tion. In these experiments there was a rise of the plethysmograph with each fall of pressure which indicated vascular dilatation, the result, we have no doubt, of the acid in the gastric fluid.

VII.

The pathology of gastric tetany has given rise to much speculation. Some observers have confined their attention to the alterations in the cerebro-spinal system, but their investigations have been for the most part negative. Slight alterations of brain structure have been observed, with thickenings of lining membranes and inflammatory exudations, but the changes in themselves cannot account for the nervous symptoms of tetany. The theory of dehydration of the tissues generally, from the constant bouts of vomiting and from the permanent hypersecretion of the gastric juice into the stomach, has now been discarded. The reflex theory likewise receives little support from recent investigations.

We are convinced that the only plausible theory is that of autointoxication. A poisonous substance is formed in the stomach, which, when absorbed into the blood stream in sufficient amount, gives rise to the tetanoid contractures and other concomitant symptoms which are well known in this disease.

Bouveret and Devic²¹ uphold this view, and although their results have not been confirmed by some other observers, we think that their elaborate investigations have done much to clear up the uncertain prevailing ideas on this affection. Their results are briefly as follows: they associate this form of tetany to that condition of the stomach in which there is a constant hypersecretion of gastric juice. Tetany, they say, results from an autointoxication, the poisonous substance or substances being produced by a chemical change in the stomach in presence of excess of hydrochloric acid.

They differ from Brieger,²² who considers that a peptotoxin is produced in the normal peptonisation of proteids, but they believe that in the presence of alcohol such a substance can be obtained. They found that the introduction of this peptotoxin, so prepared, caused violent tetanic convulsions of a nature precisely similar to those observed in man, and if introduced in weaker doses produced the ordinary contractures of tetany. They believe that in a dilated stomach, where there is an excess of free acid, this peptotoxin can be obtained, which when injected into animals causes convulsions. They proved this on several occasions. They do not know the nature of this peptotoxin, but they do not consider it to be either an ethylamine hydrochlorate or an ethylenediamine. They believe it to be a form of syntonin to which new properties have been added by the action of alcohol.

Gumprecht²³ declines to believe that autointoxication is the underlying cause in such cases, as he has failed to produce convulsions in animals by intravenous injection, although he has been able to separate an albumose which had toxic properties, but which he could not discover in the urine.

Ewald²⁴ has recently described a case where there was an entire absence of hydrochloric acid; and Sievers,²⁵ after making a survey of 27 cases, believes that hyperchlorhydria is not essential for the causation of gastric tetany.

We have found that a toxic substance existed in the stomach in the case under consideration, which when injected into an animal produced a marked fall of blood pressure and slowing of the heart beat. This substance was not present in the normal gastric contents of health; and, moreover, after neutralisation of the tetany fluid, practically no fall of pressure was obtained. We do not know what this toxic substance is; it has a marked acid reaction. Whether the brown colour and chloroform-like smell of the alcoholic solution are due to this material we cannot say. It is soluble in alcohol and in normal saline solution. By its injection into animals it causes either direct or reflex excitation of the cardio-inhibitory centre. It is, therefore, by no means improbable that other centres in the brain or cord are similarly excited.

As we mentioned before, the gastric contents were obtained after the patient was in a state of partial convalescence, and it is possible that had the fluid been obtained during the period of coma with contracture, this poisonous substance would have been present in greater amount, and would, when injected into the veins of an animal, have produced convulsions and death.

VIII.

We append a bibliography in chronological order. This is necessarily limited, and is confined almost entirely to reports on cases of gastric tetany. Special attention has been given to the more recent works on this interesting subject.

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CHANGES IN THE NEURONAL CENTRES IN
BERI-BERIC NEURITIS.

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By most writers on the subject of beri-beri the statement is made that there are no changes in the proper cells of the central nervous system. It seems incredible, in view of the profound changes in the spinal sensory and motor cells, that are now known to accompany the peripheral neuritis of alcoholism, which in so many respects resembles beri-beric neuritis, and experimental section or destruction of the axonal portion of neurons.

Scheube,¹ who appears to have missed no important point in the clinical and pathological aspects of the disease, has in a table of 20 cases recorded no lesion of the spinal cord more marked than "im mittleren Dorsalmarke Atrophie und theilweiser Schwund der Ganglienzellen der Vorderhorner." And, further, in his last published work on the subject² he has not changed his opinion in the matter. In fact the only authorities who appear to have observed central changes are Pekelharing and Winkler,³ although they failed to investigate the extent and to appreciate the significance of them. The relatively imperfect methods of their day for the investigation of cellular changes in the central nervous system was undoubtedly the reason.

My experience leads me to differ from most observers who have studied this disease. For in a careful examination of 8 cases of beri-beri I have found changes in those posterior

spinal ganglion and anterior horn cells which give origin to the degenerated nerves, and in the combined and hypoglossal nuclei of the bulb in those cases when the fibres from these parts were atrophied. The lesion is scarcely distinguishable from that found in cases of alcoholic polyneuritis.

It will not add to the usefulness of the present observation to give the clinical history of each of the 8 cases, or to detail the changes found in the muscles and nerves. The clinical aspect and morbid anatomy of the disease has been so thoroughly handled by the above-mentioned authors that it is not necessary. But there follows a complete history of Cases i and viii, and a tabulated statement of the main clinical and pathological features of the other cases. I have adopted Scheube's clinical divisions of the disease as being the best.

It may be well to say that these 8 cases are not picked, but are the first 8 cases that I have had an opportunity to do a necropsy upon since my arrival in the Malay Peninsula. I shall state as briefly as possible the nature and distribution of the changes in the central nervous system of the 1st and 8th cases to come under observation.

