

such a case, it is very easy to repeat either the alcohol or phenol injection but to do another laminectomy is, of course, much more difficult, especially if one has to operate in a scarred area of the cord resulting from the previous operation or in traumatic lesions as a result of the initial injury. I agree with Mr. Sutcliffe Kerr that it is very difficult to control the extent of the effects of alcohol or phenol following intrathecal injection. That is the reason why I have always warned not to use the intrathecal alcohol block in incomplete lesions with good voluntary or reflex functions of the bladder and sexual function. This applies, of course, also to phenol. I have always been very sceptical to the claims—and Dr. Nathan knows my views—that with intrathecal phenol injections one can single out special spinal roots for blocking without affecting the bladder function in incomplete lesions. In fact, we have admitted patients after this procedure and found the bladder permanently paralysed.

With regard to surgical procedures on tendons and muscles, I was very impressed by the scepticism and disappointment as expressed by a very experienced orthopaedic surgeon like Mr. McSweeney with regard to transplantation of tendons in spasticity, in particular in the upper limbs. I can only amplify what Mr. McSweeney has said from our experience. Some patients have been admitted after these transplantations. In theory these are very beautiful, but in the end the disability in the upper limbs was much worse than before the operation, especially after transplantation of flexors of the wrist on to the extensor digitorum. One should take notice of what Mr. McSweeney has said.

I come to the last point—the local injection of phenol into the peripheral or motor points. This seems to me a practical way in the treatment of spasticity especially in incomplete lesions. I think proper examinations before and afterwards and follow-up are absolutely necessary to give us in the future a clear picture about the value of this procedure. Here I have a little criticism of the film Dr. Glass has shown us. Unfortunately, the degree of contracture of the long flexors and flexor pollicis longus before the injection was not shown in the film, and this would have been important for comparison. I am delighted that these two procedures have been taken up because it will improve greatly the clinician's knowledge of the anatomy of the entry of the branches of peripheral nerves into individual muscles as well as their functions. In this connection, clinicians should become familiar with the classical work of Duchenne de Boulogne and Otfried Foerster.

Finally, it is essential to ascertain if possible electromyographically, before injecting peripheral nerves or motor points for the treatment of spasticity which muscle or muscle group is preponderant and after blocking this muscle examine the effect on the whole flexion or extension synergy.

### (B) ANKYLOSING SPONDYLITIS

#### TRAUMATIC PARAPLEGIA AND TETRAPLEGIA IN ANKYLOSING SPONDYLITIS

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SINCE Bechterew (1894, 1897), Strümpell (1897) and Pierre Marie (1898) recognised ankylosing spondylitis as a systematic inflammatory disease of the spinal column resulting in progressive rigidity of the spine and the thorax, a considerable literature on aetiology, incidence, sex, symptomatology, pathology and therapy,

has accumulated on this subject. Good reviews on this disease have been published in recent years (Böni & Kaganas, 1954; Aufdermaur, 1957; Sharp, 1965). However, there are relatively very few reports on fractures of the ankylosed spine, in particular those resulting in injuries to the spinal cord (Abdi, 1904; Stiasny, 1933; Bergman, 1949; Lob, 1954; Sharp & Purser, 1961). In Abdi's case, a fracture of the lumbar spine resulted in a cauda equina lesion, Stiasny reported a case of fracture between the 5th and 6th cervical vertebra with little neurological changes, and Lob (1954) reported another case who, following a fall on his head from a height of 3 metres sustained an angulation of the rigid cervical spine between the 6th and 7th cervical vertebrae leading to localised separation of the ossified anterior ligament and fracture of the articular joints of C6, apparently without any cord involvement. The fracture healed completely within three months, the angulation of the vertebra was reduced and the elevated anterior ligament formed a bony fusion between the previously angulated vertebrae. In the four cases of cervical fractures reported by Bergmann—most of them between the 5th and 6th cervical vertebrae—the fractures occurred in what had formerly been the intervertebral space rather than through the vertebral body itself. There was no relationship between the degree of cord damage and the anatomical displacement of the spine. In one of these cases in spite of a profound separation of the 5th and 6th cervical vertebrae in extension the neurological symptoms were negligible. On the other hand, in the third case, who while intoxicated fell and struck the shoulder region against the ground, there was an immediate paralysis of all four limbs, in spite of a very slight displacement between C5/6. However, this man made a complete recovery. The time necessary for bony consolidation in all cases was short, what the author ascribed to the underlying tendency to ankylosing spondylitis. Sharp and Purser (1961) observed damage to the spinal cord in seven out of 22 cases, with spontaneous dislocation of the atlas on the axis in 2 per cent. of the patients. The ankylosing spondylitis was in an advanced state.

**Clinical Material.** In the present paper seven cases—six male and one female with long-standing ankylosing spondylitis—are reported who were involved in accidents and sustained injuries to the spinal cord. Six of these patients were admitted to the National Spinal Injuries Centre, Stoke Mandeville, the seventh to my unit at the Rookwood Hospital, Cardiff. The age of the patients at the time of accident varied between 38 and 60. Two of the patients had injuries to the lower dorsal and lumbar portions of the spinal cord. In the remaining four, the cervical cord was involved. One of the patients, the female, is of special interest as she may be described as 'Scandinavian' form of the disease *i.e.* the combination of rheumatoid arthritis and ankylosing spondylitis.

