BRITISH MEDICAL JOURNAL

LONDON SATURDAY JULY 12 1958

SITTING, STANDING, AND WALKING*

BY

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In the terms of the original bequest the Oliver Sharpey Lectures are required to have some physiological leaning, and physiology in the widest sense is the basis of medicine. A clinician, particularly a clinical neurologist, is conscious, all the time he is practising his art, that he is using the work of the physiologists and anatomists who preceded him, whether these were labelled as such or were in fact clinicians. There is now perhaps a tendency to swing away from the technique of meticulous clinical observation as a basis for determination of function, whether in the nervous system or elsewhere. Instead there is often the substitution of the results of investigations carried out by technical experts who may see only a part of the patient. The former is said to result only in the description of disease; the latter to fundamental observations on the nature of disease. In spite of this, whatever may be the fate of descriptive neurology, which must surely be the same as that of descriptive clinical medicine of any kind, there is still room for the elucidation of physiological mechanisms by the careful clinical study of disease processes aided, wherever necessary, by planned laboratory investigation. This was true of the past and it is still true to-day. One has only to watch a Symonds, Critchley, or Carmichael at work elucidating a clinical problem and then (perhaps this is what the critics fail to realize) applying these results to the interpretation of normal function and its breakdown to see that the clinical techniques still have much to offer to physiology.

It is with a little hope of following this well-established principle that I am offering the following clinical studies, and as a support for what may appear to be a narrow presentation of a few clinical phenomena as the basis for the analysis of function I will quote Karl Pearson (1911). "To decry specialization in education is to misinterpret the purpose of education. The true aim of the teacher must be to impart an appreciation of method and not a knowledge of facts. This is far more readily achieved by concentrating the student's attention on a small range of phenomena than by leading him in rapid and superficial survey over wide fields of knowledge."

Posture

The apparently simple physiological procedures of adopting and maintaining a posture can be regarded as simple only until an analysis is made of the various mechanisms which must be brought into play to initiate, continue, and close the underlying motor activity. The first or original action, as every parent knows, is always an uncertain one—needing constant repetition before a pattern is established; beginning as so many learning processes do by a series of attempts which gradually achieve success, until firstly sitting, then standing, and finally walking become fixed patterns which are followed with consummate ease, no longer requiring more conscious thought than is necessary to set the pattern in action. It is a commonplace of daily observation that complicated performances can be carried out while walking. One has only to see a messenger boy walking down the street, biting an apple held in one hand while reading with apparent complete concentration some book held in the other, avoiding oncomers, stepping on or off kerbs without pause, to realize that the youth is walking without the least effort of body or of mind and that all the co-ordination of muscles under sensory guidance is taking place with no apparent correction, intervention, or supervision.

It is not my intention in this lecture to make any analysis of the peripheral or spinal physiological phenomena of posture or of the partial contraction which enables a muscle to remain active indefinitely. This has been so well covered by Sherrington and his followers that it is now standard teaching, though I hope in the next lecture to discuss modifications of these views. What I will attempt is to show, by consideration of some patients with dissolution of powers of sitting and standing, some of the phenomena other than those of spinal cord activity that are necessary to the efficient performances of these motor functions.

Some Elementary Principles

First, however, as many of my patients have suffered brain damage, it is necessary to restate certain elementary principles which are sometimes overlooked in the desire to localize a lesion or, what is so much worse, a "function" of the brain. When a part of the brain is damaged a disorder of function may be produced. What we observe is the result of the rest of the brain making an effort to adjust for the deficiency, with greater or less success. It is never a simple arithmetical state of affairs. The situation is further complicated by the fact that men are not born equal. One brain is better than another in many ways, not the least of which is the power of adjusting for loss of part of the structure. Further, a poor original equipment may appear to suffer out of proportion to the rest. As a simple example one can take the individual who has normally the greatest difficulty in knowing his right from his left—the recruit

*The first of two Oliver Sharpey Lectures delivered before the Royal College of Physicians of London on February 11 and 13, 1958.

who breaks the drill instructor's heart because he will turn the wrong way as often as not. Let such a person sustain a closed head injury of only moderate severity and as a result lose some brain activity as an overall deficit; then the lowering of his ability to recognize his right from his left will appear now to be great, perhaps even glaringly obvious, and may well give the impression of an apparent focal disorder.

