itself at an advanced period of life, when the metabolism is at a low ebb. They would therefore represent a degradation or dissolution of the cell whereby it reverts to an immature form.

The melancholic condition does not depend upon the presence of these cells, but the cell condition is due to the cause which on the psychical side manifests itself in depression.

Although both in imbeciles and melancholiacs, therefore, it is supposed that the immediate factor which acts on and affects the cells is similar, there is no necessity to postulate any psychical parallel between the two conditions. Whether the above-mentioned factor operates on a fully developed nervous system, or on one which is not fully developed, will determine the respective psychical results following this change in the cells.

In the other class the cell change is due either to direct lesion of the cell bodies or to a lesion of their axons. I am inclined to think that, at any rate in the cases with an alcoholic history, the second alternative is the correct one. And as we are able to definitely assert that lesions to the axons will set up the change in their cells of origin, I prefer to accept this explanation in those cases where we find such a condition rather than invoke another—a problematical cause about which we have no certain knowledge.

Reference.

(1) Lugaro, 'Riv. Speriment. di Freniatria,' 1902, f. i, p. 981 (account taken from abstract by W. Ford Robertson, *Review of Neurology and Psychiatry*, vol. 1, No. 1).

On the Action of the Rolandic Cortex in Relation to Jacksonian Epilepsy and Volition.

By A. B. Kingsford, L.R.C.P.Lond., M.R.C.S.Eng.

The feature of Jacksonian epilepsy to which I wish to call attention is the periodicity of the discharge. Whether we regard the lesion as "irritative" or "discharging," it is, at all events, *chronic;* and whether we regard the discharges as going

direct from the cortex to the efferent nerve-cells, or as causing convulsions in a more roundabout way through subcortical systems, they are, at all events, *periodic*. How, then, do chronic lesions cause periodic discharges? The answer which I wish to put forward is suggested by certain passages in Mercier's *Psychology, Normal and Morbid*, p. 283, which run as follows: —Speaking of how organic bodies may contain a store of motion which can be liberated by the impress of motion from without, and after likening this property to that of animals and their power of movement, the author continues, "But animal organisms have a further property which most inorganic bodies have not. They are continually adding to their stores of motion, and by these continual additions their store at length becomes surcharged. The tension of the contained motion reaches such a pitch that the *containing resistance* is no longer sufficient to keep it in bond, and it breaks out, possibly without the provocation of added motion, certainly with minimal provocation." Later on, when discussing "Will and Desire,"(1) the same author writes of nervous mechanisms, "There are many machines used in the arts, which depend for their actuation on the gradual filling of a vessel with water. The vessel is of such a shape and so supported that, as it fills, the centre of gravity shifts, until, at a certain degree of fulness, the vertical at the centre of gravity falls without the base; the vessel then capsizes, empties its contents, regains its previous distribution of weights, rights itself, and begins to fill once more."

Let us extend the analogy a little.

Suppose the overturning of the vessel to be partly regulated by an elastic string so as to allow of its filling a little more full than it otherwise would without capsizing, then we can imagine the arrangement and power of the string to be such as to allow of the vessel discharging a little when the limit of stability was passed, and to effect the righting of the bucket again before much of its contents are emptied out. Suppose, moreover, the upper end of the string to be fixed to the arm of a lever capable of moving towards and away from the bucket under the impress of external circumstances, *e.g.*, the wind.

Some such an arrangement as this may be taken to represent the Rolandic mechanism and its controlling action over (all) subcortical centres in health.

Now let us suppose the elastic string to become weakened

as by "perishing," then it might happen that the bucket could never fill as full as it did at first without discharging some of its contents, and that an excessive discharge would follow any sudden inclination of the lever towards the bucket.

The action of a faulty control mechanism such as this would be fairly analogous to that of the diseased Rolandic cortex.

This point of view involves a twofold assumption :

1. That the discharges, causing convulsions, start from sub-cortical centres, and represent in fact a spontaneous overflow of their continually accumulating energy.

2. That the function of the Rolandic cortex is to control such discharges, and to determine their direction when allowing them to issue.

As these assumptions are not warranted by any great authority it will be necessary to examine the grounds for them, commencing very briefly with the current theories of action of the Rolandic area and the main evidences upon which they are based. At the outset of our inquiry we are confronted by considerable difference of opinion. Thus Schäfer (2) says (of the Rolandic areas), "In spite of the fact that movements have resulted from their stimulation, we are *not* justified in terming these portions of the cortex *motor*, but may regard them as sensory, and may look upon the movements as being set up by a motor-discharging centre elsewhere as the result of nervous impulses reaching it from the sensory region of the cortex." While Ferrier (3) writes, "Sensory and motor centres are not coincident, or at any rate not co-extensive, in the motor area. Sensation may be abolished by lesions altogether outside the Rolandic area. Paralysis of cortical origin may be, and frequently is, independent of impairment of cutaneous or muscular sensibility in the paralysed limbs." And Bastian,(4) after denying the existence of motor centres in the cerebral convolutions, says, "To argue that groups of cells have motor functions merely because stimuli issuing from them evoke movements when they impinge upon motor ganglia is quite on a par with the argument that an organ has sensory functions because fibres come to it from sensory cells." Dr. Bastian then continues, "The centres in question are rather sensory in nature, and are probably intimately concerned with certain groups of kinæsthetic impressions, whatever other functions

they may subserve or with whatever other centres they may be in intimate relation."

These extracts illustrate sufficiently for our purpose the well-known controversy about the functions of the Rolandic cortex, which might almost be called the battle-field of the nervous system. The case for the existence of motor centres, or even of sensori-motor centres, rests on the evidence afforded *post mortem* by Jacksonian epilepsy and on the results of experimental stimulation of the so-called centres in the higher animals and in man.

On this point Sir William Gowers ([5]) says, "Of all the regional diseases of the brain in man, lesions of the convolutions stand almost alone as a cause of convulsion, and experiments demonstrate that irritation of the cortex in the motor region has the same effect." "The results of experiments seem, indeed, conclusive." Accepting Gowers' facts, I suggest that the results of experiments are susceptible of another interpretation. Concerning these results Sir Michael Foster ([6]) says, "In considering this point" (*i. e.*, the question of localisation) "it must be remembered how rude and barbarous a method of stimulation is that of applying electrodes to the surface of the grey matter compared with the natural stimulation which takes place during cerebral action. The one probably is about as much like the other as is striking the keys of a piano at a distance with a broomstick to the execution of a skilled musician." Now if thumping a piano is at all likely to damage it, much more must similarly "barbarous" treatment be likely to damage the working of a far more delicate mechanism such as the Rolandic cortex. And so Foster's simile warrants, I think, the hypothesis that the results of experiments on the Rolandic cortex are attributable to the injurious effect of the stimulation. Moreover Hitzig ([7]) is reported as saying that he "found that simple exposure of the pia is followed by marked injury to the convolutions lying below, and that there is often implication of those contiguous to them. The uncovering of the membrane of the motor zone led not only to motor impairment in the extremities, but also —save in one case—to impairment of vision, and in all the cases to impairment of the reflex movements of the eyelids." It is generally agreed among physiologists that whatever other functions the Rolandic cortex may have, it has certainly some

inhibitory power. In 1892 Dr. James Shaw ([28]) wrote, "Inhibition is primarily a function of the motor area," and since that date Sherrington (and others) have brought forward evidence sufficient to prove it.([27]) Whatever the exact nature of inhibition may be, it must mean exertion of force through some distance, involving a continuous expenditure of energy while it lasts. Any damage done to the Rolandic mechanism is likely to impair its working. As with disease, so with injury—in either case there may be, as Mercier says, increase of process; but there must always be defect of function, and this, I believe, is the true explanation of the apparently active response to electrical stimulation on the part of the Rolandic cortex—namely, diminution of its inhibitory action leading to over-action or discharge of the lower automatic centres. That even a minimal stimulation is on the way towards causing damage serious enough to interfere with function is extremely probable when we consider that prolonged stimulation causes visible hyperæmia with convulsions, and that a condition of disturbance quite invisible is likely to interfere with the function of so delicate a mechanism as the Rolandic cortex. But to regard the case as one of defective action of the Rolandic cortex only is, I think, to take an incomplete view of the relation of the upper and lower centres. If the latter are constantly being charged with motion, as suggested by Dr. Mercier, derived chiefly from the food assimilated, how is this motion distributed? What becomes of it more especially when the organism is at rest? Now the upper centres are often spoken of as storehouses of motion or nervous energy, and from whence (apart from the food) is their energy stored up? I would suggest that when the supply of energy in the lower centres exceeds the demand, during the intervals of quiescence, such excess may pass off to the upper centres and there be stored up for future use, undergoing perhaps some change of form in the process—just as the energy of an electric current may be stored in the form of energy of chemical separation in a secondary battery. But the body itself presents a still more suggestive analogy. Sugar is intermittently thrown into the portal blood-stream in larger quantity than is required at the time, and is accordingly stored up in the liver, undergoing a change of form into glycogen in the process, and is restored to the blood in a

continuous, though variable stream, as required by the organism. Now, if matter may undergo this kind of storage, why may not motion, since both are forms of potential energy, and the storage of either is an economy? For I take it that the inhibitory action of the upper centres on the lower is a steady output of motion or energy tending to preserve and build up those lower centres when partially exhausted, in return, so to say, for the surplus motion intermittently received from them when replete. So much for a possible mode of inhibition, considered as a nervous economy. There is yet another point of view from which inhibition appears an economy in the scheme of the nervous system. It has been shown, as already stated,(8) by Sherrington and others that there are certain definite inhibitory centres in the cortex, and, moreover, that some of these are in close proximity, not to the supposed excito-motor centres for the same group of muscles, but to those of their respective antagonistic group of muscles. From the point of view that all the centres are fundamentally inhibitory, it would seem that agonist and antagonist centres are so coupled together that on receipt of a volitional stimulus the inhibitory action of the agonist is lowered, with a corresponding increase in the inhibitory action of the antagonist. The experiments of Dr. Charles Beevor (9) favour this view, for he has shown that while directly antagonist muscles are relaxed, the synergic muscles may be, and generally are, called into play. Now it is obvious that in any such exercise as walking it is an economy of force to lower the resistance against which the agonist group has to work, and still more of an economy if the very process of lowering the resistance of antagonists is, from another point of view, a storing up of energy in them ready for their turn to become agonists. Such, I believe, is what actually occurs, for when antagonists are relaxed there must be a diminished output of that form of energy from lower, perhaps spinal centres, which maintains muscular tone, and such diminution implies a damming back of the ordinary continuous outflow, *i. e.*, a storing up of motion. The calling into play of synergic muscles along with the directly agonist muscles is thoroughly in harmony with the action of lower centres educated (so to say) by volitional methods, as will be shown later. Furthermore, if it be true, as I suggest, that even minimal stimulation of the Rolandic areas disturbs them

