NEUROPATHIES CAUSED BY BONY SPURS IN THE CERVICAL SPINE WITH SPECIAL REFERENCE TO SURGICAL TREATMENT

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Osteophyte formation, in association with prolapsed cervical disc, has previously been reported. The disc was thought to be the only important factor from the clinical point of view until Bull (1948) studied the radiological evidence and measured the size of nuclear material at various cervical levels. He doubted the prime role of the displaced disc in the production of neurological symptoms and stressed the arthritic changes in the neurocentral joints. Philip (1950) further emphasized the importance of osteophytes in syndromes of root pain in the upper extremities.

The difficulties and dangers attendant on the surgical approach to the cervical encroachment syndrome have previously prevented full recognition of the deformities responsible for the neurological syndromes found at these levels. This paper is based upon operative, radiological, and clinical observations, and will emphasize the importance of bony spurs compared with the disc prolapse. A more exact scrutiny of the deformity and its anatomical and mechanical relationship to nervous structures has resulted from improvement in surgical technique. Numerous cases of cervical root and cord disturbances have been studied clinically and radiologically. Of these, 19 failed on conservative treatment and operation was performed to prevent progression of symptoms. These patients showed intractable brachialgia or progressive root and cord dysfunction, or both together. A minute portion of prolapsed disc tissue was found in only one of 19 cases, while in all cases there was an osseous spur at one or more levels, with varying degrees of hypertrophy of the overlying posterior common ligament. In all cases the spur formed a bridge, traversing the thecal canal from one intervertebral foramen to the opposite and the adjacent margins of each body contributed in the formation of the spur. The morphological features of the bridge and its anatomical relationship to the multiple nervous structures crossing it will be described later. Such spurs

are so placed that they may interfere with many neural elements. Eight cases are reported, illustrating the more common syndromes and the possible resemblance to other conditions, such as progressive muscular atrophy or subacute combined degeneration of the cord. They demonstrate too the variability of cervical root disorders, occurring alone, or with cord involvement. Either nerve root or spinal cord signs may appear first and either may remain predominant throughout. With regard to brachialgia, operation was undertaken when root pain resisted all forms of conservative treatment, or when it merely represented one part of a wider syndrome with cord involvement. In all 19 cases the bony bridge was removed in its major part, or in its entirety. Post-operative follow-up is recorded for periods of up to 27 months. A posterior laminectomy alone was carried out in one case (No. 8) at the level of the spur, which at this time was thought to represent a venous malformation. Temporary improvement resulted, followed by further deterioration. Case 7 demonstrates that spurs in the low dorsal and cervical regions can produce two separate disabilities.

Case Reports

Case 1 (No. 3797).—A man aged 38 years, a salesman, on April 6, 1949, complained of pain in the right arm since 1942, and of weakness and wasting of this limb, with wrist drop since 1945. He was involved in bomb blast in 1942, and dates the onset of pain in the arm from this time. He was then in the Forces. He developed a schizophrenic psychosis and was given full insulin therapy, which aggravated the neuritic symptoms. There was a good recovery from the psychiatric condition, but suddenly in 1945 he found that the right hand was weak in its grip and clumsy in handling small objects. Later he could not write and noted fibrillation in small muscles of the hand. He was seen at the National Hospital for Nervous Disorders, Queen Square, London, and was examined by three consultants. Extracts from their reports read :

"... I can but agree with the diagnosis of progressive muscular atrophy (made first in 1945 on muscular weakness, wasting and fibrillation of arm muscles), and I think he is, in addition, developing a certain amount of spasticity as shown by increased reflexes. The prognosis is, of course, extremely bad and I have been disappointed with all forms of therapy advocated in this disease."

"... It has been suggested that his symptoms are due to herniation of an intervertebral disc, but I could not discover clinical or x-ray evidence of this. The minimal sensory symptoms are also against this diagnosis. My own impression was that he has an anomalous form of progressive muscular atrophy and Dr. ... came independently to the same diagnosis. I must admit that I am not wholly satisfied with this diagnosis, but after careful investigation of his case I could come to no more probable conclusion."

He consulted us on April 6, 1949. Pain was now down the ulnar side of the left arm and over the lower right ribs at the back. The neck movements were full, but extension produced pain in the right side of the neck, radiating to the shoulder tip.

Cranial nerves were normal.

Motor Functions.—There was wasting and weakness in the right pectoralis major, supra and infra-spinatus. The shoulder girdle drooped slightly on the right. Weakness and wasting were well marked in the triceps, the forearm, and hand muscles on the right, and most marked in the extensors of the wrist and fingers, the thenar, and hypothenar muscles. It was less marked in the biceps, deltoid, and spinati. The right scapula was winged. Fasciculation was continuous in all the affected muscles, and muscle tone was reduced on the right. In the left upper limb there was subjective weakness of the hand, but little objective cause could be found. Sweating was profuse over the ulnar borders of both hands.

In the lower extremities power and tone were normal and symmetrical.

	Right	Left
<i>Deep reflexes</i>		
Biceps	++	+
Triceps	—	±
Supinator	±	+
Knee	++	++
Ankle	+	+
<i>Superficial reflexes</i>		
Abdominal	+	+
Plantars	flexor	flexor

With these should be compared the findings in 1947 at the National Hospital that "all the reflexes in his right arm are absent and those in the left depressed".

Sensation.—Pain, touch, vibration, and joint sense were normal throughout. A radiograph showed narrowing of C.5-6 and less of C.6-7 interspaces. In the right oblique there was narrowing of the nerve canal at C.5-6.

Operation.—On April 20, 1949, under pentothal, gas, and oxygen, a midline neck incision was made and laminae C.5 and 6 were removed. A bony deformity, anterior to the cord and producing kinking and posterior displacement, was found. There was marked constriction of the mixed roots C.5 and 6 on the right. The deformity was seen to be due to an osteophyte formation anterior to the roots at both levels. Anterior and posterior decompression was carried out on the right and this freed the two mixed roots. On the left only a posterior decompression was done. On this side the bony spurs, although present, did not reach the same height. On both sides the dentate ligaments were cut and the dura was closed. At operation the cord was seen to be compressed and immobile.

No new signs were detected in the immediate post-operative period. Twenty-four hours later the patient volunteered that his fingers were "freed". Six days later there was subjective improvement in abduction and adduction of the right fingers and extension of the right thumb. On discharge on May 18, 1949, improvement was still mainly subjective.

Six months later he reported a gradual improvement in "freeness and power" of all movements of the right arm and hand. Wasting and fasciculation were still present in this hand, but thenar and hypothenar muscles were fuller and power had improved. Pronation was still very weak. Extensors of the wrist, pectoral muscles, biceps, triceps, deltoid, and supraspinatus were almost normal. In both arms the reflexes were all present and slightly increased. In the left arm power, tone, and facility of movement were normal.

The lower limbs were normal and no abnormality of sensation was discerned.

This man's condition had been diagnosed by three eminent neurologists as progressive muscular atrophy. There is, therefore, no need to stress the similarity of the syndrome to this condition, but it might be noted that pain was perhaps too prominent. The fluctuation in the reflex findings over four years and the complete absence of sensory abnormality despite involvement of the mixed nerve roots is of interest.

Case 2 (No. 4335).—A man aged 73 years, an ex-railway guard, was examined on August 20, 1949, and complained of loss of normal movement and sensation of the hands and legs.