In his most comprehensive account of the parietal lobes, Macdonald Critchley (1953) takes great pains to stress the impossibility of precise anatomical localization of the multitudinous fractions of disordered functions evident in brain damage. He makes clear that it would be wrong to attempt more than the most general distribution of higher functions—perhaps not more than lateralization, perhaps not as much as that in many cases. No one will deny that permanent damage to certain fibre tracts will produce permanent defects of function. Damage to the visual pathway will produce permanent defects in the visual field which are so clear because no adjustment is possible—a final path has been affected. It is true that occasionally the defect is disregarded or filled with images that do not exist, but no adjustment can be made to produce actual vision in the defective part of the field. In higher functions, however, constant and varying adjustments are being made which must account for the fluctuations in the results of examinations from day to day as pointed out by Head and Holmes (1912).

It is true, then, that the study of brain damage gives results which are difficult of interpretation and which may be valueless for anatomical localization. They must, however, be useful for pointing to the functional organization of the individual.

An infant usually sits up at about 6 months old, unless it has profound muscular hypotonia, or disease of the central nervous system, or mental defect, when it may take much longer. When it sits up at first it sways readily, and unless propped it falls in any direction very easily. Once it succeeds in sitting steadily it makes efforts to pull itself up or if held will take its weight on its legs for increasing periods. When it has succeeded in standing it sits down anywhere by letting go and ending sharply in the sitting position. Once it has reached independence in standing, then it gradually tries to achieve a more gentle translation into the sitting position. But when it can get around the room and needs to sit on something, whether a stool or even its pot, one may find in the early stages that it does not manage very easily to seat itself in exactly the position it wants, and has to manœuvre itself round to get to the desired relationship *vis-à-vis* the outside world.

Trunk Apraxia

A similar situation can be found in the so-called rumpe apraxie or trunk apraxia, first described by Sittig (1929, 1931) in his monograph on apraxia, and further commented upon by Lange (1936). In this condition the sufferer is unable to sit up, lie down, sit down, or roll over on command, though he may do so spontaneously. Only one case is described, though in considerable and most revealing detail; but, as the underlying pathology was a vascular degeneration, there were numerous other defects which complicate and confuse the picture.

Nielsen (1946) briefly describes three cases of trunk apraxia. In the first a man of 40 was unable to rise from his bed or lie down upon it. He raised his arms and legs in the air in an effort to rise, but never succeeded in the attempt. Permission for a necropsy was not obtained.

The second patient could not read but could understand when spoken to. He could not undress himself; he tried to unbutton his clothes, but never succeeded in getting anything done. He acted like a child who had not learned how to do it. When on the examination couch, he turned and rolled like an infant and had to be carefully watched. When asked to sit down he walked across the room to the chair as though uncertain whether he was acting properly. When he finally reached the chair he turned round and regarded it from all angles. He tried to get himself into the chair but bent his knees while facing it. When this was not successful he turned half-way round, decided that was wrong, turned all the way round to the proper position, bent his knees a little—again decided that was wrong, and never succeeded in sitting down. At necropsy a diffuse glioma was found almost filling the left hemisphere, stretching from the tip of the frontal horn of the lateral ventricle to the occipital pole. It is remarkable that his defects were so few.

The third patient had difficulty in sitting up or lying down, yet he had no paralysis. He could not slide down the bed, but raised his pelvis in all sorts of clumsy ways, and finally put it down where it had started from. Necropsy was not performed, but the patient was regarded as suffering from diffuse cerebrovascular degeneration.

I have quoted almost all the details given in Nielsen's book. They are relatively few, but do indicate a difficulty with orientation of the body. Unfortunately it is all too clear that these patients had massive cerebral disease with great dysfunction of many parts of the brain, and it is impossible to use this sort of material to analyse the nature of the particular disturbances I wish to discuss.

To get a clearer view of the disorder I will describe two patients who showed this difficulty but who recovered, who were able to talk about their disorders, and whose general intellectual level was high. They were the best sort of subjects to help in working out the disorder, and, though it will be seen that this was not the only defect from which they suffered as a result of brain damage, it dominated the situation.