enough to check or even stop their action for a time, it becomes easy to understand why mechanical stimuli should have so little effect. Such gross forms of stimulation can hardly disturb, without destroying, so delicate a mechanism. To borrow again Foster's simile, it is like thrusting a broomstick among the wires of a piano in search of harmony. Thus the inhibition hypothesis of Rolandic action has advantages over current doctrines as regards both economy and simplicity. It is now necessary to see how far any such theory can account for the phenomena of volitional, and of involuntary or automatic, actions, and for the paralysis due to removal of the Rolandic areas. We may take the latter first, as the study of paralysis will necessarily help us to distinguish the volitional components of actions, the execution of which is largely involuntary in detail. That the paralysis caused by removal, destruction, or physiological isolation of the Rolandic areas in the lower animals is a purely volitional one is evident from the behaviour of such animals after operation. Broadly speaking, such animals are capable of gratifying their natural desires; the lower the animal in the vertebrate scale the less the removal of even the whole of its cerebrum seems to interfere with its daily routine. All the actual movements of such higher animals as the rabbit seem perfectly adapted for the gratification of its ordinary desires; what it has lost with its cerebral hemispheres is mainly memory of special movements acquired by education, and of the complex perceptions which help to control both them and conduct generally. Thus a rabbit runs heedlessly past a heap of carrots, only avoiding it as an obstacle, while a dog is for ever on the move, and wastes rapidly in spite of feeding ravenously. As far as the actual movements are concerned, Foster says that a dog whose Rolandic cortex has been removed "can, after recovery from the operation, carry out voluntary movements so well that it is difficult to detect any deficiency in this respect."([29]) Again, if in the dog, says Foster, "the pyramids in the bulb be divided without injury to the cortex, but with consequent degeneration of both pyramidal tracts below the section, such a dog is able, apparently, to execute all the ordinary voluntary movements of which a dog is capable, though no regeneration of the pyramidal tracts takes place."([29]) And Schäfer, speaking of removal of one cerebral hemisphere in dogs by Goltz, says, "Such animals . . . show in their ordinary move-

ments an extraordinarily slight amount of motor paralysis, though apparently rendered incapable of performing such a purely volitional acquired action as the giving of a paw."([9]) In monkeys which are subjected to similar experiments like results have been obtained, but generally with more extensive and more permanent loss. Moreover in them a wholly new phenomenon makes its appearance, *viz.*, the *contracture* which follows removal of their Rolandic centres, and which follows also lesions of the corresponding parts of the brain of man. "This state of hypertonicity," Schäfer suggests, "may be due to the cutting off from the lower centres of the inhibitory impulses which they habitually receive from the cortex cerebri, while excitatory impulses which reach them from the cerebellum are still passing."([10])

These facts suggest that the mainspring of all movements is essentially automatic, but with a constantly developing volitional control as we rise from the fish to man;

That the controlling or volitional elements become the predominant feature of the most highly organised animals and man, and is proportional to the complexity of their environment;

And that in *form* the volitional element is entirely inhibitory, what is called an act of will being simply a special relaxation, diminution, suspension (or failure) of inhibition.

The facts both of physiology and pathology point to the Rolandic cortex and pyramidal tracts as the organs through which such control or volition is exercised.

Both Foster and Schäfer agree in stating that there are undoubtedly two paths of volitional impulses, and so far as I can find this is not disputed. Foster, in 1897, left the question of the function of pyramidal tracts quite open.([11]) After discussing the effect of section of the pyramids, he says, "We can hardly doubt that while the pyramidal tract was intact the animal made use of it, and we may further infer that the movements of a dog without the pyramidal tracts are different from those of a dog in which these are intact, though we cannot state exactly what the differences are." And Schäfer ([12]) quotes Donaldson as saying, "The activity of the lower level cells is in all animals brought about by two sets of impulses, the one set derived from the sensory nerves passing from their termination in the grey matter of the lower level centres either

directly to the motor cells, or more probably through intermediary cells, which play an important part in effecting the co-ordination required for purposeful movements. The other set of impulses, also in the first instance derived from the sensory nerves, pass to the cortex, and are thence sent down (perhaps along the fibres of the pyramidal tracts) to the motor nerve-cells, or rather probably also to the intermediary co-ordinating mechanism. In the lower animals this second set plays an insignificant part in producing the ordinary co-ordinated movements of the animal ; in the higher animals an important part, so that, in them, the cutting of it off from the lower centre cells removes a great part of the impulses by which they are normally stimulated."