CASE I.

Clinical History.—C. S., male, Chinese, aged 31, married, a mining coolie, was admitted to the District Hospital, Kuala Lumpur, on August 7th, 1900; died on August 28th, 1900.

The following notes were taken on August 21st, 1900. Patient was born 31 years and 3 months ago at Wai Chow, China, and lived there for thirty years. He had an attack of dysentery for six days at the age of 20, and fever occurring every second day for one month at the age of 24. He went to school for six years. He married when 26. No children. Lost

Fig. 1.—Nerve cells showing chromolytic changes from the combined glosso-pharyngeal, pneumogastric, and spinal accessory nucleus, and the hypoglossal nucleus in beri-beri. Case I, C. S., aged 31.

his mother when 23 and father when 28; does not know of what they died. He is farmer by occupation. His usual diet is rice, dried fish, sweet potatoes, and pork once in ten days. He came to Selangor in the Malay Peninsula one year and a-half ago as an immigrant coolie. When he arrived in Simpang (a mining district in Selangor), he was 30 years of age. After working there for three months in the deep mine he felt some pain and heat in the heart region which lasted for four days, but he recovered after the use of Chinese drugs; he then continued his work for another eleven months without being ill. He then contracted fever which lasted for four days; he was treated by a native doctor. When he got better of fever a slight numbness was noticed in his hands and feet. Daily he grew worse, and after fifteen days he lost power in his hands, forearms, feet, and legs; and further his voice grew weak. He can answer questions to-day, but it seems as though he will lose his voice.

August 24th, 1900. Patient has lost all power of speech.

August 27th, 1900. Patient is speechless to-day. When he attempts to speak there is only a gurgle in his throat and associated movements of

hands and shoulders. He lies on his back, legs extended, feet turned outward, arms and fingers flexed, respiration laboured, cardiac impulse marked.

Nervo-muscular System.—Patient cannot speak, but is quite clear in his mind, understanding all questions put to him and recognising all articles shown to him. Protrudes his tongue not further than his lips, but straight. There is no observable paralysis of the lips. Hearing is normal; sight also. Internal and external ocular muscles are normal. He cannot walk or sit upright; can flex his toes voluntarily, but extension is slightly *minus*. Extension and flexion of ankle are *minus*. Extension and flexion of knee and flexion of thigh are markedly weakened. There is also marked weakness of all muscles of hip-joint except the rotators. Abductors of thighs are slightly weakened. Adductors are almost powerless. Plantar, cremasteric, and abdominal reflexes absent; knee-

Fig. 2.—Nerve cells showing chromolytic changes from the lateral and median groups of the anterior horns of the lumbar and sacral regions of the spinal cord in beri-beri. Case I, C. S., aged 31.

jerks absent; Achilles absent. All muscles of legs react sharply to tap of pleximeter. There appears to be delayed tactile sensation over whole of lower extremities. Sensation to pain is present over lower extremities, but it is delayed for quite a second. Tactile and common sensibility of trunk are normal. Sensation to temperature is apparently normal. His leg muscles are flabby, but there is no specific wasting. There is no ankle or rectus clonus. Hand grasp on both sides very much weakened; cannot spread his fingers. There is diminished power in all movements of hands and wrists; can only oppose thumb and first two fingers. Flexion and extension of wrists and elbow markedly weakened;

shoulders also are weakened. There is considerable tenderness in muscles of forearms, hands, legs, and thighs, but not of shoulder or trunk muscles. Deep reflexes of forearm increased. Hand reflexes slightly *plus*. Triceps reflex seems to be absent. Biceps is *plus*. Muscles of shoulder girdle react smartly to direct stimulation. The vocal cords are in the "cadaveric position" and motionless. There appears to be complete paralysis of all vocal muscles; when he attempts speech he makes a blowing, laryngeal sound.

August 27th, 1900. Heart beat is very rapid; pulse full and bounding. Twelve ounces of blood were abstracted this morning at 11.30 A.M. to relieve circulation. 3 P.M. Patient breathing easier; pulse full but softer than this morning. Is resting easier; no pyrexia.

August 28th, 1900. Patient died suddenly at 4 A.M. this morning.

Fig. 3.—Ganglion cells showing chromolytic changes from posterior root ganglia (fifth lumbar) in beri-beri. Case 1, C.S., aged 31.

Post-mortem Notes.—Date and hour of death: 4 A.M., August 28th, 1900, at District Hospital. Date and hour of necropsy: 10.30 A.M., August 28th, 1900, at District Hospital. Numerous papery scars on shins, leg, and ankles; inguinal glands hard and shotty; old scar on penis. Fingers flexed; feet everted. Pupils 4 mm., but equal and regular. Marked oedema over dorsum of both feet; slight over both shins. Calvarium asymmetrical; the left parietal eminence is higher than the right; sagittal suture is bent well to the left; right half less capacious than left; right hemisphere weighs 13½ ozs.; left, 14 ozs.; convolutions are well marked; striæ of cortex well-marked; cerebellum, pons, and medulla show nothing to naked-eye observation; left pleura adherent throughout by moderately recent stiff adhesions; surface of upper lobe of right lung shows numerous patches of collapse; lower lobe in advanced stage of broncho-pneumonia; bronchi of left upper lobe filled with fluid, bubbly mucus; its tissue floats; lower lobe is in a stage of red hepatization; its tissue sinks; right ventricle and auricle of heart have flabby thin walls; walls of left ventricle and auricle are slightly fatty and very flabby; no hypertrophy; wall of left ventricle measures 12 mm.; cavity is slightly dilated, and contains small amount of dark clot, weight 11 ozs.; coronaries are normal; the pericardium contains a moderate amount of clear fluid; stomach is moderately dilated; gall bladder is full of dark green bile; liver is slightly fatty, soft, and friable, weight 2½ lbs.; spleen is large and round-edged, weight 17 ozs.; large intestines are moderately congested throughout; capsule of left kidney strips readily leaving slightly eroded surface; right kidney and capsule same as left, weight 4½ ozs.; cortex 4 mm.; pyramids are congested, weight 5 ozs.; muscles of lower limbs and feet very oedematous, pale and flabby.