Case 1

A tank commander aged 28, while fighting in the battle of El Alamein, was struck in the back of the head by a shell fragment. He sustained a penetrating wound of the occiput from which brain was protruding when he was first seen. The first notes available to me were made 18 hours after the wounding, at a field surgical unit. Here he was found to be responding to questions but confused. His only complaint was of blindness. The evidence given for confusion was that when asked to move his right or left leg he moved either arm indifferently and insisted that he was moving his legs. A primary operation was performed with removal of some bone fragments and necrotic brain, and he was evacuated to the head centre, where I saw him 60 hours after wounding. He was then moderately confused, and his amnesia, both retrograde and post-traumatic, could not yet be estimated. He could now see, though he was very hazy in the lower half of each visual field. The only other abnormality was that he would not or could not move the correct limb on command, and this was out of proportion to the degree of confusion. *X*-ray examination of the skull showed a midline high occipital bone defect a little more to the left than right with radiating fissures and indriven bone fragments to a depth of 4 cm. Two weeks later a small abscess was evacuated through the wound, after which he progressed uneventfully, and a month later his wound was soundly healed. He was rational and his amnesia was now clearly about half an hour retrograde and 18 days post-traumatic. There were, however, numerous islands in these 18 days. It is interesting, and perhaps worthy of note to those who have to estimate the duration of a patient's amnesia, that later this intelligent young man gave his own views on the estimated time of loss of memory. He said that, lying in bed in hospital with few visitors, one day is so like another it may be quite impossible to be

certain when consecutive memory begins. In his case he marked it by the ward sister appearing one day wearing a Flanders poppy on her uniform as something different that he could hang on to in his mind.

Once he emerged from his confusion he showed a difficulty with visual imagery or visual recall. He would picture a uniform and try to attach the rank badges without finding the proper place for them. As he said himself a little later, "I knew I had two pips, but I couldn't see where I could wear them—wherever I pictured them seemed odd and absurd. It all came back to me quite suddenly, and I felt how stupid I had been ever to be uncertain." By the end of that first month his visual fields had improved and he then had only a homonymous defect of the right inferior quadrants which did not approach the fixation point nearer than 10 degrees. This remained as a permanent defect.

He was then allowed up and at once became aware of a queer inability to place himself correctly when getting into bed or trying to sit down. On his second day up he went into the lavatory and found he could not work out which way he must face in order to end up in the conventional position. He began by bending his knees and found them on the seat. He knew this was wrong, but no matter what he did he could not combine a knees bend with his posterior to the seat. This went on until he anchored himself by placing his hands on one of the side walls and keeping them there while bending the knees. This enabled him to sit sideways, which was a satisfactory compromise to begin with, though he soon shuffled round to the front. As this happened on several days he could not dismiss it, and tentatively mentioned it during a ward visit. Like so many patients with these bizarre disorders he was afraid that it meant insanity. When reassured he talked freely about this and applied himself to overcoming the defect. He soon worked out a technique of holding on to the lavatory door handle, instead of the side wall, before bending his knees.

At this time careful testing showed no visual agnosia, no visual disorientation; he could read, though he might miss a line now and again. He had no difficulty with his own right and left nor any finger agnosia, but he could be confused about the examiner's right and left by rotating through 180 degrees.

Two months after the wounding he discussed his difficulty in getting into bed. If he was sitting on the side of the bed and was told to get in, as often as not he would end up with his feet on the pillows and his head at the foot. He would get out to rectify this and often repeat the error. The same thing would happen if he told himself to get into bed, whereas if while undressing he started talking to another patient, then he would get into bed without thinking about it and find himself the right way up. He noticed that the action was also much easier if he was thinking with concentration about something such as the next day's activities. One day I asked him to get into bed to be examined; he was obviously in great difficulty, and helped himself by head and tongue movements such as may be seen in a child trying to solve a difficult problem. When asked what the difficulty was he said, "Once I take my eyes off the bed I'm lost, because I can't picture myself and the bed at the same time." It was certainly true that if he faced the bed and climbed in he achieved the correct ending more often than when sitting on the side. No defect of visual imagery for pictures, objects, or topography could be found at this time.

The following episode disturbed him greatly because it illustrated his difficulty in bodily orientation.

Because of the scar on his occiput he had become sensitive about the appearance of the back of his head. In places where he took off his hat he liked to sit with his back to the wall. In a favourite restaurant frequented by his regiment there were only round tables with as many as a dozen chairs. He could never decide which chair to sit on in order to have the back of his head towards the wall. As a rule he gave up trying to work this out, but sat on any chair and moved round until he got into the right place.

Another interesting defect this patient showed was a difficulty in rapid visual interpretation. He had no visual agnosia as ordinarily interested, but in the cinema he found that he could follow only slowly paced action. Any rapidly changing scene or much activity on the screen was completely beyond him. After a short time he would abandon the effort of following visually and hope the accompanying sound would keep him in touch until he could once more see what was happening. Like other patients with this disorder he preferred the theatre, where as a rule most of the action is static, to the cinema.

This patient was seen again a year later and improvement had continued. The visual fields were unchanged. Now he could usually put himself into a desired chair, though on important occasions, or where fear of failure or ridicule was present, he would resort to trial and error. He had no trouble with lavatory seats, though occasionally his knees would begin to bend too soon, but this he could control. He could now manage the cinema. He had no apraxia, constructional or otherwise, and no defect of the body scheme as applied to himself or the examiner. Formal intelligence testing gave an I.Q. of 120 on the Wechsler battery of tests.

Case 2

A sergeant was struck on the head by a shell fragment during the invasion of Europe. He sustained a penetrating wound of the skull a little posterior to the vertex from which brain protruded. He had a retrograde amnesia of a few seconds and a post-traumatic amnesia of an hour or two. He was at once aware of a loss of power in both lower limbs, because when stood up by a stretcher-bearer he at once fell to the ground. He was seen by a neurologist 36 hours after his wounding, when he had a partial right homonymous hemianopia with some diminution in power and sensation in both legs. It was then noted that he had a curious difficulty with the initiation of movements of the legs, though power was reasonable once the movement was under way. Unfortunately no pre-operative *x*-ray films exist, but an operation note is available. A bone defect was present just anterior to the apex of the lambdoid suture extending 2–3 cm. to the left of the midline and just to the right of it. The superior wall of the sagittal sinus was missing for 2.5 cm. under the bone defect, and bleeding could be stopped only by a large muscle graft. The dura was tense on the left side of the sinus, and from below it some subdural clot and lacerated brain was removed, leaving a cavity just to the left of the falx and extending down for about 3 cm.

He recovered quickly and was transferred to the Hospital for Head Injuries at Oxford four days after being wounded. On arrival he was alert and rational but noticed difficulty in judging distances, in that he would put things down short of his locker. He complained also that he didn't know where his legs were in bed or how to start them moving. Either or neither might move, and he could not be certain what would happen.

At this time he showed a right homonymous inferior quadrantanopia to 10 degrees short of the fixation point. The upper limbs were normal, but the lower limbs showed a moderate increase of tone with marked weakness of dorsi and plantar flexion of the feet and toes, more so on the right; the ankle-jerks were exaggerated, ankle clonus was well sustained, and the plantar responses were extensor. Sensation was normal except over the feet. Here he had a gross loss of all forms of sensation, both cutaneous and kinaesthetic, over the toes and almost to the ankle-joint. He had no dyphasia or visual agnosia. He had a confusion between right and left in himself and the examiner. He had no finger agnosia so long as lateralization was not required, and could copy finger postures, though he produced a mirror image of the examiner's fingers. He could sit up on

command, but on being asked to roll over he had difficulty, and, after trying and failing, wanted to know whether he had to roll to the right or left. Whatever the reply, or if told it didn't matter, he still couldn't manage until given a shove, when he continued quite smoothly.

He improved steadily and was allowed up some three weeks after the wounding. The visual field defect had cleared, but he still had marked sensory loss in the feet. He then noticed, as did the first patient, difficulty in getting into bed—ending up across the bed or with feet on the pillow. He also said, "The more trouble I take the worse I seem to be." In spite of saying this he attributed his trouble to absentmindedness. As a further example of "absentmindedness" he said that the first time he went to the up-patients' dining-hall he stood with a plate in each hand and his knife and fork and spoon in his pocket. He had no idea what to do next, whether to put a plate down or to get the cutlery out of his pocket first. This inability to carry out a complicated movement pattern persisted for another two weeks, yet from the first he had no evidence of dyspraxia in carrying out simple actions or pretending so to do.

