generally it stops either during a condition of full inspiration with the diaphragm spasmodically contracted, or in full expiration with the diaphragm completely relaxed, though this is not invariable, and a condition is met with which is midway between the two extremes. Arrest in full inspiration with spasmodic contraction is produced by irritation of the vagus trunk in the lower part of the neck, or by galvanising one of the lungs. Complete inhibition of the inspiratory centre and consequent paralysis of the inspiratory muscles occurs when the cervical branches of the vagus, viz. the superior and inferior laryngeal, the pharyngeal, and œsophageal nerves are galvanized, or when the teacheal branches are irritated by a tight ligature round the windpipe.¹ The same result follows irritation of the nostrils and lips, mechanically, electrically, or by the sudden application of hot or cold water to them, by irritation of the foot, of the skin behind the ear, of other parts of the body also, and of the chest by immersion in cold water. In all these cases the stimulus applied to the afferent nerve is conveyed to the medulla, and there inhibits the inspiratory centre.

To a certain extent vocalisation depends on respiration, but it requires something besides, and vocalisation may be arrested although respiration goes on. This is seen in Goltz's croaking experiment or Quakversuch.² If the frog from which the cerebral hemispheres have been removed is gently stroked between the shoulders or along the flanks, it will croak once at each stroke with machine-like regularity. In this it differs from a frog in its normal condition, for its cere-

¹ Rosenthal, 'Die Athembewegungen und ihre Beziehungen zum Nervus Vagus, Berlin, 1862; and Du Bois Reymond and Reichert's 'Archiv,' 1862, p. 226.

² Goltz, 'Centralblatt d. Med. Wiss.' 1865, p. 705.

bral hemispheres enable the latter either to control or to strengthen the reflex, and sometimes it will not croak at all, while at others it will croak several times at each stroke. But even those frogs which will not croak at all so long as they retain their cerebral hemisphere croak as readily and regularly as the others whenever these nervous centres are removed. It is not every form of stimulation however which causes frogs thus prepared to croak, for although they do so regularly when stroked by the finger or with any broad surface, which answers best if it is also smooth, yet if touched or stroked with a sharp instrument they do not croak, but execute defensive movements. Electrical or chemical irritation of the skin of the back never makes them croak, and when any nerve trunk is irritated they sometimes utter a sound indeed, but it is the cry of pain, and never the croak of contentment. Here in the frog is an example of a similar kind to the different effects of tickling and scratching in man, in whom tickling produces laughter and violent reflex movements, while scratching does not do either the one or the other. Laughter is usually a sign of pleasure in man just as croaking is in the frog, but as anyone knows who has been severely tickled when a child, the laughter caused by titillation is purely reflex, and may not only be unconnected with pleasure, but may be associated with a feeling of intense pain, so great indeed as to excite a sense of impending dissolution. Indeed, the common phrase of tickling a person to death might not unfrequently find warrant in fact if the pain gradually excited by the spasmodic laughter did not arrest the reflex process and thus effect its own cure, the child which has been laughing till it was black in the face suddenly ceasing its convulsive movements, uttering a loud yell, and bursting into tears. In the frog a somewhat similar instance of inhibition is observed, for by strongly irritating any sensory nerve the croaking may be completely arrested, and no amount of stroking will produce any effect.

In the instances of inhibition already given we have seen the action of muscles voluntary and involuntary, and of glands arrested by the influence of nerves, but there is another kind of inhibition no less important and still more

complicated. This may be termed inhibition of inhibition, or inhibition of inhibitory centres as distinguished from the inhibition of motor centres which we have hitherto been considering. At present our notions of nervous action seem to be getting as involved as the Ptolemaic system of astronomy, and just as epicycles became heaped upon cycles so nerve centres are being added to nerve centres. And yet, clumsy though the system may be, it serves at present a useful purpose, and may give us real aid until a better is discovered.

We have already seen the power of inhibitory centres to arrest the action of motor centres, but these inhibitory centres themselves may be kept in check by other centres, so that the original motion is allowed to go on. One inhibitory centre kept in check by another may in fact be compared to a coachman who, when restraining or pulling up a horse, may be himself restrained by his master's command to let the animal go on. It is evident that the same result may be obtained by allowing the man to do as he pleases, while applying the whip to the horse in such a way as to render restraint difficult or impossible. And so, the cases of what I have called inhibition of inhibition may be due either to arrest of the functions of inhibitory centres, or increased power of the motor ones to such an extent that the inhibitory ones cannot restrain them. It is often very hard to say which of these two is the real cause of arrested inhibition. Frequently, no doubt, there is a mixture of both, just as the master may apply the whip at the same time that he prevents his horse from being held in. In many instances, however, it is probable that the inhibitory centres are simply arrested without the motor ones being stimulated, as we find the effect of the former disappear without the appearance of any evidence of action in the latter, although motor action may become superadded if the stimulus which arrested inhibition be increased. We have already seen that the normal vaso-motor action of the ganglia in the penis may be arrested by inhibitory centres in the cord, so that the vessels dilate when these centres are stimulated by means of certain afferent nerves. But these inhibitory centres are

themselves checked by means of irritation applied to certain other afferent nerves, and the vaso-motor ganglia are then allowed to act undisturbed. Thus the dilatation of the vessels in the penis of a dog, whose spinal cord has been divided, may be at once stopped and the organ restored to its normal condition by pinching the foot of the animal or irritating the scrotum or anal region by a faradic current. Indeed, it appears that here also, just as in tickling, a greater irritation counteracts the effect of a slighter one, for while touching or rubbing the foreskin induces erection, irritation of the same part by an electric current at once arrests it.¹

The inhibition of vaso-motor centres which occurs when the intestines are struck may be prevented, and the heart and vessels allowed to perform their functions as usual, by pinching the toes of a frog at the same time that Goltz's experiment is performed.² The pinching alone causes the vessels to contract, at least, in the higher animals, but when the intestines are struck it probably only prevents them from dilating without producing any actual contraction. It has already been mentioned that certain emotions depress the circulation in much the same way as irritation of the intestines, and their effect may be counteracted by similar means. I believe that these means are frequently employed instinctively, for I well remember doing so myself while watching a frightful operation, during which no chloroform could be given to the patient. My eyes were beginning to grow dim, I felt very faint, and longed exceedingly for a drink of cold water. None was to be had, but I found that by biting my lip and pinching my fingers I could succeed in sitting upright though I could not prevent the perspiration from breaking out on my forehead. My emotion had depressed my arterial tension, but the irritation I applied to my lips and fingers partly counteracted this effect, and the cold water, had I been able to obtain it, would have done so likewise, by causing the vessels of my stomach to contract, and thus raising the pressure of blood in my arteries.

The mention of this incident leads me to consider another of the ways in which the effect of nervous action appears to be

¹ Goltz, Pflüger's 'Archiv,' *loc. cit.*

² Goltz, 'Centralblatt d. Med. Wiss.,' 1868, p. 593.

prevented, viz. by the action itself being diverted into another channel than the usual one. Some time afterwards I was assisting in the capacity of dresser at a similar operation. The duty of restraining one of the patient's legs was entrusted to me, and I was obliged to exercise considerable force in order to prevent movement. Although I could see the operation perfectly I felt no faintness whatever. The impression made by the operation on my visual nerves seemed to find an outlet in the muscular exertions I was compelled to make, and it produced almost no emotion and no effect whatever upon my circulation. A friend of mine, who was looking on, and feeling, as I had formerly done, very uncomfortable, wondered how I could be so unconcerned, and being ignorant of my former experience concluded that I was different from himself. The difference however really was not in our nervous systems, but in the circumstances (totally overlooked by him) which directed the impressions made on our sensory nerves along different channels.

It is to this power of slight differences in external conditions to divert a stimulus along different nervous channels, and the different facilities with which a passage is afforded to it by each channel, that many instances of apparent inhibition are due, and this serves to explain how the same stimulus may produce very different effects in different individuals, or in the same individual at different times.

The nervous system is no doubt a most complicated piece of mechanism, and certain differences in its structure are found in different individuals, so that one cannot predict with absolute precision the effect which an external impression will produce in any one person. Notwithstanding this, the agreements between the structure of the nerve centres are so infinitely greater than their differences, that in a general way we are quite sure that similar impressions made upon afferent nerves, either of general sensation or special sense, will excite in nearly all individuals somewhat similar reflex actions, sensations, emotions, or ideas. These will generally express themselves in similar acts. The lower the nerve centre is through which the impression reacts, the more constant are the results, and the differences between

them increase with the higher position and complication of the centres concerned. To recur to an instance I have already employed, the simple reflex closure of the eyelids is certain to occur in almost everyone when the cornea is touched ; it will happen almost, though not quite so invariably when an object is merely approximated rapidly to the eye without touching ; a blow on the eye will excite in almost everyone a feeling of anger more or less transient, but whether the feeling will find expression in a yell, as in the child, in expostulation, or a return blow, as in most adults, or in the injured man turning his other cheek to the smiter, as in certain exceedingly rare instances, depends on the nature of the nervous path through which the stimulus must travel before it excites motor ganglia, and through them the muscles to action. The nature of this nervous path again depends on the state of development of its different parts, on their comparative size and connection with other parts as determined by hereditary transmission, and on the modifications they have undergone through that assemblage of external influences to which the individual is constantly subjected during life, and which constitutes his education. In the child which has been struck we may often see irregular convulsive movements of the hands and arms at the same time that it is yelling, but its motor centres have not yet been sufficiently developed to co-ordinate these movements into regular acts of defence or retaliation, as in the boy. The boy again will ward off or return a blow at once, but will generally forget any angry feelings very shortly afterwards, the whole having been expended in action. In the man the only trace of the yells and convulsive movements seen in the child may be a deep inspiration and involuntary clenching of the fist, while the stimulus of the blow and the angry emotions it has awakened, instead of discharging themselves at once, may excite ideas of future revenge, which although more or less constantly present to the mind may find no expression in action until years after the occurrence which originated them. In other instances the emotion of anger excites the idea that its indulgence is wrong, and the individual strives to suppress it. In some instances he may apparently

succeed in annihilating it, but in others he may only be able to restrain a sudden explosion, the emotion expressing itself in a prolonged irritability and captiousness, while in yet others it may be converted into a stimulus to benefit the wrong-doer.

In all these examples we see a general resemblance, and at the same time very striking differences, between the results produced by the application of stimuli to certain afferent nerves. On examining these results more closely, we find that they may be divided into two classes, viz., the effect of the stimuli on what we may term the receptive or sensory centres, which occurs internally, and secondly, the external manifestations which these receptive or sensory centres induce through the medium of motor centres. The resemblances occur chiefly in the first class of effects, and the differences in the second. More or less pain, and at first more or less anger, succeeds a blow in everyone, whether child or man, and the differences between the kind of reaction it induces are almost entirely confined to the ways by which the pain or anger expresses itself externally.

The action which any nervous centre excites when it is itself stimulated through an afferent nerve depends upon the efferent nerves with which it is connected either directly or indirectly through other centres. Thus irritation of a frog's foot produces movement because the reflex centres which it stimulates are connected with motor nerves, a touch on the dog's foreskin produces dilatation of vessels because the nerve centre it stimulates is connected with vaso-inhibitory nerves, and a sapid impression on the tongue produces secretion because the nerve centre it stimulates is connected with secretory nerves. Sometimes we have these actions on muscles, on vessels, and on secretion occurring alone, because only one centre is stimulated, at others we have any two of them or all three occurring together. Thus a gentle touch on the eye will only cause the eyelid to close, a sudden emotion may cause a tear to fall, but a grain of sand may not only cause spasmodic closure of the lid as it falls into the eye, but a copious flow of tears and dilatation of the conjunctival vessels if it remains there. The eye may be closed

and the tears flowing while the vessels are hardly dilated at all, or the eye may be closed and red with hardly a tear, or, again, the eyes may be open while the vessels are dilated and the tears flowing freely. The sensory nerves of the eye can thus excite motor, vaso-inhibitory and secretory nerve centres, and sometimes one effect predominates and sometimes another.

The same thing is true of many other afferent nerves, and Brown-Séguard says that three kinds of reflex phenomena may be due to stimulation of centripetal nerves, firstly, a contraction of muscles or of any kind of contractile element; secondly, a secretion; thirdly, a change in the nutrition of some part of the body.¹ Although he thus classes contraction of muscles voluntary and involuntary together in one class, he gives a diagram to show what he means, and according to this the structures affected are first, muscles; second, glands; third, vessels. The division given in his diagram is more convenient than the other, for in the latter he separates contraction of vessels and dilatation of vessels, both of which may result from stimulation of an afferent nerve. To these three classes we might add a fourth, viz. nerve centres, for sometimes a stimulus seems to have no effect either on muscles, glands, or vessels, producing instead a change in the nerve centres, which may very considerably modify all their future actions.

Although a stimulus to an afferent nerve may sometimes stimulate a single nerve centre and produce a single effect, it generally acts on several. Indeed, it may be compared to an electric current passing through a branching wire. Part will pass through one branch, part through another, and a large portion will traverse any branch which presents little resistance, while only a small fraction will pass through one which offers a great resistance. In one branch the electricity may be transformed into mechanical work, in another into heat, in a third into light,² and in a fourth it may be made to decompose water, and thus be stored up as potential energy, which may be expended long afterwards in the ex-

¹ Brown-Séguard, 'Physiology of the Central Nervous System,' p. 152.

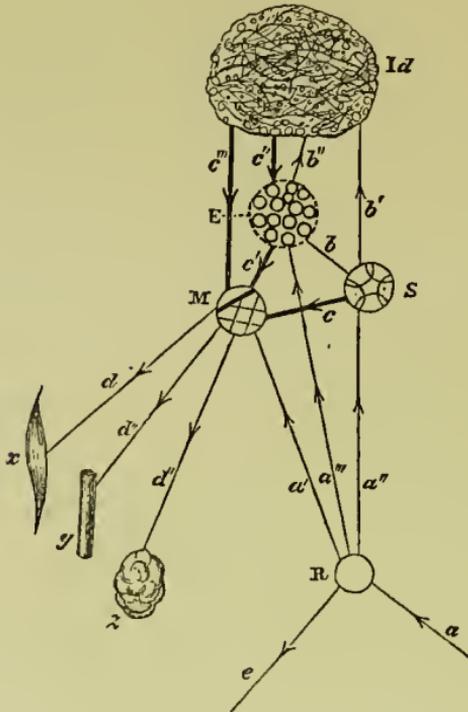
² Marey, 'La machine animale,' p. 6.

plosion of the gases formed from the water. If the current passes through all the branches at the same time, less work will be done by any of them than if the current passed through only some of them or through one alone. And if one of the branches be too small for the current which passes through it, it may be melted and its conducting power forever destroyed.

Likewise a stimulus to a nerve centre will excite action which may pass off partly in motion, partly in secretion, partly in vascular change, and partly in alteration of the nervous system. The sight of a dear friend in great danger, for example, may excite in the emotional centres through the optic nerves a certain nervous action, which may expend itself partly in muscular efforts to save him, partly in tears, partly in vascular changes, which induce paleness and fainting, and partly in such an alteration in the ideational centres that the scene is deeply impressed on the memory, and never can be recalled without a shudder. The proportion in which the muscular, glandular, and vascular systems and the ideational centres are affected by the stimulus depends on the ease with which the action excited by it in the emotional centres can expend itself upon each. Now nervous action, like an overflow of water, seems to wear a channel for itself, and the oftener it passes along any nervous path the more readily does it find its way along that one in preference to any other. If a stimulus excites to action once it will tend to do so again, and if it affects the vessels, glands, or ideational centres once it will have an increased tendency to affect these structures on a subsequent application. These tendencies are transmitted to posterity, and the son of a man in whom a stimulus leads to action will have more chance of possessing this quality than the progeny of a man whose leanings are towards reflection. They may of course be modified by the influence of the maternal parent, and by the circumstances under which the life is spent. The alteration of the hereditary tendencies effected by these may be very great, so much so that it is possible that they may be diverted almost entirely into a new channel, and the dreamy child may grow into a promptly energetic man. Sex also modifies the channels, and although

much depends upon training, and it may be to a great extent the education of girls which tends to make the emotions, excited by such a scene as I have supposed, vent themselves on the secretions and circulation and on the muscles of respiration rather than those of the extremities, leading in them to tears, fainting, and screams or sobs rather than to action; though the tendency is probably to be ascribed much more to the different nervous organisation of the two sexes than to the

FIG. 9.



alteration produced in it by the education of the individual. How much is due to the modifications produced by training during many generations is a different matter.

The ideas I have been attempting to convey may be rendered more intelligible by the accompanying diagram, rude and imperfect though it may be (Fig. 9). R may be supposed to represent a reflex centre, S a sensory centre, E an emotional, Id an ideational, and M a motor centre. The latter is supposed to represent an agglomeration of several motor centres, capable of producing co-ordinated actions, vascular changes, and secretion. This power is indicated by the

three efferent nerves, d , d' , and d'' , proceeding from it to a muscle x , a vessel y , and a gland z respectively. a is an afferent nerve which branches off in a' , a'' , and a''' to the motor, sensory, and emotional centres, s is connected by b and b' with the emotional and ideational centres, and E with $\text{I } d$ by b'' . Efferent nerves c and c' pass from s and E to M , c'' from $\text{I } d$ to E , thus connecting $\text{I } d$ indirectly with M , while c''' connects $\text{I } d$ and M directly. Let us now suppose a to be the nerves of the eye, including both the optic and the fifth. A gentle stimulus applied to it, such as dryness of the cornea or a touch upon it, may proceed to a reflex centre R and cause a simple reflex action, such as closure of the eyelid, without any perceptible sensation, as in ordinary winking. A stronger stimulus, such as a grain of sand in the eye, may not only produce this reflex action, but it may be conducted along a to other centres M , and produce a flow of tears, redness of the conjunctive, and contraction of the facial muscles through the nerves d'' , d' , and d . It is just possible that all this might occur without any sensation being felt, but generally the stimulus would pass partly along a' to M , inducing actions of the kinds just mentioned, and partly along a'' to s , where it would excite sensation. The action excited in s may in its turn stimulate M through c , and cause movements of the facial muscles expressive of pain. The stimulation of a by the sand, however, would excite little or no emotion, although a stranger unacquainted with the cause of the weeping might possibly attribute it to grief. The stimulus of a blow on the eye may not only pass up a' and a'' to M and s , but also up a''' to the emotional centre E , and there produce the emotion of anger in addition to the motor and sensory effects already enumerated. The action thus set up in E might discharge itself along c' on M , exciting it to renewed and increased activity, or along b'' on $\text{I } d$, exciting there ideas of various kinds, the nature of which would of course depend on the constitution natural or acquired of $\text{I } d$. If the motor centre M is undeveloped, as in the child, neither the movements excited in it reflexly through a' , nor by a stimulus from the emotional centre E , will be sufficiently co-ordinated to serve any definite purpose. If they are well developed, as in a boy, the

reflex through a'' may lead to useful movements of defence, while the emotional stimulus transmitted through c' may excite movements of offence. In the boy, as I have already said, both stimuli, the secondary one from the emotional centre ϵ as well as the primary one from the blow, will expend themselves almost immediately through motor centres. In a man, however, it may pass almost entirely up a''' to the emotional centre, hardly producing a movement of the face or a feeling of pain, but expending itself almost entirely on ϵ , and exciting most intense anger. The action thus set up in ϵ , instead of discharging itself in motion through m , as in the boy, may in its turn act only on $1 d$, exciting there the ideas of injury done and of revenge to be cherished. These may lead to action by-and-by, as in a Corsican vendetta, where the avenger pursues his intended victim for years until a favourable opportunity occurs, and by a thrust of his dagger he satiates his thirst for vengeance. Or they may excite ideas of the sinfulness of revenge and the advantages of peace, and thus lead to pacific actions. Or finally they may be nearly effaced by the effects of other stimuli of a different nature, arising from the pressure of daily work and daily care, and lead to no action at all, though it is probable that they always leave an alteration more or less slight in the ideational centres.

The fact that the direction taken by a stimulus depends to a great extent on the frequency with which other stimuli have passed along certain nervous paths, and thus, as it were, prepared the way for it, is admirably illustrated by Herbert Spencer in his essay 'On the Physiology of Laughter.'¹ The muscular actions constituting laughter are distinguished, he says, from most others by being purposeless, but yet they are regulated by rule. For an overflow of nerve force undirected by any motive will manifestly take first the most habitual routes, and if these do not suffice will next overflow into the less habitual ones. The jaws, lips, and tongue are most frequently used to express emotion, and thus certain muscles round the mouth are first affected. Then follow the

¹ Herbert Spencer's 'Essays, Scientific, Political and Speculative,' 2nd series, p. 111.

muscles of respiration, which are, next to those of articulation, most readily affected by feelings of all kinds, they being more constantly implicated than any others in the various acts to which our feelings impel us. Should the feeling to be expended be still greater in amount—too great to find vent in these classes of muscles—another class comes into play, and the upper limbs are set in motion, and the hands are clapped or rubbed together. Last of all, when the other channels for the escape of surplus nerve force have been filled to overflowing, a yet farther and less used group of muscles is spasmodically affected, and the head is thrown back and the spine bent inwards. The principles to which the phenomena of laughter conform, according to Mr. Herbert Spencer, are that feeling excites to muscular action, that when the muscular action is unguided by a purpose the muscles first affected are those which feeling most habitually stimulates, and that as the feeling to be expended increases in quantity it excites an increasing number of muscles, in a succession determined by the relative frequency with which they respond to the regulated dictates of feeling.

The direction taken by stimuli is determined by the channels in which a nervous current happens to be passing at the time the stimulus is applied, as well as by those in which they habitually run. Thus a kick on the shins is extremely painful when one is standing still, and it is somewhat apt to arouse a feeling of anger even when the injury has been unintentional, but if received during a struggle at football it does not cause so much pain nor has it the same tendency to excite anger, but it does increase the vigour of the recipient's exertions.

The sensory stimulus, instead of strongly affecting the sensory and emotional centres and exciting pain and anger, seems hardly to act on them at all, and to pass instead to the motor centres, where it expends its force in originating muscular action. In this case the motor centres which preside over the movements of the body are in full action, and the additional stimulus supplied by the kick is almost unconsciously directed into the same channel, and simply adds to the force of the stimuli they supply to the muscles. The use of whip

and spur by jockeys during a race is an example of the same sort.

But sensory stimuli may be also diverted voluntarily into a motor channel, not for the purpose of increasing muscular power but of diminishing pain. A sailor when about to be flogged prepares himself by placing a bullet between his teeth,¹ and the more heavily the lash descends the harder does he bite the lead. Most people do something of the same sort when in a dentist's chair. Either they writhe about, or clench their hands, or catch hold of something which they press, and I think most persons will agree that the muscular exertion does ease the pain. The writhing may be considered to be the involuntary reflex action, in exciting which a part of the sensory stimulus is expended; the clenching of the hands or compression of any object held within them is also reflex, but its exact nature has been determined by the will directing the stimulus into a particular channel. The emotional centres may also be protected from the effect of a stimulus in much the same way as the sensory ones by diverting a part of it to the motor ganglia. Indeed, it has become proverbial that violent emotional expression is to some extent incompatible with permanency of the emotion. The ordinary motor impulses too, by which the muscles are excited to action and express an emotion, may be directed voluntarily into a different channel, and a man may simply bite or compress his lips instead of allowing vexation or anger to manifest themselves in speech, act, or facial expression.

Whenever emotional excitement is prevented from discharging itself externally by motor channels it is very apt to vent itself upon the internal viscera, and the principal channel through which it does this seems to be the vagus. Almost everyone is familiar with the feeling of compression around the chest induced by grief, especially if associated with suspense. It is not peculiar to civilised peoples, for negroes from the interior of Africa, when taken to Zanzibar to be sold as slaves, often die without any apparent cause except grief and home-sickness. On being asked where their trouble is, they

¹ Darwin, 'Expression of the Emotions,' p. 72.

lay their hands on their bosoms and say it is there. This feeling of compression, as if by the clutch of a giant's hand, is well expressed by the German word *Beklemmung*. It may exist apart from the emotion which has caused it, for I have myself experienced it after the transient sorrowful thoughts which had originated it had quite vanished from my mind. It is in all probability due to irritation of the vagus, as a similar feeling is produced when the trunk of the nerve is irritated artificially. This was discovered by the late Professor Czermak,¹ who had over his right vagus a small indurated lymphatic gland, by pressure upon which he was able to irritate the nerve and observe upon himself the effects of its irritation. The vagus is an extremely complex nerve, and it would take too long to discuss its functions here, but they may be briefly summed up by saying that it regulates the depth and frequency of the respirations, the force and rapidity of the cardiac pulsations, and influences to a great extent the secretions and movements of the stomach and intestines.

Under one or other of these three heads may be grouped most of the effects which emotions produce upon the viscera, and thus we are, I think, perfectly justified in thinking that it is through this nerve chiefly that emotions influence them. The bated breath of suspense, the sigh of relief, the sob of sorrow or the laugh of joy, the quickly throbbing heart of pleasure or the slower pulse of pain, the improved digestion of hope, the impaired appetite of anxious expectancy, the nausea of disgust, and even the movements of the bowels, significant of sympathy in man² or love in parrots, may all be referred more or less to various branches of the vagus. The sudden death which sometimes follows the reception of very agitating news is probably due to arrest of the heart through the cardiac branches of the vagus, and the slower departure sometimes occasioned by grief may be due to the combined action of the cardiac and gastric branches. It is true that grief seldom kills, but the absence of fatal results is probably due to a considerable extent to the emotional stimuli being diverted into other channels. Where this is not the case

¹ Czermak, '*Jenasche Zeitschrift*,' bd. ii. 1865, p. 384.

