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SPINAL CORD TRAUMA IN CHILDREN

By DAVID C. BURKE, M.B., B.S.(Melb.)
Senior Medical Officer, Spinal Injuries Centre for Victoria, Austin Hospital, Melbourne, Australia

SPINAL cord trauma in children below the age of 13 years (excluding those due to gunshot wounds) is very uncommon relative to the incidence in teenagers and adults. Melzak (1969), reviewing the 4470 patients with spinal cord lesions who had been admitted to the National Spinal Injuries Centre at Stoke Mandeville Hospital since 1944, reported the admission of 93 children, but only 29 were due to traumatic lesions. He noted that no fracture of the vertebral column could be detected in 16 of the 29 traumatic cases. Audic and Maury (1969) included 21 traumatic lesions in their report of 60 children who became paraplegic under the age of 16. Of the 21 of traumatic aetiology 'very often' no fracture was detected.

Between 4 April 1968 and 29 July 1969, seven children aged between 13 months and 12 years were admitted to the Spinal Injuries Centre for Victoria with spinal cord injuries. Vertebral injury was absent, or minimal in all of these children. In the 10 years prior to this, between January 1958 and April 1968, there were only three admissions of children with similar lesions. It is intended to present, in some detail, the clinical findings in these cases of spinal cord trauma in children, and to discuss them in relation to some of the problems of spinal cord injuries in this age-group; in particular the pathogenesis and pathology of these injuries, and the life expectation of the patients.

MATERIAL

The clinical details of the seven children admitted to the Victorian Spinal Injuries Centre between April 1968 and July 1969 are summarised in Tables I and II. There were three babies under 2 years of age, and the others were aged 6, 8, 10 and 12 years of age, respectively. There were four males and three females. Six of the children were injured as a result of motor vehicular accidents, and one was injured in a light aeroplane crash. Two of the babies were injured in the same way. They crawled in the path of their fathers' vehicles as the vehicles were being garaged, and were injured by impact against the moving vehicle, probably a wheel. The two older children were injured by hyperflexion over a lap seat belt, providing further proof of the desirability of lap and sash belts in cars and aeroplanes instead of the simple lap belt.

The five youngest patients presented with a dorsal paraplegia, except Patient 1. whose cord lesion was oblique, resulting in an incomplete tetraplegia below the sixth cervical segment on the left side. X-rays of these five children failed to demonstrate a vertebral injury, except for slight separation of the third and fourth dorsal spinous processes of Patient 1. The two older children (injured by hyperflexion over a seat belt) presented a slightly different picture to the younger children. Both had a complete paraplegia below the upper lumbar segments, and both had a very slight, forward subluxation of the second lumbar on the third lumbar vertebra.

The subluxation was more marked in the case of Patient 7, as illustrated in Figures 1 and 2.

Three of the children whose X-rays were normal had fractures of at least one rib at about the level of their neurological lesion. Lumbar puncture was

TABLE I

Details of Patients and Accidents

Patient	Age	Sex	Delay in Admission	Cause of injury
1. B. W.	13 months	M	19 days	Ejected from motor car in collision
2. B. K.	15 months	F	8 days	Run over by father's truck at home
3. L. G.	18 months	F	28 hours	Run over by father's car at home
4. D. Z.	6 years	M	6½ hours	Knocked over by speeding car in street
5. D. McP.	8 years	M	7 hours	Hyperflexed under dashboard of car in collision
6. V. C.	10 years	F	48 hours	Hyperflexed over lap seat belt of car in collision
7. C. A.	12 years	M	5 hours	Hyperflexed over lap seat belt in light plane crash

performed in three of the patients. This was blood-stained in two cases and clear in the third. Queckenstedt test was negative in all three cases. A myelogram was performed on one of these patients, and this was normal.

RESULTS

All seven children were managed conservatively, the five youngest by nursing flat in bed for six to eight weeks with regular turning, and the two older children by postural reduction of the subluxation by extension over a lumbar pillow. The left sided subluxation of the second lumbar vertebra on the third of Patient 7 was not reduced by extension of the spine, but after a period of immobilisation has remained stable and he has had no symptoms from his back since admission. The other six children have had stable, symptomless spines since their period of immobilisation.