From the time he was allowed out of bed he had difficulty in sitting in a chair. If he started facing the chair then he would turn a complete circle and end up facing the chair. If he turned only 180 degrees, he usually sat down by the side of the chair and landed on the floor. This difficulty persisted for six weeks longer than any of the other difficulties and awkwardness. He was quite certain it had nothing to do with the loss of sensation in his feet, because he said, "It isn't the numbness; that only affects me in the dark, and it's quite a different feeling." At a later stage he said, "My legs are queer creatures and I am often confused about them. I've mixed them up with someone else's. I sat in front of another patient to help him off with his boots, and it wasn't until I had unlaced and almost got one off that I realized I was handling one of my own." I must emphasize that at this time he had no defect of his visual field and that the sensory defects had receded and were confined to the toes and distal parts of the feet, but here they were dense.

This man was intelligent, giving an I.Q. of 130 on the Wechsler series. He had some memory disturbance in that he could not remember more than 50% of the details of a short story. He did poorly with Koh's block patterns and also in visualizing tests. For instance, he gave 16 and 8 respectively for the number of corners and surfaces of a cube. This was not due to any impairment of reasoning, as he did well in the standard tests for abstract thinking. Six months later his body orientation was normal, while the sensory defects in the feet were unchanged.

Comment on Cases 1 and 2

Both these patients showed a disorder of movement which can be summed up as an inability to achieve a desired relationship between the body and the external world. This was most dramatically shown when they tried to sit down, but it is also clear in other actions. These movements were at their worst when commanded voluntarily, at their best when automatic. In this they resemble the classical case of Hughlings Jackson's (1931–2) of apraxia of the tongue and lips in which it was impossible to protrude the tongue on command but where it went out quite smoothly and dexterously to lick a crumb off the lips. It is clear, therefore, that, whatever the disorder, it is not one which has disorganized completely the orientation of the body, but one in which the voluntary drive has been blocked so far as this function is concerned.

Sittig (1931), in the analysis of his own case, felt that it was usually associated with apraxia for other parts of the body and that the order of development of the apraxia was face, upper limbs, lower limbs, and trunk. This he advanced as an example of Hughlings Jackson's (1931–2) law that the more highly differentiated functions show the first evidence of dissolution in a large or general cerebral lesion. Sittig regards all forms of apraxia as really being bilateral, but that the hemiplegic weakness usually present covers up one side of the apraxia. In his view apraxia is primarily a disorder of movement and actually a form of paralysis. This he calls a paralysis of "higher potentiality."

A superficial view of the two cases here presented might class them as examples of ideational apraxia. This would mean that the disorder was due to a faulty conception of the movement as a whole; but this must be incorrect, for even the brief analysis here given makes it obvious that we are dealing with perceptual as well as movement disturbances.

"Body Image"

It is usual to attempt to explain these orientation defects in terms of disturbances of the body image, or schema, and this view has so many implications for my argument that I would like briefly to outline the major points of the hypothesis and its development. The term "body image," or schema, was introduced as such by Head (1920), though some of his views had been foreshadowed by Bouier, about 20 years earlier. This development was probably due tc the failure in Head's (1920) view of the theories on sensation and its relation to motor activity propounded by Munk (1890). The latter argued that the parietal lobe is directly concerned with appreciation of touch, pressure, temperature, and muscle sensation. Apart from muscle sense, these sensations can be reactivated centrally in the form of images, and though muscle sense could not be revived by imagination it could in spite of this combine with the contact or pressure images. These provide definite and distinct images of the actual position of the parts of the body and of their changes, and served as the initiator of movements. By extirpation experiments he produced a mosaic of function, each fragment having sharply defined capabilities.

I cannot go further into this view, which was severely dealt with by Head both directly and by implication. Head suggested that three schemas existed. The first is a visual model and the second a surface or tactile model on which localization took place. Both of these can readily be brought into awareness. The third is a postural model, usually not part of our awareness or conscious introspection, the means of ascertaining the position of the body or of movement of its parts. Which of these body images or schemas, if they exist, is the most important is still being argued. Head is clearly in favour of the postural model, whereas Lhermitte (1939) and Schilder (1935) favour the visual model.

Head says: "The image, whether it be visual or motor, is not the fundamental standard against which all postural changes are measured. Every recognizable change enters into consciousness charged with its relation to something that has gone before, just as in the taximeter the distance or time is presented to us already transformed into shillings and pence. So the final product of the test for the appreciation of posture or passive movement rises into consciousness as a measured postural change. For this combined standard against which all subsequent changes of posture are measured before they enter consciousness we propose the word schema. By means of perpetual alterations in position we are always building up a postural model of ourselves which constantly changes. Every new posture or movement is recorded on this plastic schema, and the activity of the cortex brings every fresh group of sensations evoked by altered posture into relation with it. Immediate postural recognition follows as soon as the relation is complete."

It is clear that this must be (if we may continue the same terminology for a time) a dynamic model, for no single element in the past is sufficient as the basis of the recognition of posture. The standard must be a continuously variable one involving all that has gone before.

It is by means of this mechanism that we are able to make the adjustments of position that enable one movement to follow smoothly on another. It is difficult, however, if we follow this view in its entirety, to see how the breakdown

of the schema can result in the breakdown of the long-standing well-reinforced fully established patterns such as sitting in a desired place.

Lhermitte (1939) sees these orientation difficulties as "a change in spatial thought, particularly in the representation of external space, the objects in it, the relations of the body to external space, and the individual's concept of his physical personality."

This is, of course, expressing the results in the most general terms of the breakdown of the whole orientation mechanism, but the mechanism itself remains obscure.

To elucidate this a little further I should like to describe some further cases which, while not directly related to the mechanics of sitting, may help to clarify this obscure problem.

Case 3

A driver was wounded in March, 1945, sustaining a penetration in the right occipital region. He was operated upon within 12 hours, and a track went forward to the falx, which was bruised but not torn. Debridement was carried out to 6 cm., but a later *x*-ray film showed that a small piece of metal had entered the posterior end of the frontal lobe. Two days later it was noted that the left limb movements, particularly of the arm and hand, were slow and clumsy, and that he often used the right limbs instead. I first saw him on the fifth day, and then he had signs of damage to the supranuclear pathway of the oculomotor muscles. There were no other abnormalities in the cranial nerves, particularly no visual field defect. There was no loss of power in the limbs, but he never used the left arm if he could avoid it. A week later he was still reluctant to use the left arm, but it was possible to demonstrate that he had no difficulty with recognition of right or left, nor was there any sensory defect at all.

Three weeks later power was full in formal testing, yet there was falling away of the outstretched arm and hand, There was no ideational apraxia, because he could comb his hair, salute, and lather his face with his left hand. There was no finger agnosia. Yet when asked to find his nose with the left hand he groped for it. He could not put the left arm into the same position as the right arm with his eyes shut. With eyes open he had less difficulty, but he had to make constant visual references to the position of the two limbs. Three weeks later he could imitate right-arm postures with the left even with his eyes shut, but he still groped for his nose and chin. Particularly impressive was the test for visual disorientation. In either visual field he could locate an object with his right hand with the greatest precision even though his eye remained fixed on the centre of the field. With the left hand, however, under the same conditions, he was inaccurate by many inches until the object was close to the fixation point. He eventually made an almost complete recovery.

Comment on Case 3

In this patient we see that, during the acute phase of his injury before adjustment had set in, he was unable to maintain so simple a posture as the outstretched arm; yet his postural sense as ordinarily estimated by tiny movements of fingers, his tactile sense tested in every way, and his visual functions were all normal. With more complicated postures or where movement must be imitated or initiated, the disability becomes even more obvious. This shows that there is a dissociation between sense of passive movement and sense of position in space, that it is possible to have damage to one function without damage to the other. Head, in his studies of sensation, recognized that these could be distinguished, but believed it to be unlikely that anything more than minor differences in degree of disturbance could occur. That this is not so is shown not only by this patient but also by a similar case described by Lhermitte and Trelles (1933) of a man of 68 who also could not copy the position of one limb with that of the other in spite of apparently intact sensorium. We must accept, therefore, that more than an intact sense of passive movement or position as ordinarily determined is necessary to achieve and maintain a posture. Can we find an example in which the converse is present—that is, an ability to maintain position when the sense of passive movement is absent?

