Mechanisms of cervical nerve root avulsion in injuries of the neck and shoulder

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The author reviews the mechanisms of traumatic spinal nerve root avulsion and proposes a new interpretation.

KEY WORDS • spinal nerve root avulsion • central mechanism

Spinal nerve roots may suffer as the result of traction generated by the violent displacement and deformation of neighboring structures with which the roots are in continuity or to which they are intimately related. If a nerve root is suddenly stretched beyond its elastic limits, structural failure occurs where it is weakest, namely, at the site of its attachment to the spinal cord. Avulsion of cervical nerve roots occurs as a terminal complication of a wide range of severe injuries involving violent and extreme movements of the cervical spine, displacement of the limb on the shoulder girdle, the shoulder girdle on the trunk, or the trunk on a fixed forelimb, all of which put tension on the roots. Fundamentally, any injury in which the normal relationships between the forelimb, shoulder girdle, and trunk are abruptly and violently disturbed jeopardizes the integrity of spinal nerves and nerve roots.

The accepted thesis is that in these injuries tensile stresses are generated extravertebrally in the brachial plexus. These are transmitted centrally to produce their adverse effects on nerve roots which may, if the disrupting forces are sufficiently severe, be avulsed from the cord. The purpose of this paper is to examine some features of this proposition and to suggest that in certain injuries an alternative mechanism could be responsible for the avulsion.

Any consideration of the mechanisms responsible for the avulsion of nerve roots requires an understanding of the anatomical features of spinal nerves and nerve roots which protect them from deforming forces and those which render them more susceptible to injury.

Relevant Anatomical Features

Subarachnoid Space

The fine connective tissue of the pia mater extends along each emerging nerve fiber to invest it with an endoneurial sheath. This connective tissue also forms a loose endoneurial framework for the rootlets and subsequently the nerve roots. The nerve fibers of the roots are arranged in parallel bundles which are loosely held together. The nerve roots lack the epi- and perineurial sheaths of peripheral nerve trunks. Although the endoneurium has strength and elasticity the perineurium and to a lesser degree the epineurium, play the major role in maintaining the integrity of the nerve fibers when a peripheral nerve trunk is stretched.
A comparison of values for strength and elasticity of spinal nerve roots and peripheral nerve trunks reveals that the former fail under tension before the latter; the perineurium is absent in nerve roots but forms a conspicuous component of peripheral nerve trunks. Furthermore, the collagen fibers covering the nerve fibers of nerve roots are fewer and finer than those surrounding the nerve fibers of peripheral nerve trunks.

When a nerve root is stretched, the increase in length before structural failure depends on its initial length (short nerve roots suffer and fail earlier than long roots) and the rate of deformation as well as the magnitude of the deforming force. Nerves tolerate greater degrees of stretch when deformation occurs slowly whereas the elastic limit is rapidly exceeded when the nerve is stretched abruptly and violently. In this respect the very short nerve roots of the brachial plexus are particularly susceptible to traction deformation.

Structurally the weakest point in the nerve root-spinal nerve system is where the nerve fibers are attached to the surface of the spinal cord, and this is the site where structural failure usually occurs when the system is stretched beyond its elastic limits. The attachments of the denticulate ligament are such that traction on the dura is transmitted to the spinal cord and vice versa.

**Intervertebral Foramen**

The intervertebral foramina for the C5-8 and T-1 nerves are short tunnels a few millimetres in length. Although the term intervertebral tunnel would be more appropriate than foramen, the latter term will be retained because of its established usage. Each foramen receives an anterior and a posterior nerve root which fuse immediately beyond the posterior root ganglion to form the spinal nerve. The nerve root-ganglion-spinal nerve junction is located at the outer end of the foramen.