Unfortunately, there was no neurological change in any of the children, except for a fall in the level of Patient 4's lesion by two segments. All seven children initially presented with a flaccid paralysis of the legs, but patients 1, 4 and 5 all had vigorous flexor withdrawal reflexes on plantar stimulation from the time of admission.

It was expected that the five children with the dorsal lesions would develop a spastic paralysis after the period of spinal shock in an upper motor neurone lesion, and that the two older children with lumbar lesions would have a lower motor

TABLE II
Clinical Details on Admission

Patient	Neurological lesion	X-ray	Lumbar puncture	Other injuries
1	Incomplete below C6 Complete below C8 (L) D2 (R)	Normal, except for slight separation of spinous processes D3/D4	Blood-stained C.S.F. No block Normal myelogram	Occipital haematoma Fracture 4th rib (L)
2	Complete below D4	Normal	Clear C.S.F. No block	Fractures 4th, 5th, 6th ribs (L)
3	Complete below D3	Normal	—	Nil
4	Complete below D2 (L) D4 (R)	Normal	—	Fractures mandible, (R) femur, (R) humerus, 3rd ribs (bilat.), (R) haemothorax.
5	Complete below D9	Normal	Blood-stained C.S.F. No block	Nil
6	Complete below L1	Slight forward subluxation L2/L3	—	Nil
7	Complete below L2	Slight forward subluxation L2/L3	—	Nil

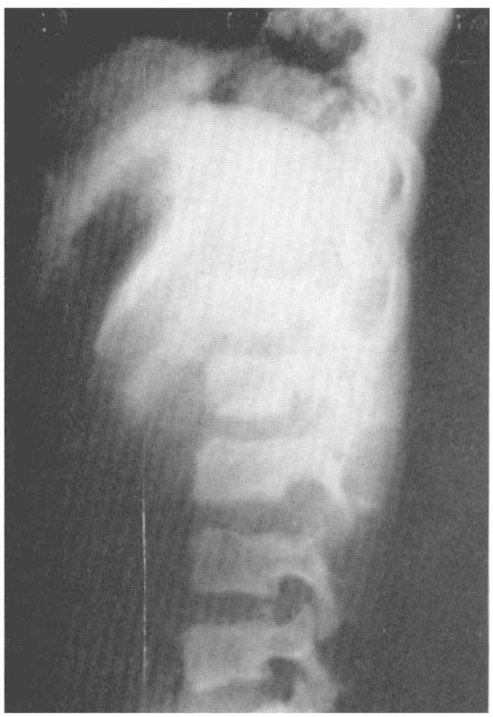

FIG. 1

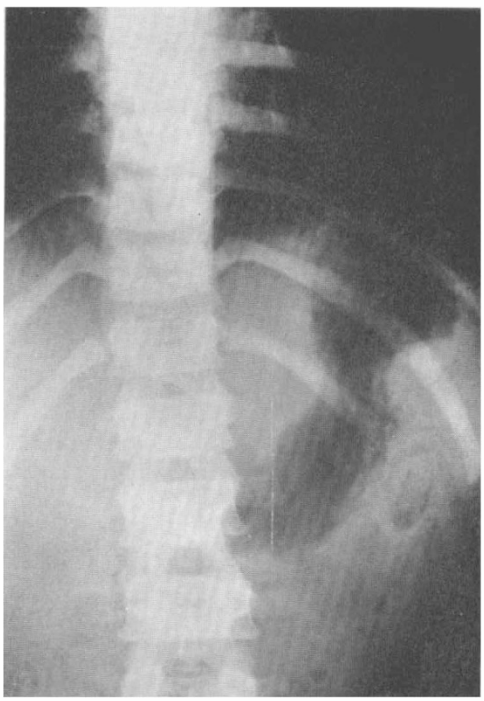

FIG. 2

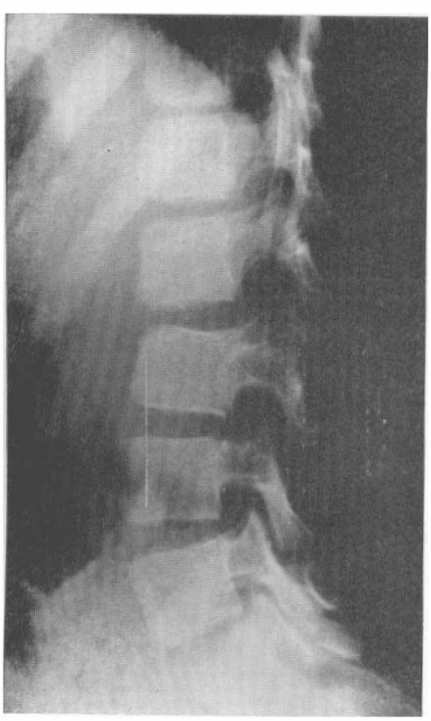

FIG. 3

Fig. 1—Lateral X-ray of the lumbar spine of Patient 7.

