Letters

Neck-abdominal reflex

Sir: Diagnosis of cerebral death depends upon the absence not only of cortical functions but also of brainstem functions. In this sense, demonstration of any kind of reflex being mediated by the brainstem becomes important. Spinal reflexes such as deep tendon reflexes, flexor plantar responses and flexor withdrawal reflexes can be observed in deeply comatose patients who have no clinical evidence of either cortical or brainstem functions. Ivan in 1973 reported a relatively common occurrence of the neck-abdominal reflex in patients with cerebral death, but this reflex has not received much attention. This communication reports a reflex contraction of abdominal muscles elicited by passive neck flexion in three comatose patients with poor outcome.

Case 1 was a 69-year-old woman with anoxic coma due to cardiac arrest. Case 2 was a 28-year-old woman, who was in the 8th month of a pregnancy complicated by toxaeemia, suddenly complained of severe dizziness, headache, nausea and vomiting, and rapidly became unresponsive probably due to intracranial haemorrhage. Case 3 was a 53-year-old woman with deep coma due to cerebel haemorrhage with ventricular rupture. All three patients were deeply comatose, and there was no motor response to painful stimulation in the first two cases, and Case 3 showed decerebrate posturing. Pupils were normal in diameter, but non-reactive to bright light or to pinching the neck. Oculocephalic, corneal, ciliary and cough reflexes were all absent. Spontaneous respiration was absent in Cases 1 and 2, but present in Case 3. In Case 1, the extremities were flaccid, and deep tendon reflexes and plantar reflexes were absent. Electroencephalogram (EEG) showed relatively rhythmic activities of low amplitude at 8 to 9 Hz during which was not altered by any kind of stimulation. In Cases 2 and 3, deep tendon reflexes were hyperactive in all extremities with extensor plantar responses bilaterally. EEG was nearly flat in Case 2.

In all three cases, passive flexion of the neck in a supine position was noted to elicit consistently a brisk contraction of all abdominal muscles. The intensity of the muscle contraction was not related to the speed or amplitude of the neck movement, although slow flexion of the neck did not produce any abdominal muscle contraction. The latency of the muscle contraction after the beginning of neck flexion was 35 to 90 ms (fig). Each muscle contraction lasted 90 to 240 ms. In Cases 1 and 3, passive neck extension or rotation did not produce any response. In Case 1, lateral flexion of the neck to the left produced contraction only in the right abdominal muscles, but the flexion to the right did not produce any muscle contraction. In Case 2, passive neck flexion also elicited bilateral foot plantar flexion and finger extension, which occurred synchronously with the abdominal muscle contraction. Muscle contractions both in abdominal and limb muscles were also produced by lateral flexion or rotation of the neck to either side. Lateral flexion or rotation of the neck to the left produced stronger muscle contractions in the right finger extensors than in the left, whereas lateral flexion or rotation to the right produced more contraction on the left. Passive movement of any limb or painful stimulation to any part of the body did not elicit any muscle contraction. In all cases, the abdominal reflexes were absent. The reflex abdominal muscle contraction was observed only at a certain stage of the clinical course in all cases, and disappeared within a few days. In spite of intensive conservative therapy, the first two cases succumbed and Case 3 remained in a vegetative state.

Since these contractions of abdominal muscles did not occur spontaneously, but were consistently and exclusively elicited by passive neck movement, this phenomenon is considered to be generated by a reflex mechanism. The afferent input of the reflex pathway may be mediated by spinothalamocortical fibres from cervical spinal and joint afferents from the cervical spine, and this reflex centre is most likely to be located at or above the upper cervical cord. The efferent pathway might be a long descending spinal tract such as reticulospinal tract, although the responsible pathway cannot be identified precisely.

Considering the underlying mechanism of this reflex, the tonic neck reflex has to be taken into account. In asymmetric tonic neck reflexes, the upper extremity contralateral to the side of neck rotation is flexed rather than extended. In symmetric tonic neck reflexes, neck flexion is usually associated with flexion of the upper extremities. In Case 1, however, rotation or lateral flexion of the neck elicited a reflex muscle contraction in contralateral finger extensors. Moreover, neck flexion elicited finger extension rather than flexion. With regard to the duration of each muscle contraction, as judged by the surface-recorded EMG discharge, the contractions both in abdominal and extremity muscles were always phasic, lasting only from 90 to 240 ms. These facts seem to differentiate this reflex from the tonic neck reflex, even though there are some similarities between the two.