² *Genesis* xliii. 30. *Isaiah* xvi. 11.

death may occur, as in a case given by Carpenter, where an orphan girl, who had tenderly nursed her only sister through a long illness, appeared altogether unaffected by her death, but was herself found dead a fortnight afterwards although free from any discoverable disease.¹ The general recognition even by uneducated persons of the effects of unexpressed emotion upon the viscera, and their instinctive attempts to direct it into other secretory or motor channels by the application of other stimuli, is so well given by Tennyson in 'The Princess,' that I shall quote the passage:—

Home they brought her warrior dead,
 She nor swooned nor uttered cry;
 All her maidens, watching, said,
 'She must weep, or she will die.'

Then they praised him, soft and low,
 Called him worthy to be loved,
 Truest friend and noblest foe;
 Yet she neither spoke nor moved.

Stole a maiden from her place,
 Lightly to the warrior stept,
 Took the face-cloth from the face;
 Yet she neither moved nor wept.

Rose a nurse of ninety years,
 Set his child upon her knee;
 Like summer tempest came her tears,
 'Sweet my child, I live for thee.'

Here the sight of her dead husband's body had excited the emotion of grief, and as this failed to find expression externally, the fears of the maidens were excited that it would cause death by its action on the viscera. They therefore tried to divert it into the channel of secretion and induce her to weep, but their efforts were vain until the old nurse succeeded in directing it in part into a motor channel, prospective if not immediate. The burst of tears was probably accompanied by embracing the child, though this is not recorded by the poet, and it certainly was accompanied by ideas of action on its behalf.

The importance of diverting nervous action into definite channels in the insane is so universally recognised by all who have the charge of lunatics that it hardly requires mention here. I may merely give as an instance the case of a youth who was subject to periodic outbreaks of violence. On being made to saw wood for a certain number of hours every day, except Sunday, the outbreaks were greatly diminished, and when Sunday also was included and an outlet given to his nervous energy on every day of the week they ceased altogether.

It would almost appear that all instances of inhibition in the higher centres might be explained by diversion of stimuli, without assuming the existence in them of an inhibitory apparatus such as we have in the lower ones. It is even possible that inhibition in the lower ones also, e.g., the action of the vagus on the heart may likewise be due to diversion of stimuli; but it is very hard to explain the facts with which we are at present acquainted on this supposition, while it is easy to do so on the hypothesis of inhibitory centres. If we thus come to the conclusion that such centres are found in the periphery we naturally extend it to the brain, and we do so all the more readily as we not only find no evidence against their existence there, but on the contrary several reasons for believing in them. It is quite true that it is much easier to divert nervous action into another channel than to arrest it altogether, and if the stimulus occasioning the action be strong it may be impossible to prevent it from producing some undesirable effect in any other way than by diverting it into a different channel.

In one of the instances I have already quoted as illustrative of inhibition we find diversion of the stimulus, for when a man is holding his hand or foot still while it is being tickled he frequently diverts the stimulus into a motor channel, and firmly contracts all the muscles of the arm or leg, so that although motionless it is perfectly rigid. But it is perfectly possible, more especially if the tickling be not great, to keep the arm or leg quite still and yet perfectly flaccid, like the legs of the frogs experimented on by Nothnagel, the motor action being arrested without any apparent diversion of the stimulus. The same seems to be the case

with emotious as with reflex aections. A violent emotion may only be restrained by opening a vent for it in some direetion where it is not likely to do harm, but a feebler one may be suppressed altogether. I am inelined to think that this is effected through the agency of iuhibitory eentres in much the same way as reflex aections are arrested. What becomes of the nervous aection excited in both the motor and iuhibitory eentres it is hard to say, unless, like motion ehecked by frietion, it is eouverted into heat. Certainly iuhibition does involve great nervous waste. It is felt to be difficult, its eontinued exertion quickly fatigues, and it is one of the first powers to fail when the nervous system is depressed by exhaustion, disease, or the aection of certain poisons.

It is always difficult completely to iuhibit the natural reaction to an external stimulus, by any exertion of the higher mental faulties, but it may be rendered eomparatively easy if another external stimulus, producing reaction of an opposite kind, acts on the organism at the same time as the first. Thus the injured infant, which if left to itself will eontinue to seream for a long time, may be quickly soothed by a few soft touches of the mother's hands or lips on the bruised part, with a few soft words of the mother's voice; and the angry feelings aroused in the man by an insult may be removed by penitent words and submissive gestures. The mother's soothing tones alone might calm the ehild, but experience shows that they are greatly aided by the local soothing impression on the injured part, and the soft touch of a woman's hand on her husband's brow seems not unfrequently to aid her words in elearing away painful impressions from his mind. The result in both of these eases depends partly on diversion of the stimulus into other ehanuels, but not entirely so, and I think we have here a stimulus of one kind eounteracting the effect of another of a different sort, and preventing its usual results, thus giving us an instance of iuhibition in the higher nervous eentres, just as irritation of the frog's toe in Goltz's experiment prevented it from uttering a croak of eontentment when its back was stroked.

The effect of a stimulus arising from the periphery of the body, and acting upon a nerve eentre, may be iuhibited

by a stimulus from the ideational centres. Thus the sobs and tears which naturally follow a fall or blow in a child a few years old will often be stopped by the idea that tears are unmanly. The stimulus which originated the idea of course came from either the tears themselves or the blow, and was thus of peripheral origin, but its action on the sobbing movements of respiration or the secretion of tears is only indirect. It sets the ideational centre in action, and from this the stimulus to inhibition proceeds. Similarly in the adult the stimulus which arouses passion may at the same time awake the idea that it is wrong to indulge it, and the stimulus then transmitted from the ideational centre may not only restrain all external manifestations, but even arrest the feeling itself. But the stimulus arising from the ideational centre probably does not act directly on the motor and emotional centres, but only through the medium of some inhibitory apparatus.

If this be so, the nature of the actions performed by any individual on the application of a stimulus to the periphery will not be regulated entirely by the proportion of the stimulus which goes to one centre or another, motor, emotional, sensational or ideational, nor yet by the size, power, and development of these parts of the nervous system respectively, but it will depend to a considerable extent on the development and power of the inhibitory apparatus through which one nervous centre may influence another.

Supposing that there are no inhibitory centres, the question whether upon a certain provocation a man will indulge in an outbreak of passion, or not, will depend upon the comparative power of his emotional centres, which impel him to do so, and his ideational ones, which tell him that such indulgence is wrong and thus tend to restrain him, as well as upon the degree to which each centre has been stimulated by the provocation. But if the ideational centre, instead of restraining the emotional one directly, only sets in action an inhibitory centre, which acts as the direct restrainer of the emotional one, it is evident that the action of the individual will depend on the power of this inhibitory apparatus. If it be weakened by any cause whatever the outbreak will

take place, notwithstanding all the desires and attempts of the person to prevent it, just as motions of the limbs often occur on tickling, however great be the wish to restrain them. Thus it is that the acts of the individual do not coincide with his intentions and wishes, and he is forced to cry out like St. Paul: 'The good that I would, I do not; but the evil that I would not, that I do. O, wretched man that I am, who shall deliver me from this body of death!' or 'dead body,' as it might be rendered.

So long as the ideational and other nervous centres worked harmoniously together he felt alive and well, but when the ideational centre became altered by the new teaching which he had listened to, and his intellectual views regarding conduct became different, while his emotional and other lower centres remained as before, he felt as if divided into two parts, one of which was either at active war with the other, or, like a corpse chained to it, acted as a heavy drag upon its movements. The ideational centres are much more readily modified by education than the emotional ones, so that a man's intellectual views are much more easily and quickly modified than his habits and character. Such a complaint as that of Paul must therefore have been extremely common among the Romans who had embraced the Christian religion; their ideas regarding their proper conduct had undergone an abrupt change, while the new training had not yet affected their emotional and reflex centres, which still remained Pagan, both by hereditary descent and early training. A similar condition is, I believe, frequently observed in the present day among savage tribes who have accepted Christianity. Their old habits being constantly at variance with their new views, they are frequently doing what they formerly considered right and proper, but now look upon as wrong, and are frequently troubled by the reproaches of conscience. On the other hand, when the same intellectual views have been held by many successive generations, the other centres come gradually to work in accordance with the ideational ones, and thus the individual does what he considers right almost without an effort, and like Paul before his conversion, or a

Chinaman who adheres to the religion of his ancestors, he rarely feels the stings of conscience.

But it is not among healthy men that we can observe in its clearest form the commission of acts not merely involuntarily, but in direct opposition to the will. For this we must look either among the insane or among those whose nervous system is deranged by poisons, alcoholic or otherwise. Such examples are afforded by lunatics who request to be restrained and prevented from committing homicide, or by a drunkard who is reported to have said that if a glass of spirits were placed at his right hand and hell-fire at his left he could not resist the temptation to take the glass, though instant perdition were the penalty of the act. In this unfortunate creature the greatest possible deterrent was supposed to be acting upon and through the ideational centres, but the inhibitory apparatus had been broken down by chronic alcoholism. It seems to be more or less paralysed also by excess of alcohol temporarily present in the blood, although not habitually indulged in, and thus a few glasses too much diminish the power of self-restraint in the individual, and not unfrequently lead to most disastrous consequences.

In small quantities, however, alcohol seems to follow the same rule as numerous other poisons, acting in exactly the opposite way to what it does in large doses, stimulating instead of paralysing. This opposite action of large and small doses is readily observed in the inhibitory cardiac ganglia, which are stimulated by small doses of nicotia and minute ones of atropia, and are completely paralysed by large quantities of the same drugs. Perhaps the same may be the case with *cannabis indica*, which calms excitement, but when taken in large doses by a healthy man seems to completely destroy all inhibitory power, the person having all sorts of ideas running through the brain without the power to direct them, and performing all sorts of antics without being able to stop, although perfectly aware of the foolish nature of his actions.

The inhibitory centres discovered by Setschenow, which regulate reflex action in the frog, are stimulated by several

drugs, especially quinine, digitalis, and morphia. The investigation of their action on inhibitory centres in the brain of warm-blooded animals is, of course, much more difficult than in frogs, and I am not aware that it has yet been attempted.

It is evident, however, that this department of pharmacology offers a most promising field for research, and it is not impossible that we may be able at some future period so to employ remedies as to alter for the better the moral character of individuals, and greatly diminish the prevalence of crime.

OBSERVATIONS ON THE
HISTOLOGY OF THE MORBID BRAIN.

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IN following up the observations I have before recorded in the previous volumes of these Reports, on the subject of the morbid histology of the brain, it will be my object in the present Paper to consider more fully the condition presented by the cortical substance in cases known as senile atrophy of the brain, as one step in the complete investigation of this affection.

Since my previous observations were published, several opportunities have been afforded me of continuing my researches in this direction, and with the increased experience thus derived, and knowledge from other sources acquired, I trust I may be able to lay down, with some show of accuracy, the histological condition we must expect to meet with in the grey substance of the convolutions. I have called this *one* step, and although an important one it is nothing more. It may, nay, more, it will be found that many of the appearances I shall describe as being present in senile atrophy may be demonstrated with equal certainty and distinctness in other forms of cerebral disease. If we expect that in the various morbid conditions of the brain we shall be able to point out corresponding variations in all the elements of which it is composed, we are wrong in theory and in fact. It may be remembered that in my previous Paper, after giving in detail the result of my examination of various

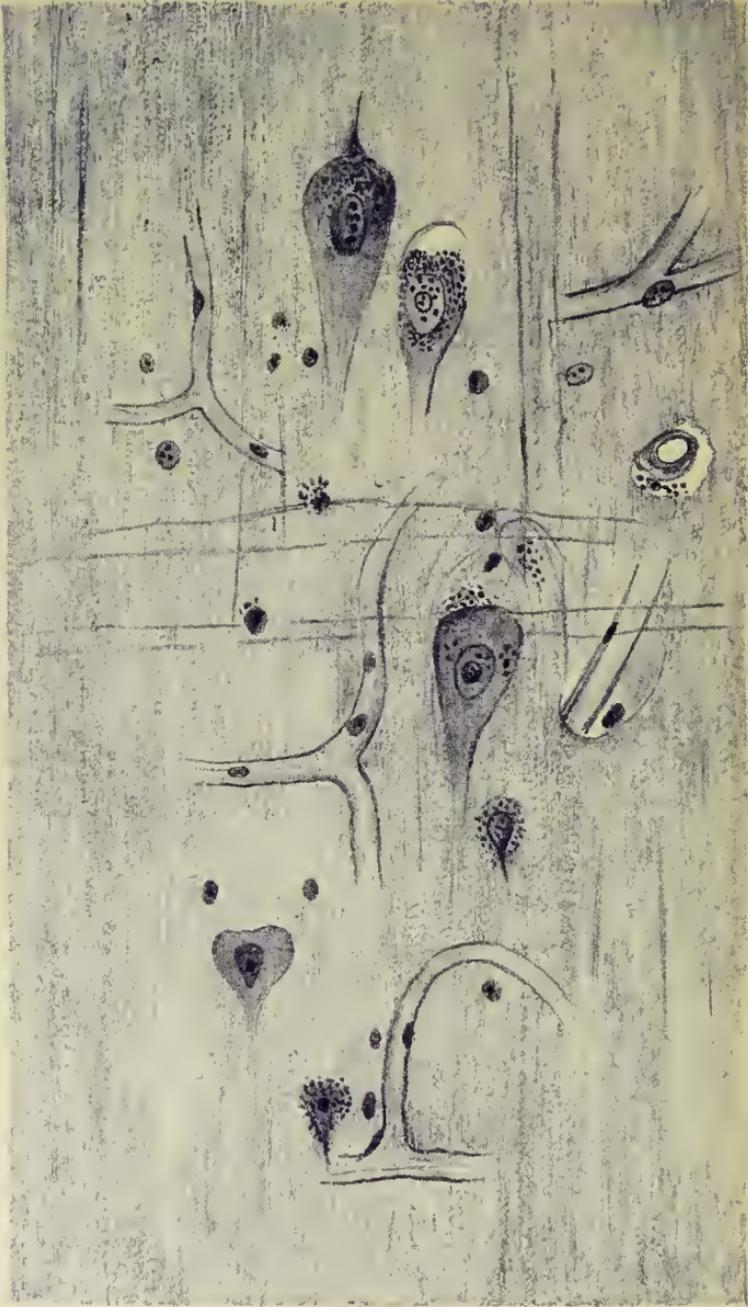
cases, I was driven to the conclusion that while in all cases I was able to discover decided pathological changes, yet that, so far as my experience had gone, in none could I put my finger on any one point and mark it out as being constant and distinctive of the affection, to the exclusion of all others; and to a great extent, as regards the cortical substance, further experience has only tended to confirm and strengthen this conclusion. So much for the fact; and with regard to its explanation, in many cases I think it is not far to seek. Thus, taking the nerve cells for example, we know that the great cause of their disease and destruction is a deficient or vitiated nutritive supply. Such deficiency may arise from various causes, but it is very conceivable that the result, as affecting the cells, may be the same. At first sight there is something very disappointing in all this; and yet, so far from being an element of doubt and discouragement, I believe it to be one of strength, and rightly considered will lead us on step by step in the road of progress. It will enable us, by a method of exclusion, to lay on one side those points which we observe to be not of essential importance, and thus more and more hem in and surround those that are. It is very necessary to bear this well in mind, and to hold fast to it with a firm grip, or we may lose heart and time in striving to find differences where none really exist, and give up in despair when we have only to work on in hope.

In this department of histology, therefore, the careful and candid inquiry into the condition present in any form of disease is of infinite service, not as helping forward the elucidation of that form only, but indirectly, also, as furthering the cause of all the others. It is with this object in view that I shall proceed to describe, so far as I may be able, the histological condition of the cortical substance in senile atrophy, and add my experience to that of those who have preceded me in the investigation of this affection. As a form of disease it is one in which we naturally expect to encounter the simplest forms of degenerative changes, but it is also one which, in its relations and bearings to others, holds a position of great interest and importance. But there is one step in this inquiry, let me not conceal it, the lack of

which, to those who apply themselves to this department of histology, offers a very serious impediment. I refer to the effect of age on the human brain, apart altogether from the question of insanity. Some facts we do know. We know that at birth, and before the dawn of those great faculties of the mind in which man stands supreme, the structure of the cortex of the brain is very rudimentary, more especially as regards the cell elements. In the brain of a child at about the eighth month, and which died almost immediately after birth, I have found the nerve cells throughout the entire depth of the grey matter to be almost uniform in size, circular in form, and, so far as I have been able to observe, entirely destitute of branches. I need hardly pause to remark how different is the condition of the healthy adult brain. Here the numerous rows of cells, their various forms and intricate connections, all tell of vigorous action. But as life goes on, and outward appearances are changed by the stamp of time, are changes also to be observed in the brain? If life is sufficiently prolonged, there comes a time when the man still hale, vigorous and strong it may be; capable also of intellectual effort little inferior to that of his younger years, will nevertheless at times be painfully conscious that the impressions which were formerly enduring are now more transient, and, to use his often repeated expression, he feels himself 'not the man he was.' Will changes be observed at such a stage in the histological condition of the elements of the brain? Everything, I think, points to the conclusion that such must in all probability be the case. I have shown that in infancy the state of the nerve cells is widely different from that in the young adult. I shall show later, that equally far removed from the latter, are the appearances observed in senile atrophy; and just, I conceive, as the distance in the former case is bridged over by stages corresponding with the growth of the intellectual faculties, so in the latter case changes occur—changes, it may be, so slight and gradual as to be hardly traceable, changes still compatible with intellectual vigour, but still change, in one or other of the elements of the brain. Of such stages, however, we are, so far as I am aware, still in want of actual

demonstration. I can but indicate as it were the extremities of the chain, and the central portion; the intermediate links have still to be supplied. But he who will now take up the subject, bringing to bear on it the patient and protracted investigation which alone can ensure success, will supply that which I believe would prove of the utmost service, and is urgently required by those who investigate the histology of the brain in the insane, to guard against fallacy or positive retrogression; for surely it is retrogression, if at any time we ascribe to the influence of special disease changes which are but the natural result of time, and which, it may be, are consistent with healthy processes.

Cells.—In proceeding to describe the condition of the elements which enter into the construction of the cortical substance, I take up first, according to my usual custom, the consideration of the nerve cells. And here I would first observe that, in my opinion, changes in these bodies can always be demonstrated throughout the entire depth of the grey matter. I am anxious to lay this down clearly and distinctly, for it is a point of the correctness of which I am fully satisfied, while it appears to be not always recognised. It is quite possible and even probable that, at the outset of atrophic change, the process may not be evenly distributed, and may tend to attack one variety of cells, leaving the others comparatively unaffected; but this assuredly is not long the case. From a careful comparison of the cells, specially directed to ascertain the relative extent to which they suffer, I am led to think that in the majority of cases of senile atrophy it will be found that the large pyramidal cells which form so conspicuous a band about midway in the depth of the cortical substance are those in which degenerative changes are most constantly and distinctly marked. I have never known an exception to this rule. With regard to the smaller varieties, however, I have more difficulty; partly because their greater minuteness renders their examination more difficult, but chiefly because, as I shall endeavour to show later, the degenerative process is often dissimilar, and when this is the case it is dangerous to venture on a strict comparison. But in addition to those layers which are made up



SENILE ATROPHY OF THE BRAIN
(PARIETAL LOBE) $\times 490$



Berjeau, Lith.

Banks & Co. Edun^r

HEALTHY BRAIN
(PARIETAL LOBE) X 230

almost entirely of caudate cells, there are others in which small round or oval cells predominate. I believe it will be found that in them, as compared with those previously referred to, structural changes are but slightly marked. But that they are affected, and that in no slight degree, is evident from the fact, that, in the process of atrophy, many of their number are entirely lost, leaving only faint traces of what were formerly broad and distinct bands of cells.

With regard to the relative degree to which the various portions of the brain are affected in senile atrophy, the broad fact is generally acknowledged that the frontal and parietal regions—regions in which the large nerve cells are in greatest number—are the chief seats of atrophic change, while the occipital lobes and the convolutions of the inferior surface of the brain are to a less degree affected. To a certain extent this is in accordance with the ordinary *post-mortem* appearances. But the fact must never be lost sight of, that in senile atrophy at least, whether to the unaided sight external wasting is visible or not, the microscope will never fail to render evident, changes incompatible with health. It is very desirable that we should go further in this direction, and be able to define, with greater nicety than has been hitherto done, the favourite seats of histological change. At present it is not in my power to do this; the subject is one of great interest, and, I may add, of difficulty also. With regard, however, to the occipital lobes, I would remark that at their extremities, in my opinion, the cells are less affected by the degenerative process than at any other part of the grey matter, although, as I have before stated, certain changes are never absent. The band of small round cells which in health forms so distinct a layer, and which I have stated is, in the frontal and parietal regions, very ill-defined when the atrophic process is at all advanced, retains to a great extent its characters in the occipital regions; and in the nerve cells of the other layers also, those degenerative changes which I shall describe in connection with the large pyramidal cells of the anterior hemispheres, are here less decided. It is quite a mistake to consider, as I fear is too often done, that the only difference between the cells of the

anterior and posterior extremities of the brain consists in the relative minuteness of those found in the latter situation. Were it so, then all that would be requisite to render the appearances identical would consist in the use of a higher power for the smaller cells. This we know is not the case. The cells of the occipital lobe have a special arrangement; they have also structural peculiarities apart altogether from that of size, and hence it is, that whether a high or a low power be employed, the cells at the extremity of this region ought never to escape recognition. But because those bodies which are most susceptible of pathological change are here almost absent, it follows that the cells of the occipital regions, while always affected, appear to be so to a less degree than those in other parts. Hence too, to some extent, is due the fact which cannot but be of universal acceptance, that in all forms of cerebral atrophy the occipital lobe is that which in the vast majority of cases is most free from external wasting.

With regard next to the morbid appearances observed in the form and structure of the cells, let me say, that in almost every case of senile atrophy I have been able to make out similar changes — not similar appearances always, but identical conditions, modified by the existing stage of the degenerative process. I have said *almost* every case, for I shall have occasion later to consider one or two cases in which I could hardly satisfy myself of the identity of the process; and although I think it may have been the same, I nevertheless consider it safer to give any such case the importance to which it is entitled.

I believe the great change, and one which is invariably present to a greater or less extent, is a granular condition of the large nerve cells. I think, also, that careful examination will reveal the fact, that in the great majority of cases the varying appearances which undoubtedly occur between individual cells, all of which are morbid, are due to the various stages of this degeneration.

The first stage in the process, as observed in the large pyramidal cells, is that in which these bodies lose their sharply defined and more or less triangular appearance, so distinctive of the healthy condition, and acquire a swollen,

or as it may be described, an inflated appearance. The most prominent continuation of the cell, viz., that which runs directly outwards towards the periphery of the grey matter, is at this stage always to be observed, but those coming off in other directions are usually destroyed, and hence the rounded off appearance produced. In one or two instances I have observed the branches to survive with their usual distinctness long after this stage in the degenerative process, but this I am sure is quite exceptional. The nucleus participates in these changes, being as it were swollen out and larger than normal, while in form it has lost the pyramidal appearance which it usually presents in health, and has become more or less rounded or oval. It is worthy of remark that at this, and indeed at much later stages, the nucleoli are invariably seen with great distinctness. In part, I think, this may be explained by the fact that the natural pigment of the cell is absent or nearly so; and, moreover, by the circumstance, which in itself is of no slight significance, that while under this morbid state the cell and nucleus are as a rule but very slightly affected by the staining solution employed, the nucleolus absorbs it to a greater extent. But beyond this, I think there must be an actual structural change in these bodies, the forerunner of that which at a later stage becomes more evident. Such then I believe to be the first step in the degenerative process: a state in which the cell is somewhat increased in size, altered in form, and usually deficient in branches, and in which also there is observed a morbid condition of the nucleus and nucleolus.

In the next stage a deposit of granules is observed, either on the exterior of the cell and pressing on its outer surface, or in its interior, in the latter case giving to the structure an appearance more and more yellow and opaque, according to the density of the granular deposit; or, lastly, both these conditions may and very often do exist together. When present only in the interior of the cell the appearance presented is often very striking. This body may be, as it were, completely sub-divided, one portion retaining the usual appearances, the other occupied by a dense mass of granules. Sometimes again these granules are collected into a mass of

varying shape, pushing aside the nucleus and producing a bulging and deformity of the cell contour. Lastly, before even any breaking down can be observed in the cell wall, its whole cavity may be completely filled by yellow granules, in some cases producing an effect which at first sight might almost be mistaken for the action of some yellow staining fluid. From this stage the process is one of gradual destruction. As the granular degeneration progresses, the cell, which in the previous stages had maintained or even exceeded its natural size, now breaks down and shrinks, leaving the nucleus with, it may be, its contained nucleolus surrounded only by a mass of granules, and forming a gap in the cerebral tissue which had formerly been fully occupied by the swollen cell. In advanced cases this appearance of numerous holes in the grey matter, each containing a nucleus surrounded by a mass of granules, is very striking. It is this stage which agrees so exactly with the description given by Dr. Lockhart Clarke as occurring in a case of general paralysis, in which the cell loses its sharp contour and 'looks like an irregular heap of particles ready to fall asunder.'¹ It cannot be too strongly insisted on that such a condition is very far from being peculiar to general paralysis. With regard to the gaps or holes I have mentioned, the objection might be raised by some, that they could be caused by a shrinking of the cellular elements consequent on the hardening process employed. Such an objection is easily disposed of. They are not found in the healthy organ. It is undoubtedly true that, in health, an examination of almost any prepared section will reveal the presence of holes in the grey matter, which are often in close relation with the cells; but, as far as my experience goes, never surrounding them, as is so distinctly the case under morbid conditions.