MICROSCOPIC EXAMINATION.

Nerves.—The trunk of the left hypoglossal contained about thirty fibres moderately atrophied. The right nerve was not examined. No changes were found in the phrenics.

The pneumogastrics in the neck (the left was more particularly examined) were found to contain only a few degenerated fibres. The appearance of the fibres, as stained by Marchi's method, teased, and mounted in cedar oil, is characteristic.

In removing the lung the right inferior laryngeal was lost. The left, however, was carefully dissected to the inferior constrictor; beyond this I neglected to trace it unfortunately. But it contains a large number of degenerated fibres; three-quarters of the whole number, I think. The majority are in an early stage of atrophy. The end of the nerve nearest the vagus contains not so many, and only slightly changed fibres. Here, as in all the other nerves examined, the grossest alteration is in the distal end of the trunk. It is in a ratio corresponding to the distance of the axons from their nutritional centres.

It is not necessary to particularise the nature or extent of the changes in the other nerves. All purposes will be answered by stating that a few fibres are degenerated in the musculo-cutaneous; a moderate number in the anterior tibials, crurals, posterior tibials, and right plantar; a small number in the terminations of the ulnars; and a more noticeable number in the lower parts of the medians. These nerves were examined by the Marchi method only, each nerve being teased out and mounted in cedar oil. Scheube, Bälz, and others have so adequately detailed the nature of the changes in the support tissue as well as in the fibres that I did not go further in my observation.

From the above it will be seen that the sensory and motor fibres of the third, fourth, and fifth L.; the first, second, and third S.; and the third, fourth, fifth, sixth, seventh, and eighth C. spinal segments have been examined and found in a partial state of atrophy, and that fibres from the hypoglossal vagal and bulbar portion of the spinal accessory nuclei were also atrophied. The chief interest, however, lies in the trophic centres for these various axons.

Cerebrum.—Neither Nissl's, Unna's, or Schäfer's tangential fibre stain show anything abnormal; the cerebellar cells are also healthy.

Medulla.—In view of the loss of speech that was a prominent feature in the case, I carefully examined the glossopharyngeal-vago-accessory nuclei as well as the fibres of the inferior laryngeals. Affection of the latter, it may be remembered, was first observed on October 21st. The voice was wholly lost on the 24th. On the 27th I made a laryngeal examination, and found the vocal cords in the "cadaveric position." The patient died early on October 28th, so that a week elapsed between the first observed affection of speech and death, during four days of which there was abundant evidence of complete paralysis of the vocal cords, and it is reasonable to infer that a number of laryngeal fibres were degenerated before the paralysis was observed.

Cells of the olivary body are normal, also those of the eighth nucleus higher up in the bulb. It is quite out of the question, therefore, that the cells shown in Fig. 1 mean more or less than that their axons are in some way unsound. The cells are from the hypoglossal and combined glossopharyngeal-vago-accessory nuclei. One or two were observed in each section of the hypoglossals and from two to five in the lower part of combined nuclei. The vagal portion of the nuclei contained never more than two in a section, and in but one section through the bulbar accessory nuclei did I find more than four. Only rarely did I observe any cells so changed in the upper part of the combined nuclei.

Cervical segments 3, 4, 6, and 8 were examined by Nissl's and Unna's stain. I may say here that when in doubt about an appearance in the cells by Nissl's stain I invariably use Unna's, as I find that though the use of the latter does not result in such fine sections as the former, it will nevertheless often show chromophilic bodies in the dendron quite plainly where the former shows them but faintly.

No positive changes were observed in C. 3 and 4. It will be remembered that the phrenics are normal.

In C. 6 and 8 a few cells are to be seen in all sections in a state of moderate chromatolysis, nuclear dislocation, and over-staining. They are almost wholly confined to the lateral

