CHEILO TOMY FOR CRIPPLING TRAUMATIC ARTHRITIS OF THE HIP-JOINT.

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I WAS much interested in the communication published under the joint names of Mr. Sampson Handley and Dr. Preston Ball which appeared under the above heading in the BRITISH MEDICAL JOURNAL of May 3rd.

I have many times, in common with other surgeons, operated upon cases of chronic rheumatic arthritis to relieve pain and deformity, but as my last case is identical in all essential respects with the two cases described in the communication, and as the operation was performed on the patient's hip on October 22nd last, three months before Mr. Sampson Handley operated upon his first patient, it may be of interest to mention the outstanding features, and thus put a third case on record. The question of priority is of no great importance, but Mr. Sampson Handley and Dr. Preston Ball are in error in thinking that the operation had not been performed prior to their first case in January, 1913.

Miss H. D., aged 21, residing in London, was sent to me in September last, the subject of polyarticular chronic rheumatic arthritis from which she had suffered from childhood. The left knee-joint, the right hip and the two wrists were disorganized with the changes characteristic of the disease. The left knee could not be completely extended without intense pain, and the right hip could bear no weight whatever, the slightest pressure on the heel causing pain in the joint. Locomotion was therefore only possible with the aid of crutches, and even then was attended with much suffering. She had been dependent on the use of crutches for seven years.

X-ray photographs, taken by Dr. Hayes on September 9th, showed lipping of the tibia to the outer side of the left knee-joint, which was the site of great tenderness. A ring of osteo-arthritis bone surrounded the right hip, impeding every movement of the joint, stretching the capsule, and on pressure evidently abutting against the rim of the acetabulum and the neighbouring bone.

The first operation was performed on the knee-joint on September 12th. (It should be mentioned that the shortening produced by the enforced flexion was corrected in London by raising the boot about 2 in.) Through an incision made along the outer side of the knee-joint the lipping of the tibia was removed with a gouge and chisel through a well-marked line of cleavage such as is seen in the new bone formed in connexion with a bunion. A weight and pulley was attached to the limb for about ten days to correct the flexion, and massage was applied for a short time subsequently. All pain disappeared immediately after the operation, and on removal of the weight and pulley the joint could be fully extended and flexed.

The result of this operation encouraged me to operate upon the hip of the opposite side on October 22nd. The joint was opened through Kocher's posterior incision. There was a well-marked "cornice" of newly-formed bone encircling the junction of the head and neck of the femur, giving it the mushroom appearance commonly seen in the specimens of chronic rheumatic hips found in the surgical museums.

The capsule having been freely opened, the hip was rotated in all directions by an assistant, and the new bone was chiselled away from about two-thirds of the circumference. The result of the operation surpassed all expectations. The following day pressure on the heel caused no pain. The stitches were removed in ten days and the patient was allowed up. She could now bear her entire weight on the right limb, and the movements of the hip-joint were free and painless. Subsequent progress was uneventful. I saw the patient when in London last week, and she is walking without crutches in absolute comfort after being a cripple for seven years. I can arrange for Mr. Sampson Handley to see the patient and the x-ray photographs if he is interested in the case.

I was glad to read the result of his two cases, which convinces me that my result was no exception, and that in selected cases the operation of "cheilotomy" is destined to give instant relief, and to change completely the outlook for a certain class of crippled patients. I think the

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title of the paper would be better if the word "traumatic" were omitted; the etiology matters little if the pathological changes are the same.

I should mention that practically every form of treatment, including the use of vaccines and serums, had been tried in my case during the nine years she had suffered from the disease. The condition of the wrists has improved considerably since the patient has been able to discard the use of crutches.

THE INTERNEURONIC SYNAPSE IN DISEASE.

BY

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I HAVE long felt that the cell-exhaustion theory does not satisfactorily explain the pathology of acute delirium. Take, for instance, such a case as this: A young woman, while walking in her sleep, falls from a height, injuring herself severely, and at once becomes acutely delirious; or a girl, owing to a celluloid comb in her hair suddenly taking fire, immediately develops acute insanity; or a similar state results in an elderly widow on finding her sister dead in the same bed. Up to the occurrence of the shock, a state of mind entirely normal to the individual apparently exists, and almost immediately after we find more or less complete inco-ordination of the higher centres and mis-interpretation of stimuli. Inco-ordination of the higher centres is the outstanding result. To my mind, this suddenly developed condition, accompanied as it is by extreme motor restlessness, cannot logically be due to cell exhaustion. For purposes of comparison I have examined my records of 17 cases of insanity occurring after surgical operation in which the undoubtedly important factor producing the alienation was exhaustion—physical and mental—plus the slight shock of the operation perhaps, and contrasted them with 25 cases of insanity immediately supervening on sudden and violent mental shock, with the following results:

TABLE I.—The Types of Insanity Resulting.