Now it is only the conclusion Donaldson arrives at which I venture to differ from. We have evidence of some inhibitory function exercised by the Rolandic cortex, and also that when lesions cause degeneration of the pyramidal tracts contractures set in (in most monkeys and in man) from over-action of lower centres due to loss of control. We are therefore entitled to assume that the pyramidal tract is the path of inhibitory influences. We have seen, furthermore, that animals deprived of their hemispheres exhibit not only loss of memory and perception, on which volitional control is largely based, but also of acquired movements ; and the destruction of either the Rolandic cortex or the pyramids deprives the animal of all the actions which he has learnt by education, *e. g.*, giving the paw. This brings us at once to the question of the part played by volition in the execution of movement. It is obvious that volition plays no part in the actual execution of movements which are effected unconsciously, but can only appear when the execution of movements is based on some conscious memory of similar movements in the past. Such revival serves the purpose of further adaptation of movement in one of two ways, *i. e.*, intensively or extensively. The adaptation is intensive when the correspondence, with an unvarying environment, is made more perfect or complete, as in learning to cycle or firing at a target. And it becomes an extensive adaptation when more variable circumstances are taken into account, as when the cyclist learns polo or the rifleman goes sniping. This is the educational function of volition in the regulation of movement. The other function is that of suspension of action, which may

come into play either when the mind is made up, pending the arrival of the right moment for action, as when the prize-fighter, with every muscle tense, watches his opportunity to deliver a "settler;" or to gain time for irritation to cool, as when a man pockets a vindictive letter at the pillar-box for re-perusal in the morning. Here the inhibitory character of the volition is most apparent, and usually adds enormously to the effectiveness of the action when it comes at last. The arrangement of the pyramidal tracts, distributed as they are to the whole series of motor centres (or their intermediary connections) in the spinal cord, strongly suggests that these tracts constitute the pathways through which this volitional control is exercised. This mechanism might indeed be compared, functionally, with those of some Invertebrata, described by Dr. Haycraft, which are so well adapted for securing sudden and effective leaping movements of the whole organism. Apart from the formation of a decision—the process, that is, of willing, with which we are not immediately concerned,—this is the only part which volition takes directly in the execution of well-adapted movements, namely, that of releasing the trigger or letting go. Indirectly volition plays a further part in the reinforcement of actions, as in volition with effort; but this is not directly associated with the execution of movements, and only clumsily so at best. It has, moreover, an explanation of its own. This view, which assigns to volition only a transitory *rôle* in the execution of particular movements, allows it much greater scope in the regulation of series of movements or actions, and a predominance over combinations of series of actions or conduct. In general, all that we are vividly conscious of is the aim in view; thus we wish to be on the opposite side of the street, and forthwith we make series after series of movements with that aim in view, and presently find ourselves at our goal—sometimes, indeed, far beyond it for want of attending to our automatic actions. Not merely are we hardly conscious of our movements, but similar series may be successfully carried out in the complete unconsciousness following an epileptic fit. Moreover any interference with the automatic processes is prone to spoil the performance. Apart from the possibility of all our ordinary movements being carried out involuntarily in certain abnormal states of consciousness, *e.g.*, after epileptic fits, in somnambulism, and when hypnotised or under the influence of alcohol or drugs,

we have the further facts that though muscles may be paralysed as far as the execution of the will is concerned, they may still be quite capable of giving expression to emotions, notably in the case of those supplied by the facial nerve when it is affected by supra-nuclear palsy. And Beevor ([13]) says that in hemiplegia you may have the latissimus dorsi paralysed as an arm muscle, but functional as a bilateral muscle of expiration, and contracting when the patient gives a voluntary cough. From which it is clear that the machinery is sufficient without volition for the execution of all completely adapted movements.

The mode of expression of emotions Mr. Darwin([14]) says is innate or hereditarily organised, and, though more modifiable by circumstances acting through the will than reflex actions, is less plastic than the instincts of recent acquisition. Such instincts are the mechanisms whose state of repletion has the conscious accompaniment called Desire, and whose overflow is controlled to a greater or less degree by the higher centres of volition according to the organisation of the instinct. Those instincts which have unduly escaped from such control and gained an independence which is unserviceable to the organism are those described by Dr. Mercier as parasitic mechanisms; and these actuate conduct or the more complex series of actions in ways inimical to the interests of the organism, just as the lower centres actuate the simpler series of movements in ways inimical to the safety of the organism in Jacksonian epilepsy. The defect in both cases I believe to be essentially defect of control of the lower by the next higher centres, the difference being merely one of the relative rank of the lower centres in the two cases, for the lower or instinctive centres of conduct would perhaps be higher than the controlling centres of mere movements. We have, then, the three modes of origin for movements—the instinctive, the emotional, and the reflex,—not sharply marked off from one another, but differing in their progressive independence of volition. The question next arises how far in volition we have another mode of originating movement. Such initiation I believe to be an indirect one only. Along with the revival in memory of ideas of actions there comes the idea of the association of pleasure or pain with the action contemplated, and in correspondence with which comes a secondary desire to realise the pleasure or avoid the pain, as the case may be. And this secondary desire may

powerfully reinforce, or inhibit, the primary one, and determine the balance between desire and control. The more vivid and extensive the memory of the associated relations of pleasure and pain, the greater will become the influence of the control over the desire, and the more elaborate the mechanism underlying concurrently progressive desire and control. That such progressive development has taken place in man seems suggested by his brain structure, with its enormous controlling agency—the last to be developed in the individual,—and by the progressive development of similar though inferior agencies in the lower animals, especially mammals, culminating amongst them in man's nearest relative, the ourang. Now the remarkable fact of there being two volitional tracts, as described by the best authorities—an indirect or subcortical, and a direct or pyramidal one,—seems quite in accordance with the view that the subcortical one serves for the direct initiation of movement through desires, and the pyramidal for inhibitory control of movements, a control which has obviously increased in man, as his Rolandic centres and pyramidal tracts have, as compared with those of other mammals. Foster speaks of the indirect route as apparently falling into disuse, which suggests that the direction of movements, becoming more and more referred to the controlling centres as man's correspondence with the outside world increased, has now become very largely a matter of relaxation of inhibition. Be this as it may, it seems that it is only because the enormous majority of men's actions are in the educational stage referred to, and worked through the Rolandic centres, that the paralysis caused by their destruction in him is so much more complete than in the lower animals, especially when we reflect that the limb muscles are for many purposes always being educated, and that those whose education is most complete are just those which escape most and recover most readily after lesions causing pyramidal degeneration.

It might be supposed that destruction of part of the Rolandic system and corresponding loss of volitional control should leave the lower centres all the more free to act with the production possibly of movements or convulsions ; but you cannot alter one part of an organisation and leave all the rest just as before.

First there is the effect of shock, which is proportionate to the size and importance of the part removed, and the brunt of which falls on the most elaborately organised of the mechanisms

associated with the mutilated part, while the simpler and more completely organised mechanisms recover more quickly, and also more completely. Thus the operation may permanently alter the relation between subcortical and spinal centres for the worse.

Secondly, the removal of a constant controlling force should result not in spasmodic actions, but in a chronic overflow of energy from the lowest and least affected mechanisms. And this is just what we see in the early and late rigidities, due largely to over-activity of spinal centres, and affecting both agonist and antagonists simultaneously.

Furthermore, on our hypothesis the accumulation of energy in the subcortical centres would especially be interfered with, *i. e.*, any excess from time to time might very well leak away and be lost as one of the consequences of the operation. Suppose, however, we could by other than surgical means suspend or diminish the controlling influence of the cortex with minimum disturbance of the subcortical centres; then we might well expect to get some display of automatic action. And so we do, as is seen in the automatisms (often very elaborate) which follow attacks of petit mal, in somnambulism, and in hypnotism. The basal elements conspicuously lacking in all these states are those of conscious memory and perception, with their joint controlling influence.

Whether the actions are due to spontaneous overflow, as in petit mal, or to suggestion, from within, as in somnambulism, or from without, as in hypnotism, the striking feature of them all is the faulty or defective adjustment to the totality of the environment. The difference between them and the convulsions of Jacksonian epilepsy seems merely the difference between the plane of combined series of movements called actions and the plane of mere movements; as we know that the Rolandic cortex is the seat of the trouble in the latter, we may fairly assume that in the former the defect is one of association systems next above.

Another mode of diminishing cortical control is by administration of anæsthetics, and the automatisms to which these give rise are distressingly obvious both to the anæsthetist and the surgeon, and sometimes to the friends of the patient.

Before passing on to see how this hypothesis harmonises with the leading features of epilepsy, it is well just to say that however doubtful may be the possibility of "stimulating" the

Rolandic cortex to action, there is no doubt that other and lower parts of the nervous system may be so stimulated. That there is some essential difference between the two is evident from the difference of the curves obtained. The difference is probably one of completeness of organisation with a correspondingly stereotyped character of response. It is quite conceivable that the responses of subcortical parts of the brain to stimulation may be nearly as stereotyped as that of a piece of nerve, seeing how stereotyped by practice become our actions and habits.

The characteristic feature of an epileptic discharge is the progressive and correlated increase of the violence of the spasms and the intervals between them. Commencing with contractions so small and so frequent as to appear fused in the so-called tonic spasm, the intermissions soon become more evident, and the separate spasms more forcible, till the last of all, which generally comes with surprising violence just when the fit appears to be over. This feature, as Dr. Mercier ([15]) pointed out long ago, is suggestive of a discharge taking place against an increasing resistance, resembling, as it does so closely, the discharge of electric sparks from a static machine while the distance between the conductors is being slowly increased.

In our case the tendency to discharge on the part of the automatic centres must lessen with every actual discharge which takes place. So an uniform resistance would suffice to prolong the intervals between the discharges. But the general fact that the discharges, as measured by the force of the muscular contractions to which they give rise, increase progressively in amount, proves that usually the resistance is an actually increasing one. If so, whence comes this gradually increasing resistance? It is difficult to believe that it also is a function of the same centres which are discharging at the moment from "want of stability." Thus Gowers ([16]) says, "The process of inhibition which plays so prominent a part in many minor attacks, and in the initial stage of many severe seizures, seems at present to baffle our efforts to explain it.

"It was formerly regarded as the result of an increase in that *resistance* in the nerve-centres which normally controls and limits nerve activity. The resistance was supposed to be a function of nerve-cells related to, but distinct from, that which

causes their discharge. But when scrutinised this is merely a translation of the phenomena observed into terms of nerve physiology. The fact of 'inhibition,' of arrest of action, is certain, but its nature is not elucidated by its description as increased resistance.' We need to have some conception of the process by which activity is permitted and prevented, and of that we have at present no discernment."

That there is indeed a difficulty is more than evident when we compare with this what Mercier said of the action of nerve-centres generally and the muscles they serve. The passage runs thus :

"That a stimulus is necessary to set the centre in action all will admit, but that another is necessary to terminate the action will be to many a new proposition. But yet it is sufficiently obvious. It is no more possible that the centre can stop of its own accord (unless, indeed, it be entirely exhausted) than that it can start of its own accord. For a centre to cease acting from sheer exhaustion is so extremely rare that it virtually never occurs in the normal organism."([17]) Further on the same author says, "We must, therefore, conclude that the action of nerve-centres is arrested . . . . by the impact of an *extraneous* force."([18])

Here is not only a recognition of the difficulty, but, as I think, a foreshadowing of the way out of it. For if we suppose that the actual discharge takes place from the parts below the cortex—the mesencephalon (and, perhaps, cerebellum),—and that the cortex exercises only the function of inhibition (which function, Gowers [[19]] says, is certainly exercised somewhere, and which Sherrington has shown to be *a* function of the cortex), the difficulty so completely exposed by Gowers vanishes. For it is easy to imagine that, though in disease the normal output of inhibitory current from the Rolandic cortex—the normal rate, *i. e.*, of conversion of potential into kinetic energy which the maintenance of such current implies—may be below par, there yet may be *some* store of potential energy available for conversion on particular occasions. It is, further, easy to imagine that such conversion may take time, whatever its determinants, just as the conversion of liquid nitrous oxide into laughing gas takes time, and can only continue at a rate proportional to the access of heat, or thermal energy, from without. Now when we consider the relative complexity of the processes

which determine a development of inhibition on the plane of action, it seems more than probable that such development may require a relatively considerable time.

On the one hand, it seems clear that the ultimate effect of the majority of stimuli must be mainly inhibitory; otherwise, with a progressive intelligence and an increasing correspondence with an ever-widening environment (and the susceptibility to its manifold stimuli which that implies), man must surely have become the most restless animal on the face of the earth.

So far is this from being the case that, while a busy man is respected as one whose activity is productive, a mere busy-body is said to run about "like a dog in a fair."

On the other hand, it seems clear, too, that stimuli can only act as inhibitory influences indirectly in the first instance, *i. e.*, by a revival in sequence of a group of nerve-processes underlying a state of incipient action, and of another group of processes, the physiological substratum of some unfavourable (or painful) memory, associated with the state of action towards which those stimuli at first directly incite the organism. As the sequence of presentation and inhibition becomes fixed by repetition, the presentation itself, from being an exciting cause of action, becomes the symbol of the unfavourable memory which lapses from consciousness as the mechanism becomes organised and the process itself correspondingly expedited.

In our case it may well be that such conversion of potential into kinetic energy, which the regain of control by the Rolandic area implies, may be chiefly determined by the sequence, on innervation impulses, of the kinæsthetic impulses (unconsciously) received from the tensely contracted muscles—a state of muscle which, if too long continued, must from time immemorial have been unfavourable to the organism.

The fact that a fit can occasionally be arrested by artificially increasing the intensity of these impulses, as by forcibly resisting the movements of the limb, favours this supposition.

Furthermore it may possibly be that the action of a ligature in arresting Jacksonian fits depends partly on the principle of pressing the muscular nerve-endings as well as on the receipt of painful impressions from the skin, which tend to excite the inhibitory action of the cortex.

Moreover it is probable (as suggested by Dr. Shaw) that these kinæsthetic impressions are (some of) the first to become

associated with impressions of pain,—to exercise, that is, an indirect inhibition by awakening a memory of unfavourable experience in advance of a similar second experience. So we may expect them to be the last to be lost in disease. A roundabout process such as this will obviously require time, and that time is not likely to be shortened by disease.

Here it is convenient to call to mind that just as the newborn infant is without control over its movements, so is the whole of its Rolandic cortex inexcitable. Not that it is by any means unable to move after birth, or, indeed, for some months before, for its movements are excessive simply from want of control—a control which develops *pari passu* with its pyramidal tracts and the excitability of its Rolandic cortex. Certain features of the anatomy and physiology of the inexcitable Rolandic cortex between the "motor" areas lend support to the view that the "motor" cortex and the pyramidal tracts are essentially *inhibitory* in function.

Thus it seems that after extirpation of these parts, more especially towards the frontal region, the time of spinal reflexes is shortened. And again, if these parts be stimulated by *very strong* induced currents, in the dog, we have prolongation of latency and diminution of intensity of such reflexes. Such inhibitory effects, moreover, appear more marked in the front than in the hind limbs, and to travel by both anterior and antero-lateral columns of the cord.

Now if these areas are a species of foci through which the complexus of nervous processes underlying perceptions and ideas are brought to bear on "sensori-motor" centres (or, as I should call them, inhibitory centres), and if the action of such processes, etc., is mainly inhibitory—as I have endeavoured to show,—there is no difficulty in understanding the loss of inhibitory control which follows their removal. As for the increased inhibition which follows their stimulation, it would seem that here we may have another rough imitation of painful or unfavourable stimuli transmitted along well-organised paths to the "motor" inhibitory centres and heightening their action. It remains now to see how far the inhibitory theory may serve to explain some of the principal phenomena of epileptic after-states.

First, the general distribution of symptoms as between arm and leg is in accordance with the hypothesis of inhibition.

