Sleep Apnea Following Percutaneous Cervical Cordotomy

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ABSTRACT: Sleep apnea (Ondine's Curse) is an uncommon but potentially fatal complication of percutaneous cervical cordotomy. The authors have had the opportunity to review the case histories of 6 patients who have died of sleep apnea following this procedure. In a series of 112 patients, 144 cervical percutaneous cordotomies were performed from 1977 to 1985 — 80 unilateral cordotomies and 32 staged bilateral cordotomies. Six (5%) patients died as the result of sleep apnea. Five patients (16%) died following bilateral cordotomy and one (1%) patient with pulmonary disease died following unilateral cordotomy. The clinical data of these 6 patients are presented and unique spinal cord pathology is described in this report. The pathophysiology of the sleep apnea syndrome is also discussed.

RÉSUMÉ: Apnée du sommeil suite à la cordotomie transcutanée. L'apnée du sommeil (la malédiction d'Ondine) est une complication rare, mais potentiellement fatale de la cordotomie cervicale transcutanée. Les auteurs ont eu l'occasion de reviser les histoires de cas de 6 patients qui sont décédés d'apnée du sommeil à la suite de cette intervention. Dans une série de 112 patients, 144 cordotomies cervicales transcutanées ont été pratiquées entre 1977 et 1985, soit 80 cordotomies unilatérales et 32 cordotomies bilatérales pratiquées en 2 étapes. Six (5 %) des patients sont décédés par suite d'apnée du sommeil. Cinq patients (16 %) sont décédés après une cordotomie bilatérale et un (1 %) patient atteint de maladie pulmonaire est décédé après une cordotomie unilatérale. Nous présentons les données cliniques sur ces 6 patients et nous décrivons des constatations anatomopathologiques uniques au niveau de la moelle épinière. Nous discutons également de la pathophysiologie du syndrome de l'apnée du sommeil.

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Since its introduction in the 1960's, percutaneous cervical cordotomy has become a popular procedure in the management of intractable pain. The destruction of the lateral spinothalamic tract in the anterolateral column of the spinal cord by a radiofrequency lesion can be a simple and effective method of producing contralateral analgesia. Complications such as respiratory dysfunction, ipsilateral paresis, sphincter disturbance, and circulatory insufficiency have been described following this procedure and are produced by inadvertent injury to other spinal cord pathways.

Respiratory dysfunction following percutaneous cervical cordotomy is the most serious of these complications because of its potentially fatal outcome. The respiratory complications were first recognized in the early 1930's, when cordotomy was performed by the open method.[1] Despite advantages of the percutaneous cordotomy, the problem continues. Sleep apnea is an unusual type of respiratory dysfunction that may complicate the cordotomy procedure especially if the patient undergoes bilateral cordotomies. Sleep apnea was first referred to as Ondine's Curse by Severinghaus and Mitchell in their 1962 publication.[2] The term Ondine's Curse relates to a mythical water nymph, Ondine, who, having been jilted by her mortal husband, took from him all automatic functions requiring him to remember to breathe. When he finally fell asleep he died.

Since 1977, 144 cordotomies have been performed by one of the authors (WST) on 112 patients. Six of these patients died following percutaneous cervical cordotomy as the result of sleep apnea. The case histories of these 6 patients will be presented and the spinal cord pathology of three of these cases will be described. Using this information, the pathophysiology of this interesting syndrome will also be discