The next morning, December 15th, the temperature was 98.2°, the pulse 68, and the respiration 22. No improvement in the movements was noticed; the bed sore was about the size of the palm of the hand, and evidently a large amount of necrosis had occurred. During the course of the next two days the frequent movement necessitated in dressing the bed sore caused the sutures in the neck wound to give way, and a large amount of cerebro-spinal fluid escaped into the dressings, but this gradually diminished day by day, and the wound slowly granulated, and was firmly healed by March 26th.

The course of the bed sore was slow in the extreme, and even when discharged from the hospital for passage to England in June, a small sinus still remained unhealed.

To trace the recovery of the various parts it will be best to describe the improvements separately.

Temperature.

There was a good reaction after the operation, from 96° in the morning to 99.2° in the evening. There was again some collapse on the fourth day and the temperature dropped to 96°, and in the evening was only 97°. From this time, except for occasional rises due to constipation, it showed an evening rise to about 100°, with a morning fall until the end of February, when it was practically normal.

Genito-urinary System.

Priapism, which had been so constant up to the time of operation, did not recur afterwards. Retention of urine lasted for a week, when he passed urine voluntarily, but cystitis ensued, and the bladder had to be washed out for five weeks. There was a heavy deposit of pus, but the reaction remained acid. Although he could pass urine voluntarily, he had only slight control for some months, but eventually recovered it.

Intestines.

The bowels were obstinately constipated, and croton oil was occasionally necessary. At times the faeces had to be removed digitally. This continued until April, when the abdominal muscles regained more power and the bowels acted naturally.

Sensation.

Marked hyperaesthesia was present over the right leg, with some exaggeration of knee-jerks and plantar reflexes, but sensation remained intact. Except for the cramps mentioned below, the only pain complained of was severe headache for about ten days after the operation.

Movements.

Four days after the operation the thighs moved together in adduction, and from this time the movements of the legs increased slowly, the left, which was originally less completely paralysed, regaining complete movement much more quickly than the right. This recovery of power was accompanied by the most acutely painful cramps.

The movements of the arms were strong, but the grasp of both hands remained feeble, with marked atrophy of the interossei; this lasted for four months, and his grasp was far below normal on his discharge after six months.

No decided improvement in the intercostal muscles was noted until February 2nd—the fifty-first day—and the return of power in these muscles was accompanied by severe neuralgic pains corresponding to the cramps in the legs. The movements of head and neck were perfect a very few days after the operation.

He was first allowed on his feet on March 27th, when he was able with considerable help to walk to an armchair. The right leg was kept closely adducted to the left, and no free movement was made with it. From this time, with daily massage, steady improvement was noticed.

On leaving hospital for England on June 2nd, six months after his accident, his condition was as follows:

With the exception of a certain amount of hyperaesthesia, sensation was normal. He had complete control over his bladder and rectum. He could walk short distances (about 30 yards) with the help of one stick, but there remained some uncertainty in the movements of the right leg. The interossei of both hands were atrophied and the grasp poor, but with difficulty he could write a few sentences. Respiration was natural, the temperature normal, the pulse 72.

He was invalided from the service in July, 1908. In his last letter to me in August, 1909, he described himself as much stronger, and able to walk fairly well, but stated that he was weak in the right leg and small of the back. His hands were quite well again.

The question of operative interference in cases of injury to the spine is a very important one. During the past two years I have had three cases in addition to that here recorded, and in none of these was any operation attempted. The first was a fracture dislocation in the dorso-lumbar region, and this patient was alive eighteen months afterwards. He was in a workhouse infirmary, paralysed from the waist downwards, leading a catheter life and passing his motions under him.

The second case was one of fracture dislocation in the mid-dorsal region; he died of exhaustion in about four months.

The third case was a fracture dislocation of the fourth and fifth dorsal vertebrae. He died suddenly on the fifth day. At the autopsy it was found that a sharp-pointed

fragment of the body of the fifth dorsal vertebra had penetrated the posterior mediastinum and caused a severe haemorrhage, as a result of which the man had died.

