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Some lesions of the brachial plexus

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Summary

Three types of lesion of the brachial plexus are discussed: entrapment syndrome; tumours; and traumatic lesions. In the first the importance of the pathological anatomy is stressed; in the second the rewarding results of accurate diagnosis and careful treatment are noted; and in the third the expanding possibilities of neural reconstruction and of specific treatment for pain are described.

Introduction

It is a very great honour for me to be asked to speak in this famous city and university, at a meeting of this society, *in piam memoriam* Reginald Watson-Jones, a famous man, in his day one of the glories of British medicine, a world figure in orthopaedic and accident surgery, one of the chief founders of British orthopaedics, and the moving spirit in the development of a famous publication—the British *Journal of Bone and Joint Surgery*. Each one of us is in his debt—either directly through association in clinical, editorial, or other work or indirectly through the influence that he exercised on the practice of orthopaedic surgery. To most men of my age Watson-Jones was at first a name—that of the author of a prodigious work on *Fractures and Joint Injuries,* a practical guide and a work of art and literature that served us well in the days before tape-slide demonstrations and approved courses at approved hospitals under the supervision of men approved by the thought-police of orthopaedic surgery. Much of what was written in *'Fractures'* holds good to this day; much has been superseded by later work. Yet that supersession implies no criticism of the writer—rather the reverse; the good teacher is he who inspires original work and whose pupils grow up to do the work that extends the frontiers of knowledge.

I came into close contact with Watson-Jones when, fifteen years ago, I started work on the *Journal of Bone and Joint Surgery*. I was at once aware of the man's power and enthusiasm and energy—those qualities that had inspired so many colleagues and juniors and that had done so much to create British orthopaedic surgery and the journal that served it. I realized then why I had never heard an ill word about Reginald from anyone who had worked with him and how it was that he had influenced so many younger men to take up orthopaedics. Watson-Jones's enthusiasm, knowledge, and energy brought him to a high position in his profession and in the country, but that high position never made pompous; he was always, for as long as I knew him, the same easy companion, critical often but always appreciative of good work and consistently loyal. The particular quality apparent to all who worked on the journal was Watson-Jones's devotion to and knowledge of the English language. This is a quality specially to be remembered nowadays, even here, at this moment in time, in a conference situation, in the context of a scientific meeting, with meaningful dialogues going on without, and with data and parameters hovering overhead. Let us hope that we can maintain Watson-Jones's standards in a world grown hideous with the proliferation of the appalling catch-phrases of the semiliterate.

The purpose of this lecture is to commemorate the man and his work and to remind ourselves of the standards that he set. The modern tendency for new advances to appear first in the advertisement columns of the journals would not, I think, commend itself to Watson-Jones—however much it appeals to me as a confirmation of my practice of having the advertisement columns of a certain journal bound and casting away the text. I am very glad to see signs of a return to the old practice of doing the work, observing the results, and then writing. It would be impertinent of me, on the strength of a limited acquaintance, to speak at length of Reginald's personal qualities. I must say this: never was there a better, kinder, or more generous host; never was there in the better sense of the word a better party man.

I chose to speak today on a neurological subject even here, in the heart of the citadel of British neurology and neurosurgery, because in connection with these matters I experienced an instance of Watson-Jones's loyalty. I wrote a trifling paper on an aspect of the surgery of the brachial plexus, omitting all but a few references to any work but my own, and was promptly and properly savaged by one of the elder brethren of orthopaedic surgery. Lion-like, Reginald sprang to my defence, well though he knew how much of the criticism was

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just. It was enough for him that he had backed me. I was saved, to speak today.

I have been studying the disorders of the brachial plexus for close on thirty years; almost but not quite immobile, making perhaps an occasional advance. None of the conditions about which I shall speak today is at all common; I have been able to have special experience of these only through the kindness of friends and colleagues who have sent patients to me. I would gladly name those friends and colleagues were they not so numerous; they will know without that how grateful I am to them. Younger men in this audience should know that as the Health Service becomes *gleichgeschaltet* the opportunity for acquiring such experience will be eroded and—eventually—removed. No more Watson-Joneses! No more McKees! Charnley no more! All will have their proper 'catchment areas', all their work-norms not to be exceeded.

