Pulmonary Function Following Percutaneous Cervical Cordotomy

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This is a report of respiratory changes following percutaneous cervical (C2) cordotomy for intractable pain, consisting of: (1) a retrospective review of 13/200 patients with respiratory complications; and (2) a prospective study of pulmonary function in 41 patients. (1) Three of 13 patients had sleep-induced apnea following bilateral cordotomy and required artificial ventilation for 1 to 3 months. (2) Following unilateral cordotomy, mean FVC, MEFR and \( \text{Pao}_2 \) in all 41 patients were not lowered. Seven of 41 patients developed dyspnea post-cordotomy; two needed temporary artificial ventilation; but 6/7 had greatly reduced pulmonary function pre-cordotomy. Two developed phrenic palsy without dyspnea. Following cordotomy on the second side (17 patients) there was a significant reduction in MEFR, FVC and \( \text{Pao}_2 \) values, greater in the 7/17 who became dyspneic. Three developed phrenic palsy with dyspnea.

Percutaneous cervical cordotomy was introduced by Mullan ¹ and modified by Rosomoff ² to provide simple, effective treatment of intractable pain. Destruction by radiofrequency of the lateral spinothalamic tract in the ventral quadrant of the spinal cord (fig 1) is carried out in the awake, cooperating patient. A properly-placed lesion results in contralateral analgesia to the desired level.³ Should the more dorsal corticospinal tract be dammed, ipsilateral paresis can occur, and respiratory and circulatory insufficiency can be produced through inadvertent injury to neural connections related to these systems. Respiratory insufficiency has been reported following open surgical cordotomy,⁴ but the effects of percutaneous techniques have not been described.

As a result of complications consequent to percutaneous cordotomy at this hospital, a prospective study of pulmonary function in another group of patients undergoing cordotomy was conducted. The two groups form the subject matter of this report.

Retrospective Review

A review of cases antedating January 1966 disclosed that 13 patients among the first 200 (total of 315 cordotomies) had some respiratory distress (table 1). Three patients in
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<th>Patient</th>
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whom apnea followed cordotomy are described (Patients I/1–3). In the remaining 10 cases (Patients I/4–13, table 1) the patient's terminal state or pre-cordotomy pulmonary pathology accounted for the respiratory dysfunction; however, cordotomy was considered a possible contributing factor.

**CASE REPORTS**

**Patient I/1 (E. K., 8/65)**

A 59-year-old bedridden woman with a 20-year history of tuberculosis had severe chronic pain in both upper and lower extremities secondary to osseous involvement. Bilateral percutaneous cordotomy was done, but levels of analgesia were not sustained. Three weeks later the right cordotomy was repeated with analgesia to C4. Six hours after cordotomy she complained of shortness of breath, and ipsilateral hemiparesis developed. Shortly thereafter she was found apneic and hypotensive. She was ventilated artificially via tracheal tube for 12 hours, but repeated attempts at discontinuing artificial ventilation were followed by recurrence of respiratory insufficiency. A tracheostomy was performed. Five days later, movement and good strength returned on the right side. She developed pneumonitis, treated successfully, but for two
weeks she required the ventilator. After adequate spontaneous breathing for a week (vital capacity, 1 liter), the tracheostomy was allowed to close. A month after the third cordotomy, she again complained of dyspnea; this was thought to be due to hysteria, but she was found dead in bed the next day.

**Necropsy.** The surgical lesion in the spinal cord included both ventral quadrants, as planned, plus an extension into the right dorsal quadrant. The latter accounted for the transient hemiparesis; the large bilateral ventral necrosis probably was the basis for the respiratory insufficiency (fig. 2).

**Comment.** This was our first recognized case of postcordotomy apnea, probably induced by sleep. Pulmonary status had not been studied in detail, and fluoroscopic examination of the diaphragm was not performed.