The encouraging results from the operation in the case recorded makes one think that the operation, if only exploratory, might be performed more often.

Certainly in this case it ought to have been done much earlier, the bed sore would have been saved and probably the cord would have recovered its functions more quickly.

I am sure the operation was greatly facilitated by the use of the local anaesthesia in the preliminary stages, it reduced the risks of a prolonged anaesthesia in a case where respiration was much embarrassed.

Although the operation wound was opened by the movements and the spinal cord must have been in danger, no sepsis occurred and the wound granulated, so that this risk can be avoided. When one considers what a life the patient will lead if left alone, with the unconscious passage of faeces and constant catheterism, it is surely a duty to make at least an exploratory incision and to see the extent of the damage.

While recommending operative interference, it should be remembered that it can only be done where ample nursing facilities are available, as in a hospital or a nursing home. The onerous after-treatment would be well-nigh impossible in a private house.

NOTES ON DIVER'S PARALYSIS.

BY

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FROM 1900 to 1908 I have had medical charge of probably the largest pearling centre in the world, including in its population upwards of 400 professional divers who are daily engaged gathering pearl shell at depths varying from 7 to 20 fathoms. I have myself seen a diver bring up shell from a depth of 25 fathoms (150 ft.), but this was an exceptional feat. This means that these men are working under pressure, roughly, from 20 to 50 lb. per square inch above normal, the shifts varying in length inversely as the depth of working. In the lesser depth a diver will remain down one, two, or more hours, in the greater generally under half an hour. The ordinary rubber diving dress with metal helmet is the apparatus used, air being supplied by three barrelled pumps worked by hand wheels. The divers are of various nationality—whites, Japanese, Filipinos, Malays, etc., the majority being Japanese, the whites being the least numerous. They work from small schooners of 10 to 14 tons, and range over many hundreds of miles off one of the most forlorn and desolate coasts in the world. Consequently, when an accident happens they may be several days' sail from port, and so have not the advantages of their colleagues the "caisson" workers, with their decompressing and recompressing chambers and immediate medical attention. Hence, many severe and often fatal cases of diver's paralysis occur.

For eight years I have impressed on these men and their employers the fact that they can with very little extra time and trouble obtain all the safety conferred by the decompressing chamber by a slow and gradual return to the surface, and when this course has been followed few, if any, cases of paralysis have occurred.

Since 1900, not counting the slighter cases characterized by the men themselves as "rheumatics," I have had upwards of 200 cases of diver's palsy; 60 of the patients were dead before a doctor could be reached and I made *post-mortem* examinations. Among the 140 odd who reached me alive I have had to deal with all degrees of paralysis, from slight paralysis of legs and inability to pass urine (always present) up to total paraplegia and loss of sensation. Clinically, the salient features of the paralysis are its bilateral distribution and the constant loss of the power of micturition. The legs are attacked before the arms and recover later. Sensory nerves seem to suffer much less than motor, but I am bound to add that many cases had to be examined through an interpreter, and it is difficult in any case to get correct replies on such a matter

from coolies. Out of these 140 cases 11 died, 8 from septicaemia consequent on sloughing and cystitis, and 3 from supervening meningitis. The rest, after a longer or shorter time, recovered, most of them completely, about 10 per cent. being permanently affected with slight paresis, generally of the anterior muscles of the legs.

I have had patients who have been twice, thrice, or even oftener paralysed, and who have more or less completely recovered. I have never seen extensive secondary degeneration of the cord follow, though one would expect this from the lesions found in the cord *post mortem*.

The treatment after the establishment of paralysis is that of all organic nervous disease—one can only wait on Nature's efforts, though in this disease Nature is kinder than usual. General hygiene, massage, and electricity, all are useful to some extent, and, of course, any complications which arise must be treated. I have been often astonished at the way apparently hopeless paraplegics have recovered in the course of many months.