Fig. 2—Antero-posterior X-ray of the lumbar spine of Patient 7, showing left sided subluxation of the second lumbar vertebra on the third, and fractured transverse processes of second lumbar vertebra (left) and third lumbar vertebra (bilateral).

Fig. 3—Lateral X-ray of the lumbar spine of Patient 6, four months after injury.

neurone paralysis. However, the two baby girls, Patients 2 and 3 remained flaccid below the upper dorsal region. Patients 1, 4 and 5 became spastic as expected, but Patients 6 and 7 developed a spastic paralysis below the first lumbar and second lumbar segments, respectively.

Further X-rays of the spines of all of the children were taken, including tomography where indicated, looking for some evidence of the mechanism and site of injury. Only the X-rays of Patient 6 gave any more information. New bone formation around the posterior intervertebral joints between the second and third lumbar vertebrae was considered to be good evidence of capsular ligament tearing around these joints (fig. 3).

The urological management, and the results of treatment are summarised in Table III. Six of the seven children had their bladders managed initially by an indwelling catheter. Patient 1, who was admitted 19 days after his accident, had good reflex bladder function on admission to the Centre. He had not been catheterised since the accident, and did not require catheterisation subsequently. His urine has remained sterile throughout. The other six children had an indwelling catheter for periods ranging between 9 and 37 weeks, but all were catheter free by the time of discharge from hospital (including Patient 2 who has not yet been discharged). Six of the children have satisfactory, balanced bladders with low residual urine. Five of the patients have reflex bladders, but the two young girls, Patients 2 and 3, have lower motor neurone bladders. They are on a regular two-hourly regime of expression by suprapubic pressure.

Patient 3 has a satisfactory bladder on this regime, as she remains dry between bladder expressions and has a low residual urine after expression, but Patient 2 is often wet between expressions although she has a low residual urine. The six children who had indwelling catheterisation for varying periods, developed an infection of the urine after a variable period. The three females had infected urine on admission to the Centre, there being a delay in admission of all three of a period varying between 28 hours and eight days. Patient 3 had not been catheterised prior to admission, but the other two children had been catheterised at other hospitals. A very pleasing aspect of the bladder management of the children was the relative ease with which the urine was rendered sterile, and maintained sterile (see Table III). There was no significant clinical urinary infection in any of the seven children.

REVIEW OF THE LITERATURE

Reports on spinal injuries and paraplegia in children, if not primarily concerned with teenagers, where the pattern of injury is similar to that of adults, appear to be preoccupied by subjects such as 'Congenital causes of Paraplegia' (Norton & Foley, 1959), 'the Management of Scoliosis' (Kilfoyle *et al.*, 1965; Audic & Maury, 1969), 'Birth Injuries' (Stern & Rand, 1959; Crothers, 1960; Leventhal, 1960), and 'Pseudosubluxation of the Second Cervical Vertebra on the Third' (Dunlap *et al.*, 1958; Vigoroux *et al.*, 1968). No specific reference to spinal cord trauma without vertebral injury in children under 13 years of age could be found, even in the specialised texts of Spinal Cord Pathology of Davison (1960) and Hughes (1966), except for the passing observations by Melsak (1969) and Audic and Maury (1969). Guttmann (1960) reported two children aged 3 years and 7 years respectively, who were knocked over by vehicles. The younger child had no visible vertebral injury to account for his dorsal paraplegia and the other child a doubtful fracture of the