Ten years previously he had noticed "numbness" of the legs, but this disappeared after two months. Seven months before examination it recurred, mainly in the feet, to be followed by weakness of the right, then the left leg. Weakness gradually progressed and he fell on three occasions. Frequently there was a feeling of "muslin cloth" wrapped around his feet. Then the arms became "numb". He became unable to write as he could not hold a pen properly, and dropped objects without noticing. The hands were shaky and he was unable to play the piano. Distinction of heat and cold in the arms and legs deteriorated. The right arm was weaker than the left. Shooting pains in the neck and

shoulders were worse on the right. Micturition was normal.

There was slight pallor of the optic discs, but cranial nerves were otherwise normal.

Motor Function.—Power was reduced in both arms and legs, more so on the right. He was unable to stand for long. There was weakness of all muscle groups in the lower limbs, with increased tone and clonus, especially on the right. In the upper extremities power was generally good, but there was slight weakness of finer finger movements, with commencing wasting of the first dorsal interosseous on the right. There was some subjective difficulty in using the hands.

Sensation.—Vibration and deep sensibility were markedly impaired in all extremities. Pain, touch, and temperature appreciation was grossly depressed in both legs. There was a variable sensory level about D.11–12. While under investigation pain loss appeared in C.6 and 7 on the right, and a sensory level became stabilized on the trunk at D.3.

	Right	Left
<i>Deep reflexes</i>		
Jaw	+	+
Triceps	++	++
Biceps	++	++
Supinator	++	++
Knee	+++	+++
Ankle	+++	+++
<i>Superficial reflexes</i>		
Abdominal	—	—
Plantars	Extensor	Extensor

Blood pressure was 165/85, and pulse rate 80 per minute. The heart borders were normal to percussion and there were no murmurs, but many extrasystoles. A blood count showed mild anaemia, but no abnormality in the red cells was observed.

A test meal (June 28, 1949) showed achlorhydria.

There was a slow rise and inadequate fall of the cerebrospinal fluid on jugular compression (1 lymphocyte per c.mm.; total protein, 63 mg. per 100 ml.).

Radiographs showed marked generalized osteoarthritic lipping throughout the vertebral column. "Encroachment" was demonstrated into the foramina on each side at C.6–7 and C.7–D.1.

Lipiodol myelography showed a temporary "flat type of cupola" formed opposite interspace C.7–D.1 and C.6–7 in the prone position. Lateral views showed filling defects at these interspaces.

Operation.—We operated on September 13, 1949, using pentothal and gas and oxygen anaesthesia.

With a midline incision a laminectomy of C.7, the lower portion C.6, and the upper portion D.1 was performed. The dura was opened. An obvious spur, bridging from side to side, and formed by contiguous borders of C.6 and 7, was found. This involved the mixed roots at C.7 and the anterior radicles of C.8, which were already discrete at C.7 level and placed

more medially. On flexing the neck there was posterior displacement of the cord, producing pallor. Posterior and anterior decompression was performed to free nervous structures.

A smaller spur was found at C.7–D.1 with involvement of mixed roots of C.8 and anterior radicles of D.1. On flexion of the neck, pallor of the cord was less marked at this level. Only posterior decompression was carried out. Dentate ligaments were cut on both sides over these segments.

Owing to the age of the patient anterior decompression of the roots and cord was less extensive than in Case 1.

There was no immediate accession of neurological signs, and 10 days later the patient felt the numbness fast disappearing from the legs and the pains from the arms. He was discharged on November 11, 1949. Superficial healing of the wound was temporarily delayed.

He was last seen in September, 1951, when he was walking unaided. He carried a stick for confidence, although this was not necessary. The legs were still tiring after much exercise. Movements of the neck were absolutely free. He was now able to play the piano with adequate dexterity.

Examination demonstrated no impairment of the long tracts. The root signs were not elicited. In the lower limbs there was depression of vibration sense only, compatible with age. There were no other sensory signs. Blood counts and test meal were not repeated.

Subacute combined degeneration of the cord was mooted at one time. The cord alone was first involved and later the roots. Only at a late stage could a clinical neurological level be determined.

Case 3 (No. 5017).—A man aged 36 years, a parks superintendent, was examined on March 9, 1950. He complained of pain down the left arm and from the shoulder referred to the radial side of the forearm and index finger. Latterly he had perceived numbness in the index finger. In 1944 he was thrown out of an army lorry, and there was some indefinite injury to the neck. Pain appeared about two months later coincident with some domestic troubles. He had previously been seen by neurologists, who diagnosed a prolapsed cervical disc. Physiotherapy, neck traction, and later collar immobilization all failed to give relief.

On examination no motor involvement was detected, but there was alteration in the quality of sensation in dermatomes C.6 and 7 on the left, and to a less extent in C.5. There was no evidence of cord dysfunction. The left biceps reflex was depressed.

Radiographs showed a massive osteophyte formation, formed by the posterior lips of the bodies of C.5 and 6. The osteophyte was very evident in the left intervertebral foramen and extended across anterior to the cord, this being well shown on the lateral plates. There was narrowing of the interspace with a cervical kyphosis.

Operation.—On March 9, 1950, under general anaesthesia a window of bone was removed, leaving virtually intact the laminae and spines at C.5–6. Mixed nerve C.6 was exposed and this was very flattened over an anterior spur, the "high-spot" of which was just medial to the axilla of the nerve. The spur extended laterally

into the foramen. The spur was removed anteriorly over its whole visible and palpable extent.

By the second day the arm pain disappeared. Between the sixth and ninth day it reappeared slightly and then again disappeared. The patient was discharged on March 28. There was still some neck stiffness, but no abnormal signs. He had no other subjective complaints. He was last seen in August, 1951. There was still some stiffness of the neck, rather less than before. Passive movements of the neck were free in all directions. There was occasionally a transient twinge of the former pain in the arm. He had returned to work which involved lifting and moving heavy objects. Radiologically the cervical kyphosis persisted.

This patient illustrates the group in which brachialgia is the presenting and dominant symptom. Again despite involvement of a mixed nerve root there was no paresis or marked objective sensory loss.

Case 4 (No. 4513).—A man aged 47 years, a business man, was examined on January 1, 1950. He complained of progressive weakness in the right leg for one year. He had suffered from flat feet for many years, and thought that this caused pain in the calves. One year before the right leg began to drag, and had become considerably weaker than the left leg. He had no pain or discomfort in the limb. He had been seen by many orthopaedic surgeons and had been given various forms of physiotherapy. The picture was one of slow deterioration, no injury, and never any lumbago or sciatica. He had no bladder or bowel symptoms.

On examination it was seen that he walked with a slight limp. The cranial nerves were normal, as were the upper extremities.

There was slight wasting of the right quadriceps, and marked reduction in power of all movements of the right lower limb, particularly, however, in the flexors of the thigh and dorsiflexors of the foot. Within limits of power there was no incoordination and no fasciculation.

	Right	Left
<i>Deep reflexes</i>		
Biceps	++	++
Triceps	++	++
Supinator	++	++
Knee	+++	++
Ankle	+++	++
<i>Superficial reflexes</i>		
Abdominal	+	+
Plantars	equivocal	flexor

In hospital both plantars became extensor in type.

Myelography demonstrated an indefinite defect at D.12. Here also was a doubtful pedicle widening. In the prone position throughout the whole cervical region there was a "stickiness" in flow.

First Operation.—On February 2, 1950, under general anaesthesia, and acting on the minimal radiological evidence, a negative exploration was performed at D.11-12. No change in his condition resulted.

In subsequent months there was a deterioration of power in the upper and lower limbs, particularly the right. The lower limbs became so weak that the patient was unable to walk. Apart from subjective numbness in the third and fourth fingers of the right hand there was no sensory disturbance. Intermittent fasciculation was noticed in the muscles of the upper limb. There was no sphincter disturbance. Objectively, weakness of all limbs was confirmed. The deltoid, extensors of the fingers, and the small muscles of the hand, particularly those of the thumb, were weak and wasted. There was no further wasting in the legs, but spasticity was present.

All deep reflexes were increased. Both plantars were persistently extensor. The jaw and pectoral reflexes were increased. A slight loss to vibration was found over the malleoli.

Radiographs showed extensive spur formation, especially at C.5-6, 6-7, and C.7-D.1, and less at C.4-5. C.5-6 interspace was narrowed.

Second Operation.—On May 1, 1951, the laminae of C. 5, 6, and 7 were removed. Intrathecal inspection showed marked pallor and immobility of the cord. Posterior displacement of the cord coincided with the anteriorly placed spurs at C.5-6, 6-7, and C.7-D.1. These spurs were entirely removed and the cord was replaced in a more natural position with resulting increased mobility.

After operation there was no accession of neurological signs, and after 48 hours there was subjective freedom of movement of the ankles and feet, and clonus was less. After three days the right plantar response was flexor. Thereafter the right plantar reflex was variable and bilateral clonus was occasionally found.

At the fourth week he started walking and could take an average good step.

After six months he reported that his hands were more powerful and he was able to use them for everyday actions. Wasting was now less obvious and deep reflexes of the arm were normal. Clonus in the legs was ill-sustained and the plantar reflexes were variable. Psychologically one felt that this man was somewhat frightened of himself and was not exerting the full available power in his legs.

This case is reported mainly to draw the moral from the rapid progress between the two operations. The increase in disability was largely motor and referable to the lower limbs. Only at a late date did clinical signs appear to suggest a cervical level. The follow-up is too short to assess the effect of the second operation.

Case 5 (No. 5143).—A man aged 45 years, a surgeon, on May 7, 1950, complained of progressive weakness of the left hand for 15 months.

Six months before the onset of disability he fell from a chair and hit his head against a wall. Some stiffness of the neck followed and he then noticed weakness of the small muscles of the left hand, at first when closing artery forceps. Weakness progressed and wasting became so extreme that he became unable to operate. There was no pain, no undue sweating, and no fasciculation.

On examination there was no Horner's syndrome and the cranial nerves were normal.

In the left upper limb below the elbow the musculature was reduced in bulk. There was marked wasting in the muscles of the thenar and hypothenar eminences, while the interosseous muscles and lumbricals were severely affected with a corresponding loss of power. Weakness of flexors and extensors of the wrist and fingers was however minimal, despite the wasting of these muscle groups. The other limbs were normal.

The deep reflexes were symmetrical in both upper and lower limbs. Plantars were flexor.

Sensation.—This was normal to all modalities throughout.

Radiographs showed C.6-7 interspace narrowed, and spur formation on lateral and oblique views at C.4-5 and at C.6-7.

Operation.—On May 25, 1950, under general anaesthesia, "windows" were made at C.6-7 and C.7-D.1. A spur at C.4-5 was ignored as incapable of producing the clinical picture. Clinically the level C.7-D.1 was more implicated, and despite negative x-ray findings a window was made here. A large spur was found at C.6-7 and was removed anteriorly. There was compression of the mixed root of C.7 and anterior root of C.8 at this site. Nothing abnormal was found at C.7-D.1.

In this case the spur had its "high-spot" beneath the upper axilla of the C.7 root. There was relative post-fixation of the emergent root. The height (or depth) of the spur reached $\frac{3}{8}$ in.

There was no immediate deterioration. The patient was seen in January, 1951, seven months after operation. He had returned to surgery, but required trick movements to handle instruments. However, before operation he was quite unable to operate. The bulk of the first dorsal interosseous muscle had decreased further but in all other movements power was improving. Particularly it was to be noted that there had been no extension of the muscle groups affected, nor had there been any increase in the weakness of the affected muscles. Before operation this surgeon had been given faradism, shoulder girdle exercises, and a course of "stimulating" x-ray therapy to the cervical spine. In spite of these measures his condition had progressively deteriorated.

In this case there were no sensory abnormalities, nor was there a history of pain. The case again demonstrates the dissociation of extreme, focal motor loss without sensory disturbance, although mixed cervical roots are involved. The progression of wasting in some of the intrinsic hand muscles without spread to other groups might indicate that the affection of C.8 motor root was too complete to be relieved by operation.

Case 6 (No. 5301).—A man aged 44 years, a farmer, consulted me on July 20, 1950.

In January, 1950, driving on a long journey, he developed pain at the back of the neck, radiating to the left shoulder. Two days later the neck became stiff.

He had physiotherapy and x-radiation without improvement. In March, 1950, the right side of the trunk and right leg became numb. Numbness in the leg was more marked below the knee. At the same time the leg became weaker and dragged when he walked. There was, however, no limp. In June, 1950, numbness of the ulnar side of the left palm and to a lesser extent of the right palm was noticed. In July, 1950, the left hand-grip became weak and again to a lesser extent the right grip. All objects touched with the palms were cold. He had precipitancy in the mornings, but no other disturbance of micturition. When the bladder was full, sensation was appreciated only on the left side of the lower abdomen. Sexual functions were normal. He was sleeping badly. Two weeks before examination there was a sudden deterioration of power in the left hand, but there had been no further deterioration since then. Pain in the back of the neck was still present, but had been much less severe for one month. It was aggravated if he leaned forward in a sitting position.

On examination the patient was seen to be obese, with no limp. There was a large port-wine stain over the left trunk and left buttock and upper thigh. This did not cross the midline and had been present since birth. The neck movements were normal in range, but extreme extension produced a pain, radiating to the left shoulder. This pain was similar in nature to that which marked the origin of his illness. The cranial nerves were normal.

Motor Functions.—Power was reduced in the left arm. There was general weakness in all muscle groups, but most marked in the small muscles of the hand. The right arm was normal, tone was symmetrical, and posture was well maintained. There was some clumsiness of fine and alternating finger movement on the left.

There was a general reduction of power in the left lower limb, but tone was normal.

	Right	Left
<i>Deep reflexes</i>		
Biceps	+	++
Triceps	++	+++
Supinator	+	++
Knee	++	+++
		(clonus)
Ankle	+	+
<i>Superficial reflexes</i>		
Abdominal	—	—
Plantars	flexor	flexor

Sensation.—Pain, touch, and temperature sense were depressed in C.8 and D.1 on both sides. On the left C.7 was slightly involved to the same modalities. Vibration was slightly reduced in the same distribution on the left. Joint sense was intact.

Pain, touch, and temperature sense were less well appreciated on the right side of the trunk below D.3. The depression extended to the midline.

Pain, touch, and temperature sense were depressed in the right lower limb. Joint sense was intact and vibration sense was absent.