The most troublesome cases were those complicated by cystitis and deep sloughing. The former complication is very frequently set up by imperfectly cleaned catheters used by the diver's friends, often for several days, while making for port. The paralysis of micturition is so well known among the men themselves that no diver would consider his outfit complete without a soft catheter. The sloughing is also the result in many cases of the treatment applied as first aid by the patient's friends, and is especially common among the Japanese, whose unbounded faith in very hot baths too often leads to parboiling and damage to the enervated tissues, forming the starting point of frightful sloughing, which often defies all efforts of nursing to restrain. I have seen the sacrum, portions of the ilium, and the capsule of hip-joint absolutely denuded, and several of the deaths in my cases resulted from this condition. A few very bad cases, however, made wonderful recoveries. One case, a Filipino diver, was admitted to hospital with total paraplegia and sloughing over the buttocks, which extended till the right hip-joint, most of the sacrum, and the posterior portion of the crest of the ilium could plainly be seen; yet after more than twelve months in hospital and several plastic operations he made a fair recovery, and was able to return to the Philippines.

Owing to the distance from port at which these boats work, I have never been able to see a case in the earliest period of attack. The general history is that the diver has worked longer and deeper than usual, and then has hurried to the surface. He has felt quite well below, and gets aboard as usual. Often he has had the dress removed and even sat down to his meal before symptoms appear. Then he suddenly drops, sometimes "as if shot," or he may remark he is not feeling well, and the symptoms come on more gradually. He may or may not lose consciousness. Some rapidly fatal cases have occurred without initial loss of consciousness, and it is practically never lost in slight attacks. Death may occur within an hour or not until after a day or two.

Clinical experience directs attention to the spinal cord in this disease, and my sixty autopsies have amply corroborated the inference that here is to be found the greatest mischief. I have never found the classical "bubble of nitrogen" in the hearts of my cases; indeed, I honestly confess I doubt my ability to recognize a bubble of nitrogen. I have, however, often fancied that during the rapid putrefaction of these bodies there has been a larger involvement of gases and greater distension of the tissues than is the case with other bodies after equally sudden death. I have only once found an infarct in the lungs, and only twice clots in the heart, and then to no great extent; and in these cases beri-beri, with its often insidious onset and clotting in the heart, has always to be remembered. Beri-beri used to be the greatest scourge known to the pearling fleets, but segregation and sanitation have greatly diminished its ravages. Almost invariably the heart, lungs, and large veins are engorged with dark liquid blood. So marked is this that on one occasion a medical man present at one autopsy frankly told me he would, from the thoracic examination alone, have been quite content to certify asphyxia; yet, on exposing the spinal cord, the characteristic "teased" appearance and minute haemorrhages were present, together with free blood, in the dural canal.

In my experience the following appearances are typical of diver's paralysis on *post mortem* examination of a rapidly fatal case. The thoracic viscera and large veins of the neck are engorged with dark liquid blood. There is nothing noteworthy in the abdomen. There is more or less venous engorgement in the meninges of the brain, markedly increasing towards the base, especially round the medulla. Section of the brain is generally negative; sometimes the blood points seem rather larger than usual. I have only once found a haemorrhage in the brain; it was about the size of a horse-bean, and situated in the left internal capsule, and was accompanied by another haemorrhage practically severing the spinal cord opposite the body of the fifth cervical vertebra. The most characteristic signs are found on exposing the spinal cord. The dural cavity contains blood or blood-stained fluid, and the meninges of the cord are congested with blood, and the congestion appears to be most intense in the cervical region, say the portion corresponding to bodies of the fourth to the sixth cervical vertebrae. This portion of the cord appears to me to be the diver's calx Achillis. It is here that the characteristic "teasing" is most apparent. I call it teasing for want of a better word to explain the appearance in the section of cord; it looks as if one had stippled the face of the section with a fine knife or needle, a semi-disintegrated appearance. With this condition is nearly always associated haemorrhage of greater or less extent, also most marked in the above portion of the cord; indeed I have never found an effusion of any size except in this part; here they may range in size from mere points of blood to, as I have seen in nine cases, large haemorrhages practically cutting the cord in two, and forming clots filling the meningeal tube for over 1½ in. I had neither time nor appliances for microscopic pathology, which in such a hot climate would need exceptional apparatus, and, I may add, exceptional skill, to obtain good results. In this respect, however, I hope my omission will soon be rectified by more competent pathologists, for I have reason to believe steps are being taken to get a specimen cord for examination.