The manner in which the connective tissue coverings of these neural structures are formed and their relationship to the wall of the foramen are of particular interest and significance in relation to the problem of nerve root avulsion. They have been investigated and described elsewhere, and the features relevant to the present discussion are shown in Fig. 1. This is a somewhat schematic reconstruction that is not to scale, but it correctly represents the relationship of structures and tissues in the foramen. Opposite the intervertebral foramen each pair of anterior and posterior nerve roots invaginate the arachnoid and the dura to form a funnel-shaped depression or indentation in the wall of the dural sac. At the bottom or apex of this funnel each root (anterior and posterior) perforates the meninges independently, carrying with it as it does so a tubular dural-arachnoidal sleeve which is separated from the nerve fibers of the root by an extension of the subarachnoid space. Some nerve roots first descend intradurally to a level which may be as much as 8 mm below the center of the foramen which they are to enter. At this level they perforate the arachnoid and dura in the usual way. They then ascend acutely, enclosed in their sleeves, to enter the foramen by passing over its lower margin. In this way the nerve roots are angulated at the site where they pass through the dura. Ascending or angulated nerve roots are common in the lower cervical and upper thoracic regions and have an incidence which is related to age. If the patient is under the age of 25 years there is an incidence of about 40%; between 25 and 40 years this increases to from 71% to 76%. These angulations could be the site of injury when the nerve root is subjected to tension.

At the inner pole of the ganglion the dura becomes adherent to the ganglion, continues over it as a connective tissue sheath and then, beyond it, becomes the tough fibrous perineurium of the spinal nerve. The extension of the subarachnoid space terminates in a cul de sac where the dura and arachnoid...
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become adherent to the ganglion. In the case of the anterior nerve root the subarachnoid extension is obliterated further medially as the meninges close around the nerve to form a definitive sheath for that structure. The distance for which the subarachnoid space extends laterally about the nerve roots is subject to some variations but these variations are not relevant to a discussion of root avulsion. There are, however, two features in particular about these meningeal-nerve root relations which are constant and which relate to this problem: medially, in the intervertebral foramen the nerve roots lie freely within their dural-arachnoid sleeves; and laterally, the dural tissues forming the sheath of the ganglion, and the anterior nerve root applied against its surface, are firmly adherent to these structures.

The connective tissue coverings add to the thickness of the neural contents of the foramen so that the spinal nerve is considerably thicker than the combined cross-sectional areas of its corresponding anterior and posterior nerve roots in the subarachnoid space. The neural structures and their circumscribed connective tissue coverings occupy 35% to 50% of the cross-sectional area of the foramen so that there is free space between these structures and the wall of the foramen. This interval is occupied in part by loosely arranged connective tissue. The neural structures and their sheaths are not adherent to the wall of the foramen except at its entrance where there is some exchange of fibers with the capsule of the intervertebral joints.

Function of Anatomical Features

The normal arrangement in the foramen, in which the neural structures and their meningeal coverings are only loosely attached to the wall of the foramen by fine areolar connective tissue, permits some sliding of the nerve complex inward and outward through the foramen so that the system can adjust appropriately to movements of the vertebral column. When traction is applied to a peripheral nerve some movement of the spinal nerve-nerve root complex outward through the foramen is also possible, and this can affect the nerve roots and spinal cord. Normally this movement does not put undue tension on the nerve roots for there are two structural features that protect the system against traction deformation.

The manner in which the dura ensheaths and attaches to the ganglion and the spinal nerve is such that lateral traction on the nerve is immediately transmitted centrally along the dural sleeves of the nerve roots to the dural funnel and the dural sac. The overall effect is that the cone-shaped dural funnel is pulled laterally into the foramen and plugs it in such a way as to resist further dislocation of the system laterally. Being attached to the dura, the denticulate ligament is also drawn outwards. This, in turn, results in some movement of the spinal cord laterally which reduces the tension that has developed in the nerve roots following displacement of the entire system outwards. This movement of the cord would, however, put tension on the corresponding contralateral nerve roots. Thus the strength and integrity of the system are due, not to any strong attachment of the dura to the intervertebral foramen, but to the continuity of the spinal nerve sheath with the dural sac.