General examination was normal.

Pre-operatively this was thought to be an angiomatous malformation of the cord, with a solid tumour at about C.6-7. Myelography was performed and showed "stickiness" of flow upwards from D.12 with a "flat type" of cupola at C.6-7 interspace. The block was, however, partial.

Operation.—On August 10, 1950, under general anaesthesia laminectomy at C.5 and 6 was performed. The dura was opened and the cord was found to be kinked at two levels, opposite C.5-6 and 6-7 interspaces, displaced posteriorly, flattened from side to side, and pale at the operation site. At each of these levels there was spur formation. The upper spur involved the mixed root of C.6 on each side and at this level the anterior roots of C.7 were implicated in front of the cord. Decompression at this level was performed anteriorly from side to side. The spur between C.6-7 did not extend laterally. Its "high-spot" was in the midline. It involved the cord and the anterior radicles of C.8. The position of this spur precluded its removal with safety at this sitting, as much handling had already occurred at the higher level. It was left, and a posterior decompression with section of the dentate ligaments performed at this zone. The posterior ligament was thickened to $\frac{1}{8}$ in. over the upper spur. An angiomatous malformation of minor degree was found over the length of the exposed cord, but considered insufficient to cause the major symptoms.

On recovery from the anaesthetic the patient moved all limbs and stated that the numbness of the right leg had disappeared. He passed urine normally. He was walking on the fifteenth day, and discharged on September 6, just under one month from operation. He returned to work after two months.

One year later numbness in the trunk and leg had disappeared, apart from a small zone on the outer side of the right calf. There was no weakness of the upper or lower limbs, or pain or stiffness of the neck. He still felt numbness in the ulnar borders of both hands.

All reflexes were normal. There was some diminution of pain in C.8 distribution on both sides, but no loss elsewhere. Bladder sensation and function were normal.

This patient finds the numbness in C.8 distribution an annoying disability. He is very willing for a second operation if this could be eliminated. It is possible that the C.6-7 spur anterior to the cord may yet have to be dealt with, especially as more serious symptoms may eventuate.

It is noted in this case that there was an abnormality of vascular supply to the spinal cord, presupposed at clinical examination and confirmed by inspection at operation; yet evidence is here adduced to show that the congenital abnormality had but little influence in the aetiology of the syndrome presented. However, one cannot but speculate on the role which this abnormality subserves, when compression of the cord is produced by bony deformity or by any other means.

In this case spur deformity was found at operation and the radiological diagnosis made only in retrospect. The case indicates the ability of a bony spur to produce a predominantly sensory defect in terms of the cord conduction tracts. Only at a late stage did sensory, motor, and reflex signs, referable to root involvement, appear.

Case 7 (No. 4573).—A middle-aged woman consulted me on January 2, 1950, and complained of pain in the back and lower limbs. She had had a serious fall four years previously, with no sequelae at the time. In June, 1949, while bending to arrange papers her back became "locked" and she was unable to straighten up. A few hours later, on attempted movement, there was pain low in the back and down the right thigh. This continued and grew more severe. There was temporary relief on resting. Pain recurred in August, 1949. Conservative treatment and manipulation under anaesthesia gave no relief. She became incapable of any occupation requiring much movement. The left leg became weak with dragging of the foot.

On examination the upper limbs, trunk, and cranial nerves appeared normal. There was considerable weakness, with wasting of the glutei, hamstrings, quadriceps and peronei, more on the left, but no fasciculation. Tone was normal. The left lower abdominal reflex was absent and the deep reflexes were depressed in the left leg. Sensory loss was apparent to all modalities to the level of D.12-L.1 on the left, most marked to pin prick. Vibration was lost to the iliac crest, but joint sense in the left great toe was good. Movements of the back and left leg were restricted by pain. There was considerable spasm of the paravertebral muscles.

Myelography showed a filling defect at the junction of D.9 and 10, outlining a medial lesion anterior to the cord.

Operation.—On January 12, 1950, a laminectomy at T.9 and 10 was performed, and a small, mushroom-shaped, bony spur was removed from the midline, anterior to the cord. In the words of the surgeon: "it hardly seemed as if it would account for her symptoms", but the thecal canal was narrow at this point.

Within one week there was disappearance of the previous pain, and improvement in sensory loss. She was discharged on February 2, 1950, with residual stiffness of the low back related to the operation wound; there was no sensory residue, or dragging of the left foot.

This case has been introduced to indicate the role that bony spur formation may play in producing progressive cord or root dysfunction at levels other than the cervical spine—here in the low dorsal region—and also in my experience at the lumbosacral region, where it may bear partial responsibility for the syndrome of a prolapsed disc.

Case 8 (No. 4424).—A housewife aged 47 years was seen on October 22, 1949. In December, 1945, she developed pain in the right arm and shoulder, radiating into all the fingers. Before this she had been nursing her father in war-time Holland, under difficult conditions

and at times carried him on her shoulders. The onset was sudden with a sense of uselessness of the hand. She was unable to write easily and dropped things. She experienced "pins and needles" and "shocks" of indescribable pain in the hand and forearm. At the end of 1946 the right leg became weak and dragged. Treatment with physiotherapy, infiltration of "fibrositic" nodules, and neck-traction was given for five weeks between January and March, 1949. Paraesthesia improved initially, but afterwards there was aggravation of pain. Thereafter she reported steady deterioration; there was also occasional pain in the left arm and attacks of clockwise vertigo.

On examination there was no nystagmus, and no evidence of vestibular or cerebellar dysfunction. Neck movements were limited, especially extension. There was weakness of the intrinsic muscles of the right hand, flexion of the wrist and fingers, pronation and supination of the forearm, and slight weakness of extension of the wrist and elbow and of the deltoid. There was wasting of the first interosseous muscle on the right. Biceps, supinator, and triceps jerks were depressed on the right. Vibration and joint sense was depressed to the shoulder, and pin prick was depressed in C.6 and 7 dermatomes on the right. In the right leg there was weakness of plantar and dorsiflexion, with increased tone of the distal muscles. There was an increase of right knee and ankle reflexes with clonus and extensor plantar responses. Abdominal reflexes were all absent. Blood and cerebrospinal fluid were normal. Manometry gave normal results.

Radiologically there was loss of the disc space with spur formation at C.4-5 and C.5-6. Spur encroachment was clearly evident in the right and left oblique views at the lower level. The whole body of C.5 appeared to be drawn backwards breaking the contour of the spinal canal at both levels.

At this time it was felt that the bony deformity was incapable of producing such a disability unless a prolapse of the disc was also present.

First Operation.—On November 3, 1949, through a midline incision laminectomy at C.3, 4, and 5 was performed. The theca was opened. The cord was displaced slightly backwards. On rotation a bulge was felt and seen anteriorly and extrathecally and more to the right. Inspection extrathecally was incomplete as intense venous congestion caused bleeding so that a venous malformation was suspected by the surgeon, who considered it was impossible to proceed. The posterior decompression and section of the dentate ligaments, it was hoped, would relieve the symptoms.

Catheterization was required for five days, after which the bladder was normal. Other symptoms improved. When discharged on November 26, 1949, there was mild pain only in the right arm, and the patient felt movements of the right fingers and foot to be freer. There was subjective improvement until April, 1950, when symptoms recurred and steadily became worse. She was re-admitted in February, 1951, and refused conservative treatment. The left arm was now involved to a greater degree and there was pain on the right in the middle finger, on the left in the fourth and fifth fingers.

