if the atomic weight of argon be 40, on subtracting 32, or twice the average difference, the number 8 is obtained, which closely approximates to $3{\cdot}9 \times 2$. Which of these views is correct time must decide.

II. "Alternate Current Dynamo-Electric Machines." By J. HOPKINSON, F.R.S., and E. WILSON.* Received April 4, 1895.

(Abstract.)

The paper deals experimentally with the currents induced in the coils and in the cores of the magnets of alternate current machines by the varying currents in and the varying positions of the armature. It is shown that such currents exist, and that they have the effect of diminishing to a certain extent the electromotive force of the machine when working on resistances as a generator without a corresponding effect upon the phase of the armature current. It is also shown that preventing variations in the coils of the electromagnet does not, in the machine experimented upon, greatly affect the result, and that the effect of introducing copper plates between the magnets and the armature has not a very great effect upon the electromotive force of the armature, the conclusion being that the conductivity of the iron cores is sufficient to produce the main part of the effect. A method of determining the efficiency of alternate current machines is illustrated, and the results of the experiments for this determination are utilised to show that in certain cases of relation of phase of current to phase of electromotive force, the effect of the local currents in the iron cores is to increase, instead of to diminish, the electromotive force of the machine.

III. "Note on the Relations of Sensory Impressions and Sensory Centres to Voluntary Movements." By H. CHARLTON BASTIAN, M.D., F.R.S., Professor of Clinical Medicine in University College, London. Received April 5, 1895.

In a recent communication to the Royal Society by Drs. Mott and Sherrington, entitled "Experiments upon the Influence of Sensory Nerves upon Movement and Nutrition of the Limbs," results of a most important and hitherto unsuspected character were brought forward. In this communication they have shown that "section of the whole series of sensory roots belonging to a limb," either upper

* The large majority of the experiments herein described were made in the summer of 1893, and a considerable part of the paper was then written. We have to thank Mr. F. Lydall, one of the student demonstrators at King's College at that time, for much assistance.

or lower, immediately produces a lasting motor paralysis in the limb thus apæsthete.

The interpretation of these results seems at first sight very difficult. The authors of the paper, after referring to views which I have put forward in reference to the fundamental importance of sensory impressions in the production of voluntary movements, give expression to the following explanation :—"We think these experiments go even further than his arguments in pointing to the influence of sensation upon voluntary movement, inasmuch as they indicate that not only the cortex, but the whole sensory path from periphery to *cortex cerebri*, is in action during voluntary movement."

This interpretation of their interesting experimental results is one which I cannot accept because it is opposed to other thoroughly ascertained facts. Clinical investigation has established the fact that in cases of complete hemianæsthesia due to lesions or functional defects in the posterior part of the internal capsule, there is not only no paralysis but little or no impairment in the ability to perform, under visual guidance, even the most delicate movements with the apæsthete limbs. This shows therefore that it is not necessary, as they suggest, for "the whole sensory path from periphery to *cortex cerebri*" to be in action during the performance of voluntary movements. It was the knowledge of these facts, made known by clinical investigation, that caused me to be much perplexed when Dr. Sherrington kindly showed me some of the animals on which he had been experimenting. The reality of the paralysis was obvious, but how were we to account for the fact that the interruption of the sensory channels from a limb in one part of their course produced this almost complete paralysis, whilst the interruption of these channels higher up (in the brain itself) gave rise to no such results? I was for a time quite unable to explain the apparent discrepancy, though further consideration seemed to remove the difficulty.

A brief exposition will, I hope, suffice to throw light upon this question, as well as upon the extent to which afferent impressions and the activity of their related centres are really needed for the production of voluntary movements.

I have for some years contended, in opposition to very generally accepted views, that there is no evidence to prove the existence of motor centres in the cerebral cortex; whilst, on the other hand, there is much evidence to show that the cortical regions supposed to be motor, are, in reality, sensory centres of kinæsthetic type.* The postulation of the existence of motor centres in the cerebral cortex had its origin in, or, at least, derived its principal support from, the

* "The Muscular Sense: its Nature and Cortical Localisation," 'Brain,' April, 1887.

doctrine (now generally admitted to be erroneous) that "feelings of movement" were, in the main, "concomitants of the out-going current."