At a still later stage the granules almost or entirely disappear from round the nucleus, leaving this body free, and in many cases hardly to be distinguished from some other nucleus-like bodies which are found abundantly in the grey substance. It will thus be observed, and the fact is one which has been laid down very clearly by others, that

¹ 'Lancet,' Sept. 1, 1866.

the nucleus is the most persistent part of the cell, surviving long after the latter has wasted and disappeared.¹ But although this is the case, it must not be imagined that the nucleus remains unaffected; and yet, so far as I can judge, the point is one which has been almost entirely overlooked by most observers. I have before alluded to the fact that it becomes, in the first place, swollen out and altered in form; but further, in the later stages, other changes are observed. Dark granules make their appearance, sometimes scattered in an irregular manner, at others aggregated round the nucleolus, increasing the prominence and distinctness of that body. Thus far I have not been able to observe the actual breaking down of the nucleus. There can be little doubt, however, that such a process actually occurs, for in some instances no trace of its presence can be detected in the mass of granules resulting from the degeneration of the cell.

With regard to the nature of these granules there has been some difference of opinion. By some, including myself, they have been termed fatty. It would seem however that the contrary opinion held by Howden, Batty Tuke, and others, supported as it is by the indecisive results of the ordinary chemical tests for fat, is gaining ground. I confess, for my own part, that I am not fully satisfied as to which is the correct view; but in the meantime, until the matter is definitely settled, it is as well not to use the term fatty. I trust that the day is not far distant when chemical analysis will come to our assistance, and throw light on many points which are now uncertain and obscure.

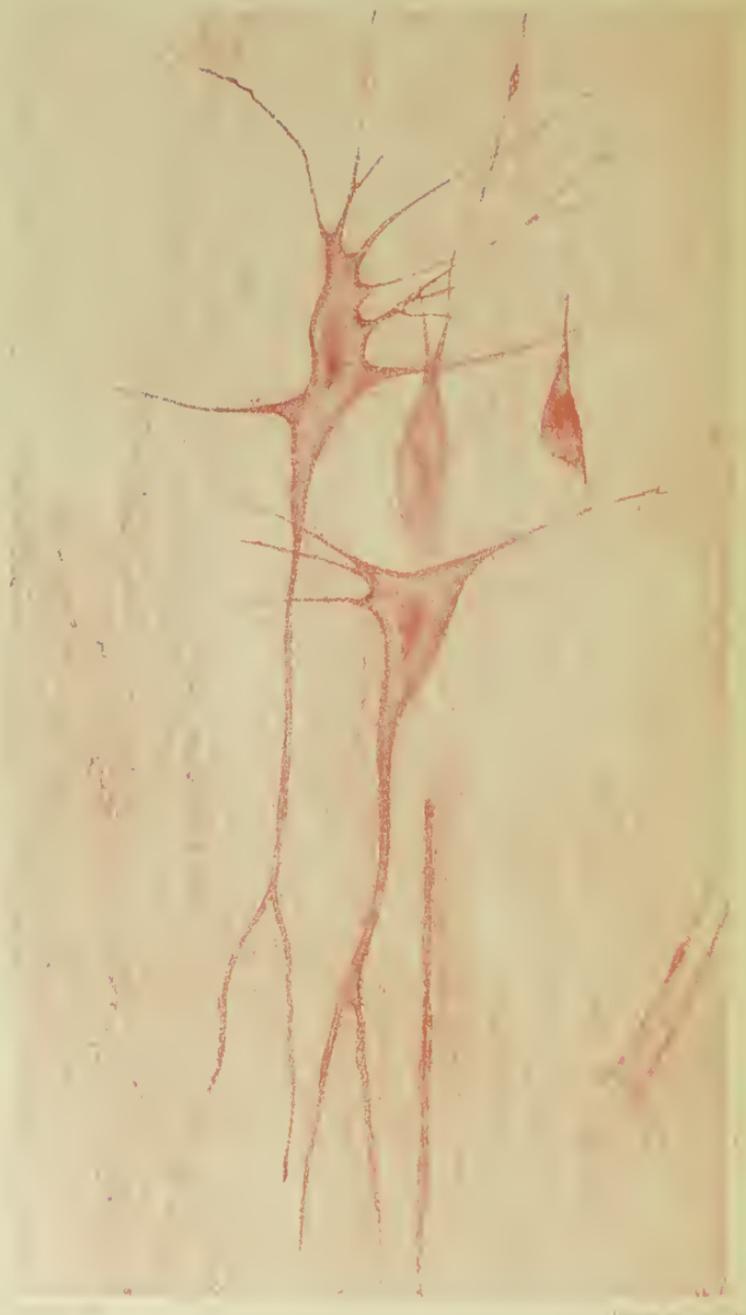
Such then are the changes in the larger nerve cells which I am satisfied will usually be traced, and a consideration of them will make clear the fact formerly stated, that according to the stage of cell degeneration which predominates, so we may have appearances differing more or less distinctly, but none the less due to the same pathological process.

To a certain extent the appearances I have endeavoured to describe will be observed in the accompanying plates, which represent corresponding spots in the grey matter of

¹ 'Psychological Medicine,' by Drs. Bucknill and Tuke, 3rd ed. pp. 630, 631.

the parietal lobe in health (at the age of 21 years) and in senile atrophy, the method of preparation and the magnifying power employed being in both cases identical (about 500 diams.). I have selected the largest nerve cells for representation, for in them, as I have before observed, the diseased process is most distinct and can be best traced. Let me say that it has been my endeavour to reproduce as accurately as I possibly could, the appearances presented, adding nothing, modifying nothing; and as, so far as they go, faithful representations, I trust they may be found not without interest to those who do and those who do not work with their microscopes. A comparison of these two drawings will in the first place at once reveal the fact, which now-a-days, I am glad to say, needs no words of mine to uphold it, that between the cells of the brain in health and under morbid conditions there may be found structural differences as certain and decided as is the very existence of the cells themselves, and will explain better than can be done by mere words some of the changes I have alluded to. I say some changes, for it is manifestly impossible that under a high power, and when so few objects are together in the field, all or even many of the conditions one would wish can be brought together; still, to a certain extent, the spot selected is not unfortunate, and several peculiarities I have indicated are sufficiently evident in the morbid cells.

I have before remarked that in the small nerve cells, and more especially in those which form the first or most external layer of these bodies, I failed in most instances to trace, in the degenerative process, the various stages above described; and indeed in them I am compelled to admit that the change seems to be rather one of atrophy and shrivelling than degeneration properly so called. In health they are very distinctly pyriform and have nuclei large in proportion to the surrounding cell structure, and it is, I think, by a shrinking and wasting of the latter, that the changes in appearance, of which there can be no doubt, are brought about. They are, in senile atrophy, decidedly smaller than in health and less pyriform, while the branch or branches in most cases are imperceptible. Now, as we approach the extremities of the occi-



HYPERTROPHIED NERVE CELLS 490

pital lobes, it is found that the cells presenting characters identical with these small pyriform bodies become more and more numerous, and hence I think it is that at the extremity of the occipital lobe the prominent change is one of shrinking and atrophy, degeneration proper being comparatively little observed.

It cannot be denied, however, that cases do occur, although I am sure very exceptionally, in which a condition of simple atrophy of the cells is not confined to certain parts, but is the prominent condition observed. In these cases the cells are greatly decreased in size and in number, while the rows which they form are proportionally deficient in depth, but why in these special instances there should be an absence of granular degeneration is not, as far as I know, satisfactorily shown.

Hypertrophy of the nerve cells was, I believe, first described in connection with senile atrophy by Drs. Rutherford and Tuke, and at a somewhat later date was detected by myself in general paralysis of the insane. I had never observed the condition in senile atrophy, and was, I confess, inclined to think that some other morbid change was intended. By the kindness of Dr. J. Batty Tuke, however, I have had an opportunity of examining some of his fine preparations, and a single glance sufficed to show me that we had discovered a similar condition. In truth, once observed, this appearance of the nerve cells can never escape immediate recognition. Standing out, as I have elsewhere expressed it, like giants among the other cells, their unrivalled size and the number and distinctness of their branches, stamp them as something totally different from every other condition. In the accompanying lithograph I have represented a group of three of these bodies as they occurred in the case of general paralysis, and their general characters are sufficiently well shown. In my experience they are found at the site of the large pyramidal cells, and seem to be formed by a peculiar transformation of these bodies; for although enlarged they frequently present in their interior large masses of granules, showing very clearly degeneration of structure. In form also they show considerable variation. It has so

happened that those I have depicted, beyond their size, show nothing very remarkable in this respect, but in some instances the form assumed is very peculiar. Sometimes they are of immense length, but narrow; or one extremity may be swollen and almost bulbous in form. In other cases, again, they are more or less stunted and globular. With regard to the number of their branches—a number in many cases far exceeding that observed under normal conditions—I would observe, that the fact would seem to be of importance with reference to the histological structure of the healthy cells. I can hardly conceive that even if we are right in considering that a condition of hypertrophy has taken place, any branch would be present which in health had not its representative. Hence I am led to think that as in the inflamed conjunctiva numerous vessels come into view which were formerly, owing to their minuteness, unobserved, so here there may be in health numerous branches of the pyramidal cells which, by reason of their extreme minuteness, pass unnoticed, but which under certain circumstances, as in that at present under consideration, may enlarge and form a very prominent feature.

I do not think I err in regarding the above facts as of real importance and value in the morbid histology of the brain. Whatever be the actual transformation undergone by these large cells, we see that they may occur both in senile atrophy and general paralysis, while in both also they are of quite exceptional occurrence. In one case only of general paralysis have I ever been able to observe this condition.

With regard to the arrangement and relation of the various rows of cells in senile atrophy, I have before pointed out that although a practised eye will not fail to detect indications of these rows, yet they exhibit a state of confusion which is not observed in health, and which furnishes conclusive evidence that some of their number have disappeared.¹ The well-defined rows, which form so beautiful a feature in health, are seen no longer, and in propor-

¹ 'West Riding Asylum Reports,' vol. ii. pp. 46, 47.

tion to the advancement of the atrophic process do they become less and less distinct.

In addition to the granules found in immediate relation with the cells, I would remark that, more especially in advanced cases, deposits of a similar nature are to be seen scattered abundantly throughout the grey substance. It is still a matter of uncertainty in my mind whether such deposits are in all cases derived from the destructive process in the cells, or whether they may have some other origin.

Vessels.—Under this head my observations need be only very brief, for the state of the vessels in almost all forms of cerebral disease has always attracted much attention, and as they admit of easier study than most of the other elements, accurate descriptions are not wanting. To make this part of my subject, however, complete, it is necessary I should refer to these descriptions, while at the same time drawing attention to a few points which may perhaps repay special notice.

In the first place I would remark that in senile atrophy of the brain there will, in my opinion, be usually found a dilated condition of both arterioles and capillaries, similar to that which I have before described in chronic brain-wasting. But, as regards the capillaries, it is the only change which in the majority of cases will be detected. In health they are so fine as almost to escape notice with a low power, but in senile atrophy, owing to their increased size, they are more conspicuous. I have never been able to satisfy myself of any shrinking or notable deviation from the course they pursue in health. I would observe here that great caution must be exercised in judging of any abnormality in this respect, for it cannot be doubted that the hardening process and subsequent preparation may produce variations which do not properly exist, and this I think the more likely to occur when the cerebral substance is soft and loose. The most beautiful examples of tortuous vessels I have ever seen occur in my prepared sections of the brain of an infant, where it is in the highest degree improbable that any such condition was present previous to the hardening process. With regard to the small vessels, in addition to their increased size, the

most prominent change consists in the deposit of minute granules and crystals of hæmatin on the vascular walls. The former are of a yellow or brownish colour, and highly refractive. They are sometimes found scattered in an irregular manner on the wall of the vessels, but very commonly are collected into masses forming small elevations under the sheath of the vessel. Hæmatin crystals do not tend to collect in such a manner, but both kinds of deposit show a decided preference to be deposited at the angle formed by the bifurcation of the vessels. The appearances thus presented are well described in a very instructive paper by Dr. Batty Tuke in the 'Medico-Chirurgical Review' for April, 1873, and in which most of the morbid appearances of the vessels at present known are discussed. But although such deposits are always very distinct, it will be found that the small vessels do not present the coarseness which is so marked under some other conditions, and more especially in general paralysis. One of the most constant changes in the latter affection consists in the presence of large numbers of nuclei in the wall of the vessel; but in senile atrophy such a condition is very uncommon, and, so far as my experience goes, is never found to the same extent. With respect to the course pursued by the arterioles, although at times slight eccentricities may be noticed, I have found nothing distinctive or worthy of special description. The perivascular canals, or at any rate the channels for the vessels, are in this, as in so many other forms of chronic disease of the brain, considerably enlarged, and the cerebral substance immediately bounding them coarse and indurated. In senile atrophy I have not observed the apparent occlusion or even destruction of the vessels which I have frequently noticed in general paralysis, and I believe it will be found that the state of degeneration before described in the cells is altogether of primary importance. It must never be forgotten that, with the advance of years, many changes occur in the vessels of the brain altogether irrespective of mental symptoms, and the remarks which I formerly made with reference to the effects of age on the condition of the cells are equally applicable to the vessels. We need more thorough know-

ledge of *all* the possible changes which may be found in the brain of the sane, and not till such knowledge is acquired can we venture to estimate, with any show of accuracy, the importance of any condition of the vessels in relation to the mental symptoms of the insane.

Fibres.—It will be remembered that, in my description of the degenerative process observed in the cells, I alluded to the fact that one of the earliest and most common changes was in many cases a loss of their branches, which became broken off and destroyed. There can be no doubt that, corresponding with this, the fibres throughout the course of the grey matter are also in many cases greatly altered or even broken up, and their course very twisted and irregular. It has been a matter of common observation that in this and in other forms of long-continued cerebral disease they are very decidedly coarser than normal. The change is a very interesting one, but so far, I am not aware that it has been satisfactorily explained.

Neuroglia.—Under this head, which includes the delicate, almost homogeneous matrix supporting the nerve elements, and the neuroglia corpuscles, I shall conclude the present division of my subject. The most decided change which this substance undergoes is one of wasting and atrophy. It loses the extremely fine, almost retiform appearance of health, and breaking down in an irregular manner, presents in extreme cases patches within the grey substance in which nothing but a mass of molecules and nuclei can be detected. With regard to the nuclei, they are subject to some variation, both as regards number and appearance. In some cases they retain apparently their usual characters, but are somewhat increased in number, and tend to collect here and there in small groups. As a rule, however, they do not, in the grey matter, present anything like the proliferation observed in other forms of brain disease. In advanced cases their natural full round appearance is lost, and they become shrivelled and deformed. It is by the atrophy of the nerve cells and the breaking down and shrinking of the neuroglia elements, proceeding *pari passu*, that the wasting and dimi-

nution in thickness of the cortex, which are invariable accompaniments of senile atrophy, are brought about.

Such then are the most important changes which I believe an examination of the grey matter in these cases will disclose, and it now remains for me to state somewhat more concisely such conclusions as I feel justified in drawing. But before doing so I would again observe, that my remarks must be held applicable to the grey cortex only, to the exclusion of all other points. There remains for examination every other part of the nervous system, and when this has been done with as much care as has been bestowed on the grey substance of the convolutions, then, and not till then, shall we obtain a proper insight into the pathology of the affection, or be able with advantage to compare it with others. Many points there must be in common; but differences, and essential differences, there must also be; and these cannot but soon be grappled with and brought to light. But with the thorough scrutiny of the nervous system in the insane there must be combined more accurate knowledge, derived from wider study of the various structural modifications that may exist, compatible with healthy mental processes.

CONCLUSIONS.

A. *Cells.*

1. That in senile atrophy of the brain the nerve cells throughout the entire depth of the cortical layer, and in all parts, are morbidly affected, although to a variable extent and in a different manner.

2. That in the large nerve cells the morbid process in the great majority of cases is one of granular degeneration.

3. That in the smaller nerve cells generally, and occasionally also, but rarely, in the large, the process is one of simple atrophy, without granular degeneration, properly so called.

4. That the nuclei of the cells invariably participate in the diseased condition, and become the seats of granular deposits which lead ultimately to their destruction.

5. That at an early period the branches of the large cells are usually atrophied and destroyed to a greater or less extent, but exceptionally are retained up to a late period in the degenerative process.

6. That the condition of so-called hypertrophy of the cells (Rutherford, Tuke) depends on a peculiar transformation of some of the large pyramidal bodies, and is not confined to senile atrophy, being also observed in general paralysis, but in both is of exceptional occurrence.

B. *Vessels.*

1. That a condition of dilatation, both in the small vessels and in the capillaries, is that most commonly observed.

2. That corresponding with this there is enlargement of the vascular canals with induration of the surrounding cerebral substance.

3. That as a rule no great proliferation of the nuclei of the vessels is observed, the morbid deposits consisting of yellowish granules and hæmatin crystals.

4. That there is no remarkable change in their course and direction.

C. *Fibres.*

1. That the fibres generally are abnormally coarse and tortuous, and in some instances seem to be broken down at various parts.

D. *Neuroglia.*

1. That the most prominent change in the neuroglia is one of atrophy and degeneration, the substance, at first loose and imperfect, breaking down ultimately into molecular *débris*.

2. That the neuroglia corpuseles are somewhat increased in number, and while at first presenting their usual characters, become eventually shrivelled and atrophied.

ON THE
HOURLY DISTRIBUTION OF MORTALITY
IN RELATION TO RECURRENT CHANGES IN
THE ACTIVITY OF VITAL FUNCTIONS.

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SEVERAL interesting observations have recently been made regarding the existence of maximum hours of mortality and the allied subject of recurrent variations in the activity of physiological function. During the present year an important contribution to the literature of the subject has been made by Dr. James Finlayson, of Glasgow, who in a couple of papers published, the one in the 'Transactions of the Philosophical Society of Glasgow' for 1873-4, and the other in the 'Glasgow Medical Journal' for April, 1874, has supplemented and summarised Schneider's searching examination into the statistics bearing on the subject, tabulated the results of Mr. West Watson, a Glasgow predecessor in the study of vital problems, and collected the scattered statistics contained in the important contributions to the literature of this interesting inquiry. In an independent summary, showing the hours at which the greatest number of deaths occurs in several Glasgow institutions, Dr. Finlayson determines the comparative cumulative mortality during successive hours of the same day, and during groups of hours

collocated on account of some marked contrasts in meteorological conditions and physiological activity. As far as regards deaths from chronic diseases, the results obtained by Dr. Finlayson are confirmatory of those of Caspar and the summarised statistics of Schneider. They show a notably increased mortality during *ante-meridian* hours as compared with those of the afternoon and evening, and especially a determinate maximum between 4 A.M. and noon. With regard to acute diseases, the figures of Dr. Finlayson show that in them the morning rise is nearly if not altogether equalled by a second rise in the extent of hourly mortality occurring in the afternoon. He accounts for this divergence from the general results by referring to the influences exerted by the *post-meridian* rise in temperature which characterises that group of diseases as a means of determining in them the modification of their hours of maximum mortality.

There can be little doubt regarding the existence of a diurnally recurrent period which in general diseases can be regarded as the maximum period of death, and the argument adduced by Dr. Finlayson and other arguments of a similar nature are quite sufficient to prove that the exception presented by acute diseases is one which only throws more light upon the causes producing an *ante-meridian* maximum of mortality in chronic diseases, inasmuch as it is dependent upon the fact that acute diseases have an independent periodicity, which enters into and modifies the ultimate determination of the period when they are most likely to end in death. The results obtained by all who have examined widely into the hours of the day at which chronic diseases reach a mortal termination are identical in one important point. They show that the hours of greatest mortality are those which follow immediately upon the period when functional activity is at its lowest, when meteorological conditions are most unfavourable for the continuance of organic life, and when the stillness of night, shorn of everything which by sensorial impressions tends to maintain functional activity, has favoured an inevitable tendency to a nocturnal depression in the performance of vital acts. Between the hours of 4 and 9 A.M. the largest number of

deaths resulting from chronic diseases occurs, and a comparison of the statistics which bear upon the subject shows that in these diseases there is an especial tendency to the occurrence of death between the more limited hours of 8 and 10 A.M. In fact, at this time the mortality from chronic diseases surpasses that of the early morning hours. This fact gains significance from its evident connection with physiological laws. This is the period of reinvigorated function, when one might say, with a mixture of poetry and physiology—

“—How mine eyes throw gazes to the east
My *heart* doth charge the watch; the morning rise
Doth cite each moving sense from idle rest—”

the period when the invigorated heart, not only emotionally as expressed by Shakespeare, but in its integrity of function, anticipates by a greater physiological activity the physical exertion which an opening day must necessitate. But when by such a deviation from normal physiological conditions as occurs towards the termination of a prolonged malady, the heart passes nocturnally through the period at which the weakness of its action is most marked, it exhibits a gradually increasing tendency to fail, either when its contractility is at its lowest, or when physiological alternation and stimulating external conditions require a renewal of its full activity. Under such circumstances, instead of anticipating ‘the watch,’ it completely ceases in the extremity of its weakness to perform its impaired function.

The intimate relation which appears to exist between the maximum hours of mortality on the one hand, and periodical changes in physical, meteorological, and social conditions on the other, opens up an interesting study, and promises to provide not only an approximate explanation of variations in relative hourly mortality, but in addition to illustrate some points in human physiology. It affords an opportunity of inquiring into the recurrence of normal depressions of physiological activity simultaneously with certain meteorological changes, leads to the question how far the nocturnal depression of the heart’s action—which must be regarded as the most probable agent in the determination of

increased relative mortality—is dependent on or independent of the nervous system, and besides justifying the deduction of several important practical inferences, suggests the existence of a law of alternation, manifested not only by external creation, but also in the intimate phenomena of physiological action.

Regarded as part of this general question, a comparison of the mortality statistics of a large lunatic asylum with those of Schneider, Mr. Watson, and Dr. Finlayson appeared to promise interesting and instructive results. It could not fail to elucidate the general question by increasing the recorded volume of statistics, and might possibly lead to the determination of some important differences in the hours of maximum mortality traceable to the influence of central nervous derangement. On these grounds, I felt called upon to accept Dr. Crichton Browne's suggestion to examine the death returns of this asylum for the last ten years, and determine how the hourly distribution of deaths compares with published accounts of the hours of maximum mortality. After a careful examination of returns which, by an exactitude extending to the smaller fractions of an hour, cannot be surpassed in the element of reliability, it appears that the statements of Watson, Schneider, and Finlayson are confirmed by them in a manner which throws the similarity of results beyond the suspicion of simple coincidence. When a striking identity of results takes the place of the variation which, by virtue of the difference in the class of diseases supplying the materials for observation, might reasonably have been anticipated, fresh authority is added to numerical conclusions based on extensive statistics. It must be observed, however, in fairness to those who have entered into the investigation of the comparative hourly statistics of mortality, that mere modification of the maximum from one hour to another, either immediately before or after it, makes only an apparent modification of the identity of results. Nothing short of a survey of all hours of death could determine absolutely the exact hour at which the fatal issue has most frequently occurred, and even such a summary would be imperfect in proportion to its deficiency in materials and its

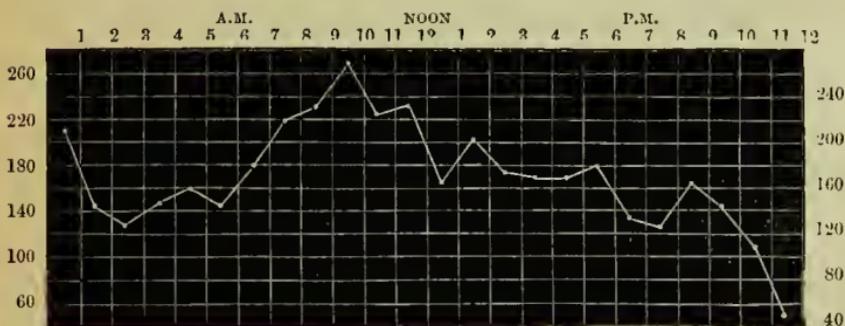
liability to alteration by fresh numerical accessions. It is sufficient for scientific purposes to find that all observations on the subject point to the existence of hours, such as 4 or 9 A.M., around which periods of highly increased mortality are constantly and closely grouped. Such an approximation, indeed, is all that can be expected from the inquiry. In individual cases there are exceptional circumstances, dependent on both inherent and extraneous conditions, which in chronic diseases are capable of determining the occurrence of a patient's death either shortly before or shortly after a period which general considerations might suggest as the most probable. Slight superiority in the remains of bodily vigour, judicious administration of stimulants, and the absence of the depressing emotions of bystanders, might be in themselves sufficient to lead to the temporary prolongation of a flickering life beyond the hours which physiology and statistics of indicate as the most fatal. On the other hand, opposite conditions to these might induce a fatal issue shortly in anticipation of such hours; but such variations do not alter the fact that there are periods of diurnal recurrence over which death seems to mount a special guard—which physical and physiological conditions combine to stamp with a special mark of fatality. And yet, for the sake of utilising the information derived from the investigations of the statistics of relative hourly mortality, it is necessary to determine as narrowly as possible the limits of maximum periods. It is physiologically interesting, but of limited practical importance, to find that the hours between 4 and 10 A.M. are the hours of greatest mortality; but that period, embracing as it does almost one-third of the entire day, is too prolonged to admit of the opinion that special remedial measures could be continuously employed to counteract the tendency to death during a period relatively too protracted to merit the name of critical. But when several independent inquiries, into sufficiently extensive data, lead to the conclusion that in chronic diseases the decided maximum of mortality daily occurs between the hours of 8 and 10 A.M., an opportunity is afforded for the practice of prophylactic and sustaining measures, and for the determination of prognosis as influenced

by the bodily state before and after those hours. When, as Dr. Finlayson has shown, there is a tendency for the mortality in acute diseases to be characterised by a double elevation, of which the first point stands at 3 to 6 A.M. and the second from noon to 3 P.M., it becomes possible to adopt practical measures based upon the knowledge of such periods, narrowed as they will doubtless be by the acquisition of further statistics.