No.	Name. Sex. Nationality. Age.	Type of Disease. Duration of Symptoms.	Nerves found Atrophied.	Cells found Changed.	Remarks.
1	L. K., male, Chinese, 26	Hydroatrophic; five months	Plantars, anterior and posterior tibials, musculo-cutaneous of legs, a few fibres in upper part of sciatics in legs, anterior crurals, terminal portions of ulnars and medians	About a quarter of the cells in the anterior horns of 2, 3, and 4 L., and of the 1, 2, and 3 S. are changed; also a few in the 4, 5, 6, 7, 8 C. and 1 T. In the 3 and 4 L. posterior spinal ganglia and in the 1, 2 and 3 S. are many changed cells; none in medulla and cerebrum	See text.
3	C. Q., male, Chinese, 28	Hydroatrophic; five months	Plantars, anterior tibials, mus- culo-cutaneous, numerous fibres in lower parts of sciatics, an- terior crurals, medians, ulnars	In 7 C., 3, 5 L., and 1, 2, and 3 S. posterior spinal ganglia many cells are changed. A large number of cells in the anterior horns of cor- responding spinal segments are also changed	—
4	T. T. H., male, Chinese, 27	Hydroatrophic; seven months	Plantars, anterior and posterior tibials, musculo cutaneous, an- terior crurals, ulnars	The 5th, 6th, and 7th pairs of C. ganglia con- tain a few changed cells; 3rd and 5th L. and 2nd, 3rd, and 4th S. ganglia contain a large number of changed cells; 6, 7, 8 C. segments contain a few altered ventral cells; the lum- bar and sacral segments a large number	In this case there was marked atrophic paralysis, hyperes- thesia, and hyperalgesia of the lower limbs
5	C. H., male, Chinese, 26	Acute pernicious; 27 days	Plantars, anterior and posterior tibials, anterior crurals, me- dians, ulnars, pneumogastrics (a few)	Intense and widespread atrophy in 3, 4, and 5 L., and in 2 and 3 S. ganglia. Most of the ventral cells in 2, 3, 4, 5 L., and in S. 1, 2 markedly changed. I discovered no changed cells in combined nuclei	This patient had only slight oedema of lower 3rd of shins. Marked hyperesthesia and hyperalgesia, complete para- lysis of movements of feet, legs, and hands. He died after a severe cardiac crisis.
6	C. S., male, Chinese, 25	First attack when 13 years old. Numerous (12) relapses up to last attack four months ago: hydro- atrophic form	Plantars, musculo-cutaneous, posterior and anterior tibials, sciatics (a few fibres), anterior crurals, median and ulnars, phrenics	All the lumbar and the 1, 2, 3, 4 S. Segments contain numerous deeply pigmented and slightly altered cells. The cervical segments not examined. Only a few cells changed in 3, 4, 5 L., and 1, 2, 3 S. ganglia	This man died of hydroperi- cardium and dilated fatty and weakened heart. There was little sensory disturbance beyond slight anesthesia, an- algesia and tender calves during life.
7	P. L., male, Chinese, age not known	Hydroatrophic: four years ago disease began: was cured apparently: three relapses since	Plantars, musculo - cutaneous, anterior crural, sciatics slight, ulnars and medians slight, pneumogastrics slight, phrenics slight	A few in the 4 and 5 C. ventral horns; a larger number in the lumbo-sacral segments 3, 4, 5 L., and 1, 2, 3 S. The ganglia were not saved in this case. In the lower part of the com- bined nuclei a few changed cells were found	I did not see this case till the necropsy, and the only clinical note I have is a "previous history."
8	—	—	—	—	See text.

groups. Only rarely is one to be seen in the median groups. In no sections did I observe any changes in the cells of the posterior horns.

Lumbar 1, 2, 4, 5, and S. 1 and 3 were observed by the above methods. Fig. 2 shows the nature of the change in many cells in the lateral and a few in the median groups. Under any power of the microscope the change is not distinguishable from that in cells I have from cases of chronic alcoholic polyneuritis. The cells are not in any instance disrupted, although in *a*, Fig. 2, disintegrating does not seem far off. The most marked chromatolysis is in the middle portion of the cell body, from which position the nucleus has shifted. I have observed no vacuolation, and no cell in median plane minus a nucleus or nucleolus.

The posterior spinal ganglia corresponding to the above-mentioned spinal segments were examined, and in them is to be seen a confirmation of the fact that change in the axon is accompanied, or soon followed, by change in the neuronal body.

Fig. 3 shows how closely the change resembles that found in a similar situation in alcoholic neuritis.

Nothing of this change was to be seen in the third and fourth cervical ganglia. In the sixth and eighth pairs about ten changed cells were observed in each section. In no case was there complete extrusion of the nucleus or vacuolation, only a moderate chromatolysis affecting chiefly the centre of the cell and the immediate environment of the dislocated nucleus, and a general overstaining of the hyaloplasm.

The lumbo-sacral ganglia contain more cells, more violently disturbed. The nature of the change is shown in Fig. 3; it is more pronounced however. But it is rare to see more than two altered cells lying close together. The rule is to find them diffusely scattered throughout the ganglia.

It appears to me that I have said enough to prove that beri-beric neuritis is no exception to the rule that governs in other neurites, and in the various forms of experimental interference with axons, namely, that atrophy or loss of function in the axon leads to or is concomitant with disturbances in its trophic cell. But for fear that some critic might suppose that in the hours that elapsed between death and the necropsy

there was time for a *post-mortem* change to intervene, which simulated the *ante-mortem* change of alcoholic neuritis or section of axons, I will introduce in detail case No. 8. The necropsy on this case was made ten minutes after death, the nerve immediately hardened in 1 per cent. osmic acid and eight hours later teased in cedar oil and mounted. Further, sections from several of the cervical and lumbo-sacral segments and ganglia were fixed immediately after death and cut and stained within twenty-four hours, so that there was no possibility of *post-mortem* change occurring, or change from too long an immersion in absolute alcohol.

The following is the clinical history and the main features of the macroscopic and microscopic changes:

CASE VIII.

C. C., male, Chinese, aged 25, unmarried; occupation, mining coolie admitted to District Hospital on October 13th, 1900; died on October 16th 1900.

The following notes were taken on October 20th and throughout. Patient was born in Canton (China) about twenty-five years ago, and lived there for eighteen years. Was told that he had fever for six days when he was 6 years of age; except for this he was in good health while in China. By occupation he was a jeweller. He was not exposed to rain or wet more than ordinarily. His usual diet was rice, fresh and dried fish and vegetables; beef and pork six times a month; duck and fowl on festival days only. Seven years ago he was brought by Towkay San Chong to Selangor in the Malay Peninsula, and worked for him in the surface mines for one year. He was much exposed to both wet and rain when at work. For the other six years he worked in various parts of Selangor in the deep mines, and was while at work much exposed to wet and rain. Four years ago he had a chance followed by bubo; he was cured by Chinese medicine. Two months ago had fever and diarrhoea; the former lasted for two days, and the latter for a month. Both were cured without taking any medicine. Twenty days ago (about October 1st) slight oedema appeared in the legs, but did not increase. Two days after the first appearance of the oedema a slight weakness and numbness was felt in the lower extremities, the latter commencing in the dorsum of the feet and extending to the knees. Shortly after the arms from shoulders downwards became numb and weak, gradually increasing in intensity. For thirteen days after the weakness and numbness came on in the legs he was able to walk slowly; during the last seven days, however, he has not been able to walk at all. He was admitted to this hospital on October 13th, quite helpless in his legs. Parents are still alive. States that this sort of disease never affected his parents. Has been an opium smoker for the last six months only. Began by using half a chee, now he smokes one chee. He says that he was a very strong stout man, but has grown rapidly thin during the last month.

October 22nd, 1900.—Patient extremely emaciated (Fig. 4) lies on his

back, legs and feet rolled outwards; the latter are dropped. Face wears an anxious expression. Seems to be suffering great pain. Speech not affected. Can hear and see all right. Says that he has no taste in his mouth when he eats. He cannot distinguish between salt and sugar. He cannot walk. He can voluntarily slightly flex his toes; movements readily overcome when he attempts to extend his toes. There is scarcely any movement in the toes themselves, but noticeable associated contraction in the extensors of the leg. He cannot spread or move his toes separately. There is no power to further extend the feet, as they are in the position of complete extension. Power to evert the feet is lost absolutely. Flexion is only slightly performed; the slightest pressure overcomes it. Extension of the leg is almost *nil*, leg falling flat from the flexed position when voluntary extension is attempted. Power of flexion is markedly weakened. Abduction and adduction of thighs are both markedly weakened. Internal and external rotation of thighs extremely weak. Internal rotation is accompanied by a coarse tremor. The thighs rotate outwards by their own weight. Flexion of the thighs upon the body cannot be performed. When the thighs are placed in the flexed position they cannot be retained so. He seems totally unable to raise himself in sitting position. There is certainly considerable diminution in the power to flex the body on the thighs. Extension of the body on the thighs cannot be tested.

He can partially spread his fingers, but movement is very weak; can slightly roll his thumbs; can oppose his thumbs to all of his fingers, but the movement is extremely weak and slow. Extension and flexion of phalanges and of wrists are both markedly weakened, and apparently equally; can make no impression on the dynamometer with either hand. Supination and pronation are voluntarily performed, but can be overcome by the slightest opposition. Flexion and extension of arms can be performed willingly, but the movements are readily overcome. Elevation of the shoulder is weakened, also internal and external rotation. The last stage in the movement of all limbs is markedly accelerated by their own weight. He seems unable to lift his head from head rest.

When he is lifted to the sitting position his head seems to be very unsteadily poised. External and internal ocular muscles are normal. There is marked wasting of all muscles of the body except the facial. The abdominal and intercostal muscles are extremely wasted. Deep pressure of all parts of legs, feet, hands, and arms is resented, and the parts appear to be extremely tender. Deep pressure of shoulder-girdle muscles does not cause pain. Deep pressure of abdominal muscles causes pain; also of the spinal muscles. Pectorals seem to be free of pain. There is slight oedema of dorsum of feet, especially at the roots of the toes. Oedema of the ankles is more marked. There is slight oedema of the back of the hands.

Face has a pasty look, but is not cedematous. All passive movements of the body are protested against, and they undoubtedly causes considerable pain. There is no inco-ordination present, only irregularity from weakness.

Right calf measures 24 cm., left 23.5; both thighs above the knee 23.5 cm.; middle of right and left upper arms 16.5 cm.; right and left forearms 16 cm.

Ocular reflexes are normal. Neck muscles react smartly to the slightest tap on the skin. Irritability of the facial and scalp muscles is slightly increased. All the deep muscles of shoulder girdle and arms react more than readily to direct stimulation. The muscle fibres heap into a small rounded mass where they are tapped by the pleximeter. The intercostals and abdominals are over active to direct stimulation. All deep muscles of the lower limbs except the intrinsic of feet are extremely irritable to direct stimulation. Knee-jerks are nevertheless totally absent; so also is the Achilles. Plantar, cremasteric, and abdominal reflexes are normal.

Tactile sensation: A very light touch by cotton wool is not felt in any part of the right leg two inches below the knee. The upper limit of this partial anaesthesia is strikingly defined. If the stimulus, however, is the weight of the Sierring's aethesimeter, it is readily felt on any part of the leg, but more so in the neighbourhood of the knee-joints. I can find no areas of total anaesthesia in either leg. He recognises the quality of the stimulus when he is touched with cotton or the smooth surface of the aethesimeter. The lightest touch of cotton is recognised anywhere on the right thigh except in the groin over the genito-crural distribution. The reduction in sensation in the latter place is the same as in the leg and foot.

Stimulation by cotton wool is felt everywhere on the left leg, except the inner hollow of the ankle, dorsum of the toes, sole of the foot, outer edge of the foot, and over the middle of the belly of the gastrocnemius. But in the enumerated areas more marked stimulation by cotton is felt and its quality distinguished. There seems to be no change in tactile sensation of the abdomen, loins, thorax, neck, back, face or scalp. The upper limbs are hyperaesthetic.

Common sensibility is absent on the plantar and dorsal surfaces of the great and second toe of the left foot. The rest of the left limb is hyperaesthetic. The sole of the right foot seems to be normal; also the plantar surface of all the toes. The rest of the limb is decidedly hyperaesthetic. There is slight hyperaesthesia of the whole and of both upper limbs and of the abdomen; the rest of the body appears to be normal.