When I first met with this disease I was greatly handicapped by the want of literature on the subject—in fact, all I had was comprised in one page of Osler's *Medicine*. Consequently I was presented with a pathological problem which I had to unravel as best I could, and was often sorely puzzled. For instance, I noticed that though the divers started work about March there were practically no cases of paralysis before September, and from thence to the end of the working season, about the end of November, cases came in almost daily. All sorts of theories are current among the men themselves accounting for this well-known fact—for example, working further out in deeper water, that the water itself "gets heavier" at this end of the season, etc.

The mystery was elucidated by the discovery in one autopsy of signs of scurvy. There was the simple explanation of the prevalence of the disease among men who live for months on small boats, eating salted and tinned foods, and also of its infrequent occurrence till the blood vessels have been somewhat weakened, as we know happens in scurvy. Acting on this knowledge I preached an anti-scurvy crusade, and noted that in fleets where my advice was taken and extra vegetables and other antiscorbutic precautions used there was a very considerable reduction in the number of cases of paralysis.

The points which have particularly impressed me in dealing with diver's paralysis are:

- (a) Signs of asphyxia are nearly always present; it would appear from this that in the rapidly fatal cases death is usually due to affection of the breathing centres.
- (b) In roughly 2 per cent. of the cases in which death was not immediate meningitis set in about a week after paralysis. In this respect, however, considering the damage done to the central nervous mechanism, the only wonder is that it is not a more frequent occurrence.
- (c) The question arises as to the cause of the congestion found in the meninges on *post-mortem* examination of fatal cases. Is it due to over-expansion of vessels by gases suddenly released from pressure, or is it simply part and parcel of the condition of asphyxia? From concomitant appearances in the thorax, it would appear that the latter is the true explanation, yet when one considers the blood or blood-stained cerebro spinal fluid, one is forced to think

of the former as being equally applicable. Probably both factors are often present.

(d) The paralysis of the bladder and loss of the power of micturition are constant, and occur in cases so slight that it is practically the only symptom calling for attention. I have had patients walk up to hospital for catheterization. Why this centre should be attacked so early and constantly is a problem I have failed to solve, more especially as the lumbar enlargement of the cord does not show nearly so much damage as higher up.

(e) The apparent point of selection for the most striking lesions of this disease is in the cervical portion of the spinal cord. The part of the cord where all the large haemorrhages were found without exception could be covered by three cervical vertebrae. Even in the cases in which apparently the disintegration of the nervous substance was the chief lesion, and no large blood effusions occurred, this portion of the cord showed the curious teased appearance more plainly than any other. Why such is the case is a problem for anatomists and physiologists. I can only make two modest suggestions: First, the comparatively large size and great mobility of the spinal canal at this point. Secondly—and this is a mere bow drawn at a venture—this portion of the cord is movable in the metal helmet of the diver, the rest being covered by the rubber dress closely pressed to the body by the water pressure. Theoretically, there should be nothing to choose from the pressure point of view. Is there anything in the movement? It must be remembered that a diver's dress at a great depth precludes bending the back beyond a very slight degree.

Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

A CASE OF TETANUS.

On October 15th I was called to see a married woman aged 20, who was suffering from inability to open her mouth. She had been married over fifteen months, and had had one child, in February, 1909. On my arrival she was up, and apparently not suffering to any alarming extent. After careful inquiry and examination I was obliged to abandon any history of accident, as she was positive that she had not within the last month hurt herself in any way. Her mother-in-law, however, told me that ten days previously she had what was considered to be a miscarriage; she had missed two periods, and the discharge occurred on the date of the third period. She had, however, quite got over this, and the day before my visit I had seen her out walking. There was no vaginal discharge, nor any sign of injury. Her jaws could be separated slightly. All her teeth were very carious, and the smell from the mouth was most unpleasant. I thought possibly this trismus was due to irritation from the teeth, and the fact that the wisdom teeth were uncut favoured this opinion. There was pain in the back from the nape downwards. The pulse 100, and the temperature 100°. I prescribed an antiseptic mouthwash and sedative medicine.