Entrapment syndromes

I plan to speak first about the 'entrapment syndrome' of the brachial plexus—the thoracic outlet syndrome, the thoracic inlet syndrome, the costoclavicular syndrome—that most difficult and elusive of entrapment syndromes.

It has for long been recognized that the subclavian artery can be distorted by a seventh cervical rib or by a 'normal' first rib—trapped against either by the snare formed by the tendon of the scalenus anterior muscle[1-3]. Under such circumstances the interior of the vessel is broken and thrombus forms and is propagated. Acutely severe or episodic peripheral circulatory insufficiency may ensue. The signs are there for all to see, clinically, radiologically, and at operation (Fig. 1). Such distortion and interference with function can, it seems to me, affect the lower trunk of the brachial plexus. The reflection is not original; other and better men[4] have made the same comment. Yet some among us stoutly maintain their disbelief in this entity of what we may call the neurological outlet syndrome. I hope today to present evidence that will finally convince these doubters.

There are five points at which the lower trunk of the plexus can be distorted, trapped, or compressed. First, by a hypertrophied suprapleural membrane (Sibson's fascia); occasionally the 'membrane' is muscular and aponeurotic—truly a deep part of the scalenus anterior running behind the subclavian artery and in front of the lower trunk from the seventh cervical transverse process to the first rib. Next, the nerve may be trapped between the first rib and the hypertrophied suprapleural membrane. Thirdly, between the suprapleural membrane and an aponeurotic rib analogue extending from the seventh cervical transverse process to the first rib. Fourthly, the distortion may be caused by a seventh cervical rib. Finally, the lower trunk may be trapped between the first rib and the subclavius muscle (Figs 2 and 3).

Diagnosis The symptoms produced are, of course, those of pain and abnormal sensations in the upper limb, principally in the distribution of the lower trunk. These symptoms are commonly made worse by use of the limb, especially by carrying heavy objects. They

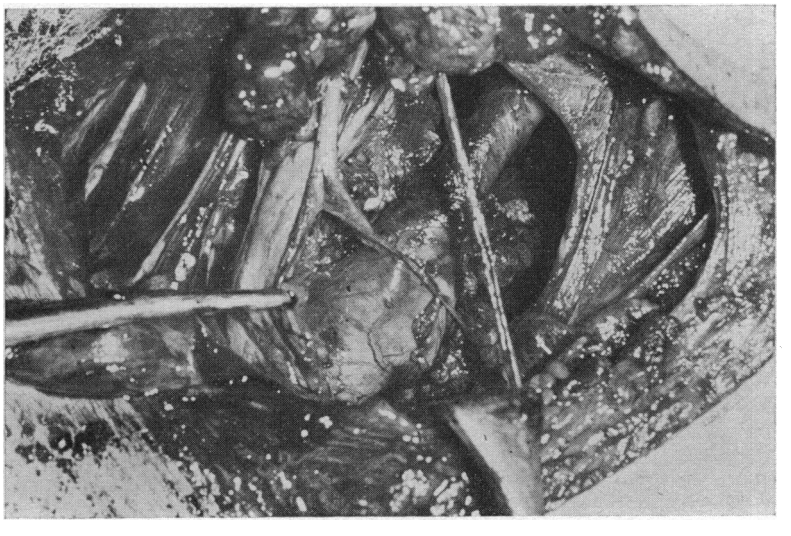

FIG. 1 *Findings at operation in a case of thrombosis of the right subclavian artery caused by distortion by a seventh cervical rib.*

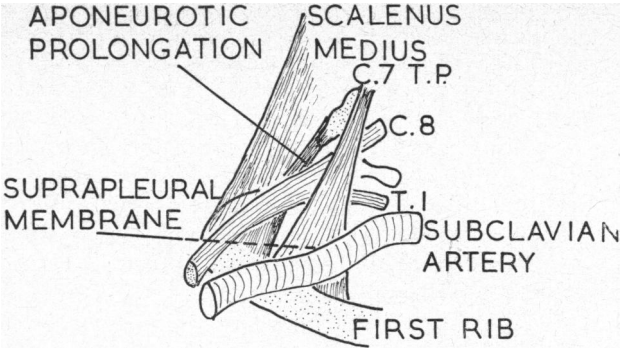

FIG. 2 *Structures at the thoracic outlet, showing some of the sites of compression of the lower trunk of the brachial plexus.*