Tactile area on point of chin $\frac{1}{2}$ of an inch, dorsum of right foot $2\frac{1}{2}$ inches, front of right leg $1\frac{1}{2}$ inch, dorsum of left foot $1\frac{1}{2}$ inch, front of left leg $3\frac{1}{2}$ inches. It would seem therefore that there was some slight anaesthesia of the lower limb.

Thermal stimulation is correctly appreciated in all parts of the body. Location of all modes of stimulation is correct.

Tongue is moist and red. Appetite poor; says that he does not care to eat. Bowels are regular. There is slight tenderness of the stomach on deep pressure. Spleen and liver not enlarged. Abdomen is very thin; abdominal aorta can be palpated in all its course, and the vertebral column is very prominent. There is pain on deep pressure in the right and left iliac regions. There is slight pericardiac impulse in the second to fifth left interspaces. Area of dullness is not increased. Under the hand the first contraction of the heart is weak, and is followed quickly by the second sound; the pause seems to be prolonged. Apex cannot be palpated; maximum of apex beat under fifth rib half an inch inside nipple line. At this point the first sound is reduplicated, and accompanied by a deep-tone, slightly roughened *bruit*. Second sound is sharper, louder, and better defined than first sound. The systolic *bruit* can be just made out in the left axilla. Aortic first sound is sharp and clear; pulmonary second is slightly reduplicated. There is slight pulsation in the veins of the neck. Pulse in recumbent position 120, full, but easily compressible; goes up to 144 on slightest exertion.

Breathing is costo-abdominal, but more costal than abdominal; the upper three intercostals scarcely act at all; the lower ones appear to do most of the work.

Here one has to deal with a clear case of atrophic beri-beri; extreme weak-

ness of the limbs and other parts of the body and atrophy of the muscles. Fig. 4 is a photograph of the patient taken on October 18th; it is characteristic of the atrophic form of the disease.

The following chief *post-mortem* notes will confirm the classification of the case:

Post-mortem Notes.—Date and hour of death, 9.30 A.M. November 15th, 1900, at District Hospital; date and hour of necropsy, 9.40 A.M. November 15th, 1900, at District Hospital. There is general emaciation; pupils greatly dilated, but equal; feet and lower two-thirds of legs slightly cedematous; also the dorsum of hands and fingers; scrotum slightly cedematous; two small scars on each side of frenum preputii; numerous small scars about ankles; dura

Fig. 4.—Photograph of C. C. (beri-beri, Case VIII) admitted to hospital October 15th, 1900, died November 16th, 1900; showing dropped feet and muscular wasting.

mater not adherent; blood vessels almost empty; there is moderate subarachnoid oedema; pia-arachnoid membranes slightly opaque; cortex pale; striæ not very plain; slightly cedematous; alkaline in reaction; white matter pale and slightly cedematous; alkaline in reaction; perivascular spaces slightly dilated; grey matter same as white; lateral ventricles moderately dilated with clear fluid. Cerebellum slightly cedematous and alkaline in reaction. Fourth ventricle slightly dilated with white clear fluid. Right pleura adherent at apex. Left pleura adherent at apex and throughout by moderately recent stiff adhesions. Right lung riddled with small tubercles; one at the apex the size of a hazel nut; bronchi of this part filled with thick yellow pus. Lower lobe slightly congested, but free of tubercles. Left lung riddled with tubercles ranging in size from a hazel nut to a pin's head. Heart normal in position and small; its blood vessels are dilated and slightly thickened. Right ventricle and auricle slightly dilated; but there is no evidence of hypertrophy. Cavity contains a small amount of mixed clot, and a large amount of semi-fluid blood. The walls are thin, and the muscle at the apex extremely fatty, some of the fat having undergone mucoid degeneration. Left ventricle and auricle contain a small amount of semifluid dark clot. Walls are not hypertrophied, but are slightly greasy. The pericardial sac contains about two ounces of clear fluid. Visceral layer contains here and there small milky patches. Stomach slightly congested. Gall bladder full of light green bile, which flows freely. Liver is soft, flabby, slightly fatty and nutmegged, also slightly cedematous. The omentum is wasted; abdomen contains about 8 ounces of clear fluid. Spleen small and wrinkled, cuts with resistance, is dark chocolate in colour, firm, and slightly fibrous. The duodenum is bile stained. Every 8 or 10 inches of ileum contains an ulcer round or longitudinal about the size of a sixpence. The caecum is riddled with ulcers ranging in size from a pin's head to a pea. Mucous membrane is congested and slightly cedematous throughout the whole length of the gut, and every inch or so contains a small crater-like ulcer. Capsule of right kidney peels readily, leaving a dark congested surface; pulp is deeply congested and slightly cloudy. Left kidney same as its opposite fellow,

Bladder is distended with urine. Muscles of the legs and subcutaneous tissue markedly cedematous. The median nerves in the palm of hands and the anterior tibial in legs and feet are markedly cedematous.

MICROSCOPIC EXAMINATION.

Nerves.—There are a few degenerated fibres in the main trunks of the pneumogastrics. In a teased-out specimen I counted only three moderately large fibres in an early stage of atrophy. There are no abnormal fibres in the peripheral portions of the hypoglossals. Quite half the phrenic fibres a few inches above their junction with the diaphragm are in various stages of early degeneration. In a teased-out specimen of the midriff the atrophy of the nerve fibres is salient.

Fig. 5.—Degenerated ganglion cells in posterior root ganglia of lumbosacral region in beri-beri. Case VIII, C. C., aged 25.

The condition of the phrenics surprised me. For when I saw the patient on October 20th the diaphragm was working well. The dresser in charge informed me, however, that abdominal respiration had practically ceased for several days before death, and that there was marked retraction of the abdomen during inspiration. The acute atrophy in the fibres of the phrenic bears out this observation.