## CASE REPORTS

Case 1. Gat, A. J., aged 60. A man with long-standing ankylosing spondylitis for over 40 years was admitted on 26 May 1960 to this Centre. Following previous treatment with bed rest, his hip joints became ankylosed in extension. However, he could manage to walk and work during World War II, and was employed as a Civil Servant working in a standing position. In April 1960 he fell at home and developed severe pain in his back with incontinence of urine and faeces. When admitted to the Centre one month later

from another hospital he showed signs of an incomplete cauda equina lesion below L5. The paralysed bladder had been treated with an indwelling Foley catheter and he soon developed proteus infection of the urine and orchitis. There was a deep sloughy sore over the right heel.

A lateral X-ray showed a fracture-dislocation through the calcified disc of the 4th lumbar vertebra which was dislocated anteriorly on the 5th lumbar vertebra. The intervertebral discs of the other lumbar vertebrae also were calcified (fig. 1A). The A.P. X-ray revealed a severely ankylosed spine of the whole lumbar and thoracic region with ossification infinitely more marked on the right than on the left. There was a fracture line visible on the right side of the 4th lumbar vertebra (fig. 2A). The sacro-iliac joints were completely obliterated. Both hips were completely ankylosed and the head of the femur showed striated structure with osteoporosis. Both tuber ischii showed some ectopic bone formation (fig. 3). The fracture was partly reduced by postural reduction (fig. 1B), but eventually healed in a more satisfactory position (fig. 1C). Figure 2B shows the dense ossification of the 4th lumbar vertebra on the right side.

The bladder and bowel paralysis disappeared gradually and the catheter was withdrawn and micturition restored with a residual urine of only 2 ounces. A cystogram and intravenous pyelogram showed no abnormality. The sore healed and he was able to walk with the help of walking aids. He was discharged on 14 December 1960 and has kept well at home apart from developing a large filiform condyloma acuminata in 1963 as a result of sogginess in the groin caused by the fixation of the legs in extension and adduction due

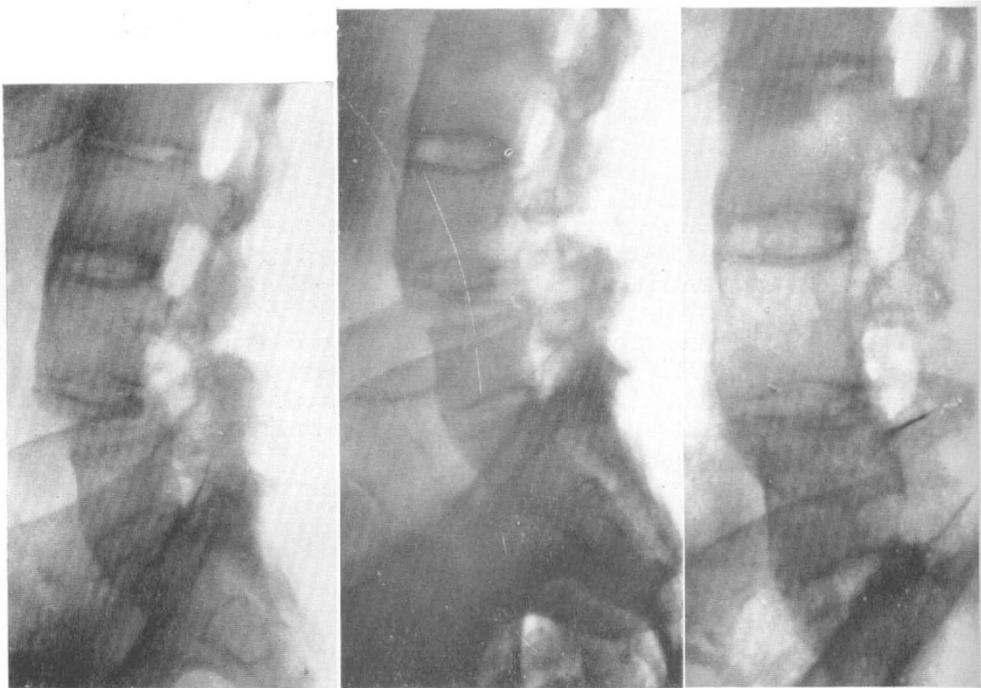


FIG. 1A

FIG. 1B

FIG. 1C

Figure 1A—Fracture dislocation through the calcified disc of L4 vertebra. Figure 1B—Fracture dislocation partly reduced by postural reduction. Figure 1C—Final stage of postural reduction.

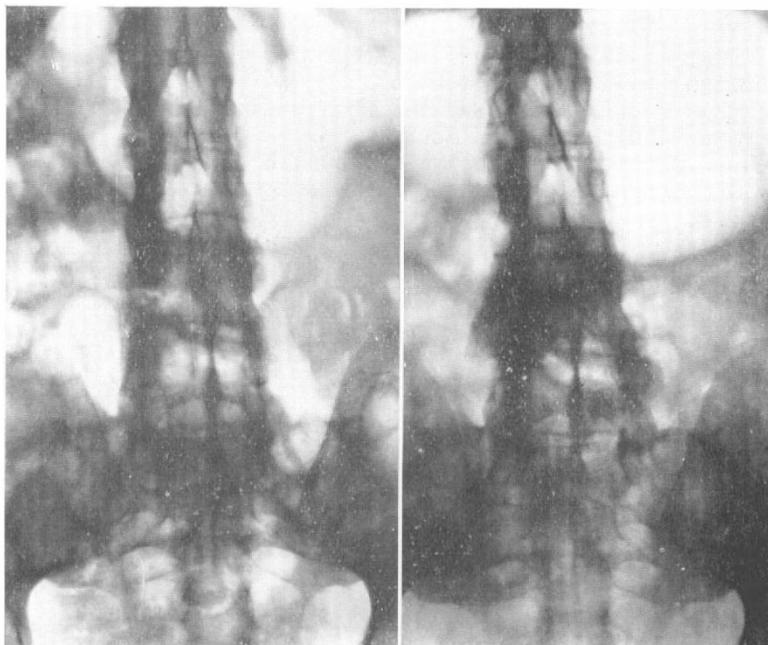


FIG. 2A

FIG. 2B

Figure 2A—Fracture line the right side of L4 vertebra. Figure 2B—Dense ossification on right side of L4 vertebra following consolidation of fracture.