Head says categorically that in order to maintain so simple a position as that of the outstretched hand and fingers one must be aware of the position of the hand in space either by kinaesthetic sensation or by vision, and in the absence of these the digits and then the hand would droop by gravity and remain uncorrected. This is standard teaching now, and we have all used the test to show the sensory defect. I have no doubt that this is true for the peripheral sensory pathways and their continuation up to the cortex, and even here it is usually true—but not invariably so.

Case 4

A Commando officer was struck on the head by the butt of a tommy-gun during a raid on the coast of France. He was rendered unconscious, and during this time he was captured. He had a post-traumatic amnesia of 14 days and on recovery he was hemiplegic on the right and aphasic. He gradually recovered power and speech, but after a few months he developed Jacksonian epilepsy. I saw him just over a year later, when he was repatriated. He then had a mild spastic hemiparesis on the right. He moved hand and fingers clumsily. He had a mild increase of tone with increased deep reflexes and an extensor plantar response on the right. Sensory testing showed a minor loss to pin-prick and cotton-wool touch over the right digits extending into the palm. All appreciation of passive movement was lost in the fingers and thumb. Movement but not direction could be perceived at the wrist and direction could be perceived at between 20 and 30 degrees at the elbow. Two-point discrimination was lost over the fingers but localization was fair. Stereognosis was absent. In the toes there was no appreciation of movement, but direction could be felt only at the knee. In spite of this gross postural loss this man could maintain his hand and fingers outstretched without any droop. I may say that this surprised me to such an extent that only after I had tried this out on several different days using blindfolds did I dare show the phenomenon to colleagues.

Comment on Case 4

If we accept this as a valid case then it is clear that the maintenance of position without use of the eyes and without the conscious appreciation of sense of position is possible. A very similar case is described by Head, and therefore we must abandon his own postulate that one must always be aware of the position of a limb to maintain that position.

This case, and also Case 3, lead me to the same conclusion, though from opposite directions—that the ability to achieve and maintain a posture is not dependent only on that part of the postural mechanism which can enter our awareness. There must be another mechanism, outside our awareness, which plays an important part, and this mechanism must receive activation in some way from the periphery. It may explain the pseudo-athetotic movements in those patients with severe tabes or sensory polyneuritis which persist even with the eyes open. There may be also in the failure of this mechanism the explanation for those tabetics who cannot be trained by re-education to use their eyes to walk with some ease. But this may be dependent on intelligence rather than anything else.

The part played by different regions of the brain in these phenomena is still very uncertain, and for reasons already given it would be difficult to attempt any formal localization and probably valueless, certainly leading to false conclusions. It is useful, however, to add some negative evidence about localization.

Phantom limbs in amputees are an example of retention within memory of previous experience—a limb continues its existence within the mind where no limb is present. It

is generally accepted that if kinaesthetic sensation is lost owing to a parietal lobe lesion, then this phantom limb will disappear. Such a case is quoted by Head (1920), and I will describe another in the second lecture, though for a different purpose. On the other hand, phantom limbs can exist without loss of the limb if the sensory pathway from the limb is destroyed. Cases of this sort have been described at every level of the sensory pathway. Whether such a phantom would disappear in a parietal lesion is probable, but I am not familiar with any such case in the literature. The suggestion, however, that such phantom limbs disappear because the post-central region is no longer functioning is rendered invalid by the evidence of the "third phantom." Such cases have been briefly mentioned by Macdonald Critchley (1953), and I would like to offer another such.