TABLE III

Urological Management and Results

Patient	Period of I.D.C.	Time until urine first infected	Clinical urinary infection	Type of bladder on discharge	Urine on discharge	Duration of sterile urine	I.V.P., cystogram
1	Nil	Sterile throughout	Nil	Balanced U.M.N.	Sterile	Sterile throughout	Normal I.V.P. 12 mth. after injury
2	11 weeks	Infected on admission	Acute cystitis X1	L.M.N.	Still in hospital	2 months	Normal I.V.P., C.U.G. 6 mth. after injury
3	11 weeks	Infected on admission	Acute cystitis X2	L.M.N.	Sterile	8 months	Slight dilatation R pelvis upper ureter (I.V.P.), and L reflux (C.U.G.) 12 mth. after injury
4	17 weeks	45 days	Nil	Balanced U.M.N.	Sterile	14 months	Normal I.V.P. 10 mth. after injury
5	9 weeks	139 days	Nil	Balanced U.M.N.	Sterile	16 months	Normal I.V.P. 20 mth. after injury
6	37 weeks	Infected on admission	Nil	Balanced U.M.N.	Sterile	11 months	Normal I.V.P., cystogram 2 mth. after injury
7	14 weeks	17 days	Nil	Balanced U.M.N.	Sterile	12 months	Normal I.V.P. 14 mth. after injury, cystogram 2 mth. after injury

TABLE IV—Summary of Progress and Rehabilitation

	Time in hospital	Final neurological lesion	Subsequent X-rays	Ambulation	Home	School
1	4 months	Incomplete below C6, complete below C8 (L) D2 (R) (spastic)	No change	Perthe's trolley standing/ sitting frame. Fair sitting balance	Good country home	Not applicable yet
2	Still in hospital	Complete below D4 (flaccid)	No change	Perthe's trolley standing/ sitting frame. Good sitting balance	Still in hospital (likely to need permanent institution)	Not applicable yet
3	6 months	Complete below D3 (flaccid)	No change	Perthe's trolley standing/ sitting frame. Good sitting balance	Poor suburban home	Not applicable yet
4	11 months	Complete below D5 (spastic)	No change	Wheelchair, semi-independent. No walking	Fair suburban home	Correspondence at home 1969. School for handicapped 1970
5	5½ months	Complete below D9 (spastic)	No change	Part wheelchair part walking, with long calipers and forearm crutches	Good country home	Normal school in 1969
6	11½ months	Complete	New bone around posterior I.V. joints between L2 and L3	Mostly walking with long calipers and forearm crutches, wheelchair occasionally	Good suburban home	Correspondence at home 1969. Normal school 1970
7	5 months	Complete below L2 (spastic)	Persistent sub-luxation between L2 and L3	Full walking with long calipers and forearm crutches. Wheelchair rarely	Good suburban home	Normal school in 1969

second dorsal vertebra to account for his complete paraplegia below the second dorsal segment. Guttmann considered that the first child's lesion was due to spinal artery thrombosis.

Bedbrook (1966) stressed the importance of longitudinal traction on the spinal cord and its blood vessels in the pathology of spinal cord injuries, to explain the much longer segment of spinal cord involved than would be expected by vascular disturbances or crushing alone.

Stern and Rand (1959), Crothers (1960), Leventhal (1960) and Attwood and Stewart (1968) all describe spinal cord injuries in new born infants, most commonly following difficult breech deliveries, while Hellstrom and Sallmander (1965) described spinal cord injuries due to abnormal postures *in utero*. Birth injuries of the spinal cord are frequently unaccompanied by fracture or dislocation of vertebral column and the most common site of these injuries is at the cervico-dorsal junction or the upper dorsal region.

It has been suggested that undue traction, possibly combined with angulation of the spine, is the cause of the injuries, as the vertebral column, composed of cartilaginous rings, is capable of considerable elongation because of its elasticity while the spinal cord and its meninges are unable to withstand the same degree of traction. The predominance of the upper dorsal and cervico-dorsal region as the site of rupture of the spinal cord has been suggested as due to the anchoring effect on the cervical spinal cord by the brachial plexus and the lumbo-sacral spinal cord by the cauda equina.

DISCUSSION

Pathology. The pathology of spinal cord injuries in adults is well documented, and the varying pattern of such lesions is well understood by specialists in the field of paraplegia. But as demonstrated by the youngest five patients in this paper, and noted by Melzak (1969) and Audic and Maury (1969) young children who become paraplegic following injury very commonly have no visible vertebral fracture or dislocation on X-ray. It is necessary therefore to consider the mechanism and pathology of these injuries and to seek a better understanding than presently exists.

The mechanism of injury of the seven children reported was clearly flexion/rotation in at least six cases. The two older children were known to have been forcibly flexed over a lap seat belt, and their X-rays did reveal a minimal forward subluxation of the lumbar spine. The 8-year-old boy is known to have suffered a flexion/rotation injury from the history of being found in a flexed position under the dashboard of the car, jammed there by the front seat which had moved forward. The two baby girls, who were run over by their fathers, almost certainly would have been forcibly flexed, although no direct proof of this is available.

The baby who was ejected from a car certainly suffered a flexion/rotation injury from clinical examination—an occipital haematoma, fractured ribs at about the level of spinal cord injury and evidence of posterior interspinous ligament rupture from the X-ray appearance of separation of the spinous processes of two adjacent dorsal vertebrae. The 6-year-old boy who suffered multiple injuries after being hit by a car provided no direct evidence of the mechanism of his spinal injury from the history or clinical examination. However, indirect evidence of a flexion/rotation injury is available from the bilateral fractures of the third rib and

the knowledge that injuries of the dorsal spine suffered by adults in this type of accident are nearly always of the flexion/rotation type.

If the clinical facts of the patients described are combined with the published material on birth injuries it seems reasonable to suggest that the elastic, cartilaginous spine of a young child may be grossly distorted by a flexion/rotation force, but that the spinal column does not disrupt, as with birth injuries, through its inherent elasticity. However, the spinal cord, and the meninges, are unable to withstand this degree of traction and torsion, and the neural tissue does disrupt. As none of the seven children discussed were subjected to surgery and all have survived the injury, there is no pathological confirmation of this hypothesis. In the absence of any published pathological data on this clinical syndrome one could postulate that the damaged spinal cord would show the effects of a longitudinal traction injury as described by Bedbrook (1966), or even possibly rupture.

Certain facets of the clinical histories of the children presented support the idea of a longitudinal traction injury to the spinal cord. The level of the spinal cord injury was higher than the level of vertebral injury in the three children in whom the vertebral injury was seen on X-ray. The two patients with subluxations of the second lumbar on the third lumbar vertebra had *upper* motor neurone lesions below the first and second lumbar segments respectively, and Patient 1 had an oblique spinal cord lesion below the sixth cervical segment on the left and the second dorsal segment on the right with definite radiological evidence of vertebral injury between the third and fourth dorsal vertebrae. The obliquity of this child's lesion also suggests a stretching injury of the spinal cord with oblique tearing of the cord. The higher than expected level of paralysis in these three children is best explained by a spinal cord lesion over several segments, above and below the vertebral injury, This, in turn, would most likely be caused by a longitudinal traction force.

An explanation of the permanent, *flaccid* paralysis below the upper dorsal segments of Patients 2 and 3 must also be sought. It is postulated that the fulcrum of their vertebral injury was in the mid to lower dorsal region, causing an extensive longitudinal traction injury to several segments of the spinal cord above and below this point. It is also possible that the great spinal artery, which according to Romanes (1963) always enters in the lower thoracic region, was damaged by the longitudinal traction, thus causing infarction of the conus.

Reviewing the seven cases critically it is apparent that the two older children in this series present a slightly different picture from the other five. Both have a definite, although minor, forward subluxation of the second lumbar vertebra on the third. They could represent a transitional stage between the infantile lesion described above and the adult form of fracture/dislocation, or alternatively they could represent an infantile form of lumbar injury, as the other five children's lesions were of the dorsal spine.

Whilst confident that our conservative approach to the management of these children's spines was correct, both on general principles and in minimising later spinal deformities, we would prefer to have a more detailed knowledge of the underlying pathology upon which to base definitive treatment.

Life Expectation. Assessment of the life expectation of a patient after a spinal cord injury is important. This may not always be an easy matter, even for adult patients, but it has been our experience recently that it is very much more

difficult with these young children, particularly the very young ones, as our experience with such patients is very limited and there is so little published data to refer to. Prior to April 1968, Cheshire (personal communication) had only seen three patients of similar age with comparable lesions to the seven described, since January 1958. One of these was a tetraplegic of 9 months of age. Dr. Cheshire felt at that time that a child of that age, though paralysed, would be better managed in a specialised paediatric hospital rather than a spinal unit, so the child was transferred immediately to the Royal Children's Hospital. Unfortunately, she died shortly afterwards of a staphylococcal pneumonia and septicaemia. Since then it has been Cheshire's policy to treat all patients with spinal cord injuries in the Spinal Injuries Centre for Victoria, regardless of age. A second child moved to England to live, and has been lost to follow up. The third child, who suffered a mid-dorsal paraplegia at the age of 22 months is now alive and well at $14\frac{1}{2}$ years of age, but has a chronic, low grade, asymptomatic urinary infection. However, his early urinary management at another hospital left much to be desired.

Excluding the three oldest children whose low lesions and sterile urine should ensure a normal life prognosis, the life expectation of the other children with high lesions would depend on three factors: the danger of inhalation of vomitus, chest, and urinary tract. All survived the acute period, although the youngest baby very nearly died of an acute chest infection soon after admission to the Centre. None of the five children have had anything more than minor chest infections, apart from that one episode, and it seems reasonable to suppose that provided they have adequate medical supervision and care until their mid teens, their life span should not be affected by their susceptibility to pulmonary collapse and consolidation. However, they must all be susceptible to the danger of inhalation of vomitus, at least until they reach adulthood.

The four young children who have been discharged home have satisfactory bladders at present with low residual urines and sterile urine, but Patient 2 remains incontinent between bladder expressions and her urine has become re-infected. This was also the experience of Guttmann (1960) who treated three young children with upper motor neurone lesions by intermittent catheterisation. However, it is not known at this stage how long their bladders can be expected to remain satisfactory, particularly those of the two young girls with flaccid bladders. Clearly the life expectation of these children past the adolescent stage will depend on the function of their urinary tract. Reference to the literature is again of little help. Survival of congenital paraplegics such as those due to spina bifida is not relevant as these children commonly have other congenital defects of the urinary tract and of other systems which only confuse the issue. No information is available in the literature about the survival of children with birth injuries of the spinal cord. Isolated reports of survival to 14 or 15 years of age are encountered, but with no details at all about the status of the urinary tract.

We feel reasonably optimistic about the continued efficiency of the three male upper motor neurone bladders, but less so about the future of the two little girls. At the moment both their bladders are being managed reasonably satisfactorily by regular Credé expression, and their urines are sterile. But it is appreciated that they cannot continue to have their bladders expressed for the rest of their lives, and as they will never be able to empty by straining because of abdominal wall paralysis it seems likely that they will require an ileal diversion at an early age. The argument for ileal diversion is strengthened by the proven reflux of one child

and persistent incontinence of the other, even though certain reservations are held about the long-term survival of patients with ileal diversions.

CONCLUSION

Possibly the reason for such a paucity of published material on paraplegia in childhood is that few of the specialised units caring for paraplegics receive children as patients. Thus congenital paraplegics and birth injuries would, in general, be treated in obstetrical and paediatric hospitals and would rarely reach a spinal unit, while many traumatic lesions of the spinal cord of children, either by gunshot or other trauma, would also be admitted to a paediatric hospital rather than a specialised centre.

As a result there has been little opportunity for the specialised problems of spinal paralysis in children to be studied by specialists who are interested in the whole patient rather than one small aspect of his problem, such as his scoliotic spine. If this is so, is there not a place for the establishment of a world survey amongst the members of the International Society of Paraplegia so as to pool information about infantile spinal paralytics, and so increase our knowledge of the special problems of these young patients? This, of course, is essentially only a short-term answer to the problem. What is really necessary is for all Spinal Centres to widen their facilities and treat all patients with spinal paralysis, regardless of age or aetiology.

Summary

A series of spinal cord injuries in children less than 13 years of age has been presented. The seven children were admitted to the Spinal Injuries Centre for Victoria between April 1968 and July 1969.

A common clinical feature of the patients was that vertebral injury was minimal or absent. A review of the literature revealed a paucity of information on the pathology of this syndrome.

It is postulated that the elastic, cartilaginous spine of a young child may be grossly distorted by a flexion/rotation force, but does not disrupt through its inherent elasticity. The spinal cord, however, is unable to withstand such stretching forces, and a longitudinal injury of the spinal cord may result.