FIG. 3

Ectopic bone formation over both tubera ischii.

to the ankylosing spondylitis of the hip joints. This healed following treatment with 25 per cent. Podophyllin in paraffin oil.

Case 2. Bri, L., aged 41, suffering from ankylosing spondylitis for 15 years, was transferred to this Centre on 11 July 1957 from the Central Middlesex Hospital, London, one day after a head-on collision with a car while riding a motor-cycle. He was not

unconscious but extremely shocked. He was found to have, apart from multiple abrasions:

- (1) A sub-trochanteric compound fracture of the right femur.
- (2) Fractured crests of the right ileum.
- (3) Fracture through the right cuneiform bone and second metatarsal.
- (4) Fracture of the left tibia and fibula.
- (5) Gross extension fracture-dislocation through the body of the 1st lumbar vertebra with marked lateral dislocation resulting in a complete paraplegia below T<sub>11</sub>/12.

The A.P. X-ray revealed a profound lateral dislocation (fig. 4A), while the lateral picture showed an extension fracture with wide separation of the 12th dorsal from the 1st lumbar vertebra (fig. 5A).

A lumbar puncture on 27th revealed xanthochromic C. S. F. and complete subarachnoidal block. In spite of three pints of blood given at the previous hospital the haemoglobin was only 59 per cent., blood urea 91 mg. per cent., which increased during the following days to 198 mg. per cent., but became normal in due course (20 mg. per cent.).

The severe fracture dislocation healed in due course as shown in Figures 4B-D and 5B-C. Further X-rays revealed presence of ankylosing spondylitis of the dorso-lumbar spine of considerable degree but only to a lesser degree of the cervical spine where the ankylosis involved the mid-cervical vertebrae, in particular the 5th and 6th. These were fused anteriorly with some though incomplete ossification of the intervertebral discs between C<sub>5</sub> and C<sub>6</sub> and just beginning between C<sub>3</sub> and C<sub>4</sub> and C<sub>4</sub> and C<sub>5</sub>. The intervertebral spaces between the other cervical vertebrae were free, and therefore the patient's mobility of his head was relatively good in all directions (fig. 6). All fractures healed promptly with the exception of the compound fracture of the femur which became infected. After the infection subsided the fracture was explored and fixed by a metal plate and healed eventually with a large bridge of callus formation. As to the paraplegia, this was of flaccid type with areflexia and hypotonic bladder. The patient learned to void every two hours by straining. With regard to the bowels, he acquired a habit to empty the bowels regularly, sometimes with the help of suppositories. He was discharged from hospital on 28 May 1958, having made a good adjustment to his paraplegia. Cystogram and intravenous pyelogram were normal, his urinary infection which he developed at first cleared up and the urine became sterile.

Case 3. Bur, G., aged 48, was suffering from ankylosing spondylitis for many years with rigidity from the hip joints to the occiput. He was admitted to this Centre on December 1962.

On 23 November 1962 he slipped on a step while walking out of a cinema. On the next day he complained of pain in neck and arms and hands and developed rapidly a tetraplegia below C<sub>5</sub>. He was admitted to the Royal East Sussex Hospital, where because of respiratory distress a tracheostomy was performed. The paralysed bladder was treated with a Gibbon catheter and the urine became infected.

On admission to this Centre he had a very severe tetraplegia below C<sub>5</sub>, with complete motor paralysis of both legs, trunk and the right arm and only a flicker of deltoid, biceps and brachio-radialis on the left. Sensory loss was severe below C<sub>6</sub>, but not complete, especially in the distal segments. Bladder and bowels were paralysed and urine infected with *B. coli*. The knee jerks were greatly reduced and the ankle jerks absent. Plantar stimulation produced plantar response.

X-ray of the cervical spine revealed gross changes of ankylosing spondylitis with complete fusion of the anterior and posterior aspects of the vertebral bodies and articular joints as well as intervertebral spaces. There was an extension fracture right through the body of the 6th cervical vertebra (fig. 7).

In spite of his rigid thorax his respiratory function greatly improved and his urine became sterile; however, on 23 December he suddenly developed an acute abdominal

distension with vomiting of blood and difficulty in breathing and died within one hour. Post-mortem revealed a complete collapse of both lower lobes and a small thrombus filling a peripheral vessel at the base of the right lung. There were small thrombi in the right calf, and an acutely perforated large chronic duodenal ulcer was found which no doubt was the cause of his death. The fracture through the body of C6 was confirmed at post-mortem.

