Short report

Observations on the innervation of the sternomastoid muscle

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SUMMARY Nine out of fifteen cases having division of the spinal component of the accessory nerve and the upper cervical motor roots as treatment for spasmodic torticollis had residual movements in the sternomastoid of sufficient magnitude to make further surgery necessary before the muscle was effectively paralysed. These observations imply a more complex innervation for the muscle than is to be found in most anatomical texts.

The dual development and origin of the sternomastoid muscle and the possibility of its double innervation has been known for some time.\(^1\)\(^2\) It contains somatic elements which are innervated via the upper cervical ventral roots but mainly it is composed of branchial muscle which is innervated from a separate nucleus that lies longitudinally within the postero-lateral grey matter of the upper four or five cervical segments. The fibres from this nucleus leave the cord in a segmental fashion, join together posterior to the dentate ligament and run upwards as the spinal part of the accessory nerve. They enter the posterior fossa passing posterior to the vertebral artery and then join lower filaments of the vagus nerve with which they travel through the jugular foramen. Outside the skull the spinal component separates from the cranial and passes downwards to the sternomastoid which, in man, it splits and supplies. Residual fibres then pass across the posterior triangle to innervate the trapezius muscle. McKenzie\(^1\) wrote that the sternomastoid muscle has a dual origin for its fibres in all those species in which it is split by the accessory nerve, namely man, pigs, dogs, and chimpanzees. In those in which it is not, for example, the rabbit, then the muscle is of purely branchial origin.

From what has been described it would appear that complete paralysis of the sternomastoid muscle in man should follow division of the spinal component of the accessory nerve as it enters the posterior fossa and the upper cervical ventral nerve roots on the same side. However, the observations that form the basis of this report show that even when this combined procedure has been carried out there remains significant residual sternomastoid function to indicate innervation separate from the spinal accessory and upper cervical motor nerves.

The operations were all carried out in patients receiving surgical treatment for spasmodic torticollis. In this condition there is involuntary rotation of the head, often associated with leaning of the head either towards the side to which it is turning or away. The aetiology of the condition remains unknown but when it occurs on its own it is thought to be a form of localised dystonia.\(^3\) There is no effective medical treatment and as the condition can be responsible for extreme distress and discomfort to the patient, surgery is occasionally necessary. The patients to be described here all had a procedure attributed, not altogether accurately, to McKenzie.\(^4\) In this operation an upper cervical laminectomy is carried out and the posterior rim of the foramen magnum removed. Both spinal accessory nerves are identified and divided where they cross the vertebral arteries. The upper three ventral nerve roots are then divided on both sides and, on the side to which the head inclines during its rotation, the fourth is also divided (this cannot be done on both sides because of the risk of impairing phrenic nerve function bilaterally). A small neural connection between the ventral root of C.1 and the spinal accessory nerve is often found\(^4\) and this is also divided.
Out of a total of 15 patients having this procedure, nine were found to have significant residual sternomastoid function following surgery. In eight this was sufficient to produce continuing head rotation to an extent that made it necessary to perform a further operation in which the accessory nerve was divided again as it entered the sternomastoid muscle.

The operations that were carried out were as follows:— (I have limited the details to the side of the "overactive" sternomastoid muscle. Residual movement in the opposite sternomastoid muscle following bilateral spinal accessory and upper cervical root divisions was often not recorded because it was not the muscle of this side that was contributing to the patient's torticollis.)

(1) Four cases with rotation of the head to the left had division of both spinal accessory nerves in the posterior fossa plus upper cervical motor roots 1, 2, 3 and 4 on the right and 1, 2, & 3 on the left. They remained with residual right sternomastoid function and in three cases the accessory nerve required division in the neck at a later date.

(2) Two patients with rotation of the head to the left had both spinal accessory nerves divided in the posterior fossa and division of upper cervical motor roots 1, 2 and 3 on the right and 1, 2, 3 & 4 on the left. They required subsequent division of the right accessory nerve in the neck because of residual right sternomastoid function.

(3) Three patients with rotation of the head to the right had division of both spinal accessory nerves in the posterior fossa and of upper cervical ventral roots 1, 2, 3 and 4 on the left and 1, 2, & 3 on the right. They required division of the left accessory nerve in the neck because of residual left sternomastoid function.