**Patient 1/2 (G. T., 5/65)**

A 40-year-old woman had had chronic pancreatitis with severe right-sided abdominal pain for ten years. She was a hysterical, and was addicted to narcotics. A left cordotomy produced only temporary analgesia. Repeated a week later, it resulted in a permanent C4 level of analgesia. Three weeks later, because of pain on the opposite side, a right cordotomy was done with a resultant T2 level. During the procedure, she was apprehensive and dyspnea. On the following day she became confused and stuporous, with shallow breathing. After she fell asleep, she was found cyanotic, apneic and hypotensive. Tracheal intubation and artificial ventilation restored consciousness promptly. After improvement to a vital capacity of 1 liter, the trachea was extubated, but she again had a respiratory arrest when she fell asleep. After resuscitation, consciousness was restored and spontaneous breathing was maintained for two hours. Subsequently, she had eight more episodes of respiratory arrest, always after falling asleep. Finally a tracheostomy was done. Vasopressors were needed intermittently for hypotension. She came to recognize the consequences of sleep and would stay awake until fatigue induced somnolence and resultant hypventilation. Fourteen days after the second cordotomy, she improved sufficiently to be weaned from artificial ventilation, with a vital capacity of over 1 liter. A month later, she fell at home, fracturing her cervical spine, and died. Necropsy was not done.

**Comment.** This was a case of "sleep-induced apnea" following bilateral cordotomy with survival after two weeks of artificial ventilation.

**Patient 1/3 (D. R., 65/66)**

A 40-year-old woman had Ehlers-Danlos syndrome and joint pain. She had a history of asthma and multiple allergies. Bilateral cordotomies were done a week apart to C3 levels bilaterally with no complications after the first, but after the second cordotomy she had fatigue and shortness of breath.

In anticipation of respiratory arrest during sleep, she was stimulated to stay awake with amphetamine and methylphenidate (Ritalin). On the second day, she had marked respiratory distress, probably from fatigue. She was ventilated with bag and mask and transferred to the intensive care unit. Here the trachea was intubated, and she was found hypotensive (pulse rate 40 per minute; body temperature 33° C.). Atropine restored the pulse to normal. On same day her vital capacity was approximately 1 liter and the trachea was extubated.

On the fifth day, she again developed bradycardia and vital capacity decreased. Diaphragmatic paralysis was confirmed by cinefluorography. She was reintubated, subsequently tracheotomized and controlled ventilation was resumed. With assisted respirations, she became apneic whenever not stimulated or upon falling asleep. On the eleventh day she had three episodes of loss of pulse. A considerable alveolo-arterial $P_O_2$ gradient was explained by pneumonitis. Doxapram intravenously, during periods of hypoventilation, resulted in only brief recovery of tidal volumes of 500 ml. On day 32, after both hemidiaphragms had recovered partially, the tracheostomy was permitted to close. The following night she had another episode of apnea and pulselessness, was reintubated, resuscitated and again transferred to the intensive care unit. She panicked whenever taken off the ventilator.

Eighty days after cordotomy, she had an episode of asphyxia while on a chest respirator. Laryngoscopy revealed weak pharyngeal and laryngeal reflexes and asynergism of laryngeal and
pharyngeal muscles; the vocal cords were relaxed and easily drawn into the trachea during forced inhalation. Paradoxical motion of the vocal cords was not found. Attempts to stimulate breathing with deliberate hypercarbia and diffusion oxygenation failed to increase minute volume, suggesting an impaired response to CO₂. Doxapram increased tidal exchange, but this was not sustained. Eventually she made an uneventful recovery.

**Comment.** Respiratory insufficiency following bilateral cordotomy for non-malignant disease, ag-

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Mean values | 72 | 68 | 69 | 67 | 76 | 76 |
Percutaneous Cervical Cordotomy

Gravitated by sleep, lack of stimuli and the patient's emotional makeup, necessitated four months of artificial ventilation. The minute volume response to increased arterial CO₂ was decreased. In spite of recovery of diaphragmatic function, reflex neural and chemical control of breathing remained impaired.

Patients 1/4-13

Ten patients (table 1) had cordotomy for intractable pain secondary to terminal cancer with metastases. Most required narcotics. The analgesic levels obtained were between C3 and C5. All became hypotensive and dyspneic, and four died, within the first two postoperative days. Six required artificial ventilation. All died within eight days. Hemiparesis and unilateral phrenic paralysis were found in several cases and Horner's syndrome in most.