may be associated with symptoms of circulatory disturbance. They may be associated with objective signs: alteration of sensibility over the medial aspect of the hand and forearm; weakness and wasting of the muscles of the hand and forearm. Only rarely does the lesion affect the vasomotor fibres. There is often tenderness over the plexus above the clavicle; often, too, there is a bruit over the subclavian artery and the radial pulse is easily obliterated by movement of the shoulder girdle. Radiographs may show a seventh cervical rib or an elongated seventh cervical transverse process. Still, the diagnostic process remains uncertain and unreliable in the absence of objective neurological signs. Even when these are present difficulties arise in differential diagnosis from neural affection in the intervertebral canal or from disease of the spinal cord. There is, unfortunately, no further reliable test that can be applied short of operation—namely, exploration at the thoracic outlet. Even that test is plainly unreliable; almost any pain is relieved for a time—and sometimes permanently—by almost any operation under general anaesthesia. Witness the widespread success of 'forage' in osteoarthritis of the hip and—in more antique days—of stretching of the sciatic nerve in cases of sciatica due to prolapse of an intervertebral disc.

It is partly this non-specificity that has led many men to discount entirely the 'thoracic outlet syndrome'. They combine it with the admittedly odd fact that most of these cases occur in women as evidence for dismissing it entirely as a clinical entity. The successes of operation are attributed to a non-specific response—the satisfaction accruing from the shedding of blood—the failures to the fact that the syndrome doesn't exist. Up to recently I

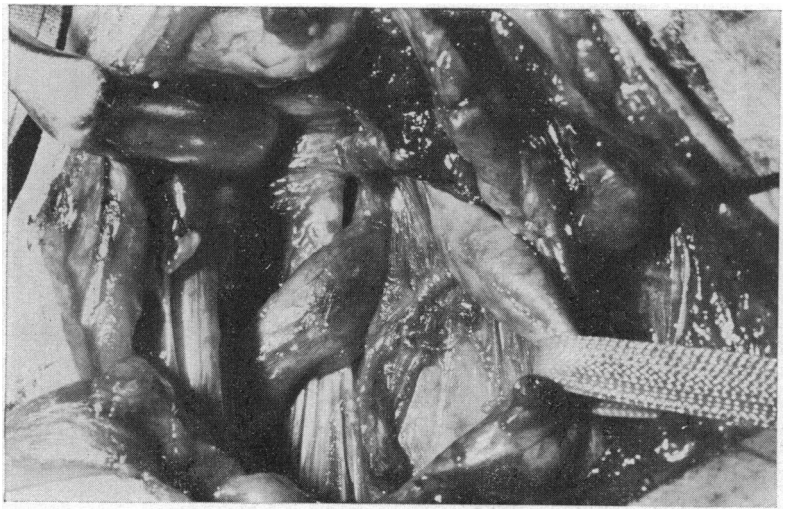

FIG. 3 *Distortion of the lower trunk of the right brachial plexus by aponeurotic prolongation of the seventh cervical transverse process.*