Further, I have endeavoured to show that sensory impressions and the activity of sensory centres are the real guides for volitional action, that they, in fact, do just such work as has been attributed to supposed cortical motor centres, and that it is a fundamental error to imagine that cortical motor centres exist for the performance of voluntary movements, altogether apart from the other motor centres that are concerned with the production of reflex or secondary automatic acts.*

I have contended, in short, that true motor centres exist only in the pons bulb and spinal cord, which may be called into activity in different ways, according as the stimulus comes, in the one case (*a*) from the cortex, for volitional movements; or as it comes (*b*) through afferent nerves and lower sensory centres, as in reflex acts.

In regard to movements of the latter category (*b*), there is no room for difference of opinion, and the results of the experiments of Drs. Mott and Sherrington are thoroughly in harmony with what is generally admitted. Section of all the sensory roots proceeding from a limb must abolish, as it has been found to do, all reflex movements in this limb.

We may turn therefore at once to the consideration of (*a*) movements initiated from the cerebral cortex (so-called voluntary movements), and strive to ascertain in what various modes such movements may be rendered impossible, or, in other words, how paralysis of such movements may be occasioned.

It may be noted here that the writer was the first, in 1869, in opposition to then prevalent physiological notions, to postulate the existence of various sensory centres in the cerebral cortex,† and that some years before any experimental evidence was brought forward on the subject. About the same time he showed how this hypothesis sufficed to throw light upon the nature of various forms of speech defects,‡ and in this he was followed by Broadbent,§ still before any attempt had been made to localise such centres.‖

* "On the Neural Processes underlying Attention and Volition," 'Brain,' April, 1892.

† "On the Localisation of Function in the Cerebral Hemispheres," 'Journ. of Ment. Science,' January, 1869; and "On the Muscular Sense and the Physiology of Thinking," 'Brit. Med. Journ.,' May, 1869.

‡ "Physiology of Thinking," 'Fortnightly Review,' January, 1869; and "Defects of Speech in Brain Disease," 'Brit. and For. Med. Chir. Review,' January and April, 1869.

§ "On the Cerebral Mechanism of Speech and Thought," 'Med. Chir. Trans.,' 1872, p. 180.

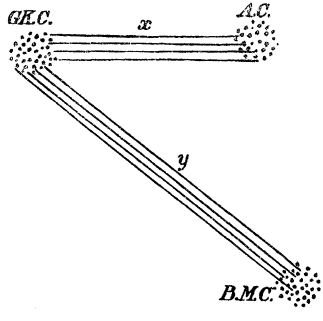
‖ See Ferrier in 'Phil. Trans.,' Part II, 1875.

From the point of view of sensory guidance, cortically initiated movements are divisible into two categories:—(I) *Speech movements*, which are now known to be produced under the guidance of co-active auditory and kinæsthetic centres; and (II) *Limb and other bodily movements*, which are equally well known to be produced under the guidance of co-active visual and kinæsthetic centres. In each case the kinæsthetic centres do not seem to act independently, but only in response to the primarily initiated activity in the auditory or in the visual centres respectively—these latter in all cases seeming to take the lead.

I. *Speech Movements.*—Words appear to be revived in thought, in the main, in the auditory centres, and, if they are to be spoken, stimuli from these centres must pass along associational fibres to related portions of the kinæsthetic centre, that is, to what I have termed the "glosso-kinæsthetic" centre, in and near the posterior part of the third frontal convolution. If we assume, as clinico-pathological evidence seems to warrant, that the portion of the auditory centre concerned with the registration of the sounds of words is situated in the posterior part of the upper temporal convolution, the associational fibres in question would pass in their course from the auditory centre beneath the Island of Reil in order to reach the glosso-kinæsthetic centre. From this region the combined stimuli would pass off from the cortex (and through the internal capsule), so as to evoke in appropriate ways the activity of the proper motor centres for speech situated in the bulb (fig. 1).

It has now been ascertained that speech movements may be paralysed by lesions in any part of this tract from the auditory centre to the bulb. Lesions in either of the two sensory centres will pro-

FIG. 1.—Diagram illustrating relative Positions of three Cerebral Centres concerned with Speech.