Type of Insanity.	Etiological Factor.	
	Shock.	Operation.
Acute delirium	11	4
Confusional insanity	5	—
Melancholia	5	9
Mania	3	3
Stupor	1	—
Delusional insanity (systematized)	—	1
Totals	25	17

TABLE II.—Ages at Onset.

Age.	Etiological Factor.	
	Shock.	Operation.
Under 20 years	4	1
21 to 30 "	5	2
31 to 40 "	3	5
41 to 50 "	4	6
51 to 60 "	4	3
61 to 70 "	5	—
Totals	25	17

As regards predisposing causes, a family history of insanity was ascertained in 28 per cent. of the cases due to shock, and in 23.5 per cent. of those due to operation.

TABLE III.—*After-History.*

	Recovered.	Died.	Permanently Insane.
<i>(a) Cases due to Shock:</i>			
Acute delirium	6	4	1
Confusional insanity ...	3	—	2
Melancholia	3	—	2
Mania	1	—	2
Stupor	—	1	—
<i>(b) Cases due to Operation:</i>			
Acute delirium	2	2	—
Melancholia	4	3	2
Mania	2	—	1
Delusional insanity (systematized)	—	1	—

The average period required for recovery was in the cases due to shock 5.8 months, and in those due to operation 16.2 months, practically three times as long. In 50 per cent. of the cases resulting from shock which became chronic a definite family history of insanity was obtained. The average period of recovery of the deliriant cases was practically the same in both, namely, three to four months.

It will be noticed that the type of insanity resulting from severe shock is decidedly different from that produced when exhaustion is the important etiological factor. Acute delirium resulted in practically half of the former cases—and, indeed, the confusional cases were verging on delirium—melancholia in more than half of the latter. In other words, as a result of sudden and severe mental shock, the tendency is to acute delirium, and when prolonged exhaustion enters into the causation the tendency is to depression. From my cases, too, I am justified in saying that the greater the shock the more the tendency towards delirium. Now if this is sought to be explained by saying that the delirium is a more intense reduction, the fact that complete recovery is both more probable and more rapid in these cases than in melancholia must be reckoned with.

Again, the tendency in acute delirium is either towards rapid recovery or death. Only one out of the whole 15 cases became permanently insane, which is in marked contradistinction to other forms of insanity. In the one case, which became a secondary dement, there was a strong family history of insanity, and she was herself of a very low mental type. One would expect profound cell exhaustion to produce a depressive—almost stuporose—type of insanity, whereas what we apparently find as a result of intense mental shock is the opposite.

In cases due to shock the age has apparently no influence worthy of notice. This is quite different from what obtains in post-operative cases, among which 8 out of the 17 occurred between the ages of 40 and 50 inclusive. The fact that 11 of the whole 17 were operated on for uterine or ovarian trouble will throw some light on this.

I think it may fairly be assumed that the delirium resulting from sudden nervous shock cannot satisfactorily be explained by the theory of cell exhaustion.

Whatever be the nature of the nerve current, the cell has apparently the power of releasing energy on the reception of a stimulus corresponding in degree to the intensity of that stimulus. The balance of evidence appears to be in favour of the nervous impulse being a physical process, and it is, I think, satisfactorily established that resistance to its passage is principally met with at the inter-neuronic synaptic membranes and finer terminal arborizations.¹

It is a matter of common observation that minor degrees of shock are accompanied by a rigor-like thrill. A weird sound in the silence of the night, the presence of a human form at a time and place when and where it is not expected, will give rise to a "start" and a conscious sensation as of an electric-like discharge throughout the system. If this is a matter of common observation on the occurrence of slight or moderate degrees of shock, is it not probable that a very intense nervous discharge might cause serious damage to the finer nerve arborizations

and synaptic junctions, and that, just as under similar conditions electrically the fusion of a wire would take place, so here, as a result of intense shock, we may have some degree of what might be termed "synaptic fusion" of the higher neurone terminals and interneuronic membranes, causing dissociation of the higher centres and misinterpretation of stimuli? These higher associative neurones being the latest to develop their terminal arborizations, are probably the most delicate.

The effect of very intense nervous shock on the memory is similar to that occurring after severe concussion—namely, there is total lack of recollection of the shock itself and of events happening shortly before, or, at best, the recollection is very indistinct. A further point of interest is the fact that the average period required for recovery in acute delirium resulting from shock is identical with that required for nerve regeneration as a result of trauma—namely, three to four months.