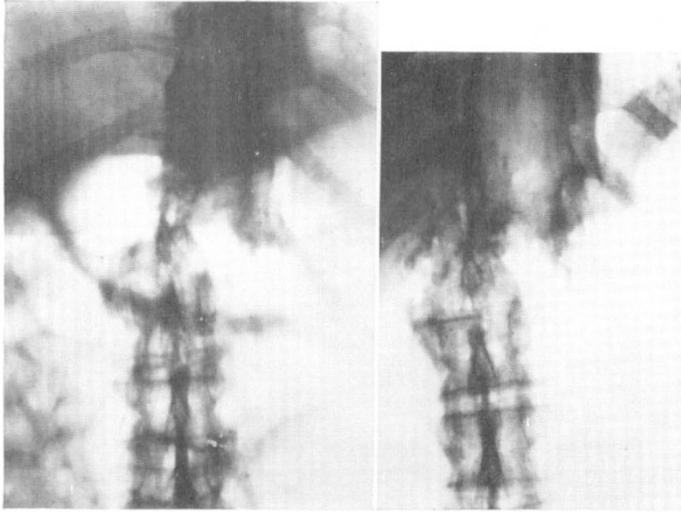


FIG. 4A

FIG. 4B

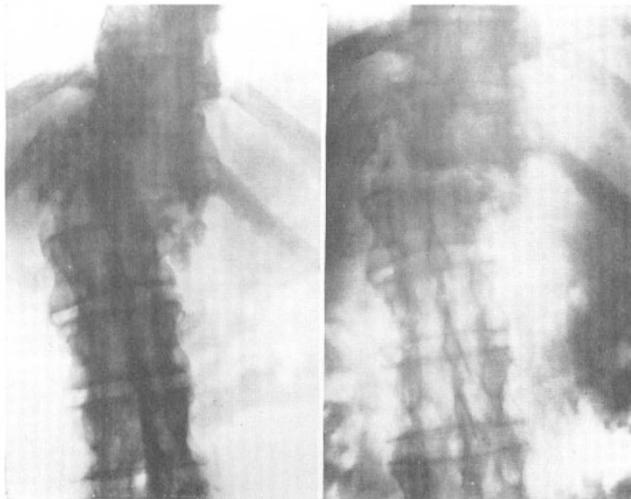


FIG. 4C

FIG. 4D

Figure 4A—Profound lateral dislocation of L1 vertebra through its body. Figures 4B-D—Stages of partial reduction of the lateral dislocation.



FIG. 5A

FIG. 5B

FIG. 5C

Figure 5A—Profound extension fracture between T12 and L1 vertebrae with side separation. Figures 5B-C—Stages of successful reduction of the extension fracture.



FIG. 6

Minor degree of ankylosis of cervical spine especially between C5 and C6 vertebrae.



FIG. 7

Extension fracture through body of C6 vertebra.

Case 4. Par, H., aged 43 with ankylosing spondylitis for six years, treated unsuccessfully by X-ray therapy.

On 23 August 1956 his small car overturned and he sustained a complete tetraplegia below C7. He was admitted to this Centre a day after injury. He had at first a complete tetraplegia below C7, which in due course became incomplete as he had some return of sensibility over the body. A lumbar puncture showed an incomplete block which became complete a few days later but the third lumbar puncture showed again an incomplete block. This man developed within a week after injury an acute paranoid psychosis which was treated by electro-shock therapy with good effect. He was discharged home 10½ months after injury. At the time of discharge motor paralysis was still complete but he had made some recovery of sensation. In February 1966 he developed a bronchopneumonia and was treated at the Whittington Hospital, London, where he died on 7 March 1966.

X-ray of the spine showed a classical bamboo type of ankylosis with complete obliteration of the sacro-iliac joints (fig. 8). The X-ray also shows the cystogram. Lateral X-ray of the cervical spine showed severe ankylosis of all vertebral bodies both anteriorly and posteriorly, the latter especially of the atlas and 2nd, 3rd and 4th vertebral bodies. However, the intervertebral spaces were wide and not ossified (fig. 9). There was the suspicion of a slight separation of the 6th spinous process but no fracture of the vertebral bodies.

Case 5. Wil, D., aged 43, was suffering from A-S for many years. At the age of 20 he had already courses of X-ray therapy. At the age of 23 he developed iridocyclitis and had two subsequent recurrences, the last in early 1965. He worked as an independent builder before his injury and was able to walk in a waddling way.

On 12 August 1965 he was on a scaffolding when the frame gave way and he fell backwards 14 feet on to the ground. He was immediately paralysed in both arms and legs. He was admitted to the Redhill Hospital, Surrey, where a complete tetraplegia was diagnosed. The paralysis in the lower limbs was of flaccid type.

X-ray of the cervical spine revealed a forward dislocation of the 7th cervical vertebra of an ankylosed spine (fig. 10). The ankylosis involved in particular the 2nd and 3rd, and even more marked the posterior aspects of the 4-5th cervical vertebrae, furthermore all intervertebral joints and the posterior ligaments. X-rays of the other parts of the spine showed a generalised A-S of combined railway line and bamboo type and osteoporosis. The sacro-iliac joints were completely obliterated and there was ankylosis of the left hip joint and rarification of both ischial rami with ectopic bone formation. He was treated for two months with skull calipers on a Stryker frame.

When admitted to the Spinal Unit at Cardiff on 21 December 1965, he had superficial pressure sores over the sacrum, both tuber ischii and left ankle. There were profound contractures of shoulders, elbows, wrists, fingers, hips and knees. Neurologically he showed an incomplete transverse spinal cord syndrome below C7, and complete below T4, with sacral sparing to touch only.

The contractures of the arms and legs have improved and all sores are now healed. However, the urine is still infected.

Case 6, Cok, V., aged 38, suffering from A-S for eighteen years, was admitted to this Centre on 17 November 1961 from the Rehabilitation Institute, Detroit, Michigan, U.S.A., with a complete tetraplegia below C7, as a result of a car accident sustained on 27 June 1961. The motor paralysis involved apart from the trunk and lower limbs all muscles of the hands and fingers, with the exception of the extensor carpi radialis and flexor carpi radialis. The paralysis was of spastic type with marked hyperreflexia. Sensory loss was complete below C7, bladder, bowels and sexual functions paralysed. The bladder had been treated with an indwelling Foley catheter and severe infection followed, and he also had a sacral pressure sore of 2-inch diameter with deep necrosis. He had a chest infection and a tracheostomy had been performed.