The transverse processes of C4-6 present special features in the form of prominent bony gutters in which the corresponding spinal nerve is lodged and to which it is strongly bound by its fibrous sheath, by reflections of the prevertebral fascia, by slips from the musculo-tendinous attachments to the transverse processes, and by fibrous slips which descend from the transverse process above to blend with the sheath of the spinal nerve below. Nowhere else in the vertebral series are the spinal nerves so confined or so securely attached to neighboring bone. Many authors have referred to the difficulty of avulsing the roots of the brachial plexus by manual traction on the exposed plexus in the cadaver. This special feature of the lower cervical region is clearly related to the mobility of the lower cervical spine and arm. The flexibility and resilience of the cervical spine and the range of movement that it normally enjoys involve stresses and strains which are greatest in its lower section. This and the great range of movement at the shoulder joint and between the shoulder girdle and the trunk provide the basis for a combination of movements that put tension on the brachial plexus. Traction stresses generated in this way are greatest in the C5-6 spinal nerves;
these have the strongest protective fibrous attachment to the transverse process. The spinal nerves at C7-8 and T-1 are not normally subjected to the same stresses, the central effects due to traction declining from above downwards. In the case of the C-7 nerve there is no gutter in the corresponding transverse process and the attachments of the nerve to bone are only moderately strong. The C-8 and T-1 nerves lack any significant attachments to neighboring structures.

**Mechanisms of Nerve Root Avulsion**

Forces stretching and finally avulsing spinal nerve roots are generated in two ways. The peripheral mechanism, which is reasonably well documented, is introduced by traction injuries of the brachial plexus which generate tensile stresses that are transmitted centrally to involve and stretch the nerve roots. The central mechanism has not received attention. It has its origin in external injuries that deform structures so that the forces created fall directly on and stretch the nerve roots between the spinal cord and intervertebral foramen. While it is convenient to examine these two mechanisms separately, it should be noted that in many injuries resulting in nerve root avulsion, the two operate concurrently to contribute to the lesion.

*The Peripheral Mechanism*

When lateral traction on the plexus reaches abnormal levels, abnormal displacements follow that result in nerve lesions. Widespread plexus lesions may occur, the severity and distribution of which are influenced by many variables, including the magnitude, direction, and duration of application of the deforming force. The sequence of changes with deforming forces of increasing severity follows a regular pattern (Fig. 2). First, weakening and tearing of the fibrous attachments binding the spinal nerve to the transverse process occurs. This is facilitated if the transverse processes are fractured. Then, with the failure of the attachment at the transverse process, the entire spinal nerve-nerve root complex is pulled forcibly outward, en masse, through the foramen. There is no resistance to this dis-

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**Fig. 2.** Diagram showing the consequences of lateral traction on the spinal nerve. **Upper Left:** Normal position. **Upper Right:** Lateral traction on the nerve. **Lower Left:** The attachments of the nerve to the vertebral transverse process are severed. **Lower Right:** Avulsion of the nerve's root. For simplification only one root (dorsal) is represented.
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placement until the apex of the dural funnel is pulled into the foramen and wedged there. By this time the nerve roots are under considerable tension. Although traction on the dura is transmitted by way of the denticulate ligament to the spinal cord, which is pulled laterally towards the foramen, the movement is limited and only partly relieves tension on the nerve roots. At this stage rupture of the nerve roots may occur without tearing of the meningeal tissues.

The final stage is reached when the dura tears at the site where it is firmly wedged in the foramen. With rupture of the dura the roots are deprived of their sole remaining and strongest line of defense. Exposed as they now are to the full violence of the deforming forces they are promptly and rapidly stretched to the point where structural failure occurs at their attachment to the spinal cord.

Certain additional features of this pattern of structural failure deserve special comment. The full significance of the attachments binding the spinal nerve to the transverse processes of C5-6, and to a lesser extent of C-7 emerges when the relative susceptibility to avulsion injury of the several nerve roots contributing to the brachial plexus is examined. While traction injuries which do not avulse nerve roots more commonly involve the upper spinal nerves of the plexus, the incidence of avulsion injuries is much higher in case of the lower nerve roots which, anatomically, are at greater risk.

Data reported by Gund and Yeoman are particularly interesting and revealing in this respect. The incidence of avulsion of individual roots in Gund's cases was C-5 (1), C-6 (4), C-7 (11), C-8 (11), and T-1 (5). The relative distribution in Yeoman's series is shown in Table 1.

It has been reported on many occasions that the anterior nerve roots are more susceptible to traction injury than the posterior. This is because the anterior nerve root is thinner than its corresponding posterior root. Moreover, stress-strain studies have shown that anterior roots have a lower tensile strength than the posterior roots. Another factor is that the dural sheath of the posterior root is thicker than that of the anterior root. Finally, the wide separation of the anterior rootlets at the surface of the cord, in contradistinction to the more condensed attachment of the posterior rootlets, weakens the system and favors the failure of some rootlets before others when the nerve is stretched.

Tears in the dura are followed by the formation of traumatic diverticula or meningoceles which extend through and beyond the foramina. These can be demonstrated by myelography. Root avulsion may, however, not be accompanied by changes in the myelogram, so that the claim that a normal myelogram in a plexus injury indicates an extravertebral lesion requires some qualification. While it is possible for a traction injury of the plexus to avulse nerve roots without tearing the meninges, this is unlikely. Consequently the finding that nerve roots can be avulsed without disturbing the normal myelogram should at least suggest that another avulsion mechanism could be responsible which does not involve tearing of the meninges at the foramen.

While it is conceivable that nerve root avulsion from an extravertebral traction injury could occur with no or minimal damage elsewhere to the plexus this is highly unlikely. Injuries that are sufficiently severe to have avulsed nerve roots will have already produced serious damage in the infraganglionic extravertebral parts of the plexus, the supraganglionic root avulsion being the final phase in the disruptive process. Furthermore, in these cases the posterior root ganglion cells will themselves have suffered along with the rest of the plexus. Thus, this mechanism of root avulsion does not give a pure supraganglionic lesion.

This raises for consideration the results of axon reflex testing in these cases. Morison, in a paper on brachial paralysis in infants and children in 1938, first suggested that a positive cutaneous flare response in a denervated area was evidence of a lesion central to the posterior root ganglion. The significance
of this observation passed unnoticed and it remained for Bonney to devise two important and useful tests, based on the preservation of axon reflexes, for determining whether the lesion is located central or peripheral to the posterior root ganglion. These are the histamine flare response and cold vasodilation tests, details of which are provided in Bonney's papers. In both tests the responses depend on axon reflexes which are based on the branching of the peripheral processes of the posterior root ganglion neurons. If the posterior root fibers have been severed distal to the ganglion the isolated peripheral section degenerates and the axon reflex is lost. On the other hand, if the posterior nerve root fibers are ruptured central to the ganglion, the parent neurons and the peripheral processes survive, the axon reflex is preserved, and an appropriate response to testing obtained. Thus the appearance of a flare response in an insensitive limb means that the site of the lesion is central to the ganglion.

The interpretation of the findings in the manner outlined assumes that the sensory neurons of the ganglion survive the injury and the traumatic avulsion of its nerve root. This, however, is an oversimplification of what are usually very complex injuries. Traction injuries of the brachial plexus that are sufficient to have avulsed nerve roots are unlikely to have done so without damaging the ganglia and plexus elsewhere, so that with stresses generated externally and transmitted centrally the distribution of the damage is infraganglionic, ganglionic and supraganglionic. Furthermore, the absence of an axon reflex, while indicative of the degeneration of nerve fibers peripheral to the ganglion does not exclude the possibility of simultaneous avulsion of posterior nerve roots.

Merle d'Aubigné and Deburge found axon reflex testing of uncertain value. Yeoman reported a fairly close correlation between the evidence provided from axon reflex and myelographic studies, although the analysis did reveal some inconsistencies. Despite these observations such tests are of value in the investigation of suspected nerve root avulsion.