AC, auditory centre; GKC, glosso-kinæsthetic centre; BMC, bulbar motor centre; *x*, audito-kinæsthetic commissural fibres; *y*, internuncial fibres.

duce such paralysis just as certainly as lesions in the bulbar motor centres themselves. If the lesion be situated in the auditory word centre (AC) it will produce "word-blindness" as well as loss of speech; though if it be situated in the glosso-kinæsthetic centre (GKC) it will produce loss of speech alone. In opposition, however, to the doctrine as to the strict localisation of aphasia, as a symptom resulting only from lesions in the third frontal convolution (Broca's convolution), I have long maintained that precisely similar defects would result from destruction of the commissural fibres (*x*) in any part of their course—thus affording an explanation of the many cases recorded by Meynert and others in which aphasia had been caused by a lesion in the "Island of Reil."

Again, it has long been known that damage to the internuncial fibres (*y*) in any part of their course would also cause loss of speech (*i.e.*, paralysis of speech movements), similar in kind to that caused by lesions in the bulbar motor centres (BMC).

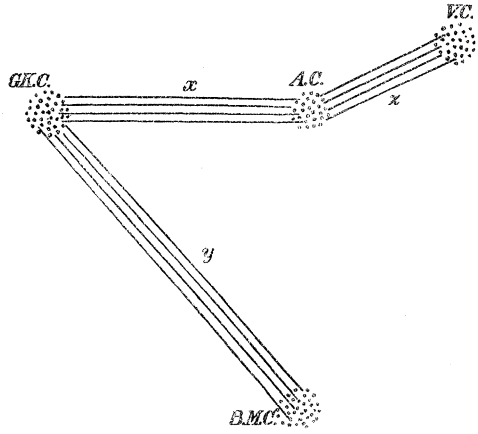
In what has been said above we have illustrations of the importance of afferent impressions and the revived activity of afferent centres for the production of the voluntary movements occurring in articulate speech. And if, instead of looking to the processes which occur when our own thoughts reveal themselves in spontaneous speech, we turn to what happens when we read the recorded thoughts of another, it will be found that a further relation with sensory centres has to be established before such reading aloud can occur.

The impressions from the printed page impinge upon the visual centre (fig. 2, VC), and are thence transmitted across commissural fibres to related portion of the auditory centre, whence the stimuli pass, as in spontaneous speech, to the glosso-kinæsthetic centre and thence on to the bulb. The consequence is, that where lesions occur in the course of these visuo-auditory fibres (*z*), the individual is unable to read aloud, and he is unable to name objects or even single letters, although he will immediately repeat words or letters as soon as he hears them. He cannot carry out the voluntary movements concerned with speech at the instigation of the visual sense, though he can do so at the instigation of auditory impressions. We get thus at the roots of Will and find them definitely localised in sensory centres.*

II. *Limb Movements.*—The visual sense in the case of limb movements performs the same office as the auditory sense in the case of speech. New movements of limbs are learned largely by aid of the visual sense, supplemented by associated kinæsthetic impressions. So that subsequently, when we desire to repeat familiar movements,

* Cases illustrating the effects of lesions to the commissures between the auditory and the visual word centres may be found recorded in 'The Brain as an Organ of Mind,' 4th edition, pp. 640—647.

FIG. 2.—Diagram illustrating relative Positions of four Cerebral Centres concerned with Reading aloud.



VC, visual centre; *z*, visuo-auditory commissural fibres. Other references as in Fig. 1.

such desire is accompanied by a "conception" of the movement to be performed. This "conception," in physiological terms, means a revival in sub-conscious memory of the visual and kinæsthetic impressions pertaining to the movement in question.

Limb movements, like others, may be paralysed either by organic lesions or by functional defects.

A. *Organic Lesions.*

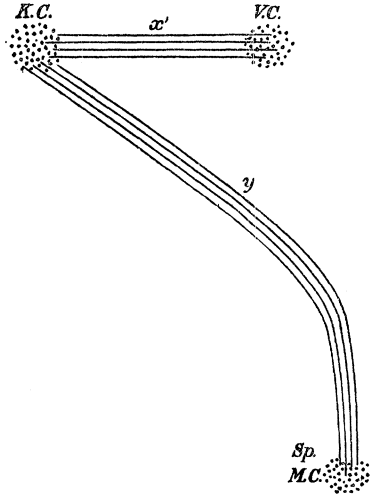
If the kinæsthetic centres in relation with the limb are destroyed, paralysis of the limb results, together with loss of muscular sense and kinæsthetic impressions generally. This has now been determined by clinico-pathological evidence, as well as by the results following excision of portions of the cortex by reason of disease occurring therein.