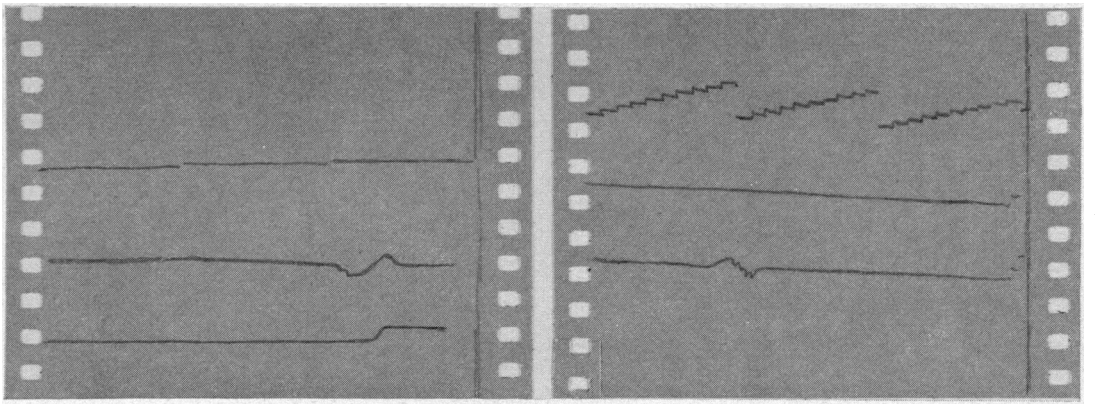

FIG. 4 *Slowing of conduction along the lower trunk of the brachial plexus in a case of distortion by an aponeurotic prolongation of the seventh cervical transverse process. Left: C6 to upper trunk (distance 5 cm, stimulus 0.5 mA, time base 3 ms). Right: C8 to lower trunk (distance 5 cm, stimulus 5 mA, time base 30 ms).*

thought these men were wrong; now I am sure that they are. In a recent case of a woman in her late twenties with pain, paraesthesiae, and objective neurological signs I was able to measure the velocity of conduction across the affected segment of nerve and across a comparable segment of the upper trunk (Fig. 4). The impairment of conduction was associated with very clear external signs of swelling and hypervascularity at the site of distortion of the lower trunk by a cervical rib analogue. I think, then, that the syndrome exists.

Treatment It follows that in cases in which persistent pain and paraesthesiae are associated with evidence of obstruction at the outlet there is a case for exploration. The exploration must be careful and systematic—there is no place here for the cautious grope and the swift slash at the scalenus anterior. All the structures must be seen and the lesion must be displayed and corrected. Under these circumstances diagnosis will be determined and symptoms relieved. It is, unfortunately, less often that motor affection can be reversed, but even that can sometimes be achieved. Great difficulties arise in cases in which previous exploration has left structures scarred, adherent, and even indistinguishable. In such cases supraclavicular exploration is difficult and sometimes dangerous. Roos's[5, 6] brilliant proposal of a transaxillary approach to removal of the first rib has helped greatly in such cases; the method allows adequate visualization and good removal, with much less danger to the neurovascular structures than that involved by the supraclavicular approach. The principal complications of such operations are of course transient deepening of the neurological affection and pneumothorax. When operations are carefully planned and well executed the results are good; they fall off only at the more speculative end of the scale.

Tumours of the brachial plexus

Very rarely in orthopaedic surgery are we given the chance of an intervention as decisive as that provided by peripheral arterial embolectomy. Such a chance arises in the case of osteoid osteoma: intractable pain; long failure of diagnosis; instant and lasting relief after operation. In the case of nerves such a chance is given by the schwannoma, the benign, enucleable tumour of Schwann cells. The symptoms are those of pain and paraesthesiae; rarely is there objective evidence of interference with neural function. Very often the diagnosis goes unrecognized; sometimes failure of recognition goes as far as the psychiatrist's couch. I have twice correctly diagnosed schwannoma of the brachial plexus, on both occasions in the upper trunk; one of these was in a young girl, the other in a man of 57. The symptoms were similar in both cases; in both cases, too, there was localized swelling and tenderness over the upper trunk