Necropsy of two patients disclosed extensive necrosis and edema in the cervical spinal cord (fig. 2, 1/4-5).

Respiratory Function Study

Between January and June 1969, 170 patients underwent percutaneous cordotomy. In 41 patients, respiratory function studies were performed pre- and post-cordotomy; the 41 patients were selected as those who came to cordotomy on a specific day of the week. They included 24 in whom unilateral cervical cordotomy was performed and 17 with bilateral cordotomy. Measurements were made the day before and approximately 24 hours after cordotomy. Bilateral cordotomies were performed at an interval of a week. When indicated, measurements were repeated at 48 and 72 hours.

All determinations were made with the patient in the semi-sitting position. Measurements included arterial P0₂, PCO₂ and pH, while breathing air (Instrumentation Laboratories Trielectrode Unit Model 127). Arterial samples were drawn anaerobically from the brachial artery, placed immediately into ice and analyzed in duplicate within 30 minutes. Inspiratory capacity was measured with a Wright ventilation meter, using a tight-fitting face mask. Maximal expiratory flow rate (MEFR) was measured with a Wright peak flowmeter and expressed as a percentage of the predicted value. Single-breath total forced vital capacity (FVC) (percentage of predicted) and 1-second forced expiratory volume (FEV 1.0) (percentage of total) were determined with a McKesson apparatus. Mea-

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Mean values 68 57 69 57 77 73
measurements were repeated at least three times until a maximal value was obtained. MEFR and FVC values were reproducible within ±10 per cent.

Differences between values before and after cordotomy were analyzed using Student's *t* test. 6

All but six patients had cinefluoroscopy of the diaphragm prior to and 24 hours after cordotomy. When diaphragmatic paralysis was found, examinations were repeated until recovery.

In a few patients, who had evidence of severe pulmonary insufficiency in the preoperative evaluation, cordotomy was not performed because of the possibility of development of respiratory complications.

**Results**

The data are summarized in tables 2-4. Preoperative $P_{a}CO_2$ values were within the normal range (35-45 mm/Hg) or lower. Most patients were slightly tachypneic, possibly because of pain. The $pH_a$ values ranged between 7.35 and 7.51. Since $P_{a}CO_2$ and $pH_a$ values showed no significant changes 24 hours after cordotomy, unless the patient was obviously in respiratory distress, they are not shown in the tables.

**Unilateral Cordotomy**

Preoperative values for FVC and MEFR in the 41 patients varied widely, but were lower in those who developed dyspnea postoperatively (tables 2A and 3). Mean FVC was 72 per cent before and 68 per cent after cordotomy, not significant. Three of 40 patients had a decrease and one an increase in FVC of more than 20 per cent. Mean MEFR was 69 per cent before and 67 per cent after cordotomy. Three of 40 had a decrease and two an increase in MEFR of more than 20 per cent. Mean $P_{a}O_2$ showed no significant change. Cinefluoroscopy (not studied in patients 2, 7, 8, 9, and 31) disclosed no phrenic paralysis before cordotomy, but paralysis postoperatively of one hemidiaphragm in two of the 35 patients. None were dyspneic.

Seven of the 41 patients developed dyspnea between one and 48 hours after the procedure (table 3). All had carcinomatosis with pulmonary involvement. None had phrenic paralysis. FVC and MEFR (pre- and postoperatively) were strikingly lower in the dyspneic group. Statistical comparison of the changes induced by cordotomy within the dyspneic and the non-dyspneic groups gave inconclusive results.

**Patient II/1 (table 2)** became short of breath 48 hours after cordotomy and required artificial ventilation via mask for two hours. He recovered. No phrenic paralysis.

**Patient II/3** had a brief episode of dyspnea at 24 hours (while spirometric values were essentially unchanged) but became apneic and stuporous on the third postoperative day. After intubation and ventilation she recovered consciousness. Vital capacity was 300 ml. Both hemidiaphragms moved well. Controlled ventilation via tracheostomy was necessary during most of the 45 days until death from pulmonary edema.

**Necropsy.** Radiation pneumonitis, pleural effusion and pulmonary edema were found. A lesion in the spinal cord involved almost the entire right ventral quadrant and the medial portion of the left ventral quadrant (fig. 2).