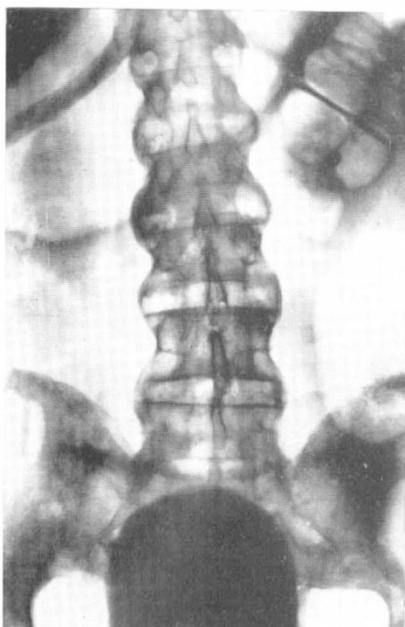


FIG. 8



FIG. 9

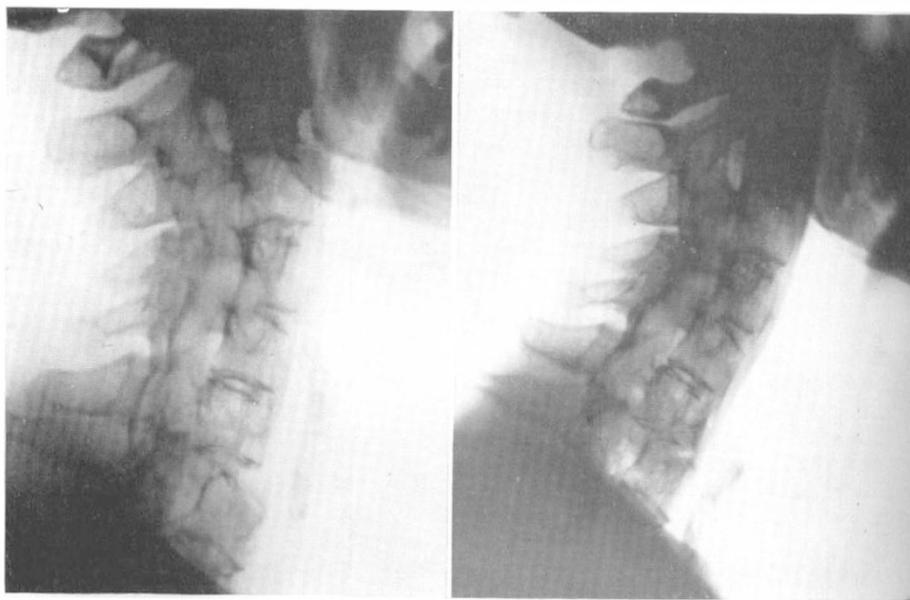


FIG. 10

Figure 8—Classical bamboo type of ankylosed spine with complete obliteration of sacroiliac joints. Figure 9—Severe ankylosis of all vertebral bodies especially of the upper cervical vertebrae. Intervertebral spaces wide. Figure 10—Forward dislocation of C7 vertebra in severely ankylosed spine involving the intervertebral joints and posterior ligaments.

X-ray of the cervical spine showed the presence of an extensive A-S with ankylosis of the bodies of the cervical vertebrae, especially anteriorly but also of the interarticular joints from the 3rd cervical vertebra downwards. Furthermore, it showed a gap in extension between the 6th and 7th cervical vertebrae with some slight forward dislocation of the 6th vertebral body on to the 7th and a rupture of the calcified anterior ligament opposite the body of the 7th cervical vertebra (fig. 11).

X-ray of the thoraco-lumbar spine revealed a combined bamboo and railway line pattern of A-S, the latter affecting the 3rd-5th lumbar vertebrae and upper part of the sacrum. Intervertebral discs were completely ossified between L<sub>2</sub> and L<sub>3</sub> bodies only. The sacro-iliac joints were completely obliterated (fig. 12).

The lung infection was overcome and the urine infection controlled, the patient's general condition greatly improved and after discharge from this Centre on 9 March 1963 he is a resident at the Star and Garter Home for ex-servicemen in London.

#### *Juvenile Rheumatoid Arthritis and Ankylosing Spondylitis*

Case 7. Miss Mur, J. M., aged 40, admitted to this Centre on 15 January 1965. This patient, with an irrelevant family history, has been suffering from rheumatoid arthritis since the age of 13, starting first in feet and ankles and gradually involving other joints of the upper and lower limbs and the spine. Seventeen years ago she had cup arthroplasties performed elsewhere in both hips. In spite of the rigidity of her legs and spine, she was able to walk with the aid of elbow crutches.

On 8 January 1965, while walking from her doctor's surgery on her crutches, she slipped and fell into some bushes. She was not unconscious but noticed immediately that she could not move her fingers or lower limbs. She was taken to Southend Hospital and from there transferred to this Centre seven days later.

The neurological examination revealed an incomplete tetraplegia below C<sub>5</sub>. While there was good function in biceps, brachio-radialis (the shoulder joints were contracted) and extensor carpi radialis, the triceps were paralysed on the right, and very weak on the left; all other muscles of the hand and fingers were paralysed and there was also a paralysis of the trunk and lower limbs with areflexia. Sensory loss was only patchy over the body, with hypoalgesia below T<sub>4</sub>, and analgesia below T<sub>8</sub> on the left, but appreciation of pin prick between L<sub>3</sub> and S<sub>1</sub> on both sides. Bladder and bowels were paralysed. The paralysis of the upper limbs gradually improved with the exception of the interossei and thumb muscles on the right. There was also return of the knee and ankle jerks, and plantar stimulation produced extensor response of the toes. There was a return of spinothalamic sensation but the postural sensibility of the feet remain absent.