As yet we have no evidence from man of paralysis of limb movements following extensive disease of the visual centre, or of the commissures existing between it and the kinæsthetic centres for the limbs, comparable with the paralysis of speech movements following disease of the auditory centre or the audito-kinæsthetic commissural fibres, except in the case of writing movements. It is, however, certainly true that with destruction of the left visual word-centre the individual is no longer able to write words or even a single letter.

But it would seem that some experimental evidence on lower

animals does exist, tending to show that a severance of the visuo-kinæsthetic fibres (fig. 3, *x*) is followed by the same kind of paralysis

FIG. 3.—Diagram illustrating relative Positions of two Cerebral Sensory and one Spinal Motor Centre concerned with Writing Movements.



VC, visual centre; KC, kinæsthetic centre; SpMC, spinal motor centre; *x'*, visuo-kinæsthetic commissural fibres; *y*, internuncial fibres.

of limbs as that which results from destruction of the kinæsthetic centres themselves. Thus, Marique* has found, and his results have been confirmed by Exner and Paneth,† that isolation of the kinæsthetic centres, by section of the fibres that connect them with the other sensory centres of the cortex, produces a paralysis of precisely the same character as that which occurs when these so-called motor centres are extirpated. Marique, moreover, found that the same kind of muscular contractions were produced on electrical irritation of the respective kinæsthetic centres after, as before, isolation, showing that these centres still retained their excitability and their connection with the pyramidal tracts.

B. *Functional Defects.*

Functional defects leading to paralysis of limbs may be either (*a*) cerebral or (*b*) spinal in seat.

* 'Centres Psycho-moteurs du Cerveau,' 1885.

† 'Archiv f. d. Ges. Phys.,' bd. 44, 1889.

(*a.*) *Cerebral Functional Defects.*—To this cerebral category belong cases of the so-called hysterical type, in which there appear to be temporary defects in the nutritional condition of the kinæsthetic centres leading to various temporary and curable forms of paralysis (either monoplegias, hemiplegias, or paraplegias), such as I have elsewhere described,* always associated with a corresponding loss of muscular sense and, more or less, of defect in common sensibility. These forms of functional paralysis are often combined with well-marked complete hemianæsthesia, either single or double, presumably due to a co-existing nutritional defect in the sensory region of the internal capsule.

Strangely enough, in one class of these cases the patients are found to be capable of performing movements so long as their eyes are open, though they are quite unable to perform even the simplest movements with the affected limbs as soon as the eyes are closed;† results which are, I think, to be explained by the existence of a minor degree of that nutritional degradation of the kinæsthetic centres which, in the other class, produces paralysis whether the eyes are open or closed. The minor degradation would seem to permit of the kinæsthetic centres being roused by a slightly stronger stimulus coming to them from the visual centres of a person whose eyes are open, though they are no longer capable of being roused by the hypothetically weaker stimulus coming from the visual centres of a person whose eyes are closed.

(*b.*) *Spinal Functional Defects.*—It is into this category that, in my opinion, the forms of paralysis fall that have been produced by Drs. Mott and Sherrington as a result of section of all the sensory roots of a limb.

We have been long familiar with forms of paralysis due to structural lesions affecting the great ganglion cells in the anterior cornua of the cord; those, for instance, due to polio-myelitis or to focal lesions of different kinds involving the anterior cornua.

Three years ago, moreover, I gave reasons for believing that there are cases of functional paralysis of spinal type, due to defects in these same regions of the spinal cord (examples of which were recorded), that ought to be distinctly separated from those of cerebral origin commonly known as "hysterical."‡ We have now, as I am inclined to maintain, in the experiments of Drs. Mott and Sherrington, an experimental proof of the existence of one of these forms of functional paralysis of spinal origin.

Instead of a lowered functional activity in cerebral kinæsthetic centres (interfering with volition), we have here a lowered functional

* 'Various Forms of Hysterical or Functional Paralysis,' London, 1893.

† *Loc. cit.*, pp. 22—28.

‡ *Loc. cit.*, pp. 96—116.

activity in the motor centres themselves situated in the spinal cord by reason of which their molecular activity is so altered that they are no longer capable of responding to ordinary volitional stimuli coming from the cerebral cortex. This, at least, is the explanation that I would suggest, instead of supposing, as the authors do, that volitional power itself "has been absolutely abolished by the local loss of all forms of sensibility" in the paralysed limbs.