Before entering further into the consideration of periodic variations in mortality, it will be well to show the similarity between Dr. Finlayson's results in the case of chronic diseases and the results obtained by an examination of 1,680 death records in this asylum. The following table, showing the maximum hours of mortality in chronic diseases as investigated by Dr. Finlayson, appears in his article in the 'Glasgow Medical Journal.'¹

TABLE NO. 1.

Hourly distribution of Mortality in Chronic Diseases, tabulated by Dr. Finlayson, Glasgow.



The next table, showing the relative hourly mortality in this asylum for a period of ten years, is constructed in a similar manner to the last, in order to bring out the striking points of resemblance between the progress of their numerical curves.

It cannot fail to strike anyone comparing these two tables how pronounced is the rise which in Dr. Finlayson's reaches its limit at 9 to 10 A.M., and in that representing the

¹ 'On the Hours of Maximum Mortality in Acute and Chronic Diseases,' by James Finlayson, M.D. 'Glasgow Medical Journal,' April, 1874.

West Riding Asylum records culminates at 8 to 9 A.M. In another table constructed by Dr. Finlayson, showing the hours

TABLE NO. 2.

Hourly distribution of 1,680 Deaths in West Riding Asylum.

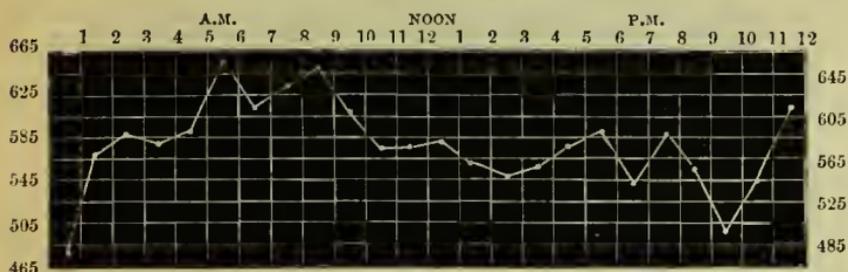


of greatest mortality in 13,000 cases examined by Mr. West Watson, the same emphatic elevation characterises the hours between 8 and 10 A.M. but it is slightly surpassed by a maximum occurring at from 5 to 6 A.M. This state of matters, however, is evidently dependent upon the fact that the table is one showing the hours of death from *all* causes, and the comparative statistics of acute and chronic diseases in relation to hours of death show that had the separation of acute from chronic diseases been effected, the probability is that in this instance also the maximum mortality in chronic diseases would have been fixed between 8 and 9 A.M. It appears to me, however, that, with regard to chronic diseases, the observation of one unvarying and limited diurnal period, characterised by a marked increase of mortality, is not the only precise conclusion which can be gathered from the statistical investigation of this question. The whole of the tables show, as Dr. Finlayson has pointed out with regard to his own and Mr. Watson's results, a gradual and persistent rise, culminating in a definite morning climax. Though in no instance is this rise undisturbed by hourly variations, yet it is very well worthy of notice that not only is the morning tendency in all cases distinctly upwards, but the *ante-meridian* fluctuation in the curve of each table is decidedly less erratic than the fluctuation which occurs in spaces indicative of *post-meridian* mortality. It is scarcely necessary

to point out how highly probable it is that this determinate upward movement towards a maximum associated with regular fluctuations is indicative of the influence of some direct agencies producing that tendency and the variations which characterise it, inasmuch as regularly intercurrent modifications in the progress towards a certain vital result are suggestive of more complex causal influences than those productive of simple tidal changes. In order to represent this comparative condition more vividly, and also to draw attention to a marked depression common to Mr. West Watson's results and those deducible from the West Riding Asylum records, and also approximately expressed in Schneider's conclusions, I shall take the liberty of reproducing from Dr. Finlayson's article his tabulated arrangement of Mr. Watson's figures.

TABLE No. 3.

Hourly distribution of about 13,000 Deaths, summarised by Mr. West Watson and tabulated by Dr. Finlayson.



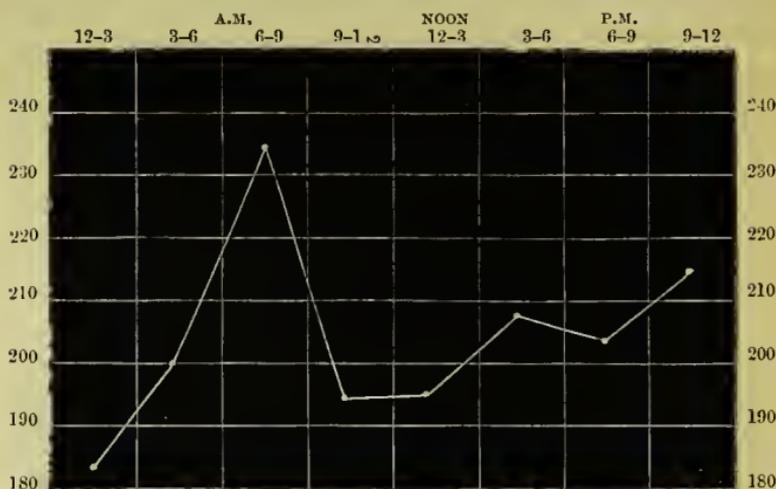
By again referring to the numerical tabulation of the West Riding Asylum records (Table No. 2), and comparing it with this table, it will be seen that in the former there is a marked decline in the number of deaths between 8 and 9 P.M., and that in the latter a similar decided fall characterises the propinquitous hours of 9 to 10 P.M. In Dr. Finlayson's researches this evening decline is not so well marked, but in the comprehensive statistics of Schneider it is of indubitable occurrence. It is impossible to overlook this great diminution in the number of deaths characterising these evening hours, especially as it can be clearly shown that the morning rise in mortality and the evening decline

are converse conditions dependent upon converse aspects of the same causation.

The fact that the action of many incalculable influences makes it difficult to determine to an hour the maximum period of death is illustrated by the regularity of the progression of the numerical curve when the mortality is represented, not for separate hours, but for triads of hours.

TABLE NO. 4.

Distribution of 1,680 Deaths in West Riding Asylum over periods of three hours.



By spreading the inappreciable influences in this manner over groups of three hours, so as to allow their operation during one hour to counteract their influence during another, the action of primary influences is shown in the production of changes which are characteristically gradual and precise. Thus the table (No. 4) dealing with deaths from chronic diseases in stages of three hours shows one steady rise towards a maximum reached at 9 A.M., a decline to a minimum from 9 A.M. to noon, maintained through another period of three hours, and followed by a persistent though limited rise towards midnight. In the same manner Dr. Finlayson's table of deaths from acute diseases, grouped in periods of three hours, shows a steady and uninterrupted progress through a maximum period from 3 to 6 A.M. a marked decline from 6 A.M. to noon, a second elevation lasting

from 12 to 3 P.M., a gradual fall from 3 to 6 P.M., and a precipitate plunge towards a minimum reached at midnight.

From a general consideration of all the information bearing on hourly changes in the rates of mortality it appears:—(1.) That there are some hours which are associated with a great liability to death. (2.) That in acute and chronic diseases the maximum hours of death are widely different. (3.) That in chronic diseases a very large proportion of deaths occurs at a period which may be said to range through one hour before and one hour after 9 o'clock A.M. (4.) That acute diseases are characterised by two daily periods of marked mortality, the first in the dead of night, the second in the afternoon. (5.) That diseases grouped without distinction as to the duration of their course are distinguished by a maximum mortality rather later than that of acute diseases, and an elevated mortality corresponding with the maximum hours of death from chronic diseases.

Conclusions so definite, in a question the principal elements of which enter deeply into vital statistics, point to the probability of a combined exercise of physical and physiological laws in the production of the conditions of which they are the summary. What are the causes productive of effects so clear in their definition? Dr. Finlayson, following in the wake of Dr. Edward Smith, who has devoted special attention to the alternation of physiological acts, and guided by independent observations, and the facts and opinions adduced by Dr. Roberts, Dr. John Davy, Jürgensen and others, sees in this recurrence of maximum periods of death additional evidence of the marked alternations of functional activity.¹ Dr. Finlayson seems to be unaware that for many years Professor Laycock, of Edinburgh University, has during the course of every winter session drawn the attention of his class to the phenomenon of maximum hours of death, both as a means of determining the existence of functional periodicity, and of showing the influence exerted by that periodicity in determining decided variations in the extent of hourly mor-

¹ 'On some Indications of a Daily Periodicity in the Vital Functions of Man,' by James Finlayson, M.D. 'Proceedings of the Philosophical Society of Glasgow,' 1873-74.

tality. But the suggestions which Dr. Finlayson puts forward, and especially the facts which, after the tedious labour of himself and his colleagues, he has added to the literature of the study, as well as his admirable collection of scattered statistics, have introduced an approximate certainty into the subject which will foster further inquiry and afford a feeling of increased security in speculation. The occurrence of these hours of maximum mortality is so intimately associated with the alternations of physiological activity, that the clear appreciation of the causation of the former can only be obtained by the aid of a knowledge of the latter. The investigations of Knox, the suggestions of Sir Henry Holland, and the experiments of Dr. Roberts, Dr. Edward Smith, Dr. Parkes, Scharling and others, have contributed largely to the determination of the exact alterations which occur periodically in the activity of physiological functions and tissue changes. It has been shown that diurnal alterations, the accession of which is capable of overcoming no small amount of artificial opposition, characterise the action of the organs subservient to respiration, circulation, and secretion, and the tissue changes, which result in the production of animal heat. With regard to the exhalation of carbonic acid, Dr. Carpenter thus sums up the evidence bearing upon the existence and extent of its modifications during night and day.

‘Independently of these variations, which have their source in the condition of the individual, there is reason to believe that there is a diurnal cycle of change in the quantity of carbonic acid exhaled; the maximum being (*cæteris paribus*) before and after noon, and the minimum before and after midnight. From the experiments of Scharling upon the human subject, it would appear that the average proportion exhaled by day to that exhaled by night is as $1\frac{1}{4}$ to 1, and this difference *does not seem to be affected by sleep or wakefulness*. Dr. Edward Smith found that the quantity of carbonic acid varied at every moment, and that there were similar hourly variations in the quantity of carbonic acid evolved as in the rate of pulsations, to which we have already referred. There was the least during the middle hours of the night, a slight increase with sunlight, a

large increase after the meals and a decrease before them, and a *prolonged and inevitable fall after about 9 o'clock P.M.*¹

With regard to the secretory function, it has been determined by Dr. Edward Smith that a marked minimum in the secretion of urea occurs during the hours between midnight and six or seven A.M. Though less definitely determined, it seems certain that the bodily temperature fluctuates daily through a variation extending to about $1\frac{1}{2}^{\circ}$ F., the maximum occurring in the afternoon, and the minimum in the dead of night.² But owing to its important, and, in many respects, independent position amongst the physiological systems, periodical modifications in the activity of the circulatory system must be expected to supply marked indications of an alternation extending to every active part of the body. Recurrent circulatory variations evince a uniformity and a comparative indifference to the action of opposing influences which point to them as probably initiatory in the circuit of general change. Recurring alterations in the number of arterial pulsations indicate the existence of a corresponding habitual recurrence of changes in the cardiac activity which can scarcely fail to lead, in the line of physiological action, to similar changes in respiratory, secretory, thermometrical, and other phenomena. According to Dr. Edward Smith and others, there are definite and gradual accelerations in the rapidity of the pulse after each meal, succeeded by a gradual decrease prior to the period of the next. But a more important alteration, as observed by him, consists in an invariable decrease after 9 o'clock P.M., comparatively unaffected by the administration of food. The lowest point in this decadence occurs between the hours of 3 and 5 A.M., and an increase results on or accompanies the rising of the sun. He observed that the difference between the maximum of the pulse during the day and the minimum during the night was in health as much as from 14 to 34, and in phthisis from 22 to 45 beats. Such an important modification in the numerical completion of the circulatory circuit must have an important action on all other physiological functions, over

¹ 'Principles of Human Physiology,' chap. vii. p. 292.

² Ibid. chap. x. p. 405; also Prof. Jurgensen, quoted by Dr. Finlayson.

and above any independent recurrent alteration in their own activity. It indicates a necessary diminution in the amount of exhaled carbonic acid, exclusive of any occult influence which diminished circulatory action may have in producing a reduction in the number of respirations consequent on an alteration of the blood stimulus on the respiratory centres situated principally in the medulla oblongata and upper part of the spinal cord. Again, with regard to secondary changes in the activity of secreting organs and the manifestations of animal heat, it seems evident that unless in abnormal instances of unnatural excitation or disproportionate disintegration of tissues, diminished vascular activity would lead directly to an impairment of both. To the marked alternation occurring in the exercise of nerve function, and characterised by the phenomenon of sleep as a prominent symptom, it is unnecessary now to refer, but the question as to whether the alternation in the activity of the nervous system is primarily productive of the recurrence of modifications in other systems must be subsequently reviewed. Thus it appears that there is a marked correspondence between variations occurring in relative hourly mortality and modifications manifested daily in physiological activity. The observation which appears unavoidable is that there is not only a resemblance in mode, but such a close correspondence in occurrence as suggests the existence of some causal relationship. Shortly after the hours when the action of the heart is at its weakest, when the index of vital heat is at its lowest, and when the respiratory and secretory activity has reached its minimum, deaths from chronic diseases crowd upon each other in such a manner as to show that the fatal decline generally commences during the hours of physiological prostration. That the same is not exclusively the case in acute diseases is, as stated by Dr. Finlayson, due to the fact that a *post-meridian* exacerbation characterises their course; or rather that the occurrence of such an exacerbation shows acute diseases to possess an independent periodicity, and that an additional period of elevation in hourly mortality is determined by the recurrence of hours rendered fatal by their idiosyncrasy. In the instance of chronic maladies

this relation between maximum mortality and minimum function might almost have been accepted *à priori*. If it is assumed that in a healthy man the fulness of life is indicated by the greatest activity of function, it follows that during every recurring period of nocturnal depression he reaches a point which, in the sphere of physiological function, is at the opposite pole, and consequently his nearest daily approach to death. So that if 'black night' is not

Death's second self that seals up all in rest,

it is the time when, by the oblivion of sleep and the extreme depression of organic functions, men are both 'insensible of mortality and desperately mortal.'

What is the causation of this daily physiological alternation, which has such an influence in the determination of relative mortality? Several probable causes suggest themselves, and among the most prominent stands the apparent influence of the change from day to night, with all the secondary meteorological variations associated with it. There is the influence of recurrent alterations in the function of the nervous system, acting as a primary agent in the production of changes in other systems. There is the influence of habit, as suggested by Dr. Finlayson, or of habit strengthened by hereditary transmission of tendencies in organs and tissues, and finally the influence of a daily diminution in the functional activity of the heart, in order that the renovation of its muscular tissue may be more persistently effected. Each of these probabilities has its own elements of weakness, and it is necessary to determine which one of them or what combination of them affords the nearest approximation to an explanation of the phenomena under review.

First, with regard to the influence of solar changes, and the meteorological modifications mainly dependent upon them. These may be adduced as the nocturnal withdrawal of direct solar heat and light, barometric modifications, and variations in the amount of free atmospheric moisture consequent on the cooling of the earth's surface. In addition to these purely physical alterations, it is necessary to ascribe a certain amount of sedative influence to the condition of

quietude and repose which, in a natural state of society, exists as a contra-stimulant to function exercise, by the absence of many of those excitants to physical and intellectual activity which characterise the busier period of the day. What relation has alteration in external heat to modification in functional activity? It has been observed (also by Dr. Edward Smith) that during the warmer seasons of the year the pulse is accelerated, countenancing to some extent the ancient idea of a spring plethora, which led to the employment of measures of depletion at that season, a practice which still holds its ground in most domestic codes of medicine. This ancient practice, based on the venerable principle expressed by Hermes Trismegistus, 'Omnia quæ in cælis sunt in terris terrestri modo,' illustrates the early belief that systemic changes were influenced by physical conditions, and is not the only example of the correctness of conclusions resulting more from analogy than syllogism. Again, independently of the action of the nervous system (as proved by section of nerves or the administration of woorara), it is determined that plunging the leg of a frog into warm water increases the frequency of arterial pulsations. Notwithstanding these observations, it is highly improbable that such declination of atmospheric temperature as occurs gradually between the early afternoon and the dead of night could in itself have much effect in depressing functional activity. It is directly proved that the amount of external temperature required to elevate to any extent the temperature of the body is much in excess of the periodical external variations. The result of long-continued observation of diurnal changes of atmospheric temperature shows that the mean variation is one extending through $10\frac{1}{2}^{\circ}$ F. The highest average occurs at 2 P.M., nearly, and the lowest at 4 A.M., nearly. But it must be remembered that in human society the means employed to counteract this normal decline in nocturnal temperature may have a more depressing action than that which would result upon the unopposed influence of the decline itself. The production of compensatory heat, by the use of hydro-carbons, or by the accumulation in the atmosphere of heated human exhalations, never

fails to have a depressing influence on physiological function. The influence which the chemical and thermal rays of the sun exert upon the growth of vegetable forms suggests the idea that their absence may have at least some tendency to repress functional activity in higher forms of life. Some countenance is given to this view by the experiments of Dr. Reid, of Edinburgh, proving that a patient recovers from carbonic acid poisoning more quickly when placed in a strong light than when merely taken into the open air.¹ And in hospital construction there is no doubt that a large supply of light is of importance as well as a sufficient supply of air. The influence ascribed to the chemical rays in the production of sunstroke would also favour the idea of their agency in the modification of physiological acts. With regard to the action of light on plants, however, it must be remembered that it is a purely chemical one—that it is an action by which the force derived from the swiftly vibrating rays enables the green colouring matter of the leaves to decompose atmospheric carbonic acid into its constituent elements in order that the carbon may be absorbed and assimilated. It is not improbable that in the instances referred to by Sir Henry Holland, showing the potency of the rays of light in depraved states, the effects were subjective, or induced not vitally, but by a stimulating action on what is spoken of colloquially as ‘the spirits.’ With regard to the influence of chemical as distinguished from thermal rays, the nature of their action must be more clearly understood before their absence can be admitted as exercising a depressing influence on vital function. Again, with respect to the action which diurnal barometric changes may have, it has only to be noted that these changes are so minute, when compared with other great barometric variations which have been experienced with almost no direct effect upon human function, that they can be safely overlooked.² It still remains to

¹ ‘Medical Notes and Reflections,’ by Sir Henry Holland, Bart., M.D., F.R.S.

² Though the inference is limited to two persons, yet it may be worth while to mention the great experiment made by Mr. Green and Mr. Rush in September 1838, in ascending to the height of 27,136 feet, or 5½ miles, above the level of the sea, the greatest elevation ever reached by man, and very nearly approaching the level of the Kinchinjunga, the highest ascertained summit of the Himalaya Moun-

refer to the nocturnal absence of the many excitants of physical and intellectual energy which during the day tend to maintain the current of physiological activity. Though it will be universally admitted that such a condition supplements other agents in inducing modifications of physiological action, it will be as generally allowed that this cause could not alone effect any regular depression of general function. And finally with regard to hygrometric changes, it is highly improbable that they exert any appreciable influence, as the observations which have determined the existence of periodical alternations have been conducted under circumstances in which those changes must have been counteracted by artificial heat. Thus it appears scarcely probable that recurrent solar changes and the associated meteorological conditions can be proved to exercise anything like an exclusive influence on the production of recurrent physiological depressions. At the same time it is not unlikely that they act as accessories to more direct agencies.

In considering how far alterations in the functional activity of the nervous system may act as agents in producing functional depression in other systems, and especially the circulatory, it is not necessary to enter fully into the observations which prove the possession of independent characteristics by the muscular system. It is not requisite to demonstrate the inherent irritability, amenable to special laws, the independent power of producing electric currents, and the possession of elasticity which distinguish the muscular tissue. A reference to the fact that the heart retains, after removal from the body, a remarkable power of continuous contraction is comparatively unimportant, as it is difficult to determine what influence the sympathetic ganglia in its own texture may exert in relation to such movements. Any observation based on the existence of cardiac movements in anencephalous monsters, and in animals whose brain and

tains. The barometer fell from $30^{\circ} 50'$ to 11° during the ascent, the thermometer from 61° to 5° . The first 11,000 feet was passed through in about seven minutes. Yet under these remarkable circumstances Mr. Rush suffered no inconvenience but from cold, and Mr. Green little other than from the toil of discharging ballast at different intervals, which hurried the respiration during the time.—Sir Henry Holland, 'Medical Notes and Reflections,' p. 184, Note.

spinal cord have been cautiously removed, are open to the same objection. It appears, however, that the latter observations are accepted as conclusive in demonstrating that the action of the nervous system is not essential to cardiac movements. It is certain that depressed states of cardiac function occur entirely independent of the state of the nervous system, and an additional proof of this observation is supplied by the fact that the time of daily circulatory depression is several hours antecedent to the occurrence of sleep. It is further confirmatory of this view that the hours of maximum mortality are the same in a large lunatic asylum, where the nervous system presents all variations of organic and functional change, as those which mark the termination of general chronic diseases. The muscular system having distinct properties and a capacity for exercising certain powers when entirely deprived of all assistance from the nervous system, and the heart being an organ more amenable to direct than nervous irritation, it is certain that, however much the controlling influence of the nervous system may modify cardiac action, the heart is quite capable of undergoing independent variations of functional activity. What share has habit, as suggested by Dr. Finlayson, in determining the occurrence of physiological alterations? The instances of similar alteration adduced in support of this suggestion are instances of modifications in bodily function induced in a manner partly voluntary. Such are the recurrence of sleep at hours closely correspondent, and the alteration which physiological changes undergo by changes in the hours of meals. It appears evident that modifications in the functions of those organs over which the will has little or no control could only be induced indirectly by habit in one way, namely, by the habitual combination of influences favourable to functional changes, and by the habitual regulation of voluntary physiological activity in such a manner as to induce as an effect a habitual diminution of functional power in the organs manifesting such physiological activity. In this manner, no permanent voluntary influence can depress the action of the heart, the lungs, or the kidneys, or modify the production of animal heat, and, consequently, cannot inaugurate a habit of

functional alternation. But the habit of fully and voluntarily exercising the bodily and mental activity during the time when nature expends the direct influence of her forces upon a given hemisphere necessitates a habitual subsequence of functional depression, following in the wake of solar changes, and extending in voluntary tissues to complete repose, while in the instance of tissues independent of the will it stops short at the stage of relative inactivity. Thus, though habit could scarcely be regarded as primarily explanatory of the production of these recurrences, there can be little doubt that with respect to accessory influences, and the tendency to automatic repetition of long-continued physiological acts, it may go far in determining the time at which these recurrent changes manifest themselves. In this manner the philosopher Kant methodically saved expenditure of mental and physical energy by bringing into nightly co-operation the habitual tendency to physiological depression on the one hand, and the agency of accessory influences on the other, by abstaining at a fixed period from all exciting trains of ideas and all physical exertion. In this manner he separated distinctly the period of functional activity from that of repose, and more fully utilised both. In the matter of recurrent physiological changes this influence of habit is so strongly marked, that the negative effect produced by such experiments as excluding sunlight for several mornings for the sake of proving its want of influence on physiological changes is the only result that could reasonably be anticipated. And owing to the same tendency in the organs and tissues to continue in an acquired line of physiological activity, it is not at all certain that even the temporary exchange of the repose of night for the activity of day, and *vice versâ*, would make anything but a very gradual change in the periods of functional alternation.¹ Still it may be regarded as certain that a gradual change, such as would transfer the period of depression to its proper place after the period of activity, would ultimately occur.

Thus though the influence of habit, aided or unaided by

¹ See Dr. Finlayson, 'On the Daily Periodicity in the Vital Functions of Man,' *ut supra*, p. 101.

meteorological and social conditions, may determine the period of functional depression, it does not account for its production. These periodical changes in functional activity must either be induced by powerful recurring influences acting from without, or they must depend upon the occurrence of a daily requirement of the system, by which the temporary depression of physiological function becomes necessary for the healthy continuance of organic existence. Already it has been shown that no external change can be regarded as the exclusive or even the main agent, and it now remains to determine what influence the known conditions of tissue nutrition and organic function exert in producing a daily recurrence of functional depression. What, for instance, are the laws which regulate the exercise of contractility in muscles, what provision is made for their restoration after activity, and how do these provisions effect modifications in the activity of the cardiac and other functions? It is well known that though the active exercise of contractility is favourable to the healthy maintenance of muscular tissue, two conditions are attached to this manifestation of activity. The muscular tissue must have recurrent periods of repose, either between contractions or during a period of rest. In addition it must be provided with a sufficient supply of nourishment, and be constantly relieved from the products of disintegration. There is reason to believe that if these conditions were perfectly maintained in the nutrition of muscular fibre, contraction could be continued as a constant phenomenon.¹

But the experience of every day, and the production artificially of muscular exhaustion by constant contraction, show that the interval between contractions is not sufficient for the nutrient requirements of muscular fibre. In both instances intervals of rest are required for the purpose of reconstruction and for the restoration of vital irritability. The manifestation of exhaustion by muscular tissue after the ordinary exercise of its contractility is a sufficient indication of this. But during the period of its action the disintegration

¹ Ranke, quoted by Carpenter, *ut supra*, p. 677.

which it undergoes in the performance of its function is not fully compensated by simultaneous nutritive processes. The opposite condition to that which is found in childhood is evinced. In childhood nutrition being in excess of function admits of continuous development, and as a characteristic a decided tendency to sleep is manifested as the most evident sign of repose and absence of functional excitement. In adult life, organic development having been completed, a larger correlated amount of force is allotted to functional activity. Functional activity involves structural disintegration and demands constant reconstruction, and when the balance between disintegration and reconstruction is lost, contractility is weakened, and the muscular system, by a daily recurrence of exhaustion, sinks into a state of depressed function in which the type of the child recurs. While a similar state of the brain substance ensures an almost complete exemption from physiological excitation, the renovation of wasted tissue and the restoration of irritability are effected in preparation for renewed functional activity. If, therefore, it is true of general muscular tissue that daily periods are required for the renovation of its wasted texture and the renewal of its inherent powers, why should the muscular tissue entering into the structure of the heart and respiratory muscles present an exception to other muscular structures? The rhythmical character of their operations may provide for a more regular and measured supply of nutritive material than is assigned to voluntary muscles; but it is impossible to believe that the disintegration of these muscular textures is so equally balanced by their reconstruction that the heart and the lungs could be capable of working with a continued uniformity of action without a period of relative repose. Consequently it is evident that some provision must exist in nature for the readjustment of such deviations from the nutritive balance as occur during periods of relative functional excitement. Everything in nature points to a diurnal alternation of activity and repose, and there is no more marked instance of that alternation in the whole round of natural phenomena than that presented by the study of muscular conditions. The constant exercise

of the whole or part of the muscular system during bodily activity necessitates diurnally a recurrent period of repose. And as the fibres of the involuntary muscles come essentially under the action of the same laws, it is reasonable to conclude that they also, after an activity more uniform than the others, should manifest a depression of function even earlier than they. Such has been determined to be the case, as demonstrated by the recurring diminution in the number of cardiac pulsations occurring about 8 or 9 o'clock P.M., and characterising the whole nocturnal period of muscular inactivity. It appears, therefore, that in all probability the diurnal depressions of physiological activity have their origin in a diurnal diminution in the contractility of the heart, common to it and to all muscular structures, and directly and physiologically dependent upon a loss of balance between the activity of reconstruction of its enfeebled texture on the one hand, and the amount of disintegration resulting from its functional activity on the other. The function of the heart, quite as much as the function of the brain, is maintained at the expense of its own material, and this must be restored diurnally for the continuance of its functional integrity. And if it appears that the case of the heart differs from that of the brain with regard to the extent to which its function is nocturnally depressed, it must be remembered that such parts of the nervous system as are, like the heart, connected with the maintenance of nutritive changes in their own and other tissues, must like it be comparatively active even during sleep. That the periodically diminished functional activity of the heart is due to a correlative storing up of nutrition in its own tissue is corroborated by the more marked nocturnal exercise of cell nutrition in the brain, a process which is believed by Dr. Hack Tuke to be conducted principally if not entirely during sleep.