In the manual portions of the medians and ulnars there is a widespread, and in nearly all of the fibres an advanced, atrophy. Near the plexus the degeneration is fully as great as in the lower portion of the nerves.

Where the sciatics divide into internal and external popliteal it is rare to see a healthy fibre. Many are reduced to strands of neurilemma. The anterior tibials and the branches to the peroneal are grossly wasted. There are also many atrophied fibres in the posterior tibials, and in both internal and external plantars. The posterior nerves, however, are not so degenerated as the anterior. In the upper part of the trunks of the anterior crurals a few sound fibres are extant. But the great majority are in some stage of atrophy.

The axonal atrophy in this case is therefore profound, and it was no surprise to find their trophic centres relatively affected.

Cerebrum.—Quite normal in spite of the slight edema and paleness of the grey matter noticed at the necropsy. The cerebellar cells are also sound.

Medulla.—The hypoglossal cells and those of the combined nuclei are beautifully stained and normal in appearance. The hypoglossal nuclei are therefore in keeping with the axonal prolongations of their cells, that is healthy. That there are no changed cells in the combined nuclei in spite of atrophy in the vagi is apparently contradictory. But it is quite possible that I may have overlooked the cells that the few atrophied vagal fibres centre in.

Spinal Cord.—In the third, fourth, fifth, seventh, and eighth cervical segments there are many changed cells. They are confined to the anterior horns, and are almost equally distributed in the various cell groups.

There are appearances in the lateral and anterior subgroups of C. 5, 7, and 8 that point to a total destruction of some cells. In the first place none of the subgroups in these parts contain more than two or three cells, all extremely wasted. In some thirty sections I did not discover a single normal cell in the lateral and anterior subgroups in either ventral horn. There are no signs such as empty pericellular spaces to suggest that the cells have dropped out. The atrophy of the muscles in this case was marked and rapid, and there is gross degeneration in the ulnar and median nerves. This may be sufficient to confirm the idea that there has been a total atrophy and removal of many ventral cells from the seventh and eighth segments. But it is contrary to experience. For it is well known that there may be atrophy of long standing in nerve fibres without total destruction of their parent cells. Here, however, one has to deal with an acute pernicious case of beri-beri, and it is just possible that in this type of disease the affected nerve tissue disappears rapidly.

None of the other cases showed signs of total cell atrophy. But they are all, except No. 1, chronic cases. Only a further study of the central nervous changes in beri-beri will clear up this point. The few cells yet extant are changed as are those in the previous case, and so need not be more particularly described.

In L. 1, 3, 5, and S. 1, 2, 4, there are many changed cells in the motor groups. None of the other cases exhibit so general or so marked a degeneration in individual cells. A large number of cells are wholly devoid of both nucleus and nucleolus. I thought at first that this was due to the knife having passed above or below the centre of the cells. But in a series of twenty sections at 10.0 from the fifth lumbar segment I could find no signs of nucleus or nucleolus in eight different cells in the lateral and anterior subgroups. The processes of such cells are greatly wasted, though scarcely ever ruptured, and they are wholly devoid of chromophil granules. The hyaloplasm is either palely or darkly homogeneous, and there are vacuoles in a few. It seems to me that this is another proof of a rapid and severe process of degeneration operating in the cells of the anterior horns in this case.

This view is confirmed by the state of the cells in the posterior spinal ganglia. I have never before seen ganglia so profoundly altered as are the lumbosacral in this case. Scarcely a cell in L. 4 and 5; and S. 1, 2, and 3 is normal. More than half the cells have dislocated nuclei, and very dark or light homogeneous hyaloplasm; a few nuclei have burst and the nucleoli have disappeared.

A large number of cells are more positively degraded. Vacuolation, shrinkage, increased density of the whole cell body, and actual disintegration are common. It seems, too, that many cells have disappeared. There appears to have been a severe and rapid cellular atrophy in operation. Fig. 5 shows the types and the extreme nature of the change in many cells. (See Table on p. 1613.)

Here, then, is evidence enough that the neuritis of beri-beri is not radically different from other parenchymatous neurites, and of the larger truth that the various parts of each neuron are interdependent, disturbance or destruction of the axonal prolongation resulting in disturbance or destruction of the nutritional centre.

The profound atrophy in the spinal anterior horn and posterior root ganglionic cells of Case VIII is no doubt the natural and usual concomitant of rapid and extreme degeneration of the nerve fibres that occurs in cases of acute pernicious beri-beri. The beri-beric poison probably works so forcibly on the neuron that its stability is wrecked and the

whole structure soon destroyed. Recovery is perhaps impossible from the first. One has only to see a strong coolie enter hospital with slight cedema, weakness of muscles, and sensory disturbance to observe the rapid progress of the paralysis and wasting of the limbs and often of the diaphragm, with sudden death from respiratory or heart failure in spite of all symptomatic treatment to feel that only a specific will be of use in such cases.

The dislocation of the nuclei and slight breaking down of the chromophilic granules in the cells of all these cases is probably a pseudo-degeneration and the cause, so far as the afferent side of the nervous system is concerned, of the hyperæsthesia, paræsthesia and algesia constantly observed in this and other forms of peripheral neuritis.

Probably the atrophy of the fibres accounts for the total and partial anæsthesia, and the changes in the cells by modifying the incoming impulses accounts for the hyperæsthesia and paræsthesia and delayed sensation.

The important point here, however, is that in beri-beric neuritis there is more or less change in the bodies from which the atrophied nerve fibres take origin. This is true of both spinal and bulbar neurons.

REFERENCES.

¹ Die Beri-beri Krankheit, Jena, 1894. ² Die Krankheiten der warmen Lander, Jena, 1900. ³ Beri-beri, trans. Jas. Cantlie, 1893.