**Comment.** This patient did not have sleep-induced apnea: she developed respiratory insufficiency because of severe pre-existing pulmonary disease.

**Patients II/7 and II/9,** without reduction in pulmonary function values at 24 hours and without
clinical evidence later of cordotomy-related hypoventilation, eventually died with pneumonia.

Patient II/10 was very ill, became somnolent after cordotomy (Pao,
28 mm.Hg) and complained of dyspnea. The great reduction in FVC
and MEFR in this patient had little meaning since she did not cooperate. She did not require artificial ventilation, recovered and was discharged.

Patients II/27 and 29 had brief periods of dyspnea between 24 and 48 hours which did not require artificial ventilation.

Two patients had phrenic paralysis following cordotomy: patient 14 with ipsilateral and patient 32 with contralateral paralysis. Neither developed dyspnea. Both had values for FVC and MEFR above 60 per cent prior to cordotomy and showed further reduction in function due to the phrenic paralysis. In both, the palsy lasted less than a week.

SECOND-SIDE, BILATERAL CORDOTOMIES

Results are summarized in tables 2B and 4. Seventeen patients (II/25—41) had cordotomy performed on the opposite side one week after the first. Mean FVC and MEFR values prior to the second cordotomy were similar to those prior to the first, but none as low as in those of the unilateral group who developed dyspnea, probably because of case selection in the bilateral group.

Mean FVC was 68 per cent (40—100 per cent) before, and 57 per cent (28—83 per cent) after the second cordotomy, a significant reduction (P < 0.025). Mean MEFR was 69 per cent (32—100 per cent) before, and 57 per cent (23—100 per cent) after the second-side cordotomy, again significant (P < 0.025). Mean Pao,
was 77 (60—94) mm.Hg before and 73 (81—95) mm.Hg after cordotomy, again significant (P < 0.05).

Seven of the 17 patients developed dyspnea following the second cordotomy, but none required artificial ventilation. Dyspnea following the second cordotomy was accompanied by a reduction in pulmonary function values in six of the seven cases, which are reported here:

Patient II/25 became dyspneic and developed right phrenic paralysis a day after the second cordotomy.

Patient II/27 became dyspneic after bilateral cordotomy. This was the only patient with moderately reduced pulmonary function who had the second cordotomy performed. There was further reduction in pulmonary function.

Patient II/28 had lumbar radiculopathy and 24 hours after the second cordotomy developed dyspnea which lasted two days.

Patient II/32 had causalgia. He developed contralateral phrenic palsy after the first cordotomy. After the second, he became dyspneic, although pulmonary function was not further reduced.

Patient II/33 had lumbar radiculopathy and normal pulmonary function with no reduction in pulmonary function after the first cordotomy. A second cordotomy failed and was redone a week later. The third cordotomy (table 2B) caused a large reduction in function: FVC from 100 per cent to 29 per cent and MEFR from 110 per cent to 23 per cent. She became dyspneic and had transient left phrenic palsy. She was kept awake at night by Ritalin and allowed to sleep during the day under observation. Diaphragmatic action and pulmonary function values returned to normal within 73 hours.

Patients I/39 and 1/40 had malignancies. Each had dyspnea and a further reduction in pulmonary function for about two days following the second operation.

OTHER COMPLICATIONS

In all patients levels of analgesia were adequate. Temporary ataxia, hemiparesis and/or lack of control of sphincters occurred in some.
The majority complained of mild headache and had ipsilateral Horner's syndrome. All patients with severe respiratory complications and some without apnea had episodic hypotension. Hypotension appeared to be related to the patient's physical status and to the analgesic level.

Discussion

Postoperative mortality from respiratory and circulatory failure following open cervical cordotomy has been well documented,2-11 although some authors were not impressed by such complications.2-14 Feen and his associates15 advised that bilateral high cervical cordotomy should not be performed, as it could lead to respiratory paralysis. Belmont and his associates4 reported on prolonged postural hypotension in most patients with variable degrees of transitory respiratory insufficiency, more pronounced if there was pre-existing pulmonary pathology.