Case 5

A young man received a left parietal wound which resulted in a right hemiplegia with complete loss of postural sensation in the right limbs. After recovery from the initial dysphasia he felt that the paralysed hand was making piano-playing movements (he was a pianist), and only by looking at it could he convince himself that it was lying immobile across his chest or under his bedclothes. When sitting up in a chair and wanting to reach a glass of water or to turn a door-knob he often apparently put out his paralysed arm, and the feeling of movement was complete until nothing happened and he looked down at the limb with the immediate disappearance of the phantom. The tendency to try to use the paralysed limb was increased because he was definitely dyspraxic with the good left arm. He could never work out why he couldn't do things with the left hand such as use a comb, blow a kiss, or use a pair of scissors. He always tried to excuse the failure by saying it was because he was very right-handed and this was the cause of the clumsy left hand. He also said that if he shut his eyes he could easily feel his right arm doing all the things at which the left was bad. He never said I can see the right arm doing all these things. With the partial recovery of postural sensation the phantom third limb disappeared permanently.

Comment.—Cases such as this would suggest that a parietal lobe lesion causing the disappearance of a phantom limb is massive and includes more than the post-central cortex and subcortex. It would suggest that the mechanism for the third phantom, the maintenance of posture, and the orientation of the body not only can take place outside consciousness but can remain efficient even when the conscious appreciation of movement is lost; further, that orientation of the body as a whole when sitting or standing can fail even though normal sensation is present.

Mechanism of Body Image

The concept of the body schema as put forward by Head indicates that it is conveniently on the edge of consciousness —usually outside it, but capable of being drawn into it and thus reaching our awareness. This use of words is unfortunate, for it creates a curiously solid picture of a body-image mechanism which may, even on this hypothesis, have a definite localization. Perhaps such a view is superficially useful to explain how one loses awareness of parts of the body in terms of disappearance or damage to the body image. Yet this concretization cannot but impede progress in determining the real mechanism.

From his studies of memory and its breakdown, Ritchie Russell (1957) suggests that the memory of movement patterns is due to constant activity within the cerebrum of neuronal circuits. These are established by constant repetition and reinforced by activity. With non-physical memory, reinforcement can take place even during sleep, indicating that a voluntary drive need play no part. The circuits producing postural tone control must be widespread, including cerebellum and brain stem, and must be capable of modification by the afferent information the body can provide. It is clear from our personal experience that this mechanism exists outside our awareness once the neuronal circuits have been well established, but it must never be accepted as fixed but rather as a dynamic state. This is capable of constant change, as will be shown in the next lecture for the much simpler spinal circuits.

If the body-image mechanism is considered as a series of neuronal circuits, then at points where these circuits are narrowly confined, as in the reticular substance in the brain stem, physical change will produce the widest alterations in the pre-existing state of the individual. This may account for the ascendancy the brain stem has acquired in the neurophysiology of to-day.

This concept of neuronal circuits which are constantly active makes the difficulties of anatomical localization clear and must mean the abandoning of mosaics of function in the hemispheres. It will account also for the widespread area in which lesions may occur with the production of similar alteration in function.

The body image therefore must be regarded as a series of long-standing well-established neuronal circuits or loops which are constantly reinforced because the circuit is self-propagating. Superimposed are the modifying influences produced by streams of afferent impulses from eyes, muscles, and joints. Spreading from this are the impulses which will affect posture and movement, both of which will be considered in more detail in the second lecture.

[*The second lecture, with a list of references, will appear in our next issue.*]

SEROLOGICAL RESPONSE OF INFANTS TO POLIOMYELITIS VACCINE

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It is now recognized that satisfactory serological response to poliomyelitis vaccine occurs in children after the age of 1 year (Medical Research Council, 1957), but as yet little is known of the response of infants to this vaccine. However, as it has been shown that even newborn infants can be adequately immunized against tuberculosis (Gaisford and Griffiths, 1951), diphtheria (Butler, Barr, and Glenny, 1954), and—with a suitable triple antigen—diphtheria, pertussis, and tetanus together (Feldman, 1954), it seemed logical to try to ascertain how early in infancy immunization against poliomyelitis could be successfully undertaken. This was the purpose of the study here described.

An additional reason for this investigation was the incidence of precipitated paralytic poliomyelitis after the injection of diphtheria and other prophylactic antigens, particularly when combined (Medical Research Council, 1956), because it was felt that if successful immunization against poliomyelitis were possible in early infancy this could precede all the other protective inoculations and might thereby eliminate the risk of provoked paralysis.

In this study, 88 mothers and their infants have been investigated in respect of the relationship between maternal serum and umbilical cord levels of polio-