AFTERNOON SESSION

Chairman: Dr. G. BEDBROOK

Sir LUDWIG GUTTMANN (*Great Britain*). The chairman of the afternoon session will be Dr. George Bedbrook from Perth, Western Australia. Most of you know of the great work he is doing for spinal cord sufferers in Australia. But for some years he was also engaged to spread the gospel of modern treatment of paraplegics and tetraplegics to various countries in the Far East. I visited him first in 1957 when his unit was small and only two years old. It was just the time when he became disillusioned with the result of surgery in fractures of the spine, especially open reduction and metal plate fixation. When I visited him again in 1962 on the occasion of the first British Commonwealth Paraplegic Games held in Perth, his unit had expanded to a first class Spinal Centre. Dr. Bedbrook was one of the first in Australia to realise the need of hostels for those paraplegics and, in particular, tetraplegics who for one reason or another are unable to return home. This is now a very important problem which concerns all of us who are engaged in work with these severely disabled people in view of the ever increasing number of these patients and their prolonged life expectancy. These patients just cannot be managed in geriatric units or similar institutions. They develop bedsores and other complications very soon and have to be readmitted to hospitals or spinal units for usually very long lasting inpatient treatment. To obviate this sorry state of affairs it is essential to erect hostels with sheltered workshop facilities for these patients in the neighbourhood of spinal units, with a skeleton staff to look after them and supervised by the staff of the spinal unit. In Great Britain the first of such a hostel of 30 beds has been introduced by me at Stoke Mandeville Hospital a few years ago and has proved highly successful. It is to be hoped that more hostels of this kind will be set up in the country. In Perth Dr. Bedbrook has taken the initiative in this respect and I am sure he will succeed.

Now it is a great pleasure to ask Dr. Bedbrook to take the chair.

Dr. BEDBROOK (*Australia*). Mr. President, Ladies and Gentlemen: Sir Ludwig would be interested to know that, as we sit here at this moment, the contracts are being finalised for a Tetraplegic Hostel in Perth for 40 beds, with a sheltered workshop, and it should be in operation by this time next year.

ASCENDING CORD LESION IN THE EARLY STAGES FOLLOWING SPINAL INJURY

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INTRODUCTION

IN the first weeks following spinal cord injury changes for the better or worse commonly occur, the most common improvements are complete lesions becoming incomplete and recovery in immediately inferior roots—the so-called 'root escape';

further recovery of incomplete lesions is also common. The commonest type of neurological deterioration is a rise in the level of the lesion usually by one segment, sometimes by two segments. This usually occurs in the first four days and the deterioration is almost always temporary, the final neurological lesion being the same or lower than the lesion found immediately after the injury. Other less common forms of deterioration are incomplete lesions becoming complete, and the delayed onset of neurological lesions in patients who were not immediately paralysed by their spinal injury.

A rarer form of deterioration is when a spinal cord lesion appropriate to the bony injury rises by several segments in the first three weeks after the original injury. We have recently treated a patient at the National Spinal Injuries Centre, Stoke Mandeville Hospital who sustained a fracture dislocation of T12 and who initially had a complete lesion below L2 segment and in whom the level rose to C7 between the eighth and seventeenth days after injury. This case prompted me to examine the records of all similar patients admitted to Stoke Mandeville Hospital within a fourteen-year period.

MATERIAL

Only patients who had originally had a complete traumatic paraplegia at a level appropriate to their bony injury and in whom the neurological lesion rose by at least four segments are included. I have been able to find seven such patients.

The patients were all male, and all except one had fractures or fracture dislocations at T11 or T12. The main clinical details are shown in Table I.

This table shows a bony lesion, the initial, maximal and final neurological lesions and the period after injury when the deterioration occurred. The last column shows the presence or absence of a pyrexia of 102°F. (38.9°C.) or over during the period when the lesion was rising.

The first three patients are very similar, all started with lesions below T12 or L1 and within a few days of injury the lesion gradually rose over the course of several more days to the mid-dorsal region, the final lesion being at or slightly below the maximal lesion and flaccid, although it should be noted that Patient 2 had originally had ankle jerks present. Patient 4 was similar except that there was a gradual recovery and

FIG. 1

the lesion ended up only two segments higher than the original lesion and he became spastic six months afterwards.

Patients 5 and 6 are similar to each other. Patient 5 had a fracture dislocation of T12 with a complete lesion below L4 segment which between second and fourteen days rose to C8 and finally left him with a complete lesion at T1 on the right and T3 on the left, his lower limbs have remained flaccid for the next

13 years. Patient 6 had a fracture dislocation of T12 with a complete lesion below L2 segment, which between days 8 and 17 rose to C7 right, C8 left and is now a complete lesion below T1 right and T2 left. His lower limbs remain flaccid. Figure 1 shows the rise in sensory level as drawn on the trunk.