Jorgensen described unilateral extension-pronation movements of the upper extremity elicited from the spinal cord. In all the thoracic sensory dermatomes in patients with brain death, this reflex, however, is different from the present reflex because the former is not elicited by passive changes in the position of the head or limbs. Ivan found neck-abdominal reflex in response to quick neck bending in 75 percent of patients with cerebral death and proposed a tonic neck reflex as its mechanism. In his paper, however, details of the reflex were not described. The reflex reported here is probably the same as the

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**Fig.** Surface EMG polygraph in Case 1. Passive neck flexion is followed by bilateral contraction of abdominal muscles more on the right at an approximate latency of 35 ms. EKG artifacts are seen in all tracings.
neck-abdominal reflex, and seems to indicate severe brainstem damage, and hence a poor prognosis.

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Spinal neck-tongue syndrome

SIR: The neck-tongue syndrome was first described by Lance and Anthony.1 Their patients experienced pain in the neck with or without numbness and numbness in the tongue on sudden turning of the head. The authors attributed the symptoms to compression of the C2 roots in the atlanto-axial space; the numbness of the tongue was caused by compression of proprioceptive fibres from the tongue through the ansa hypoglossi, the cervical plexus and finally the C2 roots. We wish to report a patient who developed unilateral numbness in the neck and tongue of spinal origin.

A 35-year-old woman developed symptoms of acute transverse myelopathy in the upper thoracic spinal cord at the age of 23 years. After recovery, she had some similar exacerbations and remissions during the following ten years. Myelography at the age of 28 years was normal. At the age of 33, she became unable to walk owing to paraplegia and was confined to a wheelchair. At the age of 35, weakness of the left upper limb developed acutely. Neu rologically examination on admission revealed positive Lhermitte sign and paraesthesia on the left between C2 and C3 segment. Marked weakness with hyporeflexia was noted in the left upper limb. Joint position sense of the left upper limb was severely impaired. There were motor and sensory deficits due to transverse cervical myelopathy which persisted since the age of 33 years. Laboratory examinations were normal except for a mild pleocytosis in the spinal fluid. An EEG and a CT scan were negative.

Tonic seizures appeared after the signs and symptoms of the neck and left upper limb had subsided considerably. Voluntary movement of the left arm provoked tingling sensation in the left hand, which radiated to the shoulder. Simultaneously the spasm appeared in the left upper limb, with fingers flexed, wrist and elbow flexed and the arm abducted. The tonic spasm lasted about 60 seconds. About 20 seconds after beginning, the neck and occipital region on the left side became numb, accompanied by simultaneous numbness of the left half of the tongue. Other parts supplied by trigeminal nerves were not involved. The numbness lasted from 30 to 60 seconds. The seizure occurred in a stereotyped fashion about once an hour. Carbamazepine, 400 mg daily, suppressed the attacks.

This patient developed unilateral numbness in the neck and tongue during the tonic seizures following the upper cervical lesion. The neurologic signs on admission suggested involvement of the left posterior funiculus and left corticospinal tract. The cause of the myelopathy was not clear. The exacerbating-remitting course may indicate demyelination. Tonic seizures are one of paroxysmal neurologic disturbances in multiple sclerosis and also have been reported in traumatic injury of the spinal cord.2 Although their pathophysiology is unknown, it seems probable that in some cases such tonic seizures have a spinal origin.3,4 Levine and Anthony1 reviewed the available data on the course of afferent fibres from the tongue and concluded that they pass via the ansa hypoglossi to the C2 dorsal roots. The pathway of the afferents from the tongue after entering the central nervous system was little discussed. Bowman and Combs5 showed that the hypoglossal afferents in the rhesus monkey project rostrally in the ipsilateral dorsal funiculus via the dorsal root ganglia of the C2 and/or C3 nerve. It seems reasonable to postulate that a similar situation obtains in humans. The numbness in the neck and tongue may be explained by involvement of the dorsal funiculus during the tonic seizures. However, an alternative explanation may be possible. Sensory fibres of the mandibular branch are distributed to the tongue and descend in the brainstem as the spinal trigeminal tract. They terminate at medullary or midbrain nuclei and thus do not interfere with the spinal segment. Disturbance of this tract may cause the numbness in the tongue.

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HLA antigens and multiple sclerosis in Greeks

SIR: While the relationship between the major histocompatibility system (HLA) and susceptibility to multiple sclerosis is well documented in northern European populations, there has been controversy over the relationship for ethnic groups of Mediterranean origin. Most workers have noted an increased frequency of HLA-A, and B, antigens in multiple sclerosis