The life expectation of these young patients is also discussed in the light of their good bladder function and sterile urine a short time after injury.

Résumé

Une série de traumatismes médullaires chez l'enfant de moins de 13 ans a été présentée. Les 7 enfants ont été admis au Centre pour traumatisés médullaires de Victoria, en Australie, d'avril 68 à juillet 69. Un facteur clinique commun était celui de l'absence complète de lésions vertébrales minimes. Une revue de la littérature a révélé la pauvreté des informations sur ce syndrome. Il a été postulé que la colonne vertébrale de l'enfant consiste surtout en un cartilage élastique et qu'au cours de la lésion il ne cède pas à cause de son élasticité. La moëlle épinière, elle, est incapable de résister aux forces qui lui sont appliquées et une atteinte longitudinale peut en résulter. La durée de vie de ces jeunes malades est discutée à la lumière de leurs bonnes fonctions vésicales et de la stérilité des urines rapidement après l'accident.

ZUSAMMENFASSUNG

Eine Serie von Rückenmarksverletzungen von Kindern unter 13 Jahre wird beschrieben, die in der Periode zwischen April 1968 und Juli 1969 im Spinalen Zentrum von Victoria aufgenommen waren.

Ein allgemeines klinisches Bild war die minimale oder fehlende vertebrale Verletzung.

Es wird angenommen, dass die elastische, cartilagene Wirbelsäule eines jungen Kindes schwer bei der Flexion/Rotation Wirkung der Gewalt verändert werden kann, ohne durchrissen zu werden. Das Rückenmark dagegen kann der Gewalt nicht widerstehen und wird verletzt.

Die Lebenserwartung dieser jungen Patienten wird ebenfalls diskutiert im Lichte ihrer guten Blasenfunktion und sterilen Urin.

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Discussion

Mr. D. HANCOCK (*G.B.*). I am sorry to report yet another cause of traumatic paraplegia in children—namely, battering. I think we must bear this in mind. We have one patient in the Unit at the moment; like the majority of children she showed no bony lesion, but myelography done elsewhere has shown the presence of a swelling behind the odontoid process, which I am sure is due to haematoma or oedema, and I strongly suspect that if we were to do more myelography, certainly only on an academic basis, we would find intrinsic canal lesions showing on the screen. The whole story is certainly not an osseous one.

Dr. F. W. MEINECKE (*Germany*). I entirely agree with what Dr. Burke has said—it is very seldom to see changes in the spinal vertebra, but on the other hand we saw a lot of young children with severe associated injuries, broken pelvis, arms, or legs, and yet there was nothing to see on the X-ray of the spine. One question we are asked by

the parents very often is about the growing up of these young severely cord damaged children and we tell the parents that there would be no trouble in growing up—they would be as the normal child. This is very often a point of discussion. What we fear is the development of deformity of the vertebra after the injury, many years later. I think it was here a year ago that there was a very impressive picture which demonstrated that. At the moment I don't know of any possible way to prevent it. I would be interested to hear the experience of others on this problem.

Dr. J. S. YOUNG (*U.S.A.*). I would ask Dr. Burke if he has done electromyography on these people. The longitudinal lesion in the spinal cord has been of interest to me. The movement of practice in the last few years of scanning the muscles by myography innervated by the various segments—and indeed we are seeing longitudinal lesions quite frequently.

Dr. D. BURKE (*Australia*). No, I didn't do electromyography.

Dr. R. HALLIN (*G.B.*). It is very interesting looking at the aetiology of the fracture in the 6th and the 7th patient, they have the same kind of cause being hyperflexed over the lap seat safety belt. It is also interesting to find that in both there was the same type of fracture and these are the only two who showed bony injury, namely slight forward subluxation of lumbar 2 and lumbar 3. I think this should stimulate discussion about the effect of safety belts in the aetiology of fractures of the lumbar spine whether the belt is of the lap type, and in cervical spine whether it is of diagonal type. Am I right in thinking that, even with lap and diagonal types of belt, the latter type fixing the upper part to the trunk, in the moment of the violence of the impact, with the upper part of the body and the cervical spine moving forward, this might result in a fracture? If this is the case, it is very, very serious to use safety belts. I would like you to discuss this, please.