Percutaneous radiofrequency cordotomy, to an extent similar to cordotomy by incision, destroys not only the spinothalamic tract, but also neighboring pathways (fig. 2). Case II/3 (table 2A, fig. 2) is an example. The patient was thought to have unilateral cordotomy, but pathologic studies of the spinal cord showed necrosis or edema of both anterolateral quadrants. Post-mortem examinations of 24 patients who died following percutaneous radiofrequency cordotomies were made by Moossy (unpublished data). Four of these cases are reported in this paper. The lesions found were 3-13 mm. long, localized between C1 and C3 (fig. 2). No lesion or edema was found to ascend into the medulla or to descend to the C3-5 motor nuclei of the phrenic nerve. In one third of the cases, the lesions extended beyond the ipsilateral spinothalamic tract. Some lesions were found in the central lateral ventral quadrants, others in the dorsal quadrants, including the area of the corticospinal tract. Extensive lesions, even when limited to the ventral quadrants, could interrupt upper neuron fibers leaving the corticospinal tract as they sweep toward the C3-5 anterior horn cells, thus resulting in phrenic paralysis.

The three documented and ten questionable post-cordotomy apneas among the 315 cordotomies performed (retrospective series) represent a smaller incidence of complications than reported after open (operative) cordotomy5,7-12,13 if the severity of the disease treated can be considered comparable. Nevertheless, when respiratory insufficiency occurred, it was a life-threatening matter.

When primary apnea occurred, it took a characteristic and bizarre form. Although patients may have felt dyspnea shortly after the cordotomy, there was no objective clinical evidence of distress at first. Patients I/1-3 had hysterical personalities leading to periods of hyperventilation before as well as after operation. This could lead the uninstructed to invoke a psychiatric diagnosis, when evaluating this subjective complaint, only to find the patient, on the second or third postoperative day, asleep and apneic. When the subject was awakened, and hypoxia had not occurred, he resumed respiration at normal levels only to become apneic when natural or fatigue-induced sleep recurred. This problem persisted intermittently for three to four weeks (Patients I/1 and I/2), in one patient for three months (Patient I/3), necessitating intubation, tracheostomy and artificial ventilation. One patient died; two eventually were weaned from the ventilator and returned to normal activity.

The mechanism of this respiratory disorder is not known. A working hypothesis has been constructed from available physiologic and morphologic data and clinical observations. All three patients of the retrospective study had undergone bilateral cordotomy and each had bilateral C3-4 levels of analgesia. By inference, the anatomical lesions must have been large, bilateral, and at the same level in the spinal cord. Since morphological studies were available only for patient I/1 (fig. 2), correlation between complications and the anatomic lesion is not possible. Available neurologic and anatomic data suggest that the occurrence of respiratory complications is related more to the proportion of ventral quadrant structures destroyed than to the phrenic paralysis. This suggests an important role of "deafferentation" in the phenomenon.

Neurophysiologic investigations have recorded potentials with both inspiration and expiration from the ventral quadrants of the
first to third cervical spinal segments. These fibers appear to have a maximum concentration anterior and anterolateral to the ventral horns. Their precise function is not clear, although there is reason to believe that they represent spinal extensions of the brain-stem reticular formation. It would appear likely, therefore, that an extensive bilateral cervical cordotomy could interrupt these fibers which may serve the respiratory centers in the same manner as the reticular formation serves other specific projection systems. This would produce deafferentation of the activating reticular mechanism so that respiration would cease unless the patient was awake and using other neural pathways for awareness.

Patient I/3 and a more recent case not reported here, who developed post-cordotomy apnea, had impaired responses to increase in \( P_{\text{CO}_2} \). This finding and the postulated deafferentation of the reticular activating system interdigitate nicely. If, in addition to the absence of neural activity, the patient does not respond to an increase in \( P_{\text{CO}_2} \), there is a second explanation for hypoventilation and apnea during the sleeping state. The latter may be aggravated by the hyperventilating hysterical patient who may start, when awake, from a subnormal \( P_{\text{CO}_2} \), and goes on to sleep with neither neural or chemical activation, resulting in apnea, hypoxia and death unless the sequence is interrupted.