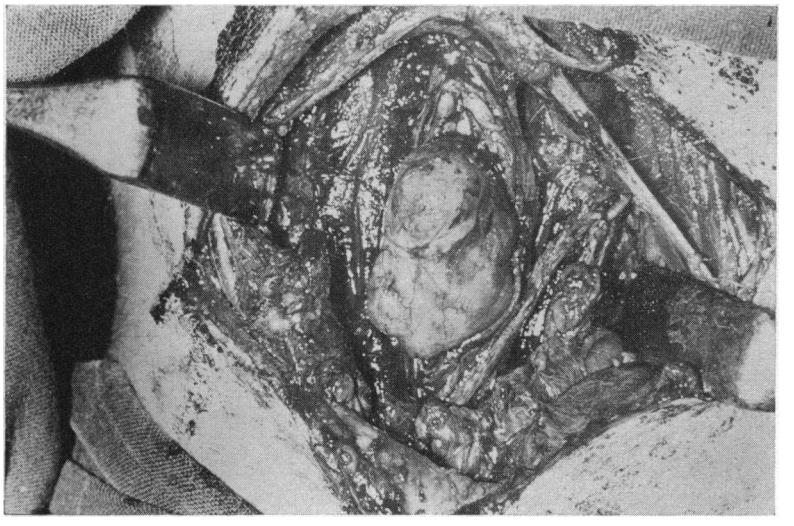

FIG. 5 *Enucleation of schwannoma of upper trunk of the left brachial plexus.*

of the plexus. The appearances at operation were similar and in both cases the tumour was enucleated without interference with neural function (Fig. 5). The relief of symptoms by such an intervention is sharp, complete, and lasting.

Other, larger, tumours of neural connective tissue are encountered. A man of 40 noticed the slow development of a tumour above the left clavicle causing peripheral paraesthesiae and later actual alteration of sensibility and slight defect of motor function. A large tumour was indeed palpable above the clavicle; radiographs showed an erosive lesion of the first rib. It was possible to remove the tumour entire, with the first rib, by a transclavicular approach without inflicting any permanent damage on the plexus or on the subclavian vessels. The tumour was shown to be a neurofibroma, almost certainly benign. Such large tumours in this area pose particular problems of removal by operation because of the proximity of important structures: the subclavian and vertebral vessels, the carotid artery and internal jugular vein, the vagus nerve, the lymphatic duct, the pleura and the apex of the lung, the phrenic nerve, and the brachial plexus. When access principally to axial and medially placed structures is required it is helpful to divide the clavicle and the first costal cartilage and to reflect the divided corner of the manubrium upwards with the sternocleidomastoid muscle. When more lateral access is required it is helpful simply to divide the clavicle or to remove a section of it. Under either circumstance the clavicle can be repaired with a strong medullary wire or, possibly better, with a plate and screws. The incidence of delayed union is, regrettably, high.

Traumatic lesions of the brachial plexus I come now—inevitably—to the matter of traumatic lesions of the brachial plexus and in particular to severe, high traction lesions. Understanding of these lesions has developed over the years; so too have methods of palliative treatment. Regrettably, much remains to be done in the matter of rehabilitation; too often these young men lie idle for years after injury, waiting for determination of a lawsuit, waiting for recovery, or prevented from returning to work by considerations of further treatment or simply by considerations of expediency. Thus although definitive diagnosis is now usually made within weeks of injury, the patient does not always profit from this early determination of the fate of his arm.

Diagnosis Diagnosis—and hence prognosis—depends on the determination of the level and of the severity of the lesion. In the case of intradural rupture the two are clearly interdependent. At this highest level the nerve roots are avulsed from the spinal cord (Fig. 6). The immediate association of this injury is the rupture of the root sleeve and the spill of cerebrospinal fluid. The consequence to the cord is haemorrhage. The formation of a false meningocele follows the spill of fluid; gliosis

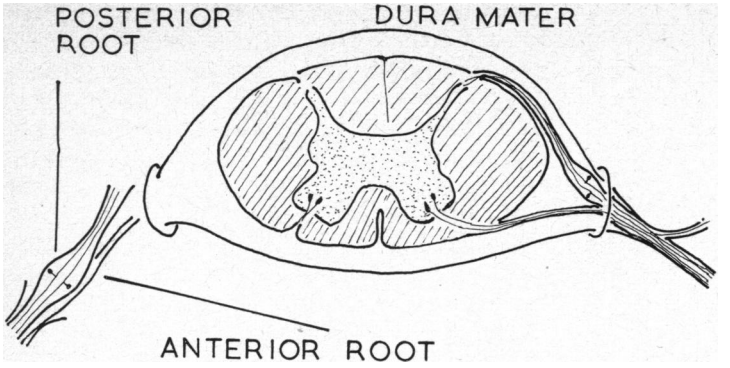

FIG. 6 *Avulsion of roots from the spinal cord.*

follows the haematomyelia. From the former springs our chief diagnostic agent, myelography; from the latter flows one of the most disabling consequences of the lesion, pain. What determines injury within the spinal canal? We know that in the severest cases all five nerves are avulsed from the cord. We know, too, that avulsion is commonest in the case of the lower nerves, principally the eighth cervical and first thoracic nerves. In contrast, the fifth cervical nerve is rarely avulsed; more commonly the damage is lateral to the intervertebral foramen. We know too that the motor root and the sensory rootlets are commonly avulsed together and that only rarely are sensory rootlets avulsed and the motor root left intact.