The right leg was weaker and apt to fall suddenly. Pain was burning in nature. Both hands and the right foot sweated freely.

On examination it was seen that the same muscles of the right upper limb were affected, but they were weaker and more wasted. There was now some wasting of the intrinsic muscles of the left hand with weakness of triceps and biceps. The right leg's condition was as before. The deep reflexes on the right upper limb were now slightly increased, and depressed on the left. There was clonus at the knee and ankle on right, with slight hyper-reflexia of the left leg. The right plantar response was extensor, the left flexor, and the abdominal responses were absent. There was diminished sensation to pin prick and cotton wool in C.6, 7, 8, and T.1 right, maximum C.7. Vibration and joint sense were markedly depressed in the right arm but intact in the left. Vibration was slightly diminished in the right ankle. Thus there was a marked increase of abnormal neurological signs.

Radiographs showed loss of cervical lordosis, exaggerating the prominence of the spurs compared with 1949.

Second Operation.—On February 22, 1951, the old incision was re-opened and lamina C.6 removed. The dura was re-opened. The cord was very pale and stretched over an area corresponding with the body of C.5. A large anterior spur was found at C.5-6 level with a "high-spot" indenting the cord medially and minimal in height laterally beneath the roots. A lesser spur was seen at C.4-5. The spur at C.5-6 was removed completely extra-theically after great difficulty with extreme venous haemorrhage. No venous malformation was seen. The cerebrospinal fluid now flowed freely, the cord was in a more normal position with improved capillary pattern posteriorly and lessening of venous congestion. The dura was closed.

The patient was discharged on March 31, 1951. She experienced a slight increase of numbness at C.7, but no other neurological change. She had passed urine normally on the first post-operative day.

On August 7, 1951, she had no pain or paraesthesiae in either arm, but some numbness of the ulnar side of the right hand. Movements of the arms and legs were free. She could write legibly and sew. Her walking was much better and the wasted muscles were filling out. There was a slight increase in the left biceps jerk; otherwise upper limb reflexes were normal. The right plantar was now flexor.

She experienced no loss of vibration or joint sense, but slight diminution of reaction to pin prick in C.6 and 7 right, especially in the tips of the fingers.

This case is possibly the most important of the series for it shows not only that an anteriorly placed, transverse bony spur, unassociated with any protrusion of disc material, can produce serious malfunction of both spinal roots and cord, but it also points the surgical moral that when the distorting abnormality is placed in front of the cord it is futile to expect permanent relief from posterior decompression with section of the dentate ligaments.

In fact it is more probable that distortion of the cord with aggravation of symptoms will result thereby.

Discussion

Bony "spurs" were removed surgically from a series of 19 cases. All but one of these spurs were in the cervical region. Eight illustrative cases are quoted to show the variability of the neurological syndrome encountered and, within the limits of a perhaps inadequate follow-up period, to record the amelioration to be expected from a surgical attack on these abnormalities. I would again emphasize that all these cases had previously had conservative treatment. This is a relatively new field in surgical neurology and this paper must, therefore, necessarily be in the nature of a preliminary communication. As further experience is gained, so one must expect modifications in approach. It is with a full realization of these considerations that I now propose to discuss the various features of this series of cases.

Aetiology.—The youngest patient was 27 and the oldest 73. Six were in the third decade, ten in the fourth, and one in the sixth.

Generalized osteo-arthritis was present in only the three oldest patients, but in the rest the spur or spurs were isolated radiological lesions. It is for this reason that the term bony "spur" is used in place of osteophyte, for the latter term conjures up a picture of widespread osteo-arthritic change.

In 17 cases an episode of sudden movement of the neck was remembered by the patient, but was often not related by them to the onset of the symptoms, which appeared in some patients months or years later. In others the onset of symptoms coincided with the trauma and the mature spur was shown to be present immediately after.

Form and Nature of the Spur.—The x-ray appearances of the spurs have been described by Turner and Oppenheimer (1936), and Philip (1950) has given diagrams showing the lipping from the margin of a vertebral body. The silhouette of the cross-section of the spur is *more often* that of a double lip from the adjacent body margins of two vertebrae and each contributes an equal share. The bases arise from varying areas and the cusps taper and curve to meet each other at the apices, $\frac{1}{8}$ in. to $\frac{3}{16}$ in. beyond the normal body contours. If the bases are large, they appear as two pyramids, each with perpendicular or curved edge (Fig. 1). The pyramids may be of denser contrast than the normal bone. In many the base is small and the cusp long and a "gargoyle" appearance may result. In others a wider base with a rounded apex show them as vague rounded hummocks. Usually, however, the

Fig. 1.—Line drawing to show diagrammatically the profile projections of the commoner shapes of the bicuspid spurs as seen radiologically.

cusps are separated in the same line as the interbody space.

At operation the linear extent of the spur formation is realized. It extends across the whole length of the posterior margin of the body (or bodies) from within one intervertebral foramen to the other. It is usually bicuspid at any point of cross section. Its side-to-side elevation is like a suspension bridge with two "high-spots" just within the lateral margins of the cord (Fig. 2*b*). Sometimes there is but one "high-spot" in the midline and here there is little ridging laterally towards the intervertebral canals and little compression of the emerging mixed nerve roots. The extent, height, and position of the spurs is usually only fully realized at operation and it is impossible to gauge their size by radiographs alone.

The spur has a brittle, sugary consistence. The bone is cancellous within a thin shell and scrapes away from its base on the body, leaving a shiny cleavage line. These features would suggest that it is no mere eburnation of the body margin, but is an ossifying structure, capable of considerable growth. The posterior common ligament, stripped from the body, covers the apex of the spur. There is a definite cleavage line between it and the bony spur. The ligament may hypertrophy over the apex to a thickness of $\frac{1}{8}$ in. or more, and thus add its thickness to the height of the spur. The annulus fibrosus retains its normal contour between the body margins.

As the apices of the cusps are strongly opposed and as the firm annulus fibres bind their bases, it is inconceivable that disc material protrudes between them. The disc material would appear to have atrophied *in situ*.

Seldom is the spur formation at one level or on one side alone. Usually the formations are at more than one level although they may vary in size. More often bilateral, the spurs may be asymmetrical, and then may show up on one side only radiologically.

FIG. 2.—Drawings of cervical vertebrae :—
(a) Cervical 7 to show spur position and profile projection in the lateral and antero-posterior views. corresponding to x-ray plates Nos. 3a, 3b, and 4.

FIG. 3a.—Right oblique view to show "pyramid" type of spur projecting into the intervertebral canal of C.5-6.

FIG. 3b.—Antero-posterior view corresponding with Fig. 3a to show the lateral extension of the spur into the vertebral arterial canal.

FIG. 3c.—Cervical 5 to indicate the osseous bridge extending across the vertebral canal as in (b). Here the high spot is single and mesial in position having an anterior relationship more to the cord than to the roots.

FIG. 2b.—Cervical 6 to show diagrammatically by the dotted line the annular zone of osteophyte formation. The spurs are indicated with two high spots, each just within the lateral margin of the spinal canal. The more lateral extension is through the intervertebral nerve canal and is even encroaching into the vertebral canal.