Patient 7 differs from the others in that his bony lesion was higher, a fracture dislocation of T4 on T5 vertebrae with a complete lesion below T5 segment; between days 5 and 7 his lesion rose to C7, three months later he had a complete lesion below T2 segment and was spastic.

TABLE I

Patient	Age	Bony Lesion	Neurological Lesion			Duration of Rise (Days)	Pyrexia 102°F. (38.9°C.)
			Initial	Maximal	Final		
1	40	fracture T12	T12-L3 flaccid	T5 flaccid	T5 flaccid	6-15	+
2	28	fract./dis. T12	T12 A.J. +	T7 flaccid	T8 flaccid	2- 7	+
3	20	fracture T12	L1 flaccid	T5 flaccid	T7 flaccid	6- 8	+
4	43	fract./dis. T11	L1 flaccid	T7 flaccid	T11 spastic	11-18	-
5	23	fract./dis. T12	L4 flaccid	C8 flaccid	T1/T3	2-14	+
6	45	fract./dis. T12	L2 Plantars	C7/C8 flaccid	T1/T2 flaccid	8-17	+
7	24	fract./dis. T4	T5 Plantars ↓ ↓	C7 flaccid	T2 spastic	5- 7	+

TREATMENT OF FRACTURE OR FRACTURE DISLOCATION

Patient 6 had had an open reduction and plating performed in another hospital the day after his injury. All the other six patients were treated by postural reduction of their fractures.

Lumbar Punctures. The results of lumbar punctures performed when the lesions were rising are shown in Table II. Patient 1 had a complete block below his fracture, the fluid from this site contained 4 per cent. of protein and 20 cells. Above the fracture (at T10) there was no block and fluid contained 200 mg. per cent. of protein and a 100 cells/cu.mm., mainly monocytes.

Patient 2 had a complete block and no lumbar puncture was performed above the fracture. Patients 4 and 5 had no block.

Patient 5 had a block at the level of his fracture demonstrated by myelography. Patient 6 had an incomplete block below his fracture, the fluid contained 200 mg. per cent. of protein and 400 cells/su.mm., all neutrophils. Above the fracture at T7 and T8 there was no block. The fluid from this site contained 60 mg. per cent. of protein and 300 cells/cu.mm. (neutrophils), this fluid had a remarkable appearance; to the naked eye it looked like dishwater, microscopy showed this to

FIG. 2

TABLE II

Patient	Lesions	Lumbar Puncture	
		Below Fracture	Above Fracture
1	L3-T5	Block Protein 3000 mg. % Cells 20	No Block Protein 200 mg. % Cells 100 (Monocytes)
2	T12-T7	Block Protein 680 mg. % Cells 20	—
3	L1-T5	No block Protein 600 mg. % Cells 0	—
4	L1-T7	No block No cells	—
5	L4-C8	Myelogram—block	—
6	L2-C8	Incomplete block Protein 200 mgm. % Cells 400 (Neutrophils)	No block Protein 60 mg. % Cells 300 (Neutrophils)
7	T5-C7	Block Protein 200 mg. %	—

be debris (fig. 2). The few strands lying extracellular are artefacts. Patient 7 had a complete block below his fracture and as he had a high lesion no higher lumbar puncture was performed.

Treatment. Patients 1, 2, 3, 4 and 7 were observed closely, but no specific treatment was given for the rising lesion. None of these patients were treated with anticoagulants.

Patient 5 had two laminectomies performed on the fracture site at the admitting hospital while the lesion was rising. The spinal cord was described as being swollen and pink. Following the second operation the lesion continued to rise and he was treated with heparin, following which there was no further rise.

Patient 6 was treated with heparin followed by dindevan when the lesion reached T6. When the lesion reached T4 he was also treated with Rheomacrodex. When the lesion reached T1 this treatment was stopped and he was treated with Arvine—this is an extract of the venom of the Malayan Pit Viper and causes total defibrination and is a very potent anticoagulant (Sharp *et al.*, 1968). Twenty-four hours later the level had risen another segment to C8 and large doses of Prednisolone were given. Following this the level rose another segment to C7 the next day, following which it gradually fell and its present level is T1 right, T2 left. Full anticoagulant therapy was maintained for a further four weeks as was the Prednisolone. His lesion is now static and he remains flaccid.

DISCUSSION

The incidence of this type of ascending lesion is low, I can only find seven cases amongst 808 admissions. However, as six cases occurred in low lesions the incidence of this type of case is higher, approximately 1.5 per cent. to 2 per cent.

The pathology in these cases is uncertain, it is also unlikely that the pathology is the same in all cases. The commonest probable aetiologies are ascending thrombosis, ascending haematomyelia, or an inflammatory or necrotising lesion which for lack of a better term I will call ascending myelitis. The possibility of compression of the spinal cord by a haematoma was not excluded in all cases. The possibility of ascending lesions being due to pressure from a haematoma has been mentioned by Guttmann (1963).