Does the reason for the vulnerability of the lower nerves lie in the direction of the angle formed at the foramen? Certainly the fifth cervical roots pass rather transversely from the cord to turn down rather sharply at the transverse process. The first thoracic nerve passes rather obliquely down from the cord and then passes rather horizontally from the foramen (Fig. 7). We think that the forequarter is pulled downwards and away from the axial skeleton during injury; that mode of injury could well predispose the lower nerves to avulsion. Unfortunately for this theory, in at least one case avulsion of the sixth and eighth nerves with preservation of the seventh

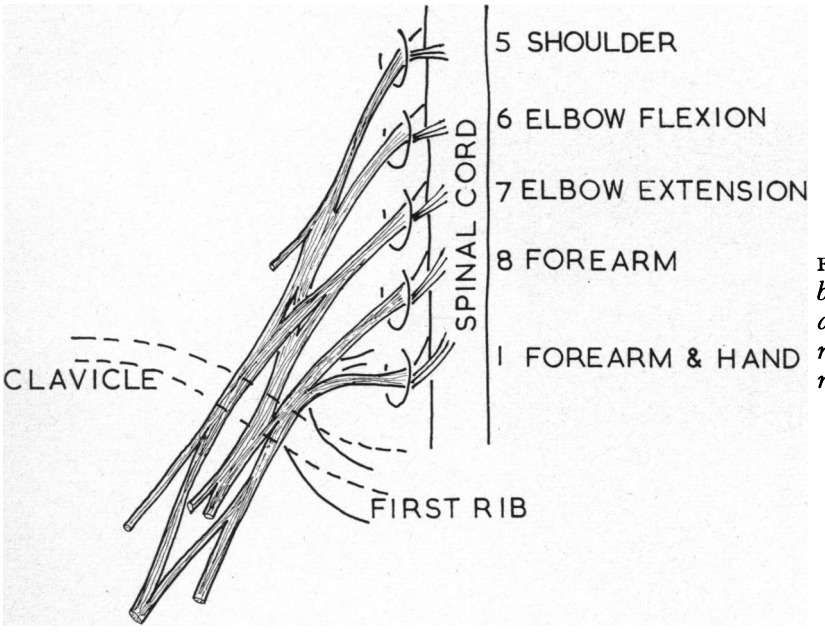

FIG. 7 *Increase of angulation between intradural and extradural parts of the cervical nerves in the lower components of the plexus.*

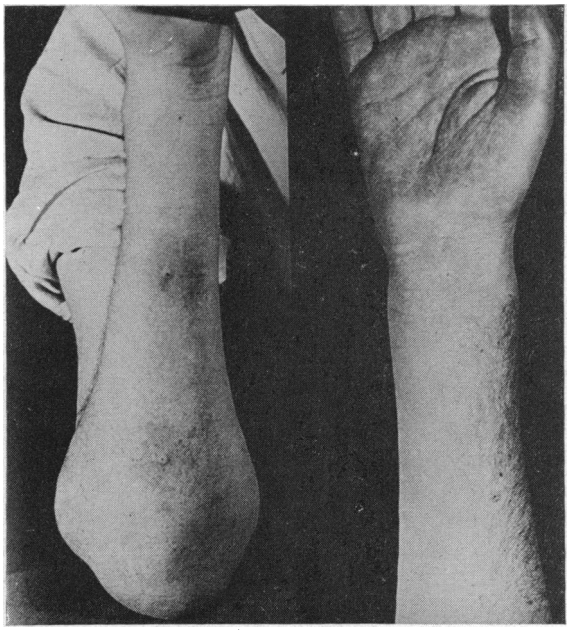

FIG. 8 *Persistence of cutaneous triple response to histamine on the ulnar side of the forearm in a case of avulsion of roots from the spinal cord.*

has been observed. Be that as it may, intradural rupture has up to now meant the certainty that there will be no recovery in the distribution of the nerve so affected; establishment of this diagnosis as early as possible is of great importance.