Thus the more frequent commencement of unilateral convulsions in the arm, its greater weakness, and, more frequently, absolute paralysis, are all explicable by the supposition that the control of the arm is more specialised in one hemisphere. That paralysis may follow a "sensory" fit without any convulsive manifestation, and that it is often in inverse proportion to such local convulsions (when they do occur), requires a further explanation.

Assuming with Gowers ([20]) that the paralysis following a sensory fit is an inhibitory paralysis, I suggest that whereas the discharge—realised in consciousness as a sensation—actually results in the liberation of motor discharges from subcortical centres, through other than pyramidal pathways, these motor discharges are overtaken and neutralised, so to say, before their exit from the spinal cord by inhibitory currents from the Rolandic cortex through the short cut afforded by the pyramidal tracts.

The paralysis seems to me as possibly a result of the reversal now obtaining of the normal relation between the subcortical centres and those in the spinal cord. The latter are now strongly inhibited, while the former are partially exhausted.

A like explanation may serve to account for the disproportion often seen between the motor spasm experienced and the subsequent paralysis. Here, again, I suppose the paralysis to be inhibitory, but that in these cases the belated inhibition is only partially successful in arresting the convulsion.

The spasm is the outward expression of the difference between subcortical motor discharge and cortical inhibitory current; the paralysis the difference, so to say, between the remaining available innervating energy of subcortical centres and the cortical inhibitory resistance against which they have to act. So, too, with the case recorded by Gowers ([21]) of a patient who, after a slight and transient spasm of the hand, "felt as if the arm were being raised above the head in violent spasm, while it was really hanging powerless by his side."

Here, on receipt of kinæsthetic impulses from the hand, there seems to have been a confused realisation in consciousnes of the innervation current, which possibly would have pro sufficient to raise his arm above his head in the m

described but for Rolandic interference. His feeling, I should say, was a kind of auto-suggestion.

Sir M. Foster has compared electrical excitation of the cortex to thumping a piano. But the Rolandic cortex seems to me more like the key-board of an organ, an instrument charged with motion or energy and called into action from time to time as that energy is released by the impress of external circumstances.

The Rolandic cortex is the organ for the execution of volition, and its influence on movement is like that of the driver's hands on the progress of a carriage and pair, or a cyclist's hands on that of a bicycle. While it has everything to do with the direction of the movement, it has nothing to do with energising it, and this is comparable with the action of the coachman who checks his horses first on this side and again on that. And just as after many repetitions the horses and the cyclist may progress without either conscious guidance or assent, so volitional acts become concurrently perfected and involuntary. The action of synergic muscles seems to illustrate the influence of volitions on movements. They shape the movement, so to say, by subtraction. Thus Beevor (32) says, "If you take hold of an iron bar and supinate as hard as you can, you will find your triceps is contracting strongly, but as soon as you flex the elbow-joint the triceps leaves off. The same thing holds good, but less so, in the case of pronation. The pronators of the arm are the pronators radii teres and quadratus; but as the former is a slight flexor of the elbow-joint, when you pronate you also flex the elbow-joint. And there, again, the triceps steps in and prevents the elbow from being flexed." The process of subtraction is clearly one of an inhibitory kind, though partial in its application, preventing one part of the action while permitting the rest.

Volition in the organism plays a part like that of law in the body politic, which, though strong to restrain, is powerless to drive, and is but a dead letter when not backed by a mass of public feeling.

In his last work, *Facts and Comments*, Herbert Spencer has insisted on the great importance of the feelings as the mainspring of action. And Mercier,(33) discussing "Freewill or Choice," says, "Granting that the willing is the choice of one mode of action rather than another, ... will is

only half accounted for, for we have yet to explain the power behind the mechanisms, the influence of which determines that any action at all shall be taken with regard to . . . . circumstances. . . . . As usually put, in every act there is the choice and the motive for the choice; and while the choice is a matter of judgment and attention, the motive is in every case a desire, an instinct, or a *quasi*-instinct." Thus desire and choice make up the process of willing, while suspension of action during the stage of judgment and attention, and "letting go" or yielding to the prevailing desire when decision is reached, make up the execution of the will. To take one more authority, Professor William James,(24) writing of "Volitional Efforts," seems unable to come to any other conclusion than that "for scientific purposes one need not give up" Professor Lipp's theory that "so far from the feeling of effort testifying to an increment of force exerted, it is a sign that force is lost, ... even if indeterminate amounts of effort really do occur." "Before their indeterminism," James says, "science simply stops;" *i. e.*, James is unable to identify any other factor in volition than the determining factors already dealt with. "The operation of free effort," says James,(25) "if it existed, could only be to hold some one ideal object a little longer, or a little more intensely before the mind. Among the alternatives which present themselves as genuine possibles it would thus make one effective; and although such quickening of one idea might be morally and historically momentous, yet, if considered dynamically, it would be an operation amongst those physiological infinitesimals which calculation must for ever neglect." If volitional education is the process of suspension and letting go, less and more, in accordance with the results of trials and error —made under the impelling force of some primordial desire for further adaptation,—and the whole execution of the will is no more than this, what need have we for assigning any such function as the term "excito-motor" implies to the Rolandic cortex and pyramidal tracts, the undoubted instruments of volitional execution?