As to the skeletal system there were typical deformities following rheumatoid arthritis in fingers, wrists and elbow, knees and ankles. The flexion of the hip was only possible to 60 degrees.

X-ray of the spine revealed gross changes of A-S involving the cervical spine affecting the vertebral bodies as well as the interarticular joints. There was also ossification of the intervertebral spaces of the lower cervical spine and there was some indication of a self-reduced subluxation of C<sub>6</sub> (fig. 13). All costal and vertebral joints were ankylosed and there was ossification in the intercostal spaces (fig. 14). respiration was almost diaphragmatic. The articular joints of the lower dorsal and lumbar spine were calcified and ankylosed. In fact the thoracic and lumbar spine showed the most pronounced railway line type of calcification amongst all the cases described here (fig. 14). There was some periostitis over both tuber ischii. The sacro-iliac joints were obliterated and there were marked articular changes in both knee joints, and the patient developed effusions in the right knee.

X-ray of the urinary tract showed no bladder or renal stones, but calcified lymph-nodes and phlebolites. Cystogram showed a ten ounce bladder capacity. There was no diverticulosis or reflux; blood urea was normal. Rose-Waaler and Slide Latex tests were



FIG. 11

Extensive A-S of cervical spine. Some separation between the 6th and 7th cervical vertebrae with fracture of the calcified anterior ligament.

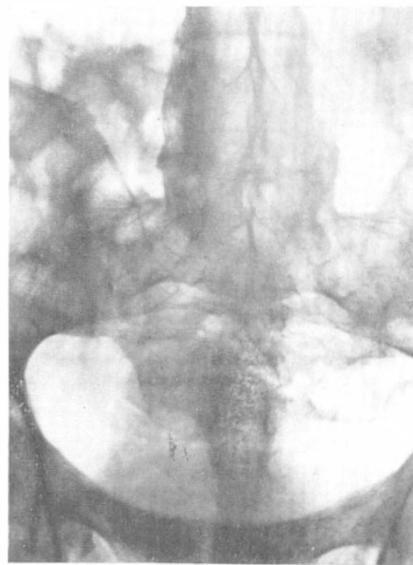
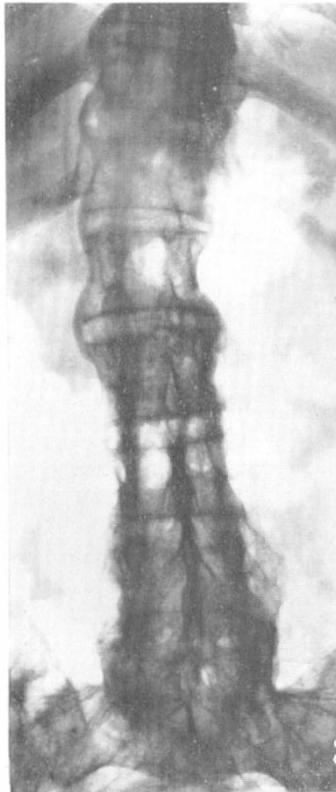


FIG. 12

Combination of bamboo and railway line type of ankylosed thoraco-lumbar spine. Sacro-iliac joints completely obliterated.

negative. Alkaline phosphatase was normal (4.9 units), phosphorus 2.96 mg. per cent., calcium 9.8 mg. per cent., ionised calcium 4.4 g. per cent., Total protein 6.90 g. per cent., Albumin 2.97; alpha (1) 0.53, alpha (2) 1.11, beta 0.61, gamma 1.63.

The muscular system shows generalised atrophy and the skin glossy appearance. There is a tendency to anaemia which is treated with blood transfusions from time to time.



FIG. 13

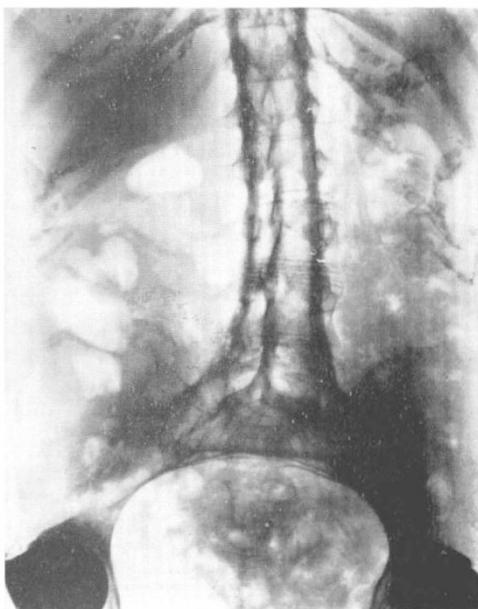


FIG. 14

Figure 13—Self-reduced subluxation of cervical 6 in severely ankylosed cervical spine.  
 Figure 14—Costal and vertebral joints ankylosed. Ossification of intercostal spaces.  
 Classical railway type of ankylosed thoraco-lumbar spine.

## DISCUSSION

The cases described in this paper show all the clinical signs of A-S and their histories reveal with the exception of the only female patient (Case 7) the systematic pattern of the Strümpell-Pierre Marie type of the disease. Although most of them also showed articular changes of the extremities these were mainly confined to the proximal joints; only Case 7 showed a clear connection with juvenile rheumatoid arthritis which started at the early age of 13 affecting first the ankles and later hand and fingers.

The degree and localisation of the ossification of the various components of the vertebral column—bodies, articular joints, intervertebral discs, capsules and ligaments—varied in the individual cases. Three types of ankylosed spine could be distinguished in the a-p view of the X-rays: bamboo type (Case 4), railway line type (Cases 1 and 7) and mixed bamboo-railway line type (Cases 5 and 6).

The lateral view of the X-rays revealed a variability of ossification of the anterior and posterior aspects of the bodies as well as the articular joints, and one of the outstanding features was in all cases the ossification of the ligaments. While

in most of the cases the ossification was most conspicuous in the anterior longitudinal ligament either in its whole length or at isolated points as described by Simmonds (1903) and Schmorl and Junghanns (1951), the ossification of the posterior ligaments was particularly pronounced in the cervical spine in Cases 3 and 5.

Great variations were also found in the degree of ossification of the intervertebral spaces. Case 2 shows only patchy ossifications in the intervertebral spaces of C4, C5 and C6 in contrast to the dense ossification of the cervical intervertebral spaces of Cases 3 and 7. The same also applies to the intervertebral spaces of the thoraco-lumbar space in all our cases.

The sacro-iliac joints were found completely obliterated in all patients and in two of them the tuber ischii showed a fluffy periostosis.

It could be assumed that the brittle spine in cases of A-S would give rise to frequent fractures, yet amongst our about 2500 traumatic lesions out of 4000 paraplegics and tetraplegics admitted to the Spinal Units of Stoke Mandeville and Cardiff only seven patients (six in Stoke Mandeville) were admitted with this condition. The reason for the rare incidence of fractures in A-S which is also confirmed by the reports in the literature may be due to the fact that these patients on account of the rigidity of their body expose themselves less to the hazards of work and traffic. Our experience is in accordance with that of other workers in this field, that minor accidents such as slipping or falls to the ground from low heights (Cases 1 and 3) may cause fractures or fracture-dislocations with serious involvement of the spinal cord.

The type of the fractures or dislocations seen in our cases was one in extension throughout, although of varying degree. The determining factor in the mechanism of this extension injury of the spine is, no doubt, the rigidity of the vertebral column which allows lateral dislocation in addition to extension depending on the direction of the violence as shown in Case 2, but prevents flexion and rotation which is the most common mechanisms in fracture-dislocations of the spine in subjects without ankylosed vertebral column. On the other hand very severe damage to the spinal cord may result in subjects with ankylosing spondylitis from accidents causing minute or no demonstrable vertebral damage at all, as shown in Cases 4 and 7. In such cases, the jarring effect of the trauma on the cervical vertebrae may result in acute damage of the vascular supply of the cervical cord causing complete or incomplete transverse tetraplegia.

#### ACKNOWLEDGEMENT

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#### SUMMARY

Seven cases of traumatic paraplegia and tetraplegia in ankylosing spondylitis are reported and their radiological and neurological symptomatology described in detail.

Three types of ankylosing spondylitis were found:

- (1) bamboo type;
- (2) railway line type;
- (3) mixed bamboo and railway line type.

The type of fractures and dislocations found was one in extension and its mechanism is discussed. Only in one case the extension fracture-dislocation was combined with lateral dislocation.

Trifling accidents may lead to severe spinal cord involvement, and the discrepancy between vertebral cord damage is stressed.

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#### Discussion

*Araluze, F.* (Spain). You have presented a case of tetraplegia without apparent dislocation of the cervical spine. It is well known that dislocation of the atlanto-axial joint is a feature of ankylosing spondylitis. As far as I can see, you have only shown an X-ray of the neck in neutral position. It is known that when the cervical spine is almost fused the only movement is at the level of the cranial-vertebral joint. In your film, one cannot see whether there is any atlanto-axial dislocation or not, so I do not think one can say that there is not a proper cause for this tetraplegia. The question is, was the tetraplegia complete or incomplete.

*Sir Ludwig.* Complete.

*Araluze, F.* I am also very interested to note that in rheumatoid arthritis it is very frequent to find people with tremendous dislocation of the vertebra with very little neurological symptoms. I have done some work in this field. I had a case with an atlanto-axial dislocation of more than 1 cm. without any neurological signs and also a lower one too. This raises the question whether the spinal cord damage is due to the impact of the victim or not; in other words, whether the spinal cord can accommodate itself to the narrowing of the canal.

*Sir Ludwig.* We could not find anything in the atlanto-axial joint but it was a very important point you made about patients who have no visible fracture and yet develop a very severe transverse spinal syndrome. The question is whether this is due to a temporary jarring of the spinal cord which seems to me not very likely in our cases. We have to think of another cause; *i.e.* the disturbance of the vascular supply of the spinal cord in this area. You will remember we had here a very interesting demonstration of the vascular supply of the spinal cord by Professor Romanes, in particular of the cervical cord, and it is possible if not likely that in these patients during the years the intra-

vertebral foramina are so narrowed that the slightest trauma produced an obstruction of the vascular supply. I see Dr. Hill, our Consultant in rheumatology here, and I wonder whether he might like to make some comments.

*Hill, A. S.* (England). I hesitate to speak at all, after hearing such a masterly exposition not only of the features of the fractured spine in ankylosing spondylitis but also of clinical features of the disease itself, on which I could not attempt to improve. We are much more familiar with the problem of the unstable atlanto-axial joint in rheumatoid arthritis. Although we know it has been described in ankylosing spondylitis, I have not come across it. We are more concerned, of course, with the prophylactic aspect of this. We have a number of patients in whom this instability has been readily demonstrated in the lateral view of the cervical spine in flexion. Some of these patients have transient neurological disturbances—I have in mind one patient who said every time she jerked her head she felt electric shocks going through her body. The problem is when to intervene in these patients and to try to press one's orthopaedic colleagues (and sometimes they do need a little pressing) to perform a fusion. I think probably you are all aware of the dangers to which these patients are exposed. We had a lesson about this quite recently, when a patient with a rheumatoid arthritis, who had about a centimetre of movement between the odontoid and the front of the atlas, had to have an operation for perforation of the eyeball by slow malacia perforans—a complication of rheumatoid arthritis. In spite of all precautions, a slight movement of the head on the operating table to get the light better led to instantaneous death. I would be very interested to hear any views—I know it is not relevant to ankylosing spondylitis perhaps—when one should intervene in patients with demonstrable instability at this joint.

*Grogono, B. J. S.* (Canada). We have had three cases like Sir Ludwig's of ankylosing spondylitis with bamboo spine who received an injury, and we are treating one particular patient at the moment—a man of 40 who had a car accident three months ago and had an almost identical injury with a hyperextension injury and an incomplete lesion to begin with when I saw him together with a neurosurgical colleague. We did a myelogram on him because his lesion became complete, and this is relevant to the vascular supply of the cord which we are discussing. Despite the fact that the myelogram did not show a block he had a complete lesion and we treated him conservatively, as Sir Ludwig has advised, and his spine has fused. One of the first patients I saw in Canada was a man who had a completely fused spine but an unstable atlanto-axial joint, and the only way he could eat was to lie down and bend his head forward and move his one remaining joint in order to get a morsel of food in his mouth. This man survived for about a year and then succumbed. I am interested in the exact pathology and what you do with a patient who has an incomplete lesion who comes to you with an ankylosing spondylitis.

*Sir Ludwig.* This seems to be a very interesting subject. That case I showed you here, where there was a fracture through the 7th cervical vertebral body in extension, was very interesting because he could walk home but developed on the next day pretty suddenly pins and needles sensation and very rapidly a complete tetraplegia. I assumed that this man developed a vascular catastrophe.

The question of operation in these cases is, of course, a rather difficult one. If there is an incomplete lesion I would not touch it; I would wait because, as in such cases, especially in elderly people over 50, if one operates one only increases the vascular shock these people already have. I would not contemplate for a moment to operate. I do congratulate Dr. Grogono that he was able to do a myelography in such a case. I have tried to do an alcohol block in a man with profound spasticity and was unable to get the needle into the subarachnoid space. This is a case (Mr. Sutcliffe Kerr will be interested to hear this) where I would agree with an anterior rhizotomy.

*Grossiord, A.* (France). Have you seen any osteomas in these patients?

*Sir Ludwig.* In the first case I demonstrated, where you saw in the A.P. the fracture

line and afterwards the enormous massive ossification, that might be an osteomatous condition. But the true osteomas I have not seen in our material at all.

*Firth, J. L.* (England). Concerning the point when one should operate or not, these are concerning cases seen at St. Thomas's and Queen Square, I hope I am not stating the obvious, but in a large number of people most of the blood supply to the cervical cord is down the anterior spinal artery which comes from the vertebrae. One can often have a very high cervical or atlanto-axial lesion at that level and yet the case presents a low cervical clinical picture. You block off the arterial supply high in the neck but it presents clinically down at the bottom end of the cervical spine. One has to be aware of a low cervical lesion, with no apparent local clinical picture in the low cervical spine of an arterial block high up, and in many cases you have to do arteriography to investigate the entire set up of the vertebral artery supply of the cervical spine before you can be absolutely certain of the set up in any one particular case.

*Harris, P.* (Scotland). In relation to the last speaker and what Sir Ludwig has said, I think the blood supply must be extremely important in these patients, and I wonder if Sir Ludwig can tell us something about the mechanism of the cervical injuries in these patients. Did they have hyperextension injuries in particular? Professor Romanes showed here just about a year ago that the blood supply comes mainly from the 5th cervical radicular artery on one or both sides and very often the lower lesion in the cervical spine will produce a higher neurological lesion. Another practical point is the importance of very careful intubation in any patient with ankylosing spondylitis or rheumatoid arthritis.

*Sir Ludwig.* With regard to the vascular supply, what our friend has said is absolutely correct. You can have the damage of the vessels much higher and yet get a lower paralysis. But I would say if you have this mechanism you will get probably in the majority of cases (of course there are very few cases so far) incomplete lesions. But, if you look at the A.P. films from these patients carefully, you might have noticed not only the ossification between the cervical vertebrae itself but also between the cervical and the upper costal area and the whole thing was just a mass of ossification, and the question is whether in some of these cases the vascular damage can be lower down. The mechanism which Harris asked me to explain is a very important question. The general views held are that extension fractures either do not produce any spinal cord injuries or minor ones. This is not true and I suggest that, in these cases, the spinal canal itself is very narrowed by the ossification and any sudden change either in flexion or extension leads to injury either of the vascular supply or the spinal cord itself.

## SPINAL CORD COMPRESSION IN A PATIENT WITH ANKYLOSING SPONDYLITIS AND PAGET'S DISEASE

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THE thoracic spinal cord is rarely involved in ankylosing spondylitis and only slightly more commonly in Paget's disease. The present case is reported because of the rarity of such a complication in these two diseases.

**Case Report.** The patient is a housewife and was born in 1903. At the age of 18 she first developed pain in the mid-thoracic region of the spine. She remained in bed for