Respiratory Hazards of High Cervical Percutaneous Cordotomy*

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A FAMILIARITY with Belmusto’s and Nathan’s clinical articles on the respiratory hazards of high cervical cordotomy and with Pitts’ classical work on the physiology of respiration in the cat will be found useful by those who undertake percutaneous high cervical cordotomy.\(^2\)\(^6\)\(^7\) We will not attempt to summarize these valuable contributions, but will draw attention to some of the known facts of the respiratory centers and pathways.

In the cat, the respiratory center lies in the medially situated reticular formation of the medulla (Figs. 1 A and B). It does not include the dorsally- or laterally-situated cranial nerve nuclei, or the ventrally-situated olivary nucleus. In its longitudinal extent it corresponds to the cranial four-fifths of the olive. It extends from just below the striae acusticae superiorly to just below the obex inferiorly. Within this area there are two types of response to electrical stimulations, which serve to divide it into inspiratory and expiratory centers. Stimulation of the inspiratory center produces tonic inspiration, and stimulation of the expiratory center produces tonic expiration, or at least tonic cessation of inspiration, in the expiratory position. The inspiratory center lies immediately dorsal to the olive, the expiratory center lies dorsal to the inspiratory center and caps it rostrally. It does not extend quite so far caudally. At and below the lower pole of the olive, electrical stimulation produces weaker responses presumed to arise from the efferent pathways, rather than from the center itself. The area concerned with inspiratory responses lies lateral to that for expiratory responses. At the level of the first cervical vertebra, degeneration produced by lesions in the respiratory centers is found in the anterior column and in the anterior part of the lateral column (Fig. 1 C). Henderson and Craigie\(^3\) in cats (Fig. 1 D) located the maximum concentration in the same region, and Nathan\(^4\) (Fig. 1 E), reached similar conclusions from a study of cordotomy in man. Belmusto, et al.,\(^5\) (Fig. 1 F) also located this area in man in the anterior quadrant of the cord, suggesting that it lay 3 mm from the lateral surface of the anterior quadrant and was 2\(\frac{1}{2}\) mm in width. Longitudinally it extended at least from C-1 to C-4, possibly to C-7.

It is well known that the cat brain may be sectioned longitudinally from the striae acusticae to the upper cervical region without respiratory difficulty. Hemi-section of the cervical cord, however, causes immediate cessation of respiration on that side, but this begins to improve within a few days, and within 3 weeks is normal again, showing that crossover takes place in the cord. There is another interesting phenomenon in some species including cats called the “crossed phrenic phenomenon.” Hemi-section of the cord, as stated, produces paralysis of the ipsilateral diaphragm. Section of the contralateral phrenic nerve causes an immediate paralysis of the diaphragm on that side, but also causes an immediate restoration of power to the diaphragm on the side of the hemi-section. Unfortunately, as Nathan has pointed out, a previously paralyzed nerve in the human does not prevent development of paralysis of the contralateral diaphragm when an extensive contralateral cordotomy is performed. One further fact of interest is that section of the posterior nerve roots through which the diaphragm establishes afferent connection with the cord will, in cats, cause paralysis of respiration on that side. This will recover within a few weeks. It thus appears that sensory deprivation might play some role in the clinical respiratory paralysis we encounter following cordotomy. (This very brief account does not include any description of the higher control of respiration in the cortex, hypothalamus, and cerebellum, of the pneumotaxic and apneic centers of the

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extensive effusion, inspiratory. A, B, pons, or of the role of the Hering-Breuer reflex and the tractus solitarius.)

It is obvious from the description above that bilateral extensive high cervical cordotomy lesions impose a serious hazard to respiratory function. More puzzling is the fact that death may occur following an extensive unilateral lesion. Here the problem is almost certainly one of pulmonary efficiency. A unilaterally intact respiratory mechanism will maintain life if the lungs are basically normal but not when there is some severe pulmonary impairment whether that be due to chronic degenerative lung disease or acute pneumonia, to an extensive tumor infiltration or an extensive surgical resection, to cardiac failure, pleural effusion, a paralyzed diaphragm, or to any combination of these.

In a series of more than 400 cordotomies, we have had nine patients in whom death was caused or accelerated by the procedure. We have had several who suffered postoperative respiratory difficulty, but survived. One of these came close to death and he too will be discussed.

**Bilateral Cases**

**Case 1.** This patient was described briefly in a previous publication. She had had a previous hypophysectomy for terminal breast carcinoma, and had died 2 hours after a second direct-current cordotomy. As the daily cortisone had been omitted on that day, we attributed her death to blood pressure failure, but in view of our subsequent experiences, it might very well be that she died of respiratory failure.

**Case 2.** This 45-year-old man with Hodgkin's disease died a respiratory death 9 months after his second strontium cordotomy. Up until then he had been ambulatory and pain-free. At the time of autopsy, it was remarked that, although there was extensive mediastinal lymphadenopathy, it did not seem to be enough to cause a respiratory death. There was extensive bilateral anterior quadrant necrosis of the cervical cord.

**Case 3.** This 63-year-old cachectic paraplegic man with extensive chordoma of the sacrum, had bilateral direct-current cordotomies separated by an interval of 4 days. A good sensory level was obtained on the left side, but faded within 2 days. On the right, despite three different electrode insertions, only hypalgesia could be obtained. Three and 8 days later strontium cordotomies were performed on the left and right side respectively. He was then virtually free of pain, but did not develop any further sensory loss. This disassociation of perception of pain and pinprick following strontium cordotomy has been noted previously. Because of some return of pain in his left leg, a radiofrequency current lesion was made 3 weeks later. The electrode was introduced 3 mm anterior to the dentate ligament. The patient experienced an immediate respiratory difficulty, a drop of blood pressure, and bradycardia, but soon recovered; he was left with right motor weakness and a sensory loss below the clavicle on the left. Thirty hours later he died in his sleep.

**Case 4.** This 56-year-old paraplegic man (trauma T-12) complained of pain in the...