In a few cases return to co-ordinated thought is apparently sudden. It seems probable that in these cases synapses of association fibres not in common use were probably uninjured by the discharge, allowing an almost sudden return to sanity once the current found a new path connecting up the higher centres. The tendency of the nervous impulse to revert to its accustomed course would account for the fact that such cases are very liable to slight relapses before ultimate recovery. In those cases which quickly result in death, may it not be due to the intensity of the discharge causing damage to many more axone terminals than would occur in a slighter degree of shock which in another case will terminate in recovery, given similar resistance at the synapses?

There are different degrees of synaptic resistances.² Sherrington states that "there is abundant evidence that synapses differ from one another" in this respect, and the action of strychnine and tetanus toxin affords further proof. Now, if in the same individual different degrees of synaptic resistance are met with, is it not probable that individuals will differ with regard to nervous irradiation, owing to the fact that in general their synaptic resistances are heightened or lowered abnormally? Strychnine and tetanus toxin have the effect of lessening synaptic resistance and of converting normal spinal inhibition into excitation.³

I have noticed that great susceptibility to the action of strychnine exists in hysteria, relatively small doses causing most unpleasant sensations, such as tachycardia and much mental uneasiness. Might not this indicate that in hysteria the neuronic trouble is synaptic—that there is normally in this affection too little interneuronic resistance, resulting in a constant leakage of current, so to speak, outside the area to which it is limited in the normal individual? Supposing this is so, what would we expect? "The hysterical temperament," to quote Starr, "is manifested by an abnormally keen sensibility to all external impressions and sensations, and a susceptibility to suggestions, by a manifest incapacity to exercise control over thought, emotion, and action, and by a tendency to act on sudden impulses."⁴ In other words, stimuli which normally would only take a certain definite course, owing to a lowered interneuronic resistance become diffused in their effects on the higher associative centres, and also concomitantly the ultimate stimulation is more intense than normal, hence hyper-ordinate and inco-ordinate reaction. The mental process resulting from sensory stimuli is in the normal person orderly, and in nine cases out of ten similar stimuli will result in a similar train of thought, but given a generally lowered synaptic resistance it is obvious that these same sensory stimuli will not confine themselves to the same orderly path, but that in addition other associated neurones will be affected, which in the normal individual would remain quiescent. Ideation is confused and chaotic and conduct illogical, distractibility is intense, the nervous mechanism is out of control and feels the need of a stronger mind, hence its susceptibility to suggestion and hence the ready obedience to the commands of one for whom the hysterical person feels respect and has confidence in.

This would probably be the result of an abnormally lowered synaptic resistance. Now imagine the opposite—an abnormally heightened resistance. Is it not possible that the periodical disorderly discharge of nervous energy in some cases of epilepsy may be due to certain neurones

acting practically as storage batteries owing to the fact that the synaptic membranes which separate them from contiguous neurones present an abnormally great resistance to the passage of the impulse, thereby practically insulating these neurones until such time as the resistance is overcome by accumulation of energy or by the reception of a violent stimulus?

An important etiologic factor in the production of acquired epilepsy is alcohol. Now, alcohol spends itself principally on the interstitial tissue; it is therefore quite likely that its circulation through the nervous system will have a more injurious effect on the interneuronic junctions than on the neurones themselves; it is also quite probable that alcohol will have a selective action on different series of synapses in different individuals. A cirrhotic liver is rarely seen in the asylum *post-mortem* room, and the moral loss of tone so common in chronic alcoholics and the peculiar memory disorder may result from dissociation of the higher association centres, and consequent lack of orderly thought and judgement.

That a sudden fright is so frequently thought to be the determining cause of epilepsy is of much interest in the light of the suggestion that acute delirium as a result of shock may be due to what I have termed "synaptic fusion." It might be possible that this fright has caused minute structural change in certain synaptic junctions, perhaps in the way of upsetting the normal polarity or causing temporary insulation, if one might use the term, of neurones, and spasmodic and irregular nervous discharge as a consequence. The idea that epilepsy may be due to faulty synaptic conduction is a fascinating subject for speculation; for instance, if we suppose the channels connecting the cerebrum and cerebellum to be affected, then as a result of spasmodic action at these synapses, and consequent brief interruption of the normal inhibitory influence of the cerebrum, a condition analogous to decerebrate rigidity would result, and the clonic spasms succeeding the tonic are what we would expect to find before the establishment of association is again complete.

It is not, of course, suggested that in every case of epilepsy synaptic disorder is present; for instance, convulsions are readily produced in children by slight irritation of peripheral neurones (phimosis, etc.). This may be due to the distracting effect of these stimuli on cerebral neurones, resulting in temporary cessation of the normal inhibitory influence of the latter over cerebellar tonus.