The results of treatment do not help to establish the aetiology. Five cases stopped ascending without specific treatment. Case 5 stopped ascending when treated with heparin. Case 6 continued to ascend when treated with heparin but stopped when treated with Arvine and Prednisolone; it is impossible to say whether this treatment influenced the outcome.

Burke and Cheshire (1968) described a case of neuromyelitis optica occurring three months after a low traumatic spinal cord lesion and they speculate on the possibility of an auto-immune aetiology. The same arguments can be used in the cases I have described except that the time interval between the primary and secondary spinal cord lesions is much less.

Without histology of acute cases I will not speculate further on the aetiology but I hope by this clinical description to have aroused memories of similar cases amongst other workers in the field of spinal injuries.

This condition is so rare that no single centre is likely to see many cases. I hope to learn something during the discussion and I invite anyone who has seen

such cases to co-operate in a joint study of previous and future cases so that we can throw some light on the nature and management of this very alarming complication of spinal cord injuries.

ADDENDUM

Since this paper was read at the Annual Scientific Meeting in November 1968, a further similar case was admitted to Stoke Mandeville Hospital. A girl of 18 sustained a fracture dislocation of T9/T10 in a gymnastic accident and was admitted to Stoke Mandeville Hospital on the same day with a complete lesion below T10. Two days later she developed a pyrexia of 103°F., five days after injury she developed severe pain in the back with neck stiffness and her lesion rose to T3 complete with hyperpathia in C5-T2. She was treated with large doses of Dexamethasone, the pyrexia subsided and the lesion stopped ascending. Full details of this patient will be included in the proposed joint paper with other spinal units.

REFERENCES

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Discussion

Dr. E. SPIRA (*Israel*). The syndrome which Dr. Frankel described is not known to me personally, but I would like to draw an analogy between this syndrome and another condition which we see occasionally, which is thrombosis of the anterior spinal artery which occurs in a pathological condition. The level of T12 is known to be the level which is crucial from the point of view of anastomosis between various branches of the anterior spinal artery. Therefore one cannot help but to think that the spinal artery is incriminated in at least part of that syndrome.

I would like to suggest further that one could, if such a case occurs again, find out the speed of the ascension and the speed of necrosis of the anterior horn cells, at least, to confirm by electromyographic investigations of both the paraspinal muscles and the other anterior muscles, so as to know what time fibrillations will occur in those cases in order to coincide it with the clinical occurrence of weakness.

Dr. P. HARRIS (*Edinburgh*). I was most interested in this paper, and have one or two points. With lumbar disc protrusions, we sometimes see—this is lumbar disc protrusions L4/5, lumbarsacral—cord involvement; the presumption is that this is vascular.

The second point is that, with brain trauma, brain oedema, and increasing neurological disturbance after brain trauma, is of course common. And in this respect I feel that the oedema vascular factor is very important. In these patients I deal with this not by decompressive laminectomy, as I have said, but by giving the patient a hypertonic agent, and we give 500 ml. of 20 per cent. manitol and intravenous dexamethasone; we give 10 ml. intravenously, and continue with 3 ml. intramuscularly, and this goes on for several days. I'd like to recommend this to you.

The third point I would like to make is that I personally feel that lumbar puncture can be dangerous in any patient with an acute spinal cord lesion. I think that the chance of infection occurring, and we see this with some of our spinal tumour cases, is very real, and I would suggest that a lumbar puncture should not be done; I don't think it is useful and I think it can be dangerous.

Sir LUDWIG GUTTMANN (*Great Britain*). Injection of hypertonic solutions to reduce oedema is an old neurosurgical procedure and I am surprised that it hasn't been done in your case. We have done it previously and it worked quite well. Also one can put in rectal infusions of magnesium sulphate solution to reduce the pressure, as this is done in brain lesions with good success.

Now, in this case which has been demonstrated here with the very nice coloured picture, there is no doubt whatsoever that this tissue which was found is debris of oedematous spinal cord, and you have seen the blown up cells which absorb this debris.