FIG. 4.—Lateral view to show typical bicuspid spur with interspace narrowing at C.5 on C.6. The cervical lordosis is straightened at this level.

Relationship of Spurs to Adjacent Nervous Structures and Effect on Nervous Function.—A bridging spur at a single level in its entire linear extent lies anterior to all nervous structures. It follows that from right to left it is related to the right sympathetic nerves in the vertebral arterial canal, the plexus from the lower cervical ganglion (Fraser, 1940), the emerging right mixed nerve in its dural sleeve, the cord and the right and left anterior root fibres destined to emerge at the level below (Fig. 5), the left mixed nerve and the left sympathetic nerves.

FIG. 5.—Line copy of x-ray plate to bring out the relationship of the radicals of the emergent root to the interspace at a level one higher.

If there are spur formations at two adjacent levels, at the lower level the pattern is repeated, but the anterior root fibres already involved at the higher level are again affected within the mixed roots, emerging at the lower.

At operation, when the dura is opened, the cord is found kinked and displaced backwards over a short distance, which corresponds to the anteriorly placed spur. The posterior surface of the cord is pressed against the dura, which is often thinned over it. The cord is pale and immobile and may even be flattened from before backwards. Transmission of pulsation by the cerebrospinal fluid over the affected segments is blocked but appears again below. The appearance of the affected area is as if it were "frozen". The displacement of the cord and the accompanying vascular effects are exaggerated by passive flexion of the head and neck.

Indeed at operation the flattening down of the nervous structures upon the anterior bridge may be so great as to prevent any access or retraction unless adequate posturing ensures a maximum cervical lordosis. In the more severe deformities as flexion is increased the "freezing" spreads centrifugally and the roots at nearby levels take up the tension. Above and below the spur there is congestion of the anterior extrathecal plexus of veins. At the apex of the bridge they are obliterated. A free flow of venous blood in these channels is re-established after the spur is removed. I do not wish to imply that disruption of venous or arterial flow is complete or constant in any but the most severe deformities. In all cases, however, there may be partial and temporary disruption proportional to the range of neck movement.

Clinical Picture.—The cases can be grouped into (1) those with cervical root involvement only. These patients almost invariably have pain in one or both upper limbs and usually there is nerve conduction loss. This almost always involves more than one root. (2) This group has involvement of the cervical cord, with conduction loss in the trunk and lower extremities. In the more severe cases and in the later stages both root and cord signs and symptoms may be present. As a rule the cases fall into one of the above two groups. Operation may be indicated in either group.

In this series the younger group—mainly the third and fourth decade—belongs initially or entirely to group 1. The older group belongs to group 2.

Group 1.—Pain is the initial and usually the dominating symptom. Frequently on one side only, it may spread to the other. The patients have difficulty in defining it, but it is usually severe. It is most frequently situated in the shoulder, scapula or upper thorax, pre-axilla or precordium. At this stage attempts to identify its origin within a certain root are impossible (compare Philip's findings). Later it will radiate into the arm; rarely does it enter any consistent finger distribution. The paraesthesiae which follow are of greater aid in root localization. These are often brought on by neck movements and felt in an index finger, or a definite diagnostic combination of fingers. Later sensory dermatome loss may be elicited. This is frequently circumscribed at first, but unfortunately often variable. More insidiously muscle wasting appears, corresponding to the affected roots. Fluctuating autonomic changes are often seen and may be confined to one aspect of one forearm or hand. To sum up: pain is of little localizing value, paraesthesiae are of slightly more value, dermatome

loss is of great help, and finally motor loss is critical to the roots involved.

In some cases there is an extraordinary dissociation between sensory and motor signs in their dominance of the clinical picture, despite the invariable involvement of the mixed nerve root in these cases. This is clearly shown by contrasting Case 3 with Case 5. The most probable explanation is afforded by postulating a vascular deficiency as opposed to a pure mechanical compression of the nervous elements. At operation, moreover, it has been consistently found that the mixed nerve root, immobilized over the bony spur, is extremely "touchy" and twitchings of the muscles innervated by it occur with very slight mechanical or electrical stimulation. The most irritable portion in terms of motor response is the antero-inferior aspect of the nerve, which is nipped and attenuated to a greater extent in those cases where muscle loss predominates. This differential anatomical nipping depends in turn upon the pre- or post-fixation of the emerging mixed nerve, relative to the spur. These observations may provide some explanation of the unpredictable neurological effects.

The state of the reflex arcs is often extremely variable in these cases. In some patients the arm reflexes are increased, despite obvious muscle wasting. In others they are reduced early and later lost. In Case 1 in 1945 reflex diminution was reported from the National Hospital, London, but in 1949 the same reflexes were increased, although muscle wasting and weakness had increased. Fasciculation was present in all cases with muscle wasting. Frequently there is a very sudden exacerbation of symptoms, particularly in the motor sphere.

Group 2.—Pain is rarely evident. The most frequent disability is referable to the pyramidal tracts. At first the symptoms remit. Later there is progressive weakness and stiffness of the legs, with bilateral Babinski responses. Over a period of months the motor weakness gradually creeps upwards. Some patients, more particularly the aged, show no sensory loss. This overall picture would suggest a systemic degenerative pathology, particularly where a lower motor neuron lesion in the arm appears. In such a case a diagnosis of progressive muscular atrophy would be reasonable. In one case the lower motor neuron lesion occurred before the involvement of the pyramidal tracts. In another case, No. 6, the spino-thalamic tracts were early involved, whereas the motor symptoms were more subjective than objective. Here the spino-thalamic tract of one side only was involved.

Clearly impairment of any tract or combination of tracts can occur. Often a remarkable and very

crisp tract dissociation occurs initially and may persist throughout. This selectivity is explicable on a vascular rather than a compressive basis. Compression is often not great, nor does it remain focal.

Case 4 illustrates a diagnostic hazard of importance. At first an ill-defined pyramidal lesion indicated no certain upper level. Only later root signs in the upper limb appeared and even then there was a "gap" between root and tract levels. This "gap" narrowed and eventually the levels coincided at the site of the spur. Premature assessment led to a useless laminectomy. In instances therefore where no definite lipiodol hold-up can be shown, and where the upper level, sensory or motor, fluctuates, surgery should be withheld until the level becomes definitive and stable.

Autonomic changes, such as increased sweating of the hands, frequently accompany the other disabilities. There may be coldness of the legs, often not noticed until after operation, when the patient will say, "Doctor, my legs are warmer. I did not know how much they were affected". This symptom may be the only one suggestive of cord involvement in the cases falling apparently into Group 1.

Diagnosis.—This rests on both clinical and radiographic evidence. At first positive radiographic findings were considered essential for the diagnosis. This still holds true where there is mixed root involvement. In some cases, where cord signs predominate, the spur has not been demonstrated convincingly radiologically. This is because it is not always easy to obtain the exact centring of the rays to throw up the spur profile, especially when the "high-spot" is medial. Frequently this "fugitive" spur does not coincide with the obvious narrowed interspace.

Even where no radiological evidence of spur is shown and even when a narrowed interspace is present, the existence of adequate clinical evidence of root involvement is more likely to indicate spur formation than disc prolapse.