CHAIRMAN. I fear we haven't time for a discussion of the philosophy and applicability of safety belts, though there is no objection to a mention of it, while we are discussing the specific topic of the peculiarities of these lesions in children.

Prof. M. WEISS (*Poland*). In our material we have 6 cases of gunshot wounds. This is a very difficult situation because children now get gunshot wounds if their parents leave arms at home without proper protection. Two of them passed through my Centre a few years ago.

I want to stress a very important problem which we found in the follow-up of the children. When we allowed them to walk, they very quickly got an excellent ability to walk even when they have very high cord lesions. We never could see such possibilities of adaptation in adults. But, unfortunately, the gait is not controlled enough, and we can see that because the gait is not very controlled there are many pressure sores on their feet, necessitating treatment. From this point of view we took many measures to protect their legs. Before we supply the basis we give them a protecting surface of soft plastic material, something like stockinettes but much wider than these, which is also very useful for spina bifida children for preventing pressure sores and then put on a base and allow them to walk without limitation.

Sir LUDWIG GUTTMANN (*G.B.*). I am also interested in the after-care of the children, in particular in their social habilitation, and I see from the chart of Dr. Burke's interesting paper that three of the seven children have been sent to an ordinary school and only one was sent to a school for disabled. We had here some trouble in the beginning with our children to get them into ordinary schools. To give you just one example of what can

happen, I mention a boy of 10 who had made an excellent adjustment to his complete paraplegia below T11 and his father came, bitterly complaining that his boy—an intelligent boy—was put into a school for disabled children. I got in touch with the medical authorities and in those years they were not very helpful at all. Had there not been an understanding headmaster who took him into his ordinary school this boy would have been kept in that school for the disabled. He became an excellent pupil and won first a scholarship to Reading University in mathematics and from there he won a scholarship in mathematics to Trinity College, Cambridge! After his exam he became a lecturer at Nottingham University in mathematics. He is now—happily married—with a fellowship at Princeton University in the U.S.A. If this boy had been kept in a school for disabled children he might be perhaps a second class clerk somewhere in an office.

Dr. D. BURKE (*Australia*). Just a few brief points—I obviously didn't have time in this paper to discuss the late problems of progress in these children. This is another subject in itself so this was not touched upon. My main reason for presenting this paper was to ask questions. What is the pathology in these lesions? Just replying to a couple of specific points that were raised: to Dr. Young, no, we do not use myography, we do not have it. We have willing workers but we don't have much equipment. Lap belts, Dr. Hallin, 98 or 99 per cent. of our patients from motor car accidents were not wearing seat belts—the seven we have now had who were wearing seat belts at the time of their accident only two were lap and sash, all the rest were lap belts. I don't think there is enough evidence in favour of seat belts. And, Sir Ludwig—the one child we have in a handicapped school is a little fat Greek boy who is not coping terribly well with his paraplegia, thanks to his parents who are not coping very well, and for these reasons and physical reasons—we need to continue physiotherapy—he needs a special school, temporarily, we hope, for a year or two.

THE RESULTS OF LAMINECTOMY IN PATIENTS WITH INCOMPLETE SPINAL CORD INJURIES

By T. H. MORGAN, M.D., GEORGE W. WHARTON, M.D., and GEORGE N. AUSTIN, M.D.
Division of Orthopaedic Surgery, University of Maryland Hospital, Baltimore, Maryland 21201

THE orthopaedic and neurosurgical literature contain clear and definite indications for laminectomy. Nevertheless, laminectomy continues to be a routine part of the early management of spinal cord injuries, both in patients with complete and incomplete lesions, in many areas of the United States. Patients continue to arrive at rehabilitation centres with records from acute hospitals which indicate that, although specific benefits could not be anticipated, exploratory laminectomies were performed to evaluate the condition of the spinal cord. The implicit suggestion is that no harm will result from this procedure.

This study is based on a review of 230 patients with traumatic spinal cord injuries admitted to the Montebello State Rehabilitation Hospital, Baltimore, Maryland, from 1953 through 1968. Seventy of the 230 patients had well-documented incomplete spinal cord injuries. The observation that significant neurological deterioration occurred in a number of patients following laminectomy suggested that a review of these patients might further clarify the role of laminectomy in the management of the acute spinal cord injury.