With regard to my case of ascending lesion which Dr. Frankel mentioned, this was one of the very few patients on whom I intended to operate but on the morning before operation he died from a pulmonary embolism. The indication for early operation I have always made in my publications is that, by progression of the spinal cord symptoms, there is at least a justification to open up and see whether there is either a localised large epidural haematoma, or one of the extremely rare subdural but extramedullary haematoma. I haven't seen such a case so far, and people with great experience, like Elsberg, have also pointed out the extreme rarity of this. But what I have seen is this: A man had an accident, got out of his car, went to the hospital; X-rays were done which did not reveal any abnormality of the spine, moreover he had no neurological symptoms. He was sent home. During the night he developed paraesthesia in both legs, and the next morning he had a practically complete paraplegia. He was taken to the nearest neurosurgical unit, where a paraplegia below T8 was diagnosed. A lumbar puncture was done, which showed a complete block with some haemorrhagic C.S.F. The complete block was confirmed by myelography. The neurosurgeon went in and found in that area where the man sustained the blow a meningioma which was bleeding, and had damaged the spinal cord. In spite of the excellent technique of my colleague—he removed the meningioma—the spinal cord remained damaged resulting in a severe incomplete spinal cord lesion below that level with very marked spasticity. He has been very well rehabilitated. With regard to the pathology in the cases described by Dr. Frankel I have no doubt that the progression of the cord lesion was due to vascular catastrophies, haematomyelia or spinal artery thrombosis, producing longitudinal lesions which in the majority of cases result in flaccid paraplegia as a result of involvement of the lower motor neuron system in the cord as indicated by the areflexia. Dr. Spira's suggestion to carry out in these cases early electromyography to determine denervation of the paralysed muscles is a good one.

Dr. HARDY (*Great Britain*). I'd like to join Hans Frankel in this study because I was not aware that he had had seven cases; I have also had six or seven cases of exactly the same sort of pattern and strangely enough, also from D12, L1 bony injuries, and not all of them were subjected to surgery.

Two comments. Twice the neurosurgeon came, who was Mr. Jefferson in Sheffield. On the first occasion he explored the region of the lower cervical and upper dorsal, and he found nothing at all. On the second occasion, which was one similar to the occasion which has just been described by Sir Ludwig, we found an angiomatous mass which was obviously responsible for part of the disturbance.

The second comment I would like to make is to go back to the paper given by Professor Romanis some years ago at Stoke Mandeville, and he did point out that it was very unfortunate that a certain number of us have only got one or two radicular vessels supplying our cord, and if you have the misfortune to have an injury then your chances of getting a larger disturbance than you would expect, than if you had more radicular vessels, were going to be very sad indeed. Romanis did point out that the radicular system was very variable, and I think one must bear it in mind when we are dealing with these uncommon types of ascension.

Now the third point I would like to raise is that our neuropathologist, Dr. Wolman, has had one opportunity to examine histologically one of these cords, and he did find a

large column of cylindrical oedematous tissue extending up from the site of the major injury going up seven segments in the cord; he also found obliterations in the spinal artery system. These are not new findings because they are already mentioned in the wonderful paper by Buss and Greenfield in 1919.

And lastly, Hans Frankel may be interested to know that we too also tried heparin and dindevan without any success.

Dr. WHATMORE (*Edinburgh*). We see this problem fairly commonly in neurosurgery, particularly with spinal cord lesions, particularly tumours. Most of these cases occur postoperatively, even with gentle surgical manipulation, and we have no doubt at all in our minds that in many cases this is due to oedema and responds very well to drugs like decadron.

To give you an example quickly. One month ago Mr. Harris had a patient with syringomyelia. This man came to us with weakness in his lower limbs, and a sensory level at about T6. Three days after the operation his level ascended to about the T1 level, and he developed a complete paraplegia. He was immediately placed on decadron, 10 ml. intravenously and thereafter 4 ml. intramuscularly for six hours, and within two days his recovery was complete—that is, he came back to his original level. He went on to recover even further, and is now walking.

Dr. BEDBROOK (*Australia*). Well, ladies and gentlemen, I think you will agree that this is a most interesting subject, and one for co-operation between us all with Dr. Frankel.

CARE OF THE HAND IN CERVICAL SPINAL CORD INJURIES

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SPINAL paralysis involving the upper extremity causes various degrees of functional loss. Trauma to the cervical spinal cord with paralysis often leads to certain patterns which are based on the presence of functioning key muscle groups. The muscle function which remains is the basis upon which proper treatment can be provided. The most important aspect of care in any paralytic situation is an accurate examination to grade the muscle strengths, list the functioning muscles, and test for sensation. This information must be recorded and repeated at intervals.

The number of patients who sustain and survive cervical spinal cord injury is increasing. A few years ago many of these patients died or became hopelessly incapacitated. With skilful management these patients now survive and are able to become useful citizens in society. It is our duty to provide the kind of care and assistance needed to achieve independence.

Proper care of the hand in cervical spinal cord injury represents a small part in the management of this kind of severely handicapped person. It also represents a small part of the upper extremity. Needless to say management of spinal paralysis is difficult and complex, requiring the help of many specialists. All aspects of the patient's mental and physical problems must be considered. Although the hand represents a small part of the patient it is extremely important, especially to the patient with severe paralysis. A systematic and organised approach to its management is essential if maximum rehabilitation is to be achieved.