What is the relation between periods of maximum mortality on the one hand, and hours of recurring depression and exaltation of function on the other? If the weakest physiological period occurs during the dead of night, why does the maximum mortality, instead of happening at that time, occur at the period ranging from 8 to 10 A.M., when in

the healthy body the capability of physiological activity is greatest? Why also does such a marked diminution in the number of deaths, as shown by the tabulated statistics of Mr. Watson and confirmed by those of this Asylum, occur at about 8 and 9 P.M., when the change from functional activity to that of tissue reintegration is commencing? Relationships disposed in such an evident antithesis can scarcely be regarded as the result of a mere coincidence in the statistics of numerous public institutions, relating to various groups of diseases, compiled independently, and summarised in different countries.

The period of functional depression is the time, diurnally recurrent, when, as far as vital energy is concerned, the body is nearest the verge of existence. But in the healthy body the tissues may at the same time be in a vigorous condition as the result of a progressing reintegration. In the body wasted by chronic disease the case is different. The organs, weak it may be throughout their diurnal course of action, reach daily the same physiological period of depression—the period most closely bordering upon death. Time after time they must pass that dangerous stage, which by the slackening of functional activity admits of the partial renovation of their wasted tissues by blood imperfect in quality or deficient in quantity. But latterly the time arrives when either by the natural progress of decay, or the action of some of the extraneous influences already enumerated, the body, constantly weak, gives up the struggle for life when at its weakest; the process of dying commences, and gradually advances towards complete extinction. On the one hand there is the precipitate advance of the organism to annihilation, and on the other a uniform perseverance in the performance of depressed function. When the hours are reached at which the habit of physiological recurrence has confirmed a tendency to increased functional as compared with nutritive activity, the disparity between functional requirements and nutritive restoration of tissues is intensified and death ensues. In this manner the hours of physiological depression are those which determine the commencement of the process of dying, and the hours at which the decided renewal of physiological activity normally recurs precipitate the decision of an in-

evitable issue. So that in the comparative hourly occurrence of deaths from chronic diseases, it may be safely affirmed that the largest number of deaths is determined by the hours of extreme functional depression, and occurs at the time when the acquired tendencies of organs and tissues have habituated them to a manifestation of increased activity which these organs and tissues in the last stages of incapacity are unable to reproduce. The first period corresponds to the dead of night, and the second to the hours between 8 and 10 A.M. In acute diseases the conditions are altered. During the time when such diseases threaten imminently to be fatal, there are periodical recurrences of intensity of the diseases themselves which determine the time of death. Thus in acute diseases there are two distinct periods of increased mortality. The first is the period when, by the physiologically recurrent depression of functional activity, the system is least able to counteract the tendency to death, and the second occurs at the time when the independent periodicity of the acute disease overcomes the resistance of the enfeebled body. The first period is that of the pulse minimum, from 3 to 6 A.M. The second period is that of the severest exacerbation in fatal acute diseases, occurring late in the afternoon. If it is reasonable to prognosticate that in the case of a feeble frame the morning hours, when a physiological exaltation of function has been habitual, would be extremely fatal, it should be conversely probable that the evening hours, during which a diminution of functional activity has become by habit a law of the organism, would be characterised by a marked diminution of mortality. It is interesting, and somewhat corroborative of the relationship between recurrent periods of physiological depression and increased hourly mortality, to find that in the tables showing the periods of death, as summarised by Mr. Watson and Schneider, as well as in those showing the hourly mortality of this Asylum, a marked diminution in the number of deaths is shown to occur between the hours of 8 and 10 P.M., when the great depression in functional activity begins. At these hours, as determined by the influence of habit, the enfeebled system begins to devote a greater amount of its energy to the main-

tenance and restoration of its own tissue than to the performance of organic function. From this point of view it appears highly probable that this nocturnal depression of function may have a converse power in warding off impending death, corresponding to the tendency which the converse morning exaltation has in precipitating it. However this may be, the opposite position of the morning and evening curves on the tabular scales, both of pulsation and mortality, supplies one of the most irresistible items of evidence bearing on the causal relationship of physiological variations and hourly modifications in mortality.

The practical features of these observations are too prominent to require detailed enumeration. They show that in chronic diseases it is necessary, by careful nursing, by judicious stimulation, and the exclusion of all depressing emotions, to assist the prostrate patient in rising buoyantly over the hours of his greatest depression, which will generally be found to occur between the hours of 3 and 6 A.M. They further indicate how important it is that by complete rest in the early hours he may have an opportunity of storing up new energies in his wasted tissues to enable him to meet the demand for that recurrent manifestation of increased activity which, as determined by physiological habit, will most commonly be found to characterise the hours between 8 and 10 A.M. In the case of acute diseases, the determination of periods of great mortality, as altered by their idiosyncrasy, shows how important it is, in a case threatening extinction, to moderate as much as possible the vigour of diseased action towards the approach of its recurrent periods of *post-meridian* intensity, and also to sustain the body by every available expedient prior to those hours (from 3 to 6 A.M.) when, in the depth of its functional fluctuation, the system is least able to react against the tendency to dissolution.



ACUTE DEMENTIA.

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THAT enfeeblement of the intellectual, emotional, and voluntary powers which constitutes dementia, and which occurs most frequently as the sequel of other forms of mental disease, may occur also as a primary disorder, and when it does so, it presents a very definite outline, and is attended by bodily symptoms of a distinctive character. Strictly speaking there are several species of primary dementia, for the fatuity which darkens declining years, and that which follows upon apoplexy, as well as that particular fatuity to which I am now referring, are unpreceded by any other kind of insanity. But senile and organic dementia have received other specific appellations, and are generally viewed in relation to a series of preliminary physical deteriorations, so that the term primary is reserved for that dementia which arises neither from age nor structural lesion, but from original functional derangement of the cerebrum in the years of its maximum activity. Dementia, thus primary, is also called acute, and as that designation, although not free from objection, has been widely received, and is perhaps less invidious than any other that has been suggested, it will be convenient to adopt it, and to apply it to those states of mental weakness which are neither congenital nor chronic, and which are not included under other headings. Acute or primary has also been called apathetic dementia, in allu-

sion to the complete torpor of feeling by which it is marked, and the same mental condition is described by Georget as *stupidité*, and is recognised by Ferrus and Belhomme as a distinct disorder.

Acute dementia attacks both sexes, but females in a larger proportion, and perhaps in a milder degree than males. It is essentially a disease of youth, being rarely seen in patients beyond thirty years of age, and it seem indeed to be often dependent upon exhausting influences operating at a period of rapid growth. Children whose powers are overtaxed at a time when the process of development is going on, and when nutrition has not merely to repair tissue waste, but to contribute to the formation of new morphological elements, often fall into a state resembling idiocy, in which they are dull, sullen, and oppressed. And the children who are thus affected by acute dementia are not always those who have displayed extreme quickness of intellect, coupled with nervous instability, who have been clever and fragile, or who have inherited a predisposition to insanity. On the contrary, they are often those who have possessed only commonplace abilities, who have been robust dunces, and who have come of a perfectly healthy stock, for it is a peculiarity of acute dementia that it is less frequently connected with a hereditary taint than perhaps any other form of mental aberration. No doubt, whenever its causes have been enumerated, neurotic tendencies have been assigned a prominent place amongst them, but that, perhaps, has arisen from force of habit rather than from accurate observation. Neurotic tendencies are the parents of such a multitude of evils that it seems one can scarcely be wrong in affiliating upon them a malady having such a striking family resemblance to their acknowledged progeny; but minute inquiry will scarcely warrant such a proceeding, for out of twelve cases of acute dementia, the history of which I was able satisfactorily to trace out, there were only three in which a hereditary proclivity to mental or nervous disease could be discovered. In nine cases there was, as far as could be ascertained, an entire freedom from any such morbid impregnation. It is not, of course, asserted that this disease

may not have its roots in ancestral mould, or in deep-laid substrata. What is alleged is that it far oftener grows out of superficial and individual conditions, and that these are themselves sufficient to account for its phenomena, without referring back to any hypothetical inheritance. Indeed, strange as it may sound, it has sometimes seemed that the absence of any neurotic inheritance was favourable to the development of acute dementia when its immediate causes came into play. These causes, such as debilitating occupations or insufficient nourishment, when operating upon neurotic subjects, have appeared to lead up to erythismic disorders, to excitement and mania, whereas when acting upon more stolid beings, they have induced a blunting of the mental powers, or acute dementia.

It might be inferred, from what has just been said, that acute dementia is oftener due to physical than to moral causes, and that inference would be correct. Moral impressions of a deleterious nature or intensity act more powerfully upon sensitive mobile beings than upon those who are duller and steadier, and consequently they are not very influential over that class from which acute dementals are drawn, unless physical conditions have previously produced prostration. Seldom do we hear of acute dementia being brought on by a fright, or a disappointment, or a joyous surprise, unless a state of extreme debility has existed when the emotional shock happened. The one moral cause which is effectual in inducing this disorder is monotony of thought and feeling or mental inanition. Man does not live by bread alone—his dietary must be varied, and if it is not varied he is starved as effectually as if he were kept on short commons. And the same is true of his mental food; that too must be varied, and if it is not varied it ceases to nourish him, and he pines into dementia. Under various conditions of life, where new impressions and ideas are not supplied, and where a tedious uninteresting routine is inevitable, does failure of mental power occur. This is especially the case when the deprivation of new impressions and the imposition of new restrictions are coincident with a period of mental evolution, when the growing mind is greedy of nourishment suited to its wants. Children who are sent at an early age into factories, where brick

walls and reiterative twining machinery surround them for two-thirds of their waking hours, and where their work consists in a series of incessantly repeated muscular movements, requiring close attention, often, I believe, pass through a phase of acute dementia; three instances of that disease in children have come under my observation in the West Riding Asylum, in whom the only discoverable cause was the laborious sameness of their occupation in the mill. Young prisoners, too, sometimes suffer in the direction of mental weakness from the wearisome uniformity of their lives, and madness may be the result of reformatory discipline. The treadmill, if used perseveringly, would indeed seem to be an infallible method of producing that affection, for even industrial pursuits, with their comparatively high interest and variation, when long pursued in incarceration are insufficient to preserve mental health. The stupidity of criminals is proverbial, and perhaps some part of it is due to the punishment which they undergo, and to its effect upon their memory and understanding. Be that as it may, there can be no doubt that a state of unmistakably morbid mental deterioration, marked by loss of memory, feeling, and activity, sometimes presents itself in criminals who are undergoing imprisonment of some duration. That state is clearly akin to acute dementia, which I have noted as being present in a marked form in several insane prisoners whom I have examined. But under many less trying phases than these found in factories and prisons, does monotony exert a debilitating effect upon the mind. Thus, as experienced by our sailors stationed on the West Coast of Africa, it is said to be very pernicious. To them no change or relaxation is possible for twelve months together, while the incessant rolling of the ship, the constant low rumbling of the heavy surf upon the beach, the tame flatness of the land, fill them with a sense of intolerable tedium. The effects of these trials are to be seen, according to the official report of Sir F. W. Grey in the invaliding of officers and others from 'mental disorganisation.'

True, under all the circumstances mentioned as productive of acute dementia by the mental inanition which they entail, physical are conspiring with moral causes to

induce a disastrous result. Children in factories suffer from the confinement which they have to undergo, and are often badly fed and housed. Prisoners have depraved constitutions, owing to their habitual vices, and are kept on a bare subsistence dietary; and sailors on the West Coast are exposed to the influence of an insalubrious climate, and see little of fresh meat or vegetables. But still, with all these, when loss of mental tonicity takes place, when intellectual weakness and disability supervene, the lack of suitable mental nourishment contributes to an appreciable extent to the production of the mental enfeeblement. It co-operates with those physical agencies which are even more potent causes of acute dementia, and which include various debilitating conditions, of which anæmia is that which first deserves consideration. Anæmia and its congener, spanæmia, are not of themselves frequent proximate causes of this disease, their immediate effect upon the cerebral functions being commonly of a different nature. It is when some other injurious influence, such as excessive fatigue, exposure to cold, or an attack of illness, is superadded to one or other of them that they conduce to acute dementia. I remember seeing a chlorotic girl, who had retained unimpaired intelligence until she was attacked by influenza, when she rapidly lost the use of her faculties, and became unable to think, speak, or move spontaneously. Symptomatic anæmia, that dependent upon other maladies, or upon cachectic affections, and sudden anæmia, that due to excessive hæmorrhage, are less apt to lead up to acute dementia than the anæmia which is tardily induced by scanty food, or such food as contains little animal matter or protein, by profuse evacuations, impure air, or uterine irregularity. The anæmia that is most protracted, and that tends most to asthenia and failure of the vital powers, is that which is most likely to eventuate in this particular mental derangement.

Acute diseases which occasion much exhaustion have sometimes acute dementia amongst their sequelæ. After severe attacks of typhoid fever there is very generally a certain amount of fatuity, exhibited in childishness of

manner, loss of memory, and universal mental inertia. This fatuity, which is accompanied by great bodily prostration, and which may proceed from changes in the intracranial circulation, or from an anæmic, atrophical, or imperfectly nourished state of the brain, is oftenest seen in those cases in which there has been much delirium during the fever, but is occasionally present when the mind has remained clear during the period of pyrexia. I was some time ago called upon to treat a lad who had passed through a sharp attack of enteric fever, in which, however, from first to last, there had been no delirium, and who when convalescent began to wander in his thoughts, to mistake the identity of persons around him, and to manifest, in fact, the early symptoms of what proved to be acute dementia. I have seen that disease also brought on by protracted chronic diarrhœa, by bleeding piles, and by leucorrhœa and menorrhagia. Further, it is probable that it sometimes has its origin in malaria or an atmospheric miasm. In one case that came under my observation it had apparently started from a peripheral cause; it came on suddenly during the cicatrisation of a lacerated wound of the hand which had been caught in machinery. There can be no question that it is often brought on by masturbation, through the channel of the physical effects of that pernicious practice rather than through the influence which it exerts upon the spirits and moral character of those who indulge in it. Anæmia, loss of strength, and nervous exhaustion result from it, and after these come forgetfulness, heaviness, and listlessness, which grow and grow until fatuity is attained. I have under my care now a young man who, from time to time, deprives himself of 'energetic reason and a shaping mind' by this abominable and inveterate vice. When he gives way to it he becomes acutely demented; when he abstains from it, or when by medical interference it is rendered impracticable for a time, he rallies quickly, and is sharp and lively, and an expert tailor. The chief tailor can always tell from the way in which he handles his needle and thread if he is conducting himself with propriety or otherwise. When the former, his fingers move nimbly and his eyes are intent on his work;

when the latter, he gazes dreamily about him and dwells drawlingly on every stitch.

Howsoever induced, acute dementia makes its incursion in one of two ways. It either steals over the patient by gradual and at first almost imperceptible encroachments, or it is ostentatiously ushered in by an attack of excitement. In the first case, some slips of memory, some relaxation of attention, some errant thoughts, some moments of blank bewilderment, are its earliest harbingers, while in the second case, an outbreak of fury, wild bursts of laughter, swift meaningless movements, and broken disconnected sentences herald it in. But however introduced, when once fairly established it is manifested by a greater or less suspension of the psychical activities. Impressions are slowly transmitted to the mind, and are imperfectly assimilated, so that only a dim fragmentary knowledge is obtained of external things and events. Comparison is curtailed, and thus cognitions cannot be adequately contrasted or integrated. Representation is feebly performed, and thus reminiscences and trains of associated ideas and beliefs drag haltingly along, instead of gliding onwards in rapid and orderly succession. Imagination has abandoned its creative work. Desire—which in health surges so tumultuously—scarcely moves within, affections and passions are dormant, and the will, whether applied to the instruments of knowledge or of movement, is destitute of strength. This inward debility is of course expressed outwardly in modifications of physiognomy, gait, and conduct. The countenance wears a perplexed or a vacant expression. The attitude betokens lethargy or irresolution. The voice loses its accustomed tone and becomes low and drawling, and the limbs perform their duties with a weighty effort. The patient is silent and self-absorbed. If spoken to he gives no heed to what is said. If roused, he may after a pause answer a few questions correctly, but when the conversation is pushed further, it is found that his ideas are turbid, and that he cannot collect or arrange them. His memory is bad, his command of language is contemptible. If he continues his accustomed occupation, it is performed in the most slovenly way. More frequently, however, all work

is given up, all exercise is renounced. At this stage some curious exhibitions of the imitative faculty and of automatic muscular activity are sometimes seen. The patient will slowly repeat any question that is asked of him, and if the sentence conveying the question be a long one, he will repeat the three or four words which close it. He never attempts to answer the question, he simply echoes it. Occasionally he will feebly echo any sound that he may hear, or will clumsily reproduce any gesture that he may witness. Occasionally one word or form of words serves as an answer to every interrogation. A girl acutely demented was admitted into the West Riding Asylum a year ago, from whom at the outset no reply could be elicited. After at least a dozen determined repetitions, however, of the first question of the catechism and of most medical examinations, 'What is your name?' being awakened and stirred into activity, or driven to desperation, she cried out 'Elizabeth,' in a shrill treble, and after that she went on crying out 'Elizabeth' as a response to everything that was said to her. For a whole month, whenever she was spoken to, without raising her eyes or changing her attitude, she shouted out 'Elizabeth' with the same sharp accentuation. Analogous to such automatic cries are certain automatic muscular movements that are sometimes seen in cases of this kind. Although there is scarcely enough conscious energy to put the limbs in motion in accordance with feelings and desires, there is abundance of a lower quality of volition to keep up for some time a definite series of movement, when these are fairly inaugurated by some external agency. Thus if the patient is made to run, he will run on, when left to himself, steadily and rhythmically, until he is stopped by some obstacle. I have seen this tendency to continue any definite movements that have been set agoing utilised in a singular manner. A few years ago there was in the West Riding Asylum a girl afflicted by acute dementia, a petrified specimen of humanity, who never voluntarily changed her position, who would not feed herself, and who gave much trouble until the nurses stumbled upon an expedient which spared them the necessity of placing every morsel in her mouth. They found that by putting a spoon

in her right hand, firmly closing her fingers over it, dipping it in the food on her plate, raising it to her mouth and inserting the point of it between her lips, they could inaugurate a process which resulted in her feeding herself. When the circle of movements upon the plate, from the plate to the lips, between the lips, and from the lips to the plate, had been several times described by the hands of the patient and the nurse conjointly, it was indefinitely repeated after the compelling hand of the nurse was withdrawn. Without looking at her plate, Jemima B—— went on drawing the spoon across it, raising the spoon to her mouth, grasping with her lips and swallowing whatever the spoon conveyed, and then lowering the spoon to the plate again. All this she did in the most machine-like manner. She always drew the spoon across the same part of the plate, in the same direction, and went on doing so long after all the food, mincemeat, rice, &c., had been removed from that part. The nurses had to turn the plate from time to time so as to bring what lay upon it within the orbit of the revolving spoon.

During the progress of acute dementia, little gusts of excitement, similar to those which I have spoken of as being in certain cases the first declaration of the disease, are occasionally encountered. In some cases there is no excitement from first to last, in others there are sharp but transient attacks, displayed in restlessness, incoherence, mischief, and destructive violence. These attacks are rather deceptive. They suggest at first an exaltation of mental activity altogether opposed to dementia, but on examination they are seen to correspond with mental weakness. The garrulity of the patient conveys no intelligible ideas, not even delusions or hallucinations; his restlessness is aimless. The inchoate excitement is not the expression of quickened or perverted thought, but rather of a state of irritation in the enfeebled brain. It subsides in a few days, and gives place again to dulness and passivity.

When acute dementia is of a severe type, and is fully developed, the mental state is one of profound stupidity. Comprehension is abolished, memory is a blank, language is lost, the sentiments are lifeless, the will is palsied, and even animal

wants are not attended to. Organic existence alone remains. The sufferer is indifferent to all that is taking place around him, and is to a great extent insensible to external impression. For now there is anæsthesia of the surface with blunting of all the senses. Pricking or pinching the skin does not cause signs of pain, nor does tickling produce movement. Loud and unexpected sounds do not startle, and irritation of the nostrils is not followed by sneezing. The indisposition for muscular exercise, which was noted earlier in the disease, has now increased into complete lethargy. The patient will sit or stand for hours in one position, lacking spontaneity to change it. Everything requires to be done for him, and while in the hands of his attendants he offers neither resistance nor aid; he is as inane and helpless as a statue. And now it is that a species of catalepsy is observed. The limbs remain for a time in any position in which they may be placed, the body in any attitude into which it may be thrown. If the arms be raised above the head they will be held there for perhaps an hour, or for such a time as would occasion intense suffering to a healthy person, and they then slowly drop to the side. Any pose in which the patient may be adjusted by a bystander, no matter how constrained or grotesque it may be, is maintained until it is modified by some external force. And yet there is no rigidity of the muscles. The limbs are flaccid and are readily flexed and extended, and it is remarkable that after being long held in positions in which great resistance to gravitation must be exerted they are still free from stiffness. I have seen a delicate girl, when sunk in acute dementia, stand for twenty minutes with her arms stretched out horizontally immovable, like a fakir.

Of the bodily symptoms of acute dementia, those connected with the circulatory system are the most prominent and significant. They consist in feeble action of the heart, small and almost imperceptible pulse at the wrist, and passive hyperæmia of the extremities. The hands and feet, and more especially the former, are cold and have a bluish-red colour, which disappears under pressure, leaving a patch of pale skin, but speedily returns. This coldness and blueness is very striking, and is often accompanied by a considerable

degree of swelling. In some cases the hands ultimately assume a deep purple colour, and vesicles form on them which give way and leave an indolent sore, emitting a thin watery discharge. The hands and feet are, in fact, affected by diffused chilblains, which form and persist even during summer, and when the extremities are kept warm and wrapped in cotton wool. With the chilblains there is occasionally a moderate degree of œdema of the ankles and legs. The same bluish-red or purple discoloration that is seen in the hands is also generally seen in the nose, ears, and cheeks, giving a chill, livid, pinched aspect to the countenance. When excitement comes on there is generally more active flushing of the face and heat of head. The pupils in acute dementia are invariably more or less dilated and very inactive.

The respiration is quiet and shallow; sometimes the patient can be scarcely seen to breathe. But although there is as a rule no pulmonary derangement, the chief dangers of the disease are connected with the lungs. In its advanced stages there is a liability to œdema of the lungs, and the only fatal cases of acute dementia that I have met with have been brought to an unfortunate termination by pneumonia and phthisis.

The temperature, as ascertained in the axilla or rectum, is always near the normal standard, but when taken in the chilled hands it may be as much as 12° or 15° F. below that point.

The tongue is tolerably clean, or is swollen, pale in colour, and marked at the edges by the teeth. There is always a copious flow of saliva, and sometimes the amount secreted is very great, so that a stream is perpetually dribbling from the lips. The flow from the mouth is not in itself a proof that the saliva has increased in quantity, for it may be that it escapes in this way simply because the patient is too lethargic to swallow it. But in some instances the saliva flowing from the mouth has been collected and measured, and in this way it has been shown that it is absolutely increased in quantity. As much as a pint has been collected in five hours, and this must be regarded as an excessive quantity, especially as no food was taken, and as the tongue and mas-

tatory muscles were at rest while this pint was poured forth. The appetite is not impaired as far as can be judged, the abstinence from food being dictated by torpor rather than by anorexia. What is placed in the mouth is swallowed, and no loathing of nourishment is manifested. In many cases, however, there are attacks of vomiting, apparently without sickness. Soon after food has been taken, without pallor or other signs of nausea, the contents of the stomach are rejected. This may go on obstinately for days or even weeks, when suddenly it ceases, to return perhaps again after an interval. The bowels are in a majority of cases confined, but brief attacks of diarrhœa are not uncommon.