The nature of the injury provides the keys to diagnosis by indirect means. In the case of avulsion from the cord the preganglionic nature of the neural lesion determines the preservation of the distal axones of the posterior root system. So we can demonstrate persistence of the cutaneous triple response, persistence of the cold vasodilatation response, and preservation of the conducting capacity of the distal axone[7] (Fig. 8).

The meningeal damage determines most of the myelographic signs—meningocele formation, loss of root pouches, and abnormality of the thecal outline. The loss of the rootlets from the spinal canal determines the loss of their shadows[8] (Fig. 9). Important decisions can be made soon after injury by these indirect means of diagnosis. In some cases, indeed, the fate of the plexus is so obviously decided that no more need be done about the neural lesion.

Treatment Is there a place for exploration of the plexus above and behind the clavicle? If so, at what stage should it be done? What are the objects of such exploration? Up to a few years ago the value of exploration was generally seen as being largely, if not wholly, prognostic. The confirmation of avulsion or —at the other end of the scale—of a plainly recoverable lesion formed the main justification for operation. Many men, myself included, had been so much disheartened by the results of repair in cases of traction lesion that they had given it up. More recently the determined and careful work of Narakas[9] in Lausanne has shown that when the improved methods of operation are used worthwhile

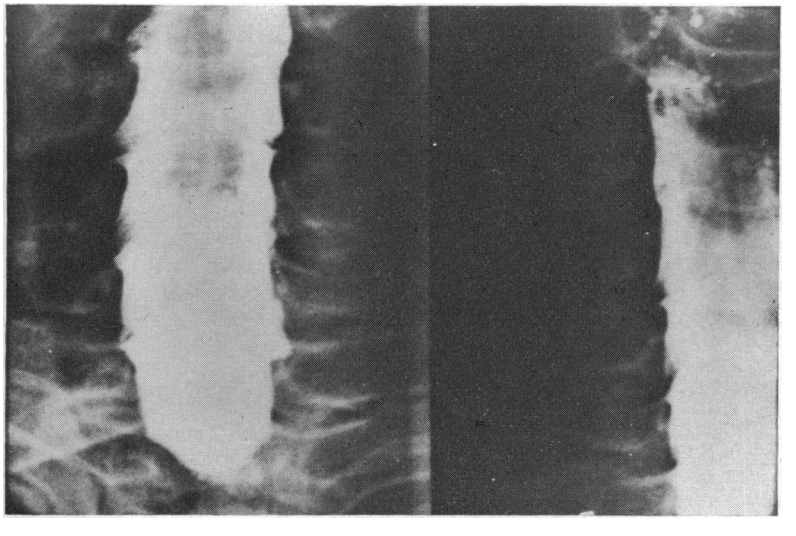

FIG. 9 *Myelograph in a case of avulsion of roots. The root shadows, clearly seen on the healthy side, are absent on the side of injury.*

results can be achieved by repair and by grafting. That such results could be achieved in favourable cases of open wounding has indeed been apparent for some time. It is perhaps regrettable that so often the chance of producing recovery in such cases is let slip. In the matter of traction lesions it is much easier to determine the nature and extent of the lesion when the plexus is seen within a week of injury than when it is seen two months after injury (Fig. 10). Again, the gap to be bridged is much less when the plexus is mobile than when it has been tethered distally by fibrosis. On the other hand delay confers certain advantages—in particular, that of definition of the extent of intraneural damage. In any case delay may be unavoidable; many of these patients have suffered other damage and are seriously ill during the first week after injury.

I think that the plexus should be explored as soon as possible after injury for determination of diagnosis and for possible repair. In most cases of severe injury the lower roots will be found avulsed and the upper nerves damaged distal to the transverse process. The extent of that damage must be measured by inspection and by electrical testing—from nerve to muscle and from nerve to nerve. If it is plain that the lesion will not recover spontaneously the damaged nerve should be resected proximally and distally and the gap bridged by a graft. If lower nerves have been avulsed they or their branches in the arm can be used as grafts. Or mobile avulsed nerves may be connected to the proximal nerve stumps. In cases in which the lower nerves are not available the sural nerve and the medial cutaneous nerve of the forearm can be used as grafts. These operations require a great deal of time and patience. Even the simplest—repair by graft of an open wound of the upper trunk—may take five hours; multiple repair in cases of traction lesion may take more than twelve hours. Haemostasis must be absolute; some magnification is necessary; maybe use of the dissecting microscope at the time of anastomosis would be helpful.