My attention has been called to the fact that Dr. Stoddart has already expressed the opinion that there is increased synaptic resistance in acute confusional insanity. As his reasons, he gives the effect of repeated subliminal stimuli in overcoming the analgesia which is present, which he compared with Sherrington's experiments on the scratch reflex of a dog, and the fact that he has met with success in the treatment of some cases with strychnine.⁵ I have not found any benefit from the exhibition of strychnine in these cases; indeed, it was this fact which led me to think that some definite synaptic lesion had occurred. In epilepsy, on the other hand, I have found decided alteration, in the majority of cases, both in the number and intensity of the fits during the administration of strychnine—in 25 per cent. there was no apparent difference, in 50 per cent. the fits were decreased both in numbers and intensity, and in 25 per cent. they were increased. This inconstant effect is to be expected, as the pathological basis of epileptiform convulsions must undoubtedly vary greatly; for instance, it may be that the neurone is irritated through its blood supply either mechanically by vasomotor action or intravascular clotting, or by the direct effect of toxins, or it may be that the cortical neurones are themselves intrinsically abnormal. In any of these cases strychnine is not likely to have any definite effect; but if the sudden discharge of nervous energy is due to faulty synaptic action, either in the way of constant or spasmodic excessive resistance to the passage of the current, then it probably would have some effect on the number and intensity of the seizures. Bearing in mind Hughlings Jackson's speculation that post-hemiplegic muscular rigidity was due to the unantagonized action of the cerebellum, which is the centre for continuous (as is the greater brain for changing) movement,⁶ it seems possible that the convulsions resulting from excessive doses of strychnine may be due to some

kind of synaptic spasm affecting the cortico-cerebellar fibres. He says: "When the influence of the cerebrum is permanently taken off by disease of the cerebrum, as in hemiplegia from the parts which it most especially governs (arm and leg), the cerebellar influence is no longer antagonized, there is unimpeded cerebellar influx, and hence rigidity of the muscles which in health the cerebrum chiefly innervates."

In this connexion also the experiments of Ferrier on the electrization of the cerebellum of monkeys are of much interest;⁷ the ocular and other movements produced were similar to those occurring in epileptic convulsions. He found, on stimulating different portions of the cerebellum, backward, forward, and left or right movements resulted corresponding to certain definite cerebellar areas; also—an interesting fact in view of the involuntary cry so common in epilepsy—he found that irritation of the adjacent testes "invariably excites a sudden bark or howl." Now if we suppose faulty action at the cortico-cerebellar synapses to exist, allowing momentary unimpeded discharge from the cerebellar neurones, the result would be a tetanic seizure, becoming clonic as the cerebral influence resumed its sway, and the nature of the seizure would indicate which cerebellar area was primarily affected. Epileptics generally fall in each fit in the same way, so that when the eyes are moved upwards and the head thrown back the initiatory discharge would probably be in the *monticulus cerebelli*, as Ferrier has shown that such movements result from irritation of this area.

REFERENCES.

- ¹ C. S. Sherrington, *The Integrative Action of the Nervous System*.
² *Ibid.* ³ *Ibid.* ⁴ M. A. Starr, *Nervous Diseases*. ⁵ W. H. B. Stoddart, *Mind and its Disorders*, second edition. ⁶ Hughlings Jackson, *The Medical Examiner*, London, 1877-78. ⁷ David Ferrier, *The Functions of the Brain*.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SUTURE OF TENDONS.

OWING to the relatively unsuccessful results frequently following the suture of tendons, especially when closely adjacent tendons and their sheaths are injured, I have adopted the plan, after suturing each tendon, of surrounding it at the line of suture with a wrapping of membrane taken from beneath the shell of an egg sterilized by boiling. The tendon sheath is then closed.

I have seen no mention of this material having been used for the purpose of preventing the formation of adhesions, and venture to bring this method to notice as one easy of application, and one from which I have obtained improved results.

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A CASE OF ACTINOMYCOSIS TREATED BY A VACCINE.

THE following case, although not unique, may help to emphasize the curability, even in advanced cases, of that somewhat rare disease, actinomycosis. As is well known, the outlook in advanced cases of this disease is particularly gloomy.

The patient, a schoolmaster, aged 40, chewed a stalk of corn whilst taking a country walk last June. Throughout July he noticed a gradual deterioration in health; in August a definite swelling appeared above the outer part of Poupert's ligament in the left groin; this was a hard, fixed mass; shortly afterwards swellings appeared also between the shoulders and at the back of the neck. The tumour in the groin was incised at the London Hospital, and was found to contain the fungus of actinomycosis. As further swellings appeared he was sent home, and the case was considered and treated as hopeless. Potassium iodide, which had been given freely, was wholly without effect.

As the condition was at one time alleged to be the result of an accident on duty, I was asked to see him and report to the London County Council. When I saw him on November 16th, 1912, he was confined to his couch; he was pale, emaciated, with a large suppurating abscess in the left iliac fossa, a second in the left costal margin in the nipple line, and a third between the shoulders. His mental