In females there is constantly amenorrhœa, rarely leucorrhœa or menorrhagia. The outbursts of excitement in them generally occur at the menstrual periods.

The symptoms of acute dementia, psychical and somatic, which have been detailed, render it an easily recognisable disorder. Dr. Hack Tuke says that it is a rare disease, but that statement is not quite in harmony with my experience in the West Riding Asylum, in which we have always several examples of it. One of these examples, has been photographed as an illustration for this Paper by Mr. George Bracey, and fairly typifies its physiognomy. The vacant expression, open mouth, dribbling saliva, and fixed attitude, which is preserved by this patient constantly, are very characteristic. Perhaps the fault of the photograph is that the expression is not quite vacant enough for a case of acute dementia, in which there is generally some drooping of the eyelids, with a half-sleepy voidness and blurred stolidity of countenance.

Having sketched the indications of acute dementia, it may now be advisable to quote short summaries of one or two cases of it, as reported in the records of the West Riding Asylum, to illustrate its progress and the mode in which its symptoms are grouped.

Ambrose G—, æt. 16. Admitted to the West Riding Asylum on March 25, 1873, from Huddersfield. He was a healthy boy until three weeks before his admission to the Asylum, never having had any illness except some shaking fits when he was two months old, which, however, have never re-

curred since that time. When thirteen years old he was sent to the mill, and there he had continued to work till attacked by illness. He often complained that the work was too hard for him, and that the spinner did not do him justice, but imposed more than his fair share of labour upon him. Towards the end of 1872 it was remarked that he was idle and inattentive, and did not perform his work well, and in the beginning of 1873 his employer felt satisfied that there was something more in his negligence than wilful indolence. Not until the beginning of March, however, did his mother recognise the fact that he was insane. Then, however, he suddenly became passive and helpless. He would not move about, nor speak, nor feed nor dress himself, and had even to be lifted into and out of bed. He also began to squint with his right eye.

His father, who was not a drunkard, died at forty years of age of some acute disease. His mother is a sober healthy woman; and there has never been any instance of insanity in his family.

On admission he was in a dull heavy state, and could not be roused. He either returned no answer to questions addressed to him, or only replied after much urging and a long delay, and in a slow, drawling, unintelligent manner. He sat wherever he was placed, and could neither be startled into automatic, nor solicited into voluntary movements. He was very pale and thin, his face was red, his hands were cold and purple. Being 4 feet 6½ inches in height, he only weighed 80 lbs. His pulse was 84 and feeble, his heart and lungs were normal. There was pronounced convergent strabismus of the right eye. There was an inguinal hernia on the right side. He was inattentive to cleanliness. The treatment adopted consisted in a liberal dietary, enforced exercise, and citrate of iron and quinine. For a time no ground was gained. During April he was as lethargic as ever; streams of saliva flowed from his lips, and vomiting occurred occasionally. Throughout the summer, however, steady progress was made, and in the autumn, when he had gained weight and strength, he proved to be a lively, active, mischievous boy. On October 28 he went home quite well.

Ellen S—, æt. 23. Single. Spinner from Bradford. Admitted February 26, 1874. She was in good bodily and mental health until twelve months prior to her admission to the Asylum, when menstruation became irregular, and when she passed into a heavy, stupid state, varied by occasional outbursts of excitement in which she sung and shouted lustily. At that time she was placed in the workhouse, and there she remained for six weeks, being discharged so far recovered that she was able to resume her work and keep at it for about eight months, when she again became dull and apathetic. In six weeks more, having again displayed excitement, she was forwarded to the Asylum. Little could be ascertained regarding her previous history, but it was alleged that she had always been a sober, steady girl, that she inherited no tendency to insanity, and that no cause could be suggested for her illness, unless amenorrhœa, from which she had suffered for some time, had something to do with its origin.

When admitted she was quiet and lethargic, but was thrown into a state of great excitement by the accidental removal from her neck, during the process of bathing, of a little medal or charm which she wore there. She sobbed, and screamed out 'Mother of God!' with painful iteration, and in a

monotonous, mechanical kind of way. The excitement was manifested only through the vocal apparatus. There was no violence nor restlessness. On the day following her admission she was alternately excited and fatuous. Giving no heed to what was said to her, she lay on her back in bed gazing vacantly at the ceiling, silent and self-absorbed, or rolling her head on the pillow and shouting out with rhythmic regularity. When roused she seemed to understand what was said to her, and made a feeble effort to protrude the tongue when requested to do so, but she answered no question, volunteered no statement, and initiated no movement. She was inattentive to the calls of nature and would not feed herself, but swallowed whatever was placed in her mouth. But everything administered to her, although even in the smallest quantity, was instantly vomited. She was observed to be much emaciated, and it was noticed that her pupils were widely but equally dilated. Her head was hot and her face flushed. On examination her lungs were found to be healthy. The heart's impulse was very feeble and slightly below its normal position. The cardiac sounds also were feeble, especially at the base, where the first was scarcely audible. The bowels were confined, and the urine was acid and contained no albumen. Sinapisms applied to the epigastrium, small quantities of food and wine, and drachm doses of liquor bismuthi speedily arrested the vomiting, so that on February 28 she retained everything that was given to her. During the next few days she improved very decidedly; she employed herself in sewing and began to take an interest in what was going on around her, but on March 10 she again became stupid. From that date she sunk down steadily into a state of dementia, until on March 27 it was noted that she was quite fatuous. Her hands and feet were blue and swollen, her face was livid. She would not speak nor move about of her own accord, but remained in any position in which she was placed. The sensibility of the skin was greatly impaired. Large quantities of saliva flowed from her mouth and she vomited occasionally. Her pulse at that time averaged about 84 in the morning and 98 in the evening, and her temperature in the axilla ranged from 97.4° to 100.2°, being generally about 2° higher in the evening than in the morning. But while the temperature in the axilla remained near the normal standard, that of the extremities exhibited a striking reduction. A thermometer, of which the bulb was placed between the fingers, and carefully wrapped in cotton wool, being retained in that position for ten minutes, only registered 85°, whereas the same thermometer, similarly applied for a like duration to the hand of a healthy person, registered 97.4°. Up till the present time (June) Ellen S— has continued such as she was in March.

William A—, æt. 16. Joiner's apprentice from Huddersfield. Admitted to the West Riding Asylum January 23, 1871. As far as could be ascertained, none of his relations had ever suffered from insanity, epilepsy, or nervous disease. It was reported that he was the eldest of four children, and that his father and mother, both steady, sober people, and three sisters were alive and healthy. Two months prior to his removal to the asylum, to the surprise of those around him, who could conceive no cause for a change in his mental condition, he became restless and excited, burst out into unaccountable fits of laughter, swore in a profane and unaccustomed

manner, and talked nonsense. In the course of a few days the excitement was replaced by sullen stupidity. He would not go to work, would not answer questions, but sat day after day brooding idly by the fireside. When brought to the Asylum he was still sullen and stupid, and no information could be elicited from him. As a rule he took no notice whatever of what was said to him, and when roused by nudging and shouting would only mutter a few meaningless words and then subside again into silence. He sat staring fixedly at the wall opposite, taking no interest in what was going on around him, and never voluntarily changing his position. When put in any posture he remained in it, unless it were a peculiarly uncomfortable one, when he changed it for some other in which he was content to remain. He would not feed himself, and was dirty in his habits. Physically he was thin and poorly nourished, though when up and dressed he looked almost robust, owing to his florid complexion. His hands and feet were blue and cold, even when warmly covered up. His lungs were healthy, but his heart sounds were exceedingly feeble. Under treatment by quinine, of which ten grains were administered to him three times a day, he improved steadily, and manifested more animation. He fed himself, took abundant exercise, and was looked upon as almost convalescent, when at the end of October another attack of excitement came on, and was speedily followed by a state of acute dementia as profound as that through which he had previously passed, and similar to it in all respects. This lasted until the month of February following, when convalescence was again established, only however to be again interrupted in March by excitement preceding a return of the dementia. During this period of dementia electric treatment was tried, a continuous current from ten cells being sent through the head daily for a month. A little brightening up was the immediate result of this treatment, and after its discontinuance a progressive change for the better went on. The patient became sharp, intelligent, and active, and was discharged recovered on December 11, 1872, having been detained thus long in order to ensure freedom from relapse.

Catherine R—, æt 13. Single. Piecer from Huddersfield. Admitted to the West Riding Asylum April 2, 1873. It is stated that about three weeks ago this girl began to talk foolishly, but that in a day or two she recovered her senses and remained well for a fortnight, when she became stupid, and seemed puzzled when spoken to or asked to do anything. She could not execute her work in the mill, and forgot how to put on her clothes. No violence, excitement, nor evil propensity had been exhibited, but she had become silly and helpless, and so was sent to the Asylum for protection and cure. It appeared that her mother died of phthisis, and her father of some acute disease, that six brothers and sisters were alive and healthy, and no relative had ever been insane. Her uncle and aunt, with whom she had resided, could not think of any cause for her illness, but a feasible explanation of its origin was offered by the fact that she had been kept at work in the mill for twelve hours per diem, at a time when she was growing rapidly. Examined in the Asylum, the girl was found to be stupid and silent; she could answer questions, but did so after long deliberation and in the most childish manner. Left to herself she would remain lying continuously in one position, or would get out of bed in a purposeless fashion, and stand

irresolutely by the bedside. Sluggish in mind, she was equally so in movement, and maintained any posture in which she was placed. She could feed herself, but was not cleanly in her habits. She was stunted in growth and presented no indications of approaching puberty. Her bodily condition was that of debility, her pulse being feeble, her extremities cold, her skin and mucous membrane pale, and her pupils dilated. Her appetite was good, her bowels acted regularly, and her heart and lungs were normal. The diagnosis in her case was that she was suffering from a slight attack of acute dementia, and the prognosis was favourable. As to treatment, it was thought that for her fresh air was the best tonic, and that good food and rest were the most potent restoratives. Accordingly she was kept a good deal out of doors, and was put upon a generous dietary, with the happiest results. In a fortnight she was convalescent, and on June 20, having been fed into plumpness and high spirits, she was discharged recovered.

These cases surely exemplify a singular and distinctively marked disorder, and yet great doubts have been cast upon the very existence of acute dementia. M. Baillarger has endeavoured to show that what is called acute dementia is generally atonic melancholia, and that the torpor which the superficial observer translates into fatuity is really the prostration of an overmastering grief. It may be at once admitted that there are many points of resemblance between these two disorders, and that they are often difficult to discriminate, while at the same time it is maintained that they are essentially distinct. A little further on we shall inquire into their pathological dissimilarity, and here it may be well to examine into the differences which characterise their progress, and which ought to help us to identify each of them. And at the very outset a difference may be detected in their mode of origin. Atonic melancholia frequently arises out of moral causes, such as grief and anxiety, and always comes on with depression of spirits, while acute dementia most often arises out of physical causes, such as starvation, or concussion, and comes on either with failure of intellect, or with a paroxysm of excitement. Then when the period of incursion is passed, and the disease is fully developed, numerous differences may be recognised. Thus there is a difference in physiognomy. In atonic melancholia the expression is that of pain or suffering, the corrugators of the eyebrows being contracted, and the angles of the mouth drawn down, while in acute dementia the facial

expression is suggestive of emotional indifference and intellectual stupidity. The general sensibility of the surface is slightly, if at all, impaired in atonic melancholia, but is greatly blunted in acute dementia. In the former the quasi-cataleptic condition, which is common to both, is much more decidedly present than in the latter. In atonic melancholia a confession of mental misery can sometimes be elicited, or, at any rate, speech, when indulged in, is coherent, but in acute dementia, on the other hand, no feeling of one kind or another is acknowledged, and speech, if made use of, is incoherent and fragmentary, and does not convey rational thought. In atonic melancholia, again, there is mostly some attention to the calls of nature, while in acute dementia the habits are generally very dirty and degraded. In atonic melancholia there is no obtrusive derangement of the digestive or secretory systems, but in acute dementia there is often obstinate vomiting and profuse salivation. Coming to the close of these two disorders, features of difference still meet us, for atonic melancholia sometimes terminates abruptly in recovery, the clouds of depression dispersing altogether in an hour or a day, while acute dementia wanes slowly, and only gradually and step by step withdraws itself from view. And even after health has been completely restored, another point of differentiation may be discovered, for after atonic melancholia there is a tolerably clear recollection of what transpired during its continuance, while after acute dementia no reminiscence, except a sense of vacuity and helplessness, remains.

What is the state of the brain in this singular disease? A clue to the correct answer to that question may, perhaps, I think, be found in the state of the hands. As we have seen, they are, in well-marked cases, blue and livid and cold. Their functions are more or less in abeyance, their muscles are weak, their sensibility is blunted. Their vessels, and more especially their capillaries and veins, are dilated and relaxed, and the circulation through them is languid. They are in fact affected by diffused chilblains. Now may it not be, to use a crude comparison, that acute dementia is dependent upon cerebral chilblains? May it not be that a

condition of atony of the intra-cranial vessels is the true explanation of all its symptoms? I think many facts may be enumerated which sanction that view. As we have already seen, acute dementia is particularly likely to occur when a state of general atony pervades the system. In most adynamic conditions its germs may be recognised. Then all those circumstances which tend to induce acute dementia also tend to weaken the tone of the vessels. And again, *post-mortem* examinations in those cases which have ended fatally have revealed that species of cerebral congestion which results from vascular weakness, with the consequences which such congestion entails. Dr. Scipion Pinel found in those cases which he examined that the dura mater and arachnoid were natural, although raised up by subjacent serosity, but that the pia mater was thickened, varicose, and infiltrated with fluid, while the grey and white matter of the brain was tumid, spongy, and distinctly œdematous. In two cases that have fallen under my observation, the morbid appearances were akin to those described by Pinel. In the first case, that of a girl, aged 23, who sank in the first stage of a partial pneumonia of one lung, and who was comatose for two days before death, the skull, dura mater, and arachnoid were quite normal, but the pia mater was very thick and enormously engorged with blood. It stripped readily from the surface of the gyri, which were not at all wasted, and displayed a pinkish purple tinge in the cineritious substance, penetrating to about half its depth. In the medullary substance the puncta sanguinæ were exceedingly numerous, and the ventricles contained a considerable quantity of serum. In the second case, also that of a girl, Mary M —, aged 19, who died of phthisis pulmonalis, supervening upon acute dementia, the dura mater was normal, but the arachnoid was slightly thickened and opaque in the upper parietal region. The pia mater was thick, and its vessels were large and tortuous, and full of dark blood, while a group of convolutions, the ascending frontal and parietal, and the postero-parietal lobules were to some extent wasted and waterlogged. The grey matter was pale, and the only other noteworthy morbid change was a remarkable hyper-

trophy of the pineal gland, which was as large as a horse-bean and of firm consistence. Now in both these cases there were evidences of atony and dilatation of the veins of the pia mater, with resulting passive congestion and phlebecstasy. In the latter, in which the disease had lasted for a much longer period than in the former, there were also signs of effusion of serous fluid in the meshes of the pia mater, and of some atrophy of a certain number of gyri, due doubtless to pressure or to defective nutrition. It is a significant fact that the œdema of the convolutions described by Pinel was in a considerable majority of cases localised in exactly the same region as that involved by the wasting in Mary M——, a region concerned in the movements of the limbs.

An atony and dilatation of the veins of the pia mater, such as that found in the cases referred to, fully accounts for the symptoms of acute dementia, and is in keeping with all that we know of its etiology. As has been already said, the conditions which most frequently lead up to acute dementia—starvation, exhausting employments, or debilitating diseases—are just those which are likely to rob the vessels of their tone, and those other conditions which more rarely conduce to it may also operate in the same manner. For it is easy to understand how in cases of intense emotional excitement, or of excessive mental application, an active functional hyperæmia of the cortical substance may pass over into venous stasis in the pia mater. An active hyperæmia culminates in over-distension of the capillaries, which within the cranium are thin, yielding, and delicate, the increased quantity of blood becomes itself an obstacle to the circulation, that leads to stasis with still greater distension, and then atony follows. The small vessels which have been overstretched cannot at once resume their former calibre, but do so only imperfectly and by degrees. And these vessels of course yield most, and recover themselves most slowly, which are least liberally endowed with tonic fibres, and hence it is in the capillaries and veins that atony and dilatation are chiefly situated.

This state of atony and increased calibre of the capillaries and veins being once established, either by constitutional

weakness or nervous exhaustion, certain effects inevitably follow. Thus there is retardation of the blood current, and diminished nutrient activity of the nerve tissues, owing to this retardation, as well as to an alteration in the relative proportions of venous and arterial blood present in the cerebrum. The activity of the interchange between the blood and the tissues is diminished. The removal of the products of combustion from the brain is retarded, and hence the functional activity of the brain and nervous tissue is interfered with. Instead of vivid ideation there is confusion of thought; instead of acute feeling there is inanimacy; instead of energy there is torpor. And then the whole body sympathises with the enfeebled brain. Every function is imperfectly performed, as the result of which, the atonic cerebral congestion is much aggravated. For as no exercise is taken, the flow of blood through the veins is deprived of that important accelerating influence which is derived from the alternate contraction and relaxation of muscles; as the breathing is shallow, the suction force of the inspiratory act is reduced; and as the heart is irritable and feeble, the propulsion of the blood through the whole circuit of vessels is less vigorous than it ought to be. The distension of the atonic vessels is thus carried a step further, and then effusion of serous fluid takes place. Whenever a want of tone in the vessels exists, œdema occurs, and that may be as decidedly present in the meshes of the pia mater, as in the areolar tissue of a limb. But œdema cannot exist in the pia mater without compression of the brain, and compression, if long continued, entails atrophy. And so it is that acute merges into chronic dementia, unless the pathological chain of events just enumerated is broken somewhere. Generally the chain snaps of itself before atrophy is reached. Often it may be broken by treatment long anterior to atrophy.

If it is asked why in these cases of acute dementia the venous congestion affects the brain, it may be replied that in atonic states of the system venous congestion is always most liable to occur in those parts where there is any special cause of weakness. It affects the hands and feet, because in them it is favoured by gravity, and by the exposure

of these parts to cold, and so it affects the brain, because there it is favoured by the tenuity of the vessels, and by the exposure of that part to sudden and considerable vascular changes.

It seems probable that in acute dementia the venous congestion affects the whole encephalon, although, perhaps, it is present to a greater degree over the frontal and parietal lobes. Its effects in wasting of the gyri and in hollowing out of the sulci into troughs are mostly confined to the upper parietal regions, where several large veins, which collect the blood from extensive districts of the hemispheres, converge to the superior longitudinal sinus, and where concentrated pressure may therefore be exerted. In acute melancholia, on the other hand, it seems probable that the venous congestion is milder in character and is limited to a certain vascular area. Ferrier's researches suggest that that vascular area corresponds with the occipital and parietal lobes. Destruction of the occipital lobes made monkeys melancholic, and it is reasonable to infer that passive hyperæmia of a certain amount, with impaired nutrition, might bring on an analogous frame of mind in human beings. Extension of that passive hyperæmia to the parietal region would account for the absence of spontaneity with reference to all the movements represented there.

Remedial measures of a moral nature are scarcely applicable in acute dementia, at least not in its earlier stages. Barriers then exist in the diminished sensibility, feeble attention, and barren imagination of the patient, which prevent stirring impressions from reaching their mark. Only when some progress has been made towards recovery, can amusement and occupation be employed as exhilarating influences. An asylum, of course, affords the best refuge for a patient attacked by this disorder, but its advantages are connected rather with its hygienic arrangements than with its system of moral treatment. It ensures adequate nourishment, regular exercise, personal cleanliness, and incessant supervision, and these avail more than pictures, concerts, and balls to expedite the passage through the

depths of primary fatuity. At the proper time, of course, pleasing diversions, and more notably some simple employment, may greatly invigorate the enfeebled mental faculties; but that time does not arrive until the depths have been traversed, and the ascent towards the level of health has been begun. In a case of acute dementia, sketched by Dr. King Chambers with his accustomed classic grace, a slowness of the nervous and intellectual power, which remained after great improvement had taken place under a course of Griffith's mixture, disappeared rapidly when occupation was provided. Some accident set the girl to work at crochet. 'She was encouraged to proceed, and to our surprise the manual occupation, monotonous and mechanical as it seems to us, acted like magic; in a few days she became cheerful, laughed and talked to the patients, and turned out a brisk girl of more than average intelligence. Doubtless the way was prepared, by the action of the iron in curing the anæmia, for a very slight extra restorative agent to enable her to regain her mental powers. Still I cannot but attribute very considerable influence to the habitual occupation, associated as it was with memories of her former happier life, taking her thoughts away from the dark, noisy back kitchen of a small London shop to the old merry days in county Cork.'

I have said that an asylum is the best place for a case of acute dementia; but I do not wish to imply thereby that such a case cannot be successfully treated in a private home. On the contrary, I have seen such a case brought to a most satisfactory issue in a private home. Wealth can command even in an ordinary dwelling most of the advantages which an asylum offers, but where limited means impose restrictions, and negative the adoption of costly appliances, a special hospital alone affords the certainty of suitable treatment. The very basis of all treatment—due nourishment—is sometimes only possible when the resources of a special hospital are available. The apathy of the patient leads to abstinence from food. What is placed in the mouth is neither masticated nor swallowed, and the œsophagial tube may have to be passed three or four times a day, in order to introduce

sufficient aliment into the system. Compulsory feeding has sometimes to be carried on for prolonged periods. One girl in the West Riding Asylum was forcibly fed for three months, never during that period having voluntarily partaken of food. She certainly owed her life to the stomach-pump, and is now a flourishing 'help' in the United States, and a goodly monument of its saving powers. Whenever forcible feeding has to be continued for any length of time, it is well to supplement it by nourishing injections. Our rule is to feed at least three times a day, and to administer two injections, composed of beef-tea, butter, and port wine, during the night. Feeding requires the presence of a medical officer, but injections may be trusted to attendants and nurses, and may therefore be serviceable in keeping up nutrition at those hours when constant medical attendance cannot be so conveniently supplied. But whatever arrangements for the administration of nourishment are adopted, the practical point is to see that abundance is supplied. Generous living is essential in acute dementia; it tends by enriching the blood to sustain the vital properties of all the tissues, and so to restore tonicity to the enfeebled vessels. Any deficiency of food greatly augments the risk of pneumonia and of phthisis which is always present in this disease.

Moderate exercise in the open air has most bracing effects in acute dementia, but in order to secure benefit from it, we must see that our patients are not merely turned out into a garden or airing court with an admonition to walk about. If left to themselves they speedily come to a standstill, and incur danger by being exposed to cold. They must therefore be given in charge of companions, who will urge them to continuous effort. I saw recently a rather curious illustration of the way in which union may be made into strength sufficient to ensure to two acute dements their daily quantum of walking exercise. There were in one ward in this Asylum two lads who were both suffering from acute dementia, and who were so sluggish that when taken out and set in motion they walked on for only a few yards, with a gradual loss of momentum, and then stood stock still until a *vis-a-tergo* again propelled them onward. By a happy in-

tuition it occurred to an attendant to link these two sluggish beings arm-in-arm, and to start them together, the result of which expedient was that the pair, instead of slowing and stopping in a few paces, as each did when started singly, went on steadily perambulating a circular path upon which their course lay.

Cold, which is so hazardous to acute dements when they are for any length of time exposed to it, may conduce to their recovery when it is properly employed. Suddenly and momentarily applied to the skin it is an effectual tonic to the vessels, and thus the shower or plunge bath, or free sponging with cold salt water, may shorten the duration of the disease which we have been considering. I give a decided preference to the shower-bath, as the best method of applying cold in this disease. It administers a healthy mental as well as a cutaneous shock, and is sometimes a powerful emmenagogue, and I am sure I have seen patients roused promptly out of their lethargy by a short series of shower-baths. As a tonic a shower-bath should not exceed ten seconds in duration, and should be followed by brisk friction.

But a still more efficacious tonic than cold is to be found in electricity, which when applied to the brain appears to act specifically in rousing its dormant energies. Dr. Clifford Allbutt's experiments upon acute dements, conducted in the West Riding Asylum in 1872, gave most striking results. The continuous current from five to twenty cells, sent through the brain, produced unmistakeable and almost immediate improvement in several cases at that time, and subsequent experience has amply confirmed the favourable impression as to its usefulness under such circumstances which Dr. Allbutt was then enabled to form. Patients suffering from acute dementia are not at all sensitive to the current, bearing it unshrinkingly when of a strength that would cause great uneasiness to other patients. They manifest, however, very definite effects from its application. Sometimes they become flushed and giddy, generally they undergo a rise of temperature, the blue coldness of the extremities being mitigated, and invariably the cataleptic tendency is lessened. I notice that

Santopadre has lately employed electricity with success in restoring tone to the vessels in cases of chilblains, which as I have hinted are in some degree analogous to cases of acute dementia. He makes use of Gaiffé's electro-magnetic apparatus, and of a current of middling intensity. The positive pole is placed in the neighbourhood of the inflamed spot, and the negative pole on the inflamed spot itself. The sitting lasts about ten or fifteen minutes, and is repeated if necessary the following day. Generally after the first sitting the itching ceases and the pain is much abated. After the third or fourth sitting recovery is complete. Faradism of the head has not yet been used in acute dementia, but it certainly merits a trial. I look to electro-therapy for a method of treating this disease, more speedy and decisive than any that has been hitherto pursued.

Of all drugs that I have tested in the treatment of acute dementia, quinine in large doses has proved most useful. Cases that have been dragging slowly along have been much expedited in their progress towards recovery when ten or fifteen grains of quinine were given twice or three times a day. I have never known any unpleasant complications to arise while quinine was being taken. It seems to invigorate the atonic vessels. Dr. Angelo Monteverdi has shown that it exerts a special action upon the grand sympathetic, whereby it causes contraction of the muscular fibres of the uterus, urinary bladder, intestinal canal, and blood vessels, and it is probably by virtue of this action that it beneficially modifies acute dementia. Guaiacum has likewise proved exceedingly useful in the treatment of acute dementia in my practice. It also gives tone to the vessels.