Myelography was unnecessary in all but three cases. In these there was suspicion of tumour or angiomatous malformation. In these cases myelography was for exclusion only. It gave no other essential information but confirmed the presence of the anterior bony spur. In the A.P. projection a "flat" cupola corresponding to the interspace was produced with both upward and downward flow of the opaque medium with the patient in the prone position. Tilting must be extremely slow or the "spill-over" will be too rapid for the recognition of the deformity, and this will be missed if the

patient is placed even with minimal obliquity. Lifting of the opaque medium over the spur will show on the lateral view and this is perhaps more useful than the A.P. viewing. Lipiodol (Lafay), 1-2 ml., shows the abnormalities more adequately than less viscid media.

Differential Diagnosis.—Case 1 shows the similarity to motor neuron disease. Pain was a little severe and the progression too slow. The history of trauma is significant. Fasciculation was seen in the atrophied muscles.

Case 2 with its age of onset, hypochlorhydria, and the suspicion of remission suggested subacute combined degeneration of the cord.

A confident diagnosis of angiomatous malformation of the cord was made in Case 6. This pathology was in fact present, but the onset of root signs and the alleviation of symptoms after operation points to the bony spur as being mainly responsible for the syndrome.

X-ray and clinical evidence serves always to distinguish from conditions affecting the more peripheral parts of the brachial plexus.

Conservative Treatment and Indications for Surgery.—On routine radiographs of the spine spurs are frequently seen. Where there are no symptoms these mean little, except that the reserve is sufficient to allow of accommodation of the nervous tissues. Naturally no treatment is required. They are a potential danger only.

In a second group of patients there are symptoms of general tiredness and aching throughout the spine. There is minimal clinical evidence of neurological involvement. Radiographs of the whole spine are taken and an incidental finding is a cervical spur, usually between C.5-6 or C.6-7. It is this type of case about which so little is known, but one supposes that just as a lumbo-sacral disc degeneration may in many cases cause chronic back disability, so a cervical spur may represent the weak spot in the mechanics of the spine, particularly where there are abnormal postural strains. These strains would theoretically be most liable to occur where high mobility changes to low, that is at the cervico-dorsal junction and between C.5-6 and C.6-7. Treatment of these cases aims at correction of the postural abnormalities and to this the majority respond.

In cases of this type, where posture plays such an important part, drooping of the shoulder girdle may well supervene, and weakness of the scalene group of muscles will result in pull and drag on the brachial plexus, this in turn being transmitted to the spinal roots, which then impinge upon the spur as "a rope upon a jagged pulley". Such an effect is particularly liable to be produced in states of

chronic ill-health where muscle tone is below par or where prolonged immobilization in collar, plaster, or traction allows the muscles to lose their inherent tone. When this muscle state exists a relatively minor strain or injury will produce the neurological syndrome, dependent on compression by the bony spur.

Conservative treatment should aim to avoid this reduction of muscle tone and should not therefore include the prolonged use of collars, plasters, or unduly prolonged neck traction. Where possible it should allow of mild muscle activity and avoid extreme disuse of the anatomical splinting muscles of the neck. Prolonged, complete immobilization, during which neck muscles atrophy or become lax, is contraindicated. I would, however, emphasize that conservative treatment, where these principles are borne in mind, results in complete alleviation of symptoms in the majority of cases, at least temporarily.

The third group of patients is that in which all conservative treatment has failed to stem the progress of the symptoms and signs. In this group it is to be noted how rapidly the neurological syndrome may progress and particular attention is to be paid to the development of cord signs. All the cases presented in this paper fall into this group. None was operated upon until it was evident that adequate conservative treatment had failed. It is further emphasized that prolonged disability may be prevented by timely surgical intervention. The size of the spur, as shown radiologically, is no indication for surgery when divorced from the clinical state.

Surgical Technique.—The aim is to obtain the fullest decompression of nervous tissue, and to restore the normal mobility of this tissue in relation to the dura and to the extradural surroundings. The effect of the spur is not entirely one of compression. It fixes the dura over a wide area, much as a pelt pegged over an uneven surface. The peripheral points of fixation are the dural sleeves of the emergent roots in the intervertebral canals of each side, usually at more than one level. Relief of symptoms is proportional to the re-establishment of the normal soft-tissue "glide". Thereby the cerebrospinal flow and the vascularity of nerve roots and cord are improved and the extrathecal venous plexus disengorged. This last engorgement may be so extreme before removal of the spur as to suggest venous malformation and to have prevented adequate surgery before (Case 8). The anterior spur is removed to an extent that will ensure this "glide" and reconstitute the fluid spaces throughout the range of normal neck movements.

The patient is postured prone on a table fitted with shoulder and adjustable horse-shoe head rests. Freedom of respiratory excursion is ensured. The head is symmetrically placed forehead down with a fairly full degree of flexion of chin to chest. Local subcutaneous infiltration of novocaine is used and the incision made in the midline from the external occipital protuberance to the vertebra prominens, the lower extremity being adjustable to the site of the spur. The large spine of C.2 is brought into the field to serve as a definite landmark but need not be fully dissected from the muscle. The laminae on both sides of the midline are exposed by means of the usual endothermy cutting technique with coagulation haemostasis.

Care must be taken to avoid tearing or cutting the posterior primary motor division. Light, cerebellar, self-retaining retractors are preferred to bulkier types, which retract mainly at depth. Manoeuvres later in the operation require that the field be not obscured by the bulkier, grid type of retractor blades.

The extent of the lamina removal varies with each case. In general, where root involvement is predominant, a "window" technique can be used. To achieve this the simplest method is the use of a $\frac{3}{8}$ in. burr centred equally on two adjacent laminae, the opening being placed laterally. Exposed ligamentum flavum is dissected away with a knife to reveal dura. Such openings can be placed at the same level on each side to facilitate removal of the spur from each side (Fig. 6). In cases where spurs are at two adjacent levels it is more rational to remove a whole lamina than to make four burr

FIG. 6.—Sketch to show diagrammatically the "window" method of laminectomy approach to a single nerve root, or the more extensive method of exposing two adjacent levels. Either exposure may have to be used on both sides. The upper and lower axillae of the roots are exposed. The cross-hatching indicates the position of the spur on the one side relative to the overlying mixed nerve root and the cord.

holes. This naturally applies to all cases where there is cord involvement and the dura is required to be opened. The upper and lower borders of adjacent laminae are trimmed with rongeurs to expose the upper and lower "axillae" of the respective nerve roots.

The head is moved through as full a range as possible to assess the degree of tightening and fixation of the dura and neural tissues and is then left in the position of maximum laxity.

A fine blunt dissector is introduced into the nerve foramina and the upper and lower nerve axillae to feel for the anterior deformity. This inspection is carried out on both sides so that the "high-spots" may be recognized. Before it is possible with safety to retract the flat, taut dural nerve sleeve, some of the posterior lip of the nerve canal must be trimmed away. The anterior deformity is then approached through the lower axilla, the spur being felt to rise rapidly to its full height quite medially, just within the lateral border of the cord. At this stage the dura must *not* be forcibly retracted over the high spot. The engorged extrathecal veins now begin to bleed and small, stringed "patties" are tucked gently between dura and bone to control them. The posterior common ligament is exposed and, stretched over the spur, it resembles very closely the tense, pale bulge of a prolapsed disc. Even on palpation it may be elastic and springy and this "give" is due to the fact that it is usually hypertrophied. As the ligament is incised crucially, the bony spur is easily seen and felt. From the point of incision the ligament is scraped away from the ridge of the spur for about $\frac{1}{8}$ in. in all directions.

Using a thin $\frac{1}{8}$ in. chisel with a slight but firm downward pressure combined with a rotary motion, a shaft is rapidly made through the exposed portions of the spur (Fig. 7). This ends upon a glistening base-plate, formed by the normal body contour. This must not be perforated. The shaft is enlarged centrifugally and the dural tension is thus freed to allow very much lighter retraction and, as the excavation of the spur by scraping and paring proceeds laterally into the nerve canal and medially beneath the cord, retraction may be dispensed with altogether. *At no time is there excessive medial retraction on the cord.* Instead the little chisel, with its very slightly curved end, is used with a progressively more acute angle to the horizontal plane. Provided the muscle and skin at the sides of the wound can give, the chisel can be used at such an angle that its tip will cross the midline anterior to the cord. With each "scrape" the chisel tip eases aside the posterior common ligament to avoid buttonholing it or nipping the dura. In this way the lower lip of the spur is removed on one side of the

FIG. 7.—Serial diagrams indicating in longitudinal section :—

- The dislocation of the cord and root over the bicuspid spur and the thickened posterior common ligament interposed between them.
 - The retraction of the root at its lower edge, incision of the posterior common ligament, and the shaft sunk through the lower lip of the spur.
 - Upper and lower lip of spur removed, all soft tissues reconstituted in normal positions.
- The posterior body contour and annulus fibrosus is unbroken and engorgement of extrathecal veins relieved.

FIG. 8.—(a) Serial diagrams indicating through a "window" the retraction within the lower and upper nerve root axillae and the cruciate incision through the posterior common ligament and the shaft into the spur (corresponding to Fig. 7(b)), from which excavation is carried centrifugally.

- Cross-hatching indicates the total spur bridge divided diagrammatically into four quadrants each to be removed in turn from the corresponding axillary approach.

midline. The process is repeated from the upper axilla to remove the upper lip. The interbody space is not entered but can be felt as a depression after the spur has gone. The procedure is carried out from the other side at the same level. The dura is opened and after cutting one or more dentate ligaments on one or both sides transthecal inspection is made. Any remaining spicule or roughness is removed, still by the extrathecal route. The dura is not incised anteriorly and therefore transthecal removal cannot be used. In the spurs with two lateral "high-spots" removal to the midline is not necessary, but in the median spur it is essential.

The floor of the spur's cavity is left as the glistening body contour above and below the interspace. The posterior common ligament is virtually intact, but bridging across the cavity. Occasionally hypertrophy of the ligament is so extreme as to continue posterior displacement of the cord. It then has to be trimmed, but if possible it should be kept in continuity.

In the first cases the dura was opened early, before the spur had been dealt with, in order to assess the degree of cord displacement and compression. The cord was found kinked and pushed against the posterior dura, which was often so thinned that the cord surface could be seen through it. The kink coincided with the spur and the posterior surface of the cord showed marked pallor for some distance above and below. In most cases flexion of the neck accentuated the kink and pallor but in one case the effect was from extension.

The sufficiency of the anterior, extrathecal decompression is assessed by the elimination of the kink and the re-establishment of a normal vascular pattern of the cord throughout a full range of neck movement. The engorgement of the extrathecal venous plexus quite suddenly disappears and it is reasonable to assume that this coincides with adequate release from the abnormal tensions. Usually engorgement persists until all levels involved in spur formation are dealt with. It has, however, happened that removal of the major spur has so eased tensions over several segments that the minor spurs were not touched (Case 6). The wisdom of this is doubtful in view of the residual symptoms in this case, referable to the adjacent segment.

Finally a thin layer of "gel-foam" is lightly tucked between the bone and the posterior common ligament to promote haemostasis. It is also placed about the nerve sleeves, where it has not been possible to preserve the normal venous or fatty cushion around the sleeve. The cerebrospinal fluid is replaced with warm Ringer-Locke solution and the dura closed with interrupted black silk sutures on an eyeless, curved, non-cutting needle. The pia-

arachnoid over the cord is not disrupted at any stage.

Haemostasis is meticulous, for scar tissue arising within excess blood clot around the nerve sleeves and dura would render pointless an operation, which is not easy neurosurgically, and replace one form of fixation or compression by another. Muscle and fascial layers are closed in individual layers, no dead space is left, and the wound is made watertight. White, Japanese silk is used for tension sutures and one layer of double black silk in the first muscle layer above the dura acts as a marker, should a later re-opening be required. The skin is closed in two layers. A vaseline dressing is applied, backed with cotton wool and gauze and the whole sealed with elastoplast strips, applied to prevent as far as possible splaying of the scapulae. No plaster of paris splintage is necessary.

The patient is nursed lying on his back on a fracture board for the first 10 days. Catheterization has never been necessary after 48 hours. Leg movements are permitted from the start and aided by mild massage and guided exercises.

Superficial skin sutures are removed on the tenth to twelfth day and the patient then allowed to roll himself from side to side. He sits up in bed on the thirteenth day, hangs his legs over the edge of the bed on the fifteenth day, and thereafter is allowed up for gradually increasing periods. Immediate relief of root pain is the rule post-operatively, but in many instances it returned after a few days, and even was accentuated for about one week. The pain gradually lessens and may re-appear on and off, only as a fleeting, disagreeable sensation, for some months before disappearing completely.

Conclusions and Summary

Characteristic cases from a series of 19, operated upon since April, 1949, are reported. From these cervical osseous spurs, anterior to the nerves and cord from side to side, have been removed. Such spurs have been recognized for some time, but their major importance in the production of neurological syndromes has not been sufficiently stressed.

In my experience prolapsed cervical disc is extremely rare and when present contributes less to the disability than the osseous spur which invariably accompanies it. In all cases reported here the symptoms were attributable to the spur.

Symptoms may be due to root or cord involvement by the spur, separately or in combination. With root involvement pain, with or without neurological signs, is always present. With cord involvement insidious, bizarre pictures with long-tract deficit arise in the trunk and lower limbs. In both types, although complex structures are in-

volved, crisply dissociated pathological effects may persist throughout the course of a progressive illness.

The striking similarity of some cord syndromes to progressive muscular atrophy and subacute combined degeneration of the cord is pointed out.

The term "spur" is preferred to "osteophyte". The spur bridges across anterior to the nervous tissues and should be removed entirely throughout its length. Reorganization of the normal anterior "glide" is the aim of the operation. Posterior laminectomy is considered insufficient.

Surgical treatment is undertaken only after conservative treatment has failed, and where the neurological disability is becoming steadily or rapidly progressive.

Removal of the anterior spur has proved safe with the technique described.

In work which evolves slowly, step by step, to a stage of fulfilment, the individual identity of contributing personalities tends to fade, but not so the value of the help and criticism of each member of our neurosurgical team. In this spirit I should like to thank my colleagues. I thank Mr. R. A. Krynauw especially for his guidance and example, and particularly for his report on Case No. 7, and Dr. Andrew Watt for his help with the manuscript and his criticism. I received encouragement from Sir Hugh Cairns, to whom I had the opportunity to present a series of post-operative cases and to demonstrate the technique of the operation during his visit to South Africa in August, 1951.

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