Interestingly, none of the three patients of the retrospective series who had post-cordotomy apnea was known to have primary pulmonary disease. Pulmonary disease could have been a contributory factor, however, in the other patients of this group, and it was definitely a factor in those of Group II (prospective study) who developed dyspnea or hypoventilation.

The study of pulmonary function did not clarify the nature of post-cordotomy apnea. However, the degree of pre-existing restrictive or obstructive lung disease correlated well with post-cordotomy dyspnea or hypoventilation, even following unilateral cordotomy. At the same time, a number of individuals with diminished pulmonary function underwent unilateral cordotomy and did not develop pulmonary insufficiency. We speculate that in those the lesion was confined to the spinothalamic tract.

In those 41 patients who had pulmonary function studies, life-threatening primary apnea was not observed. Two patients required artificial ventilation, but survived. Unilateral cordotomy did not ordinarily produce a further decrease in pulmonary function. Following the second cordotomy, dyspnea was more clearly related to phrenic palsy and to postoperative reduction in pulmonary function. None of these patients, however, required artificial ventilation. Perhaps this is explained by the fact that none had severely reduced pulmonary function preoperatively. Phrenic paralysis was invariably temporary, lasting three to seven days, and did not become a source of concern.

These observations provide a guide for selection of patients: unilateral percutaneous cordotomy, performed with accuracy, is safe in patients with near-normal pulmonary function; it has even been suggested for the intractable pain of benign disease. In patients with severely reduced pulmonary function, exacerbation of respiratory insufficiency introduces a calculated risk. Mean pulmonary function values were reduced significantly by a second-side cordotomy even in patients with near-normal pulmonary function preoperatively. Therefore, patients with a greatly reduced preoperative FVC and/or MEFR should not be subjected to bilateral cordotomy unless the considerable risk is balanced against the expected gain. All patients should undergo measurements of FVC and MEFR preoperatively to detect early any need for ventilatory assistance. Following cordotomy, patients with any suspicious respiratory symptoms should be observed during sleep.

Hypotension was uncommon and not severe following percutaneous cordotomy, as compared to operative cordotomy. The higher incidence associated with surgical cordotomy may be the result of hypovolemia from operative blood loss and the effect of general anesthesia.

Summary and Conclusions

Of 200 patients undergoing percutaneous cervical radiofrequency cordotomy 13 had re-
spiratory complications in which the effect of
cordotomy was suspect. Three of these pa-
tients had sleep-induced apnea following bi-
lateral cordotomy and required artificial ven-
tilation for one to three months.

Subsequently, in 41 unselected patients,
pulmonary function studies, including cineflu-
orooscopy of the diaphragm, were performed be-
fore and 24 hours after cordotomy.

Following 41 unilateral cordotomies the
changes in mean FVC and $P_{\text{O2}}$ were not sig-
nificant, although in a few instances these
values decreased appreciably. MEFR re-
mained essentially unchanged. Seven of the
41 patients developed dyspnea post-cordotomy
without phrenic palsy. Two of the seven re-
quired artificial ventilation transiently; and six
had had greatly reduced pulmonary function
values preoperatively. Two patients developed
phrenic palsy, but did not have dyspnea. Of
12 patients with either FVC or MEFR less
than 50 per cent of the predicted value pre-
operatively, six developed dyspnea, two died
of pneumonia and two required artificial ven-
tilation postoperatively. In unilateral cordot-
omy, therefore, postoperative respiratory insuf-
siciency is a possibility in patients with pre-
existing lung disease.

Following a second-side cordotomy (17 pa-
tients) there was a significant reduction in all
pulmonary function values, more in the 7/17
who became dyspneic. None required artificial
ventilation. Three who developed phrenic
palsy had dyspnea.

Most cases of dyspnea were related to pre-
existing pulmonary disease and/or bilateral
cervical cordotomy.

Clinical and morphologic observations sug-
gest that the degree of respiratory insuffi-
cency is related to the total number of afferent neu-
rons interrupted.

Pre-operative spirometry, postoperative res-
piratory monitoring and the availability of
prolonged ventilator care are mandatory when
a cordotomy is done in a patient with greatly
reduced pulmonary function.

Dr. John Moosy made valuable suggestions and
provided Figure 2. Dr. Floyd Taylor gave advice
about the statistical data.

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