Should anæmia appear to be the condition most urgently demanding treatment, it may be advisable to prescribe iron at once. The sulphate and the per-nitrate are the preparations which are I believe most serviceable. If there is any tendency to diarrhœa, the latter is, by its astringent property, particularly indicated. When great prostration occurs the mineral acids do good, and during a tardy convalescence a course of tincture of capsicum is beneficial.

Not much need be said here as to the modes of dealing

with the complications of acute dementia, as these must all be managed according to general principles. The vomiting which sometimes occurs is nervous in its origin, and is not to be treated by spare diet, by milk and soup, but by solid food and wine. Should these fail to arrest it, bismuth or nitrate of silver may be prescribed. Bed-sores, or chair-sores, ought to be prevented by scrupulous attention to cleanliness, and by frequent changes of position, but should they unfortunately form they will be induced to heal rapidly by alternate poultices of pounded ice and warm linseed meal, applied in the manner recommended by Dr. Brown-Séguard, or by the sprinkling of iodoform over them.

OPHTHALMOSCOPIC OBSERVATIONS IN ACUTE DEMENTIA.

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IN my former contributions to the 'West Riding Asylum Medical Reports' I have alluded to the great assistance we have obtained in the diagnosis of cerebral diseases by the employment of the ophthalmoscope, and have urged at the same time its more extended use, not only as an aid to diagnosis, but as a means of obtaining a knowledge of the action of drugs upon the intra-cranial circulation, and also its value as a means of determining when death has taken place.

It is, therefore, gratifying to find that in the most recently published work upon nervous diseases, viz., that of Dr. Hammond of New York, the ophthalmoscope takes precedence of all other instruments as being most important to the physician who makes nervous diseases his special study. Finding this set forth in the introductory chapter, it is no matter of surprise to see that the appearances of the optic disc and retina are given under the heading of the different diseases treated of in the book, insanity excepted. When the value of the ophthalmoscope is duly recognised in a systematic work like Hammond's, a large increase in the numbers who employ the instrument may be expected, so that we shall no longer be able to say, with Dr. Allbutt, that the

number of physicians who are in the habit of using the ophthalmoscope can be counted on the fingers of one hand.

In former Papers I have dealt with the ocular conditions observed in two well-known forms of insanity, viz., general paralysis and epileptic insanity. In these instances I have had the advantage of a large number of cases, as the diseases are very common, but in the disease which forms the subject of this present essay so large an induction has not been possible. Acute dementia, although by no means a rare affection, does not occur nearly so frequently as many other forms of mental disease. At least the well-marked cases do not. For an inquiry such as the present I take it that only well-marked cases should be selected, for in slight cases the well-marked condition might not be observed, and any little variation from normality might not be noticed, or if noticed might be passed over as of little moment. If on the contrary we find a well-marked state of things at the back of the eye in very bad cases of the disease, we are better able to appreciate slight variations from the normal condition in milder cases. During the last four years I have made ophthalmoscopic examinations in almost every well-marked case that has occurred in the West Riding Asylum. Acute dementia is a most interesting disease, and one the pathology of which is but little understood. This no doubt arises from the fact that it is a very curable disease, but seldom terminating in death, and when recovery does not take place a chronic condition supervenes, so that death taking place after the chronic state has continued for a lengthened period, any pathological observations can throw but little light upon the state of the brain during the acute period of the disease. With the hope that the ophthalmoscope might be of some use in elucidating the morbid processes going on within the cranium in the various stages of well-marked cases of this disease, I have put together the cases at the end of this Paper, and shall in the following remarks try to draw some conclusions from them.

It is not my purpose to give a full and minutely faithful account of the etiology and symptomatology of this disease, but only of one set of symptoms, viz., those that can be

observed at the back of the eye with the ophthalmoscope. It will, however, be convenient to give a short sketch of the most prominent symptoms, in order that a correct idea may be formed of the general state of the patient at the time when a certain condition of the fundus oculi is observed, and that the mutual bearings of the states indicated may be considered.

In the first instance then we shall generally find that our patients, who are always young, have for some time been in failing health. They will perhaps have suffered from headaches, neuralgia of the fifth, or perhaps in childhood have been the subjects of infantile convulsions. If the patient is of the male sex we shall very frequently find that he has been in the habit of practising masturbation; if a female some irregularity of the menstrual function, generally amenorrhœa, has been noticed, and will certainly exist during the attack of dementia. The patients have for long been moody and sought solitude, either reading, or, it may be, not occupying themselves in any way. Forgetfulness of personal duties, of meals, also great inattention in the course of conversation, so that answers are given showing that the person has not been attending to what was said, seeming to be pre-occupied are common symptoms. This state of things, if observed by the relatives of the patient, is seldom attributed to disease, and afterwards, when the case has become fully developed, is put down as a cause rather than as an effect of disease. An attack of excitement of short duration, but often of some violence, after which the patient is seen to be stupid and bewildered, is generally the occasion of their being sent to an asylum. The disease proceeds, if not checked by treatment, and the state of slight dementia indicated by inattention, reserve, loss of memory for recent events, etc., etc., deepens. The patient will remain for hours in one position, it may be with his face to the wall, or perhaps in some very uncomfortable position. If spoken to he does not heed, and cannot be got to do anything that is required of him, unless he is put into the way. Thus, if he is requested to move from one side of the room to the other, he

requires to be led, or at least set going, and should any obstacle be in his way he will walk right over it, making no effort to avoid it. Or if food is to be taken he requires to have the spoon placed in his hand, and perhaps to have it guided up to his mouth once, when it may be that he will automatically carry the spoon to and from the plate to his mouth, until, all the food being removed, no sense of resistance is offered to the spoon. In this the higher centres residing in the cerebral hemispheres seem to take no part, and the patient seems to be in much the same state as a decapitated frog, who if irritated will make efforts to remove the cause of irritation. Only sensori-motor actions remain. A stage further on, and the patient no longer stands, nor can he be induced to take food, even in the mechanical manner he did at an earlier period. The food must now be placed well back in his mouth, so as to come under the influence of the pharynx, where reflex action still remains. He now loses power over the sphincters, so that both the bladder and the rectum discharge their contents without his knowledge; and at this stage it becomes our duty to look well to the bladder, lest it become distended, and the distension not be noticed owing to the overflow constantly dribbling away. But a further stage than this may be reached, when we find that food lodges in the pharynx, reflex action being almost extinct, and now we require to feed the patient by means of that most valuable instrument, the much maligned stomach-pump.

The general anæmia observed in the first instance has become profound; the coldness of the extremities which indicated the feebleness of the circulation has become slowly more marked. First there is a blueness of the hands, feet, and, it may be, of the ears and nose, which shortly are noticed to be almost black from venous stasis, and should the time of year be winter, or one of the colder months, chilblains will form. Indeed, the extremities seem to be in the same state as we find them in a hemiplegic limb, which through some severe lesion is cut off from almost all nervous influence. At this period there is but feeble if any reflex response to irritation, and except that we can feel a feeble pulse, and see a

faint heaving of the chest, we should hardly know that our patient lived, so much does his body seem to have fallen under physical laws. Having sunk so low, one of two events takes place: either our patient dies, or he improves very slowly, and the symptoms observed during his recovery are such as we have noted above, only in an inverted order. On reaching a stage on the road towards recovery at which he feeds himself, assists to dress himself, and pays some little attention to what passes around him, the disease may take a chronic form which will last for years, and has only one termination, viz., death. The happier event, however, may take place, so that as the patient's body piles on flesh, and regains the appearance of health, his mind slowly clears up, and he perfectly recovers. When restored, however, he finds a great hiatus in his memory, he has passed through the valley of the shadow of death, and the shadow clouds his mind; his brain seems to have laved itself in the waters of Lethe, and perfect oblivion, extending over a longer or shorter period, remains. Indeed, we could not expect any other result. All his acts for some time showed that only his spinal cord and sensory ganglia were in action. Impressions upon these ganglia never reached the cerebral ganglionic cells to be perceived, and by being registered become part of the memory. The machine was out of gear and no work was done.

Having thus briefly alluded to a few of the more prominent symptoms which strike the eye of the observer, I must now allude to a very analogous condition, and one which in many of its external features bears a close resemblance to the disease under consideration. I refer to atonic melancholia, melancholia atonita, or the *melancholie avec stupeur* of the French.

In this latter disease the patient passes through stages characterised by many of the symptoms detailed above. He requires to be fed by the stomach-pump, but generally at a much earlier period, for he will seldom eat food when it is put into his mouth. He is silent and given to remaining much in one position, and is seemingly inattentive. The expression of his countenance is, however, unlike that of acute

dementia; instead of having an absolutely blank and stolid expression of countenance, the face of our patient is more or less indicative of distress and mental suffering. He makes no noise, does not moan, or actively give expression of sorrow. His condition in this respect resembles one who, having suffered great loss, can find no vent in tears for the sorrow that fills his mind; 'his grief is too deep for tears.' The patient takes no heed of his body and its wants, but, as we are told by him on his recovery, it is from paralysis of will, and not from blunted perception. He feels that his motorial system is chained, and is no longer amenable to his will or to the influence of his emotions. On recovery there is no hiatus in his memory, no waters of oblivion have passed over his brain; on the contrary, he has passed through the fire, and although it has purified and cleared up his difficulties and doubts, has yet left an indelible mark behind. The differential diagnosis between cases of melancholia atonita and acute dementia is not always easy; it is true that the previous history of the case may give us some clue, but then various circumstances may have occurred which have tended to liken the symptoms, or at least to have obscured signs which would help to differentiate the two diseases. Can the ophthalmoscope help us here? I fear not, unless the stage of the disease is an advanced one, and then other and general symptoms will help us perhaps as much. By a careful perusal of the cases detailed at the end of this Paper, the reader will observe that in the early stages of both acute dementia and atonic melancholia the condition of the retina is similar, viz., that a state of retinal anæmia exists. The optic discs are pale, but it is not the brilliant pallor of atrophy, the tint of the choroid is lowered, and there is a want of that distinctness and sharpness in the picture which is such a marked feature of atrophy. Another distinguishing condition between this anæmic state of the disc and atrophy is to be found in the uniform greyish-white appearance of the disc, and the fact that one disc is in exactly the same condition as the other, and also that no partial anæmia of one optic disc can ever be seen. So that should there be vascularity of a portion of

the optic disc, and whiteness of the remainder, we may be pretty certain that we have not to deal with anæmia. In observing the retinal vessels we find that they are small and shrunken, but no trace of previous tortuosity exists, as we can so often see in atrophy. I have dwelt, perhaps, unnecessarily long upon this distinction between anæmia and atrophy, because the distinction is of such paramount importance, and at times presents great difficulty.

In melancholia atonita the anæmia of the disc is never complicated by œdema, as we find is the case in the more profound instances of acute dementia.

In several cases of the latter disease, where a very advanced stage had been attained, a certain amount of œdema of the disc, and of that portion of the retina which closely borders upon it, was seen, and it is curious and interesting to notice that this condition was only observable in those cases where the dementia was so profound that the patient required artificial feeding, where the general circulation was at a very low ebb, and where the reflex sensibility was greatly diminished. As the disease progresses towards recovery, it is interesting to observe that this œdema clears up and leaves no trace of its having been present. At the same time the anæmia decreases, and the normal state of vascularity is established.

We must now endeavour to connect these retinal appearances with concomitant intra-cranial conditions, in order that we may judge how far the morbid state of the retina may be regarded as an index of the cerebral changes.

From the mental torpor in these cases we are unable to ascertain how far the morbid state of the retina has impaired its function. But that function must be seriously interfered with we may judge, because we find that in cases of anæmia after hæmorrhage we get dimness of vision, black spots before the eyes, and at last it may be total blindness, which continues until vascularity is restored. The same thing occurs in certain cases of blindness coming on after delivery. If this anæmia of the retina is sufficient to disturb its function, we have a strong fact in favour of the inference that the suspension of the cerebral functions noticed in this disease

may have its origin in a like state of anæmia of the brain, more or less profound. And this supposition gains force from the fact that the disease is very rarely fatal, and that as it progresses towards recovery a restoration of vascularity is noticed in the disc and retina, and the œdema of these parts clears up.

In recalling the symptoms one cannot fail to be struck by the many points of resemblance that acute dementia has to catalepsy. For setting aside the physical resemblance, what do we find to be a prominent mental condition in catalepsy?

Hammond,¹ in the last edition of his work, speaking of catalepsy, says, ‘In none of these cases was there any knowledge of what passed during the paroxysms, and no consciousness of there having been any mental activity.’ In another place he says, ‘Consciousness was entirely abolished.’ And is not this very much the condition of the mind of a patient who is the subject of acute dementia? We ask, what is the condition of the retina during an attack of catalepsy? Here I must again quote Hammond, for from my own experience I am unable to speak. He says, ‘I likewise in one case repeatedly examined the fundus of the eye with the ophthalmoscope, and invariably found the choroids pale and the retinal vessels straight and attenuated.’ This description of the retina in catalepsy corresponds very closely with that seen in acute dementia, only it does not seem ever to be so profound, for no œdema has been noticed in catalepsy.

In what other morbid cerebral states do we get this condition of the retina or one approaching it?

In the first stage of the epileptic fit, as I have elsewhere shown,² a state of profound anæmia of the retina is found. In partial cerebral anæmia, either from embolism or thrombosis, a state of anæmia of the retina in one eye may be noticed, and in general cerebral anæmia we have the same condition in both eyes. Hammond says that in this latter disease, which he describes minutely, ‘the vessels of the

¹ Hammond’s ‘Diseases of the Nervous System,’ p. 593. New York, 1873.

² ‘West Riding Asylum Medical Reports,’ vol. i.

retina are seen to be small and straight, and the choroid is paler than normal.'

In all these diseases a greater or less degree of paralysis of the cerebral functions is traceable to one and the same cause, a deficient supply of blood to a part or the whole of the brain, and for a longer or shorter period.

If then in these diseases we find a retinal always co-existent with a cerebral anæmia, we have some grounds for supposing that when we find œdema of the optic nerve and retina we may have at the same time some œdema of the cerebral texture, in addition to a profound anæmia of it. A very interesting case bearing upon this point, and one which would have been more conclusive had a *post-mortem* examination been added, but the patient recovered, is quoted by McNamara, where there was paralysis of three limbs and total blindness, although but little impairment of mental power. Simple œdema of the disc was found, and from this fact McNamara conjectured that there might be a similar condition of the cerebral structures without any structural lesion. And as perfect health was restored under treatment by iodide of potassium, he considered that his supposition was justified. Mr. Fearnly, formerly of Leeds, now Principal of the Veterinary College, Edinburgh, a veterinary surgeon of great ability, who has for some years used the ophthalmoscope as an aid to the diagnosis of nervous affections in animals, has noticed a similar condition in the eyes of horses and cattle the subjects of temporary paralysis.

I think, then, that we may conclude that a very prominent condition in cases of acute dementia is profound anæmia of the brain often accompanied by œdema. This being the pathology, many of the symptoms are elucidated, and, to a certain extent, the line of treatment is defined. It does not of course follow, even if thus much of the pathology can be regarded as proven, that there is not some morbid condition beyond standing in a causative relation to the cerebral state. The sympathetic nervous system may have, and probably has, something to do with the causation, for its influence upon the cerebral circulation is too well known to need any exposition here. The little excitement which is noticed in the

early stages of acute dementia probably has its origin in some intermittent action of the vaso-motor nerves, permitting sudden rushes of blood into the previously half-filled vessels. For during these periods of excitement we find a relatively increased vascularity of the disc and retina, which only holds as long as the excitement lasts. The only *post-mortem* record that I have been able to obtain bears upon this point. A patient suffering from acute dementia died from pneumonia; during the few hours preceding death, there was more consciousness than there had been previously, and at the *post-mortem* we found an injected state of the superficial vessels of the brain and of the grey matter, but a very exsanguine condition of the bones of the skull. It is, of course, no uncommon thing for a considerable degree of enlightenment to occur in cases of chronic secondary dementia during the course of any acute disease, as pneumonia. And the lightening before death in such cases shows how anything that affects profoundly the whole system overrides the usual morbid state. One of the cases reported at the end of this Paper showed this tendency in a marked degree. The patient had been in a state of acute dementia, and had come round to a certain point, and then made no progress, so that the case had become to be regarded as chronic. During an attack of pneumonia she seemed to wake up, and delighted those about her by singing to them 'the old songs,' and conversing about events that had occurred years before. As her bodily health became restored she sank back into the old apathetic condition; her cold extremities, and the chilblains that appeared upon her blue hands and feet during summer weather, indicated the feeble condition of her circulation.

I must now conclude by appending the cases from which my deductions have been drawn; and although my observations may not have done much to elucidate the pathology of this interesting malady, I must rest satisfied with the hope that they may be of use to some other investigator in building up the true pathology of the disease.

CASE I.—M. I., æt. 18. Has suffered from acute dementia for some three months. She is now in a feeble state of bodily health, and is

anæmic in appearance. She takes food when it is placed in her mouth, but otherwise is perfectly passive. She will remain in one position for many hours, and seems to be quite oblivious to all that goes on around her. Sometimes she speaks in answer to an often-repeated question, but her answer consists on all occasions of 'Yes,' and seems to have no relation to the question. She is able to walk about, but requires to be led, and is inattentive to the calls of nature. Her hands and feet are very blue and cold, and have chilblains upon them, although the weather is warm. Her eyes are of normal prominence, the irides of a hazel-brown colour, and the pupils, which are sluggish, are also dilated but equal, each measuring six degrees of the pupilometer. Examination with the ophthalmoscope:—Right eye—optic disc is of a very pale greyish tint, there being only faint traces of vascularity about it. The arteries are very straight and attenuated, and the veins are rather under the normal size and not tortuous. The choroid is pale. There are no signs of œdema about the disc or retina. Left eye—the same description applies.

One year after the above date, when the patient had improved in many respects, her bodily condition being improved and her mind a little more active, but the case having assumed a chronic character, the eyes were again examined. The discs were still pale, although not having the grey-white tint observed at the former examination. The vessels were attenuated and straight, and the choroid still paler than natural.

Six months after the last report the patient had an attack of pneumonia, during which great improvement in her mental state was manifested. She spoke of events in her past life and asked after her friends; she also sang several of her old songs, but not with the same expression as of old. During this period I examined the eyes, and found a condition of restored vascularity in both discs, retinae, and choroids. When she had thoroughly recovered from the pneumonia, and was able to get about, she sank into the old state of inaction, and her extremities again fell a prey to chilblains, etc. I again examined the eyes, and found them in nearly the same condition as before the pneumonic attack.

CASE II.—H. G., æt. 20, female. When first admitted the patient was in a state of dementia varied by occasional outbreaks of excitement, which did not last for more than a few hours; during these attacks she manifested considerable eroticism. At this time the ophthalmoscope gave evidence of anæmia of the discs, retinae, and choroids, not, however, very profound, and there was no œdema. Three months after, during which time she had retrograded, and when a state of great dementia, with feeble circulation, blue extremities with chilblains, had become developed, a second examination of the eyes was made, and the following condition found in both eyes. Optic discs are of a grey-white tint, and are swollen, the retina near to them is also œdematous, so that a clear sharp picture cannot be obtained. The arteries are small, shrunken, and straight, the veins are not at all well filled, but are darker than usual, and not tortuous. Pupils dilated and very sluggish in action. This patient's general health improved, and her mental condition also, but only up to a certain point, when an attack of mania was initiated, characterised by erotic propensities and filthy language. During this attack the eyes were again examined, when the œdema was seen to be

gone, and the anæmia had given place to a normal state of vascularity. No hyperæmic or atrophic changes were noticed.

CASE III.—L. J. R., æt. 17, female. Patient rapidly sank into a state of the most profound dementia I ever witnessed. For three months she required to be fed by the stomach-pump, reflex action being so feeble that the food placed in the back of the mouth remained there until symptoms of suffocation required its removal. The body became a prey to extensive bed-sores, and chilblains. Reflex action was almost nil.

At this period the eyes were examined. The pupils were found to be dilated and inactive. Discs in both eyes were grey-white and much swollen, the retina near them being œdematous. The arteries were very fine and not tortuous, and the veins dark in colour but not tortuous. The choroid was pale and the whole picture indistinct.

Three months after the above report, the patient had so far recovered as to be able to feed herself and answer questions, but was not able to originate a conversation. Her bodily health had improved, all sores had healed, and the circulation was more active. On examining the eyes, the pupils were still dilated, but they acted to light, although sluggishly. The optic discs had undergone considerable change, the œdema had disappeared, but there was still a white appearance. The arteries were small and straight. Veins much as before, although not quite so dark.

Nine months from the commencement of the attack, and three months after the above report, the patient is almost perfectly restored and is to be discharged. Her eyes now present an active pupil of normal size, and the ophthalmoscope reveals a clear transparent optic disc of a medium capillary tint. The arteries are fuller and not so straight in their course.

CASE IV.—E. N., æt. 25, female. First examination made when the dementia was at its deepest, when the patient was being fed by the stomach-pump, and when blue extremities with chilblains on the fingers and toes existed. The pupils were dilated and were almost insensitive to light. The ophthalmoscope showed optic discs of a grey-white tint and somewhat cloudy, as was also the retina close to the discs. The arteries were very small and straight in their course and the veins full of dark blood; choroids pale, and the whole picture wanting in clearness and definition. Six months afterwards, when the patient's mental state had become considerably restored, but when she had become the subject of softening tubercle, her eyes were again examined. The pupils were moderately dilated, but sensitive to light. The optic discs were clear and transparent, but no great vascularity existed. The arteries were of a normal size and not so straight in their course as had been noticed before. The choroids were still rather pale. The whole picture was however clear and well lighted up, and no signs of œdema existed.

CASE V.—A. I., æt. 20, female. Was in the same advanced stage of dementia as the last case when the first examination was made. The pupils were widely dilated and inactive to light. The optic discs were both pale grey and ill defined, being swollen, and the same dim appearance of the retina existed. The arteries were far straighter in their course and shrunken. Veins dark in colour, not tortuous.

Several examinations of the fundus oculi were made in this case during

the next six months, at the end of which time she was discharged perfectly well. I have not space to give the details of each examination, and must therefore content myself with a brief account. First the œdema cleared up and left the picture clear, then vascularity became slowly restored. The arteries increased in size, and small ones, which had not before been noticed, appeared. The blood in the veins was not so dark, and no signs of atrophic change could be detected. The pupils became smaller and more sensitive to light. On her discharge no sign of anything abnormal could be detected in the discs or retina.

CASE VI.—E. L., æt. 30. The first examination was made soon after her admission, when the disease was not quite so advanced as it afterwards became, but when she required to be fed by means of a spoon and had very blue and cold extremities.

The pupils were dilated and sluggish even in a bright light. The ophthalmoscope showed greyish-white discs with but a very faint capillary pink tint. The retina was transparent and the choroid pale. Arteries not numerous. Veins of normal aspect.

A month later the disease had advanced slightly, so that the patient required to be fed by the stomach-pump, and chilblains had formed on the hands. The pupils were dilated and more sluggish in action than before, and now there was some cloudiness of the optic discs and retinæ near them. The arteries were smaller than before and very straight in their course.

This patient recovered, and upon her discharge another examination was made, when the condition of the fundus was quite normal, and no signs of atrophic changes were to be seen.

CASE VII.—M. H., æt. 30, female. When the eyes of this patient were examined she had advanced to the most severe stage of the disease, and so low were her vital powers that she suffered from dry gangrene, which deprived her of several toes. The pupils were dilated and sluggish in action, and there was profound anæmia of the optic disc and retinæ with œdema, giving a veiled cloudy appearance to the picture. The arteries were very much shrunken, and the veins were fairly filled with black blood but were not tortuous. During the progress of this case towards recovery I made a few examinations of the fundus oculi, and had the pleasure of seeing the œdema clear up, so that each time I looked at the eye I obtained a clearer picture than before, until at the last examination, just previous to her discharge, no sign of previous mischief remained save a little paleness of the choroids.

CASE VIII.—M. S., æt. 21. In this case the dementia never reached the same stage of severity as in the preceding case. The patient kept up a fairly good bodily condition, although some coldness of the extremities could be observed, yet no chilblains existed. The pupils had a tendency to be somewhat dilated, but they acted fairly well to light. The fundus oculi in both eyes presented a state of anæmia, the discs were of a grey-white, and the arteries were smaller than natural. Veins of normal appearance. No œdema of the discs or retina.

The patient did not improve, and the case assumed a chronic condition. Several examinations of the eyes were made, but no change observed beyond a slight restoration of vascularity in the disc. This patient developed tubercular symptoms.

CASE IX.—H. H., æt. 23, male. A milder case than many of the others. Required to be fed by the pump, but did not get chilblains or hed-sores. Was inattentive to the calls of nature and had some cataleptic symptoms. A masturbator. Pupils dilated, but acted to a strong light. Optic discs grey-white, with small arteries and a scanty number. No œdema. This case recovered to a very considerable extent, but again relapsed, and finally ended in a chronic condition. The optic discs assumed a normal appearance, but again became pale on the return of the symptoms. No œdema ever observed and no signs of atrophy.

CASE X.—T. H., æt. 20, male. Masturbator. A very similar case to the above, only ending in recovery. The ophthalmoscopic signs were those of anæmia, grey-white discs and small arteries with pale choroids, but no œdema. On restoration to health the normal condition of the fundus was observed.

CASE XI.—R. G., æt. 16, female. This case did not advance very far, never requiring the stomach-pump; indeed, she was only in the Asylum for a few days when she got a double pneumonia, which resulted in her early death. No ophthalmoscopic examination was made in this case, but at the *post-mortem* some engorgement of the brain was found, resulting no doubt from the great amount of lung mischief.