The division of the accessory nerve as it entered the sternomastoid muscle in the neck was sufficient to abolish all residual movement totally in each case. In one case a section of the accessory nerve in the neck was sent for histological examination. This operation was carried out one week after the division of the nerve in the posterior fossa and histological examination revealed Wallerian degeneration among the fibres.

Discussion

It is not the intention of this report to describe in any detail the results of surgical treatment for spasmodic torticollis, nor is it possible to comment on the innervation of trapezius. This muscle does not provide an important component to the movement disorder of "pure" torticollis and its function following surgery was not sufficiently recorded.

The observations demonstrate that in at least nine out of 15 cases (and possibly more as some of the remaining six patients were left with slight residual head movements that could have been the result of continuing sternomastoid function) significant innervation of the sternomastoid muscle was derived from sources other than the spinal component of the accessory nerve and the upper four cervical ventral nerve roots of that side. Yet the additional innervating fibres must join the accessory nerve after the entry of its spinal component into the posterior fossa, because division of the nerve close to the muscle is capable of abolishing all residual activity.

From where are these other fibres derived? There are two possibilities.

(1) The fibres could come from cervical ventral roots below C4. This implies the presence of a major cervical motor plexus below this level which is capable of supplying effective innervation from the lower cervical region up to the point of entry of the accessory nerve into the sternomastoid muscle, approximately 5 cm below the mastoid process. However, when the nerve is exposed prior to section, no obvious macroscopic evidence of any such neural connections is seen and the anatomical texts give no support for such a structure.

(2) The innervating fibres are derived from those filaments of apparently vagal origin that join the spinal accessory component for their passage through the jugular foramen. Once in the neck, these fibres would then have to continue with the accessory nerve to their destination in the sternomastoid. This latter explanation appears the more attractive although it too lacks an obvious anatomical reason to explain why fibres innervating the sternomastoid muscle should be issuing from the brainstem rather than the cervical cord. Perhaps in seeking an explanation one should remember the role of the sternomastoids as accessory muscles of respiration. The nucleus retroambiagalis is important in the automatic control of respiration and consists of a longitudinal column of respiratory neurons extending from the vagal rootlets to the upper cervical region and lying just ventrolateral to the nucleus ambiguous.5 If there exist developmental connections between this respiratory region of the brainstem and the spinal nucleus of the accessory nerve then it is not difficult to imagine that division of the spinal component alone may reveal a functional innervation from the brainstem.

Another possible explanation is that the division of the spinal accessory and cervical motor roots at the initial operations was carried out so inexpertly that in a high proportion of cases only partial paralysis of the sternomastoid muscle was achieved. This seems most unlikely. The operations are carried out with the magnification and illumination provided by an operating microscope and the anatomy is not difficult to perceive. Also, the observation has been made by oth-
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The late LS Walsh, describing 33 cases in an address to the Indian Neurosurgical Association (1974) said, "This operation does not usually stop the sternomastoid muscle contracting and so, on the side of the neck where the sternomastoid is in spasm, the spinal accessory in the neck is cut as well." He found it necessary to divide the accessory in the neck in 15 of his cases.

Probably in only one of McKenzie's cases (the third out of a total of 12) was a bilateral intradural section of the accessory nerves carried out with a bilateral section of the upper cervical ventral roots. He wrote of this case "slight function remains in the sternomastoid probably because the nerve was not cut high enough. (intradural section of the spinal accessory nerve should be below its medullary branches to insure against interference with the pharynx and vocal cords.)"

It should be remembered that none of the patients in these series was "normal". All were, by definition, suffering from spasmodic torticollis and it remains a matter of speculation as to whether residual sternomastoid function would be observed in patients having an identical operation but who were not suffering from the same underlying involuntary movement disorder. To say that they could react differently (that is, have immediate abolition of sternomastoid function), would be to imply that patients with spasmodic torticollis have an anomalous innervation of one of the muscles responsible for rotation of the head. In my own experience the muscles most commonly involved in "pure" rotational torticollis are one sternomastoid muscle and the contralateral segmentally innervated cervical muscles. Although both muscle components of the abnormal movement may derive much of their cerebral supranuclear control from the same side of the brain (ipsilateral to the sternomastoid muscle and contralateral to the cervical muscles) there is as yet no evidence of any anomalous innervation (over an area sufficiently wide to include muscles of such different developmental origins and on both sides of the neck) that could provide a clue as to the aetiology of this mysterious and distressing condition.

References

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