FARADISATION OF THE HEAD IN THE TREATMENT OF CHRONIC INSOMNIA AND ASSOCIATED NEUROSES.*

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HITHERTO electric treatment applied to the head has practically always been by the galvanic current. All drugs, even tonics, are to be stopped during the treatment here recommended. For a few hours after each *séance* all mental excitement and mental work should as far as possible cease, and no physical fatigue should be permitted. The cause of the symptoms, if known, should be, at least in part, removed. Tobacco and alcohol, except in rare cases, need not be wholly omitted; but no excess should be sanctioned. In cases where the cause is in acute operation at the time, and cannot be removed, I have found very little benefit result. Patients must be discouraged from talking while the current is in action, and must sit quietly for a few minutes after the electrodes are removed from the head, otherwise alarming, though not dangerous, symptoms are apt to follow. Nearly all my cases—46 in all—have been not of disease, but of disordered cerebral function. Coexisting disease has sometimes materially modified the result, since the insomnia has been in some of these cases what Sir James Sawyer has called secondary insomnia, as distinguished from psychic or emotional insomnia.

Amongst the neuroses associated with the insomnia are what patients have called nervousness, headaches, a nervous fear, lightness in the head, fearful dreams, night startings, a feeling of confusion when adding up figures, a feeling of thickness in the head, creepy feelings in the head, etc.; whilst in nearly every one of them the primary complaint has been loss of sleep or imperfect and unrefreshing sleep: in all the cases the insomnia had lasted for a considerable time, from a month to a year or two.

The results I have tabulated under five heads:

1. Cases in which the patient—he or she being judge—has been absolutely cured or practically cured, 21 (45.5 per cent.).
2. Cases in which the improvement has been so great as to make the treatment well worth the trouble, 15 (32.5 per cent.).
3. Cases in which improvement, though present, has been slight or of short duration, 5 (11 per cent.).
4. Cases in which no appreciable result of any kind was obtained, 4 (9 per cent.).
5. Cases in which some harm was done, though this was of a temporary nature, 1 (2 per cent.).

Under column 1 are included cases in which the sleeplessness,

* Extracts from a paper read at a meeting of the Glasgow Medical-Chirurgical Society.

or other nervous distress, has never returned since the treatment, or has returned only after a considerable time, and its return has been due to a return of the primary cause; and even then only when the cause has been in operation over a considerable time. The more pronounced the coexisting conditions, such as pain, the less satisfactory has been the result; whilst in the purely psychic cases every one was either absolutely cured or had so materially improved as to make the good result well worth the trouble involved. Some of the unsatisfactory cases had only one application.

No "shock" is felt by the patient, only a soothing feeling being experienced. In some cases the improvement has remained, but has not increased when the treatment was stopped, and the patient has come back anxious to have it resumed. The first application has been sometimes attended with unfavourable results, but a smaller dose at the next sitting has proved successful as a sedative. In one case the treatment was unsuccessful on account of the patient taking large doses of quinine for influenza. A successful result followed when the quinine was omitted.

No result followed in neurasthenia without the Weir-Mitchell treatment; but where the latter failed to relieve the insomnia faradisation was followed by most satisfactory results.

Regarding the probable *modus operandi* of this treatment, I believe that the influence is only temporarily due to the action of the current on the vessels of the brain. As in the case of shampooing of the head, however, the action of the current on the scalp would, I think, cause some transient contraction of the vessels, and thus induce a sleepy feeling whilst the faradisation was in operation. I believe that the real influence arises from the restful action induced in each molecule of the brain substance by the gentle rhythmic impulses of the current, reckoning these at over 100,000 during each sitting, and resembling the calm cerebration following change of scene and of occupation. These impulses are numerous enough to produce more than a mere transient improvement in the metabolism of the molecules; and the alternations are slow enough to give time for this trophic action to take place, by which the finer nutrition of the brain is improved.

An essential to this treatment is an instrument for measuring the amount of the current employed. For this purpose I have been lately using the newest form of my faradimeter. I exhibited the original instrument at the Glasgow Philosophical Society.¹ The instrument is now more sensitive, more reliable, occupies less than one-fourth of the bulk of the one then shown and described, and is perfectly portable.

Dosage of current alone, however, is not sufficient for success or even for safety. A special kind of secondary coil must be used, and a means of completely controlling the current in series with the patient is essential. The coil used in these recorded cases has eight to nine thousand windings; the greater the number of windings the coil has the greater being its sedative effect. Coils with fewer windings I have found are more stimulating; those with still fewer being relatively irritating. The patient should be under the influence of the current, as evidenced by the faradimeter, before it is possible to feel it; and the amount should be very gradually increased. The maximum amount of current given is 1 milliampère.² The dose at the first sitting should not exceed one-third of a milliampère. Not only must the secondary current be controlled by powerful rheostats, but the battery current to the primary should also have a rheostat controlling it, say of 50 ohms. The faradimeter is of course in series with the patient. The electrodes are soft, and are applied to the brow and to the nape of the neck. The former electrode should be about 15 square inches in size, and should be connected to the positive pole, that to the nape of the neck about 10 square inches.

The vibrations of the rheotome should be the fastest obtainable. The current should be applied for fifteen minutes each time; three times a week for two weeks, or twice a week for three weeks; six applications in ordinary cases generally proving sufficient.

NOTE AND REFERENCE.

¹ Transactions, 1897 and 1898. ² By 1 milliampère faradic current is meant the electromagnetic equivalent of an amount of sinusoidal alternating current, the galvanometric measurement of whose sinusoidal galvanic equivalent is 1 milliampère.