The following day the patient was suffering all the torments of acute tetanus, fully conscious, and bathed in profuse perspiration; the spasms increased, and the temperature rose. Pleurosthotonos was very marked. Between the clonic exacerbations the legs were rigidly extended, the arms were close to the side, the forearms flexed with wrists extended and fingers flexed, the thumbs protruding between the first and second fingers. She died at 4 p.m. on October 17th, three days after the first onset. I was unable to use antitetanic serum, as it arrived too late.

Tetanus was only too easy to diagnose, but the path of infection was less easy to discover. There were two possible routes—the genital tract or the mouth. The first I do not think likely, for there was no discharge or evidence of injury. I believe the entrance of the germ was through the mouth, for these reasons: Having known this young woman for some time, I was aware of the state of her teeth, and also that she used a pin or splinter to

clear out the cavities and crevices after eating. In this way probably the bacillus was introduced, and, being of an anaerobic nature, thrived amidst the many aerobic micro-organisms which infested the tooth cavities and gums.

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JOS. WM. GILL, M.D., D.P.H.

THE DIAGNOSIS OF DIPHTHERIA.

THE following two cases may be interesting, both to the bacteriologist and the general practitioner:

On October 24th I was called to see a boy, aged 4, who had been sick and feverish through the night. The attack evidently came on suddenly with vomiting. His temperature at the time of examination was 100.4° F. and the pulse 110; the tongue was coated and he was slightly flushed. The history suggested a gastro-intestinal attack. I made inquiries as to sore throat, but did not make an examination, as he said his throat did not hurt him, and there were no glands to be felt. On the following morning I found the temperature still 100° F., though the boy looked better. He still denied having a sore throat, but on examining it I found a characteristic yellowish-white slough on the right tonsil and two or three small discrete patches on the left tonsil. The remainder of the fauces was clear but congested. I administered 3,500 units of antitoxin about noon and took a swab of the throat. I then learnt that his brother, aged 10, had slept with him on the night of October 23rd, and as he was going to school, I took a swab of his throat also, although he was apparently in perfect health. At about 7 p.m. the same evening the younger child's temperature was 101.6° F., but the membrane seemed somewhat less. Next morning the temperature was normal and the throat almost clear.

The bacteriological report was negative in the case of the younger boy and positive in the case of the elder. Thinking that there might possibly have been some mistake, or that the swab was not properly applied, I took another of the younger boy on October 26th, but this too proved to be negative. On October 27th a swab was taken from the elder boy and it was still positive. No antiseptic had been used in either case, and there was no other known case from which the elder boy might have been infected. I have perfect confidence in the bacteriologist.

The question naturally arises whether the younger boy was suffering from a genuine attack of diphtheria, and if not, what was the source of infection in the elder.

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Reports

ON

MEDICAL AND SURGICAL PRACTICE IN THE HOSPITALS AND ASYLUMS OF THE BRITISH EMPIRE.

ESSEX COUNTY HOSPITAL.

LARGE RAPIDLY-GROWING FIBROMYOMA OF THE UTERUS:
HYSTERECTOMY: RECOVERY.

(Under the care of ED. CHICHESTER, M.B.Lond.,
Honorary Surgeon to the Hospital.)

MRS. B. was admitted on account of a large abdominal swelling which she had noticed for two years. It caused her much difficulty in getting about, together with dyspnoea, and occasional attacks of pain. She was 47 years old, had been married twenty-eight years, and had one child aged 11 years. She stated that menstruation had always been regular, and perfectly normal. She never lost more than an ordinary amount.

Nothing abnormal was found in the chest. The whole abdomen seemed occupied by a large solid tumour, reaching from the costal arch to the pelvis, where it seemed to be fixed, and whence I thought it arose. Although elastic, the growth did not seem to be fluid anywhere, no sense of fluctuation or thrill being obtainable.