CASE XII.—E. T., æt. 16, female. A mild case of acute dementia, which ran a short course and ended in recovery. The ophthalmoscope showed the same anæmic state of the retina and optic discs, and on recovery this was found to have completely cleared up. No œdema of the fundus was observed in this case.

CASE XIII.—A. S., æt. 17. A case of melancholia atonita. Early on in the disease the eyes were examined, and the optic discs found to be pale but having still some capillary tint. No œdema noticed. Several times the eyes were examined, but no advance in the anæmia took place, and as she recovered a deeper tint of the disc was observed.

CASE XIV.—B. W., æt. 30. Also a case of melancholia atonita. Was observed soon after admission, and the same amount of anæmia observed as in the last case. At subsequent examinations no advance to the grey-white condition was observed; but on recovery, which was rather sudden in this case, a restored state of vascularity was observed.

CASE XV.—E. S., æt. 19, admitted March 9, 1874. On admission she is said to have been more or less excited, but from time to time became stupid and lethargic. At the time of the present observation (April 5, 1874) she is in a state of acute dementia; her circulation is very feeble, the hands and feet being blue and cold. It is with difficulty that an ophthalmoscopic examination can be made on account of her stupidity and the tendency to keep the eyes shut. In the right eye the optic disc is seen to be of a greyish-white tint. The arteries are fine and thready, and the veins if anything smaller than natural. There is no œdema or cloudiness of the retina. Left eye—the disc has a faint tint of pinkness, otherwise is as the right.

CASE XVI.—T. C., æt. 26. Admitted April 9, 1873. An extreme case of acute dementia, which is at the time of examination somewhat improved, although she has cold and blue hands and feet. Right eye—optic disc is very pale but has some capillary tint, the arteries are thin, straight, and thready. Veins of medium size. In the left eye the condition is almost identical with that of the right.

THE ACTIONS OF NICOTINE.

BY WILLIAM T. BENHAM, M.D. Abdn.

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MUCH has been already written on the effects, immediate and remote, of tobacco upon the animal economy. There is, however, room for further investigation, and it is at least open to question whether many of the alleged effects of this seductive drug are not in some degree chimerical.

So difficult is it to bring an unbiassed mind to the consideration of this subject, that we are tempted, involuntarily perhaps, to attach more importance to certain facts seeming to point in the direction we wish than such properly deserve. Facts, however, are sometimes so self-evident as to what is indicated by them that it is impossible to misconstrue their meaning.

I propose to put on record some actions which I have observed in the course of the experiments I have made with nicotine; which, although they do not altogether accord with those stated to have been observed by former writers on this subject, are nevertheless worthy of being noted. The result of my experiments on the action of nicotine when topically applied to the external surface of the heart, or to its internal cavities, certainly indicates that, instead of that organ being arrested in its action by nicotine, it beats more vigorously and for a longer period after the cessation of all other functions in the animal experimented upon, than in others not subjected to the influence of nicotine. And I have

also found, in animals poisoned with this drug, the heart beating more vigorously after the cessation of the respiratory function, and continuing to beat for from thirty to ninety minutes. These facts are quite opposed to the statement made by a former writer of some repute on this subject, when he says, 'In the lower animals the death is invariably due to the arrest of the beating of the heart, and in the human subject, if fatal symptoms supervene, they would be due to the same cause.'¹ My experiments negative this statement in so far as the lower animals are concerned, for in nearly all of them the heart was found to be beating long after respiration had ceased. But, on the other hand, in no case did I observe, nor was I able to produce by simple mechanical irritation, any motion of the muscles of respiration.

It is impossible in a Paper of moderate length to consider with any amount of completeness more than a very few of the effects of nicotine. I shall, therefore, at present, only put on record some of the experiments referred to above, with some others made on the human subject, in which doses of nicotine, from the $\frac{1}{50}$ th to the $\frac{1}{20}$ th of a minim, were administered internally, and also some observations made on the effect of this drug on the iris, when topically applied to the eyeball, as well as when given internally by the mouth or by subcutaneous injection.

And that by experimenting upon the lower animals we can as a rule—reasoning by analogy—predicate to some extent what the action of the same drug will be upon the human animal economy is, I think, evident. But experience teaches us that this is not invariably the case, and, indeed, when some drugs are administered to a human being, phenomena result quite different in character from those observed in the lower animals. It follows, therefore, that all experiments that can be made on the human subject without undue risk should be performed whenever possible, as being of more value in the results obtained than those observed by any other method. But we must bear in mind that in the human subject we cannot push the experiments so far as we often

¹ 'For and against Tobacco,' by Benjamin Ward Richardson, M.D. London, J. & A. Churchill, 1865.'

wish to do, and that, therefore, when we desire to observe the effect of poisonous doses, we have no other alternative than to do so in the lower animals, unless, indeed, we should happen to witness a case of accidental poisoning by the very drug whose effects we are anxious to note. We can, however, easily observe the effects of small and non-poisonous doses, and these must always prove of the greatest importance in estimating the true therapeutical value of the drug or drugs under observation.

I am quite sure the effects of the alkaloid nicotine are not the same in all subjects: and that whilst in some the administration of the $\frac{1}{30}$ th of a minim will decrease the number of the beats of the pulse, in others the effects will be just the reverse, and I have with the sphygmograph obtained tracings totally opposite in character from different individuals under the influence of the same dose of nicotine. Smoking will in some persons quicken the heart's action without, as shown by the sphygmograph, lessening the vigour of the heart's impulse, whilst in others undoubtedly the reverse takes place.

The preparation of nicotine which I have used in these experiments was obtained from Messrs. Davy, Yates and Routledge, Southwark, London, S.E., and certainly has given me every satisfaction. They inform me that it is procured from the tobacco plant in the following manner.

The leaves are infused in water acidulated with sulphuric acid, the infusion is evaporated until it is of a syrupy consistence, a strong solution of potash is then added, and the mixture is distilled in an oil bath of 280° F. The distilled product is saturated with oxalic acid and evaporated to dryness. It is then digested in boiling absolute alcohol, and the oxalate of nicotine thus obtained is decomposed by the addition of potash in a closed vessel, and agitated repeatedly with ether until the whole of the nicotine is dissolved out. The ethereal solution is then distilled over an oil bath, the nicotine coming over after the ether and water. So powerful is the sample that I have been using, that one minim of it in a gallon of water imparts to it an acrid bitter taste, which is quite perceptible to anyone

moistening their tongue with a few drops of this very weak solution.

Action of nicotine upon the heart.—When administered internally, by the mouth or by subcutaneous injection, in a poisonous dose, the effect produced on the animal in my experiments was a marked affection of the respiratory function, the inspirations becoming hurried and shallow, quickly getting less and less frequent, until with one last forced gasp the respiratory act finally ceased altogether. On immediately opening the thorax the heart was invariably found pulsating regularly and powerfully, which it continued to do for many minutes after, and if care was taken to keep up the temperature and a proper degree of moisture, the pulsations of the right auricle might be observed for hours. The following experiments will speak for themselves.

EXPERIMENT I.—Five minims of pure nicotine were injected beneath the skin of the back of a rabbit. In fifty seconds the respiratory function had completely ceased. On the thorax being opened the heart was found pulsating vigorously. The pulsations of the left side ceased in eighteen minutes, and of the right side in thirty-three minutes.

EXPERIMENT II.—One minim of nicotine was injected beneath the skin of the back of a rabbit. The respirations gradually became more and more affected, until at the end of twenty minutes they ceased altogether. On the chest being opened the heart was found pulsating rapidly and with considerable force. In ten minutes one minim of pure nicotine was painted over the heart's surface. The pulsations were at once rendered more perceptible. The body was now covered with a moist cloth and placed in a warm place near the fire. In thirty minutes the ventricles were still pulsating regularly. In fifty-seven minutes the left side of the heart ceased to pulsate. In one hour and forty minutes the right side of the heart was still pulsating, but ceased to do so immediately after.

EXPERIMENT III.—A rabbit was put well under the influence of chloroform. The thorax was then opened and the heart exposed. One minim of aqueous solution containing $\frac{1}{6}$ th of a minim of nicotine was injected beneath the pericardium. The action of the heart was at once increased in rapidity and in vigour. In five minutes respiration ceased, but the heart continued pulsating as before. The body was wrapt in a moist cloth and placed near the fire. The left side continued to beat for one hour and twenty minutes, and the right side for one hour and fifty-five minutes after.

EXPERIMENT IV.—A fatal dose of chloroform was given to a full-grown rabbit. The thorax was opened and the heart found to be beating very feebly. Two minims of distilled water were injected into the left ventricle, but no change was produced in the character of the pulsations. One minim of aqueous solution containing $\frac{1}{6}$ th of a minim of nicotine was now

injected into the left ventricle. In one minute the action of the heart had so increased as to appear to be, as expressed by an attendant who was assisting, 'actually jumping, sir!' The effect was undoubtedly very remarkable.

EXPERIMENT V.—A guinea pig was injected in the back with $\frac{1}{6}$ th of a minim of nicotine. At the cessation of all respiratory efforts, at the end of two minutes, the thorax was opened, and the heart was found beating rapidly and regularly. Another $\frac{1}{6}$ th of a minim was now injected into the left ventricle, without at all lessening the pulsations of the heart. In two hours after the pulsations on both sides of the heart were quite visible, the temperature in the thorax being at that time 90° F.

EXPERIMENT VI.—In this half a minim of nicotine was placed on the tongue; the remainder of the experiment closely resembled the preceding one.

EXPERIMENT VII.—A rabbit was decapitated, and on the thorax being opened the heart was found to be contracting regularly but somewhat feebly. In thirty minutes after in the right auricle only was there any perceptible motion. The $\frac{1}{6}$ th of a minim of nicotine was then injected into the left ventricle, immediately the pulsations on both sides of the heart recommenced, and in two minutes it was vigorously at work again. In one hour and twenty minutes after this the heart was still in perceptible motion.

EXPERIMENT VIII.—A pigeon was injected with the $\frac{1}{18}$ th of a minim of nicotine. In seven minutes the chest was opened, and the heart found without the slightest motion. It was stimulated by being pricked with the point of a syringe, it then pulsated twice, but on again being irritated in the same way it remained motionless. The $\frac{1}{18}$ th of a minim of nicotine was now injected into the left ventricle, and immediately pulsation recommenced on both sides of the heart. In one hour and forty minutes the pulsations of the right side of the heart had not quite ceased.

EXPERIMENT IX.—A rabbit was killed by the destruction of the medulla oblongata. In three minutes the pulsations of the heart numbered fifty-four to the minute. The $\frac{1}{3}$ rd of a minim of nicotine was injected into the left ventricle. In one minute the pulsations were increased to sixty-two in a minute. In one hour and ten minutes after the right side of the heart was still pulsating.

In the preceding experiments the heart was not separated from the body. But in the following one, the moment that death took place the heart was quickly and carefully removed from the thorax.

EXPERIMENT X.—A young rabbit was decapitated, and the heart was immediately removed from the body and placed in a plate before the fire; it was moistened every few minutes with a twenty per cent. solution of nicotine. Its pulsations were in no way lessened, and, indeed, when compared with another heart beside it kept moistened with distilled water, its action appeared to be the more vigorous of the two. In thirty minutes the right auricle of the heart moistened with nicotine was still pulsating, but the other one had stopped.

It is worth while contrasting the effects of nicotine in the above experiments with that of the calabar bean (*Physostigma*

venenosum), as observed by Dr. Fraser, of Edinburgh, in some experiments he made with that drug on the action of the heart under its topical application, as described by him in his most elaborate paper on the action of calabar bean.¹ He records how two grains of the extract, injected into the right auricle with five minims of water, caused the action of the heart to cease instantaneously, and also that the effect of painting the surface of the heart with a strong solution of it, or removing the heart from the body and placing it in such strong solution, was a cessation of the heart's movements in a short space of time.

Notwithstanding that both these drugs act in a similar manner on the iris, causing its contraction, their effect on the heart is widely different.

From a perusal of these experiments it will be at once seen that although the heart was subjected to the influence of nicotine, both directly and indirectly, in various ways, in no case can it be said that cessation of the heart's action, or even diminution in the vigour of its pulsations, was the result. Even when the animal was poisoned with a dose that produced a fatal termination in a few seconds only, the heart was invariably found pulsating on the thorax being opened. I cannot therefore think that failure of the heart's action is the cause of death in poisoning by nicotine, and must disagree with former writers on this subject who state that as an ascertained fact. If such were the case, surely in the above experiments the heart would not have shown such vital activity after death and for so long a period.

For if death from nicotine resulted from arrest of the heart's action, we ought to find an immense decrease in the number and vigour of the heart's pulsations following the administration of it, and after death we should find that the heart had been arrested in diastole. I conclude then that the effect of nicotine is not to cause death by arrest of the heart's action, my experiments having shown—

1. That after death from large doses of nicotine the

¹ 'On the Physiological action of the Calabar Bean,' by Thomas R. Fraser, M.D., 'Trans. of the Royal Soc. of Edin.'

heart was invariably found beating with as much vigour and rapidity as it was in other animals of the same species not killed by poison.

2. That the application of various strengths of nicotine, including the pure alkaloid, to the surface of the heart after death did not arrest its action or diminish the time such action would continue, as compared with other hearts not subjected to the influence of nicotine.

3. That acceleration in the number and vigour of the pulsations of the heart, rather than diminution, was the effect produced after death by the internal administration of nicotine during life, or its application to the surface of the heart after life had ceased.

4. That in two cases in which the heart had ceased pulsating, the injection of a strong solution of nicotine into the left ventricle was followed by an immediate recommencement of the pulsations, and that, too, when the injection of water had had no effect whatever in that direction.

From six *post-mortem* examinations I have made on animals poisoned with nicotine, not less than six, or more than twelve, hours after death, I am inclined to think that there is a considerable analogy between them and the *post-mortem* conditions described by Dr. McKendrick¹ as being present in animals poisoned with bromal hydrate—namely, congestion of the thoracic and abdominal viscera, with dark blood filling the venous sinuses of the dura mater, and congestion of the pia mater, distension of the right side of the heart and pulmonary vessels with dark fluid blood, but *contraction*—which was not the case with bromal hydrate—of the left ventricle. These appearances are the more marked the smaller the poisonous dose that has been administered. Taking this into consideration, and the fact that death ensues from the cessation of the respiratory function whilst the heart is still vigorous, I am inclined to believe—but express my opinion with all due deference—that it is to the respiratory apparatus we must look for the

¹ 'Comparative Observations on the Physiological Action of Chloral and Bromal Hydrates, and Iodoform,' by John G. McKendrick, M.D. 'Edin Med. Journal,' July 1874.

cause of death, and not to the heart, as stated by most observers, following the administration of a poisonous dose of nicotine. This opinion is greatly strengthened by my observations on animals in whom the dose of nicotine has only just escaped proving fatal. Coincident with the muscular relaxation that sets in a few minutes after the administration of this drug, the respirations become laboured, less frequent, and at last purely abdominal, whilst the heart is beating violently. As the effect of the poison passes away, the respirations gradually become more and more natural, the chest walls again participate in the respiratory act, and by the time that the animal has sufficient power to stand upon its legs, respiration is again normally performed.

The effect of nicotine then on the heart, when administered internally or topically, is not to arrest its action, but rather to accelerate it and increase its power. That this is to some extent partly caused by the paralyzing influence which nicotine is known to have upon the vagi nerves is probable. For we know that the effect of division of the vagi in mammalia is to cut off the inhibitory nerves contained in them, and thus at once to prevent their exerting that inhibition which is their constant function on the heart. The manometer also shows that increased blood pressure follows the administration of a considerable dose of nicotine, as is the case when the vagi are divided.

But that the effect on the heart is *solely* produced by the removal of all inhibitory action cannot be the case, because, when the heart has been removed from the body and released from all control of the inhibitory nerves, the application of nicotine to its surface, or the injection of it into its cavities, has still the effect of accelerating and increasing the power of its pulsation. Therefore nicotine must, in addition to any effect that it causes on the heart through its action on the vagi nerves, exert some special influence on the intrinsic nerve centres of the heart itself, producing an increase in their function, resulting in its accelerated and more forcible action.

This double effect, which I have shown nicotine to be possessed of, should, when more fully worked out, prove to be of great therapeutical value.

The effects of nicotine upon the iris.—Although it has been known that contraction of the iris follows the administration of nicotine, I am not aware that anything very definite has been published upon this subject; as I have observed the duration and extent of this action with some degree of accuracy in the course of my experiments with this drug, I now put them on record.

In every case in which I have administered nicotine internally, by the mouth or by hypodermic injection, or have applied it topically, the effect has always been the same, namely, contraction of the pupil; and besides contraction of the pupil, increased lachrymation and congestion of the vessels follow its external application. The $\frac{1}{250}$ th of a minim of pure nicotine in two drops of water, when applied to the conjunctiva in the human subject, will cause a perceptible contraction of the pupil, but it gives rise to extreme pain and increased lachrymation, followed by congestion of the vessels. I have three times applied this quantity to my own eyeball, and each time with the effect of contracting the pupil. The effects of nicotine on the pupils, when given internally, are not so marked as when it is topically applied, unless it be given in sufficient quantity to quickly prove fatal, in which case pin-point contraction may take place. In most cases the contraction follows its external application very quickly, and soon passes away. I have not noticed any extreme dilatation following this contraction. The following experiment will show the average effect of the $\frac{1}{250}$ th of a minim when applied to the eyeball. The figures refer to the number of $\frac{1}{50}$ ths of an inch measured by the pupilometer.

EXPERIMENT XXIX.

	Right pupil measured	Left pupil measured
	16	16
The $\frac{1}{250}$ th m of nicotine was applied to right pupil which measured in 20 seconds	15	—
60 „	12·6	—
10 minutes	11	15
15 „	12·6	15
30 „	14	16
50 „	16·6	16

Observation.—It will be seen that the greatest effect took place in the first minute or two, which I found was the case in most of my experiments. The left pupil sympathised

with the right to the extent of $\frac{1}{50}$ th of an inch. I shall not record each experiment in full, but the accompanying table will show the maximum results obtained.

TABLE I.

Showing the minimum contraction of the pupil, and the time it occurred after the administration of Nicotine internally.

Experiment	Animal	Average before poisoning in $\frac{1}{50}$ th of an inch	Minimum after poisoning	Time after poisoning	Dose	Observations
XXIV.	Pigeon	Not noted	{ Marked contraction }	Not noted	$\frac{m}{16}$	By the mouth
XXV.	"	"	"	"	$\frac{1}{16}$	"
I.	Rabbit ¹	17	14	30 secs. ²	$\frac{1}{16}$ v	"
II.	"	16	14.6	20 min. ²	i	"
III.	"	16.8	18	10 "	"	Chloroform
IX.	"	16	14	10 "	$\frac{1}{6}$	Injected
XI.	"	15	14	10 "	$\frac{1}{6}$	Injected
XII.	"	16.8	15	15 "	$\frac{1}{6}$	On tongue
XIII.	"	16	13	18 "	$\frac{1}{3}$	Injected
XL.	Frog	Not noted	{ Marked contraction }	Not noted ²	$\frac{1}{6}$	On tongue
XXI.	Man	11	9	15 min.	$\frac{1}{33}$	By the mouth
XXII.	"	12	10.6	20 "	$\frac{1}{5}$	"

¹ In the case of rabbits the larger diameter was the one measured.

² Taken at the time the animal died.

TABLE II.

Showing the minimum contraction of the pupil, and the time it occurred after the topical action of Nicotine.

Experiment	Animal	Average before application of drug in $\frac{1}{50}$ th of an inch	Minimum after application of drug in $\frac{1}{50}$ th of an inch	Time after application	Dose	Observations
XXVI.	Pigeon	Not noted	{ Pin-point contraction }	50 secs.	$\frac{m}{12}$	The nicotine was of course applied in solution, one or two drops containing the amount specified.
XXVIII.	"	7	3	20 "	$\frac{1}{30}$	
XXIX.	Rabbit	16	11	1 min.	$\frac{1}{360}$	
XXX.	"	12.6	11.6	2 "	$\frac{1}{360}$	
XXXII.	"	16	12	4 "	$\frac{1}{360}$	
XXXIV.	Dog	20	18	5 "	$\frac{1}{250}$	
XXXV.	"	18	17	5 "	$\frac{1}{360}$	
XXXVI.	Man	12	10	5 "	$\frac{1}{250}$	
XXXVII.	"	13	11.6	7 "	$\frac{1}{400}$	
XXXVIII.	"	13	11	6 "	$\frac{1}{500}$	
XLV.	Ra	Not noted	Pin-point	2 "	$\frac{1}{30}$	
XLVI.	Frog	"	{ Marked contraction }	3 "	$\frac{1}{60}$	

Nicotine then contracts the pupils whether given internally or applied topically, and thus differs from the bromal hydrate, which causes contraction of the pupil only when administered internally; but its action closely corresponds with that of calabar bean, as recorded by Dr. Fraser. Its effects, however, do not seem to be of so long duration, nor is the action so intense when small doses are used as when extract of *Physostigma venenosum* is given in about equal quantity.

The internal administration of nicotine in the human subject.—The results I have obtained by the internal administration of small doses of this drug have been very contradictory, but tend to prove what I have before stated, that the action of nicotine is very different in different subjects. I have found so much difficulty in getting volunteers to take this very nauseating drug, even in minute quantities, that I have only been able to administer it to seven persons who are non-smokers.

I am a moderate smoker, and have taken several doses of nicotine, varying in quantity from $\frac{1}{20}$ m to $\frac{1}{5}$ m. Besides experiencing a burning pain in the back part of the throat for some time after, I have found it produce little other effect. The smaller doses had a tendency to increase the frequency of my pulse, but the $\frac{1}{5}$ m had an opposite effect, and decreased the frequency of the pulse twelve beats fifteen minutes after I had taken it.

In the case of the non-smokers the $\frac{1}{5}$ m dose produced a nauseating effect, which lasted for hours after, in fact, as long as the taste of it remained; and some of the smaller doses produced a slight feeling of sickness about a quarter of an hour after taking it, which lasted for a few minutes. In one case the dose of $\frac{1}{5}$ m was followed by a slow pulse and considerable lassitude, with a feeling of great muscular relaxation.

The smaller doses either exerted no appreciable effect at all or caused a slight increase in the number of the heart's contractions, without any decrease in the power of the cardiac impulse recognisable by the sphygmograph; but in two cases a marked increase in the power of the cardiac impulse was clearly shown. The temperature cannot be said to have been affected.

TABLE III.

Showing the effect of Small Doses of Nicotine upon the Pulse and Temperature in Man and the lower Animals.

Experiment	Animal	Pulse before taking Nicotine	Pulse after taking Nicotine	Time after	Temperature		Time after	Dose	Observations
					Before taking Nicotine	After taking Nicotine			
I.	Man ¹	84	110	Min. 25	Deg. F. 98·4	Deg. F. 98·3	Minutes 25	$\frac{1}{33}$	Sphygmograph showed increased cardiac impulse. Felt sick twenty minutes after. Sphygmograph showed increased cardiac impulse. Sphygmograph showed decreased action. Sphygmograph normal. Smoked a pipe of strong cavendish. Sphygmograph normal. Injected, animal partially paralysed. On tongue, animal partially paralysed. Injected, animal partially paralysed. On tongue, animal partially paralysed. On tongue, animal partially paralysed. Recovered.
II.	"	80	96	20	97·7	98·2	35	$\frac{1}{66}$	
III.	"	78	74	20	98·4	98	30	$\frac{1}{6}$	
IV.	"	76	78	15	98·4	98·4	20	$\frac{1}{50}$	
V.	"	76	62	50	98·4	98·3	40	$\frac{1}{6}$	
VI.	"	80	74	25	98·3	98·3	25	$\frac{1}{50}$	
VII.	"	77	90	15	98·4	98·1	20	$\frac{1}{33}$	
VIII.	"	76	84	30	98·6	98	35		
IX.	"	80	80	15	98·6	98·6	20	$\frac{1}{33}$	
X.	Rabbit	130	105	20	103	99·6	20	$\frac{1}{6}$	
XI.	"	—	—	—	101·2	99·4	15	$\frac{1}{6}$	
XII.	"	126	140	35	102	101	35	$\frac{1}{6}$	
XIII.	"	—	—	—	101	102·8	25	$\frac{1}{6}$	
XIV.	Pigeon	—	—	—	107·8	106	15	$\frac{1}{6}$	
XV.	Rabbit	—	—	—	103	102·4	4 hours	$\frac{1}{6}$	
XVI.	"	—	—	—	103·6	102	5 hours	$\frac{1}{6}$	

¹ All these were non-smokers, and in no case was there any irregularity in the action of the heart.

In the lower animals the effects were much the same, excepting that in almost every case the temperature was sensibly diminished; the doses, however, were relatively much larger, as the $\frac{1}{50}$ th m produced in the rabbit no appreciable effect at all.

The number of respirations was not affected, except in those cases in which a large dose of the drug was given, which, of course, was only to the lower animals. Then, as has been before described, the disturbance of the respiratory function was such as in most cases to prove fatal.

By comparing the results in the above table, it will be seen that collectively they indicate that the temperature in man, as well as in the lower animals, is sensibly diminished, and that the pulse is in a majority of cases quickened rather than lessened in the number of its beats by the administration of minute doses of nicotine. That this increase in the number of the heart's pulsations is not accompanied by a diminution in its vigour, is in nearly every case capable of demonstration by the sphygmograph; and it therefore follows that nicotine in small doses does not act as the powerful depressant it has long been stated to be, but that, on the contrary, its action must be regarded as moderately stimulating and invigorating. It should consequently recommend itself to all who are open to conviction, as a valuable therapeutical agent, of too great importance to be set aside without due trial of medicinal doses of it in all suitable cases.

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