The preservation of axon reflexes in the paralysed insensitive limb is evidence of the survival of at least some sensory ganglion neurons and their peripheral processes, and points to a lesion localized at a supraganglionic level. When this finding is examined in relation to the widespread distribution of the lesions caused by extravertebral traction injuries, it becomes difficult to visualize centrally transmitted tensile stresses generated by lateral traction being responsible for the production of a lesion confined solely to the supraganglionic level. This, in turn, suggests that in these cases the "pure" supraganglionic lesion might perhaps be caused by some other mechanism which is operating intradurally. The case for such an alternative mechanism is discussed in the next section.

**The Central Mechanism**

The proposition to be examined is that abnormal movements of the cervical spine could put tension on the nerve roots, lead to overstretching and finally to their structural failure.

The lengths of the nerve roots of the brachial plexus are shown in Table 2. Soulie measured along the upper border of the nerve root and Hovelacque along the lower; in this series measurements were made along both borders. Despite variations, the nerve roots under consideration are very short. This means that the amount of elongation possible with rapid loading before the elastic limit is reached and exceeded is of the order of a few millimeters only. With violent abrupt loading measured in milliseconds it would be very much less.

Medially the attachment of the nerve roots to the spinal cord is weak and they are readily avulsed from its surface by strong traction. Laterally the roots are united to form the spinal nerve which, along with the ganglion and the terminal parts of the nerve roots, is encased in a tough fibrous tissue sheath which is the continuation laterally of the dural sleeves of meningeal tissue. Of the two attachments the lateral is by far the stronger. This can be demonstrated in the dissected specimen by exposing a section of the cord and its nerve roots intradurally and then tugging the cord away from the foramina in the line of the nerve roots. In the case of the "angulated" nerve root there is no direct pull along the roots to the foramen, the line of traction being broken at the angulation where the roots enter the dural pouch before turning upwards to ascend to and enter the foramen.
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### TABLE 2

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* Upper/lower border of root.

An examination of these anatomical features of the cord-nerve root-spinal nerve system reveals that violent displacement of the cord in the spinal canal could stretch the nerve roots with structural failure finally occurring at the weakest point of the complex, namely at the spinal cord. Furthermore, this failure could occur without the development of significant lesions affecting the ganglionic and extravertebral infraganglionic parts of the system. This then becomes a pure or almost pure supraganglionic lesion without any tearing of the dura (Fig. 3). It remains to be shown that traction stresses can be generated primarily in the nerve roots in this way.

Reference has previously been made to the flexibility and mobility of the cervical spine, particularly the lower part. Flexion of the head and neck has been shown to stretch the dura and cord and put tension on the nerve roots.1,11,12,19,23,26,41,52,54,57,58,60 With flexion of the neck and trunk, Smith64 found this stretching greatest in the cervical part of the cord and least in the caudal part with the first three cervical segments being stretched slightly more than those caudal to this level. On the other hand, Reid47 found this effect to be minimal in the case of the nerve roots at C-5 and greatest in the case of the nerve roots at C-8 to T-3 inclusive. With extreme lateral flexion of the spine the contralateral nerve roots are tightened and Roal69 has reported that lateral flexion forces injuring the cervical spine are “often complicated by a brachial plexus lesion as well as a lesion of the spinal cord.” Rotation of the head and cervical spine normally puts tension on the cervical nerve roots on the side opposite to that to which the head is turned.66

The flexibility and resilience of the cervical spine and the considerable freedom of the movement that it normally enjoys involve stresses and strains which are greatest in the lower cervical region. This raises the possibility of extreme movements at the cervical joints rapidly reaching levels in severe injuries to the neck to which the stretched nerve roots could not accommodate, with structural failure occurring at their attachment to the cord. Under these conditions the pathological changes would be maximal central to the ganglia, the latter even escaping because of the fibrous sheath attachments to the gutter of the transverse process.

Head and neck injuries with displacement but no fracture or persisting dislocation of the cervical vertebrae are known to result in cervical spinal cord injury,67,73,74 and it is conceivable that similar injuries could result in the avulsion of cervical nerve roots. Such supraganglionic lesions would be associated with a normal myelogram and preservation of axon reflex responses.

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