For my own part, I suppose that the feeling of effort is the mental accompaniment of the nervous friction (if one may be allowed such an expression) entailed by the rush of nerve-currents from many associated areas towards one centre through tracts which are as yet but little pervious. Pain seems like the

friction of organic life, and the feeling of effort seems somewhat allied to it, while both friction and effort imply waste. The essence of volition with effort seems to be suspension of action, perhaps (through the pyramidal system) by the secondary, but, as we say, higher desire, pending the arrival of reinforcements of associated memories, etc., which ultimately may secure its satisfaction and its triumph over the lower desire.

Is there, however, any evidence more directly in favour of my hypothesis than that hitherto brought forward? The case published by Oebeke, and quoted by Gowers,([28]) and perhaps a somewhat similar case, observed by Gowers himself, seem to furnish such evidence. Sir William's description of Oebeke's case runs as follows:—"A patient who had been liable to general epileptic fits from birth was seized in adult life with left hemiplegia, due, as was afterwards discovered, to hæmorrhage in the central ganglia of the right hemisphere. The epileptic fits continued to occur after the onset of the hemiplegia, but affected only the unparalysed side." That is the description of the case, referring to which later on Gowers again speaks of the lesion as occurring in the *central ganglia*. Interpreting the meaning of the case, Gowers says, "The arrest of conduction from the right cortex prevented the effects of its discharge, showing that the convolutions of one hemisphere cannot act on the limbs of the opposite side, at least to a considerable degree, through inferior commissural connections." But Gowers does not say whether the internal capsule was involved by the lesion, and in the absence of that evidence it seems to me as likely as not that the "*fons et origo*" of the convulsive discharges was destroyed by the hæmorrhage, or, if not wholly destroyed, was so weakened as to more or less restore the balance between its tendency to discharge and that of the presumably weakened Rolandic cortex to restrain such discharges.

Lastly, has this theory any bearing on treatment? Now certain convulsive diseases, like rickets, chorea, and hysteria, are all markedly benefited by a high proportion of fat in the diet, with massage and rest to promote its assimilation and retention as useful auxiliaries. All these diseases are characterised by a defect of control. If epilepsy shares this feature in common with the others it may be that like treatment would prove beneficial for it too in early cases. The striking value

of a fatty diet in curing rickets seems especially suggestive, as the convulsions of rickets, when neglected, seem so often to pave the way for the permanent epileptic habit.

(1) *Psychology, Normal and Morbid*, p. 301.—(2) *Text-book of Physiology*, vol. ii, p. 723.—(3) *Allbutt's System*, vol. vii, p. 304.—(4) *Brain as an Organ of Mind*, 1890, 4th edit., p. 587.—(5) *Epilepsy*, 2nd edit., p. 215.—(6) *Text-book of Physiology*, p. 1132.—(7) Review of Hitzig's book, *Y. Med. Sci.*, January, 1903.—(8) Schäfer, *Physiology*, p. 712.—(9) *Clin. Journ.*, August 13th, 1902.—(10) Schäfer, *Physiology*, vol. ii, p. 731.—(11) *Physiology*, p. 1149.—(12) Schäfer, *Physiology*, p. 703.—(13) *Clin. Journ.*, August 13th, 1902.—(14) *Expression of Emotions in Man and in Animals*, p. 351.—(15) *Nervous System and the Mind*, p. 54.—(16) *Epilepsy*, 2nd edit., p. 225.—(17) C. Mercier, *Nervous System and the Mind*, p. 73.—(18) *Ibid.*, p. 74.—(19) Schäfer, *Physiology*, p. 712.—(20) *Epilepsy*, 2nd edit., p. 122.—(21) *Ibid.*, p. 123.—(22) *Clin. Journ.*, August 13th, 1902.—(23) *Psychology, Normal and Morbid*, pp. 323, 324.—(24) *Psychology*, vol. ii, pp. 576, 577.—(25) *Ibid.*, vol. ii, p. 577.—(26) *Epilepsy*, 2nd edit., pp. 103 and 218.—(27) Sherrington, *Spinal Animal*, pp. 19, 20.—(28) Shaw, *Epitome Mental Diseases*, p. 223.—(29) Foster, *Physiology*, p. 1149.—(30) Schäfer, *Physiology*, ii, p. 704.

Further Clinical Observations in Cases of Acute Mania, particularly Adolescent Mania.

By LEWIS C. BRUCE, M.D., Physician Superintendent, Murthly.

FOLLOWING up my observations made upon the blood of patients suffering from acute continuous mania read before this Association at the autumn meeting, I have been able to observe three cases of acute continuous mania in adults which relapsed while in the asylum. The results of the first series of observations were that in every case of acute continuous mania there existed a leucocytosis which persisted after recovery indefinitely. I advanced the theory that this leucocytosis was a protective leucocytosis. In the three patients who relapsed the leucocytosis was found to have fallen to below 13,000 per c.mm. of blood, instead of being nearer 20,000 per c.mm. of blood, which is characteristic of the recovered cases of mania. The polymorphonuclear leucocytes averaged 60 per cent. in two of these patients, and 47 per cent. in the third. In one of these patients the attack passed off in two days, and the leucocytosis at once rose to 25,000 per c.mm. of blood. The other two patients passed into a definite second attack, and their leucocytes averaged 15,000 to 16,000 per c.mm. of blood, with a polymorphonuclear percentage of 60 or below