As I have already stated, a local loss of all forms of sensibility caused by a lesion in the brain in the region of the internal capsule causes no such paralysis. There is therefore no ground for supposing that the animal's power of willing is interfered with by section of all the posterior roots coming from the limbs; while, on the other hand, all the details furnished by Drs. Mott and Sherrington are quite in harmony with the interpretation that the animal's ordinary will power is unable to excite the spinal motor centres to action when their molecular condition has been altered by cutting off all the different stimuli proceeding to the corresponding region of the cord. The immediate result of this may be presumed to be a lowering of the habitual sub-activity of the motor centres upon which the condition of *tonus* in the muscles depends.

The fact that the results are produced only when *all* the sensory roots are cut, and that "the defect in motility increases from the attached base to the free apex of the limb," so that "the independent and more delicately adjusted movements which employ preponderantly the smaller and more individualised muscular masses of the hand and foot" are those which are most severely affected or abolished, although they may appear confirmatory of the interpretation of Drs. Mott and Sherrington, are, in fact, no less in accordance with that here given. This will be seen to be so if we bear in mind the overlapping of the fields of distribution of the sensory roots in the spinal cord (as shown by Sherrington), and the fact that the most delicate stimuli going to the smaller muscles might be expected to be those which would prove most impotent to rouse the sluggish spinal centres into activity. Again, when the authors say: "We find, however, that forcible and rapid movements, even of the fine joints at the end of the limb, can be induced in the animals by causing them to 'struggle;' for instance, while recovering from ether inhalation or while trying to free themselves on being held awkwardly, the whole limb at all its joints may exhibit movements," we have facts quite compatible with my interpretation. Muscles may not respond to ordinary volitional stimuli, and yet may respond when the stimulus is strengthened under the influence of emotion. In a similar manner may we explain the fact that when the kinæsthetic centres corresponding with an apæsthete limb are stimulated by electricity, movements of the previously paralysed limb are produced just as easily as they are in a normal

limb.* Such results may, as the authors say, be considered to point to the "profound difference between the production of the fine movements of the limb in volition, on the one hand, and by experimental stimulation of the cortex, on the other." This explanation is as valid for the one as for the other interpretation.

The stimulation by electricity may, and probably does, involve a very different kind of stimulus from that which normally emanates from the cortex during a voluntary act, and how subtle are the differences that exist between the conditions leading to paralysis or not in different instances may be judged from the functional cases of paralysis of cerebral origin, previously referred to, in which there may be in the same person, in immediately successive periods, complete paralysis of the limb so long as the eyes are closed, and no such paralysis when the eyes are opened.

What has been said above shows, moreover, how much removed we are from the position assumed to be true about twenty years ago,† when centres "immediately concerned in effecting volitional movements" were considered to be "as such truly motor."

IV. "The Fasciola Cinerea; its Relation to the Fascia Dentata and to the Nerves of Lancisi." By ALEX HILL, M.D., Master of Downing College. Communicated by Prof. A. MACALISTER, F.R.S. Received April 2, 1895.

In my paper on the hippocampus, published in the Philosophical Transactions of the Royal Society for 1893 (vol. 184, B, pp. 389—429), I stated as a subsidiary conclusion, resulting from my investigation of the brains of marine mammals, that "there is no reason for associating the fascia dentata with the striæ longitudinales (nervus Lancisii), gyrus supracallosalis, and gyrus geniculi, or for supposing that all these four structures belong to a single organ, which forms a part of the cortical centre for the sense of smell."

This conclusion was based upon the following observations:—A. In

* This fact shows that the cutting off of the afferent impressions by section of the sensory roots does not entail a lowered excitability of the kinæsthetic centres in the cortex, but rather the reverse, if we look to the really lowered activity in the spinal centres which the absence of tonus implies. In the case of complete cerebral hemianæsthesia, however, there is, over and above the absence of any such lowered activity of the spinal centres, another important difference between the conditions existing and those which obtain in the experiments of Drs. Mott and Sherrington, viz., no cutting off of cerebellar influence, so that the activity of the cortex and of the sub-cortical centres is less interfered with. These differences tend, I think, to explain the presence of paralysis with section of the posterior roots, and its absence with cerebral hemianæsthesia. (*May* 4, 1895.)

† Ferrier, "Functions of the Brain," 1st Ed., 1876, p. 200.