Is it conceivable that reimplantation of avulsed roots will ever be achieved? Could recovery of worthwhile degree follow such reimplantation? Certainly we know that the axones of the posterior root system escape damage and continue to function for years after avulsion—uselessly, of course, because they are detached from central connection. Certainly, too, the regenerating centripetal axones would have to traverse only a short distance. Is the damage to the cord sufficient to prevent proximal reconnection? The formidable difficulties associated with reimplantation may prevent the answer to these questions ever being given.

It has to be admitted that even with early diagnosis and early treatment the results of repair of traction lesions of the plexus show up poorly in comparison with the amount of work and the use of resources. Is it justifiable to

FIG. 10 *Findings at operation within a week of injury to the brachial plexus. The extent of damage is clearly visible: the plexus has been avulsed from the spinal cord.*

do so much for so small a result? I think so; these are young men to whom the restoration of even a moderate degree of function in the damaged upper limb means a great deal.

Pain The question of pain after intradural injury to the brachial plexus has attracted little attention. It has been called 'causalgia' and has in other ways been misunderstood. This is of course no matter of causalgia; the pain arises in the spinal cord as a result of the damage and subsequent gliosis. The pain is not that of causalgia; it is, typically, a chronic pain little affected by external circumstances, rarely of a burning character, never relieved by sympathectomy. It comes on within weeks of injury and persists for years. It responds scarcely at all to drugs, addictive or non-addictive. It may indeed be a problem for the patient as severe as that posed by causalgia.

Unfortunately, treatment is far more difficult. In early attempts at treatment I progressed from the periphery to the centre, discarding on the way neurolysis, sympathectomy, repair of the meningeal lesion, and separation of the cord from intrathecal adhesions. I was brought finally to tract section but was soon aware of the limitations of this method. One is intervening on the healthy side of a cord unilaterally damaged in a person with a good life expectancy and risking interference with respiratory and sexual function, and of course with the function of the corticospinal tract. One is inviting, too, the possibility of later recurrence of symptoms through redevelopment of pain pathways or through recovery of function in pain tracts only lightly damaged by the intervention. Even the admirable development of percutaneous cordotomy by Lipton[10] in Liverpool and by others nearer here has not succeeded in avoiding these risks. Most promising of all is the work that followed the experimental studies of Melzack, Wall, and others[11, 12], and that has led to the application of stimulating electrodes to the posterior columns and to more central areas of the sensory apparatus. I have been privileged to see this development in the hands of Lipton and his neurosurgical colleague Miles[13]. The method is truly physiological, aiming as it does to replace the afferent impulses torn away by injury and so to close the 'gate' whose opening has led to the abnormal transmission of painful impulses. We may consider in this connection a comparison with the mechanism of action of acupuncture and may go on from there to reflect on an application to other painful states. For men whose chief business is the relief of pain these reflections may not be out of place.

Conclusion

I return to the theme of this lecture—the commemoration of a great man. I hope that we and our successors will never forget what we owe to the work and foresight and enthusiasm of such men; I hope that medicine in this country will never fall into such a state—never wobble off into such meaningless disarray—that it will be incapable of producing such men and unable to accommodate them and their work. I wish to say to the younger men, in the words of Hans Sachs: *'Verachtet mir die Meister nicht!'* Do not despise the Masters! They too have known the limitations of their knowledge and have looked forward to the work that is yet to be done.

I wish to thank Mr R J Whitley, formerly Head of the Medical Photographic Department at the Institute of Orthopaedics, for the original photograph reproduced in Figure 8; Dr David Sutton, director of the Department of Radiology at St Mary's Hospital for the myelograph reproduced in Figure 9, and the members of the Photographic and Film Unit of the Royal Society of Medicine for the photographs reproduced in Figures 1, 3, 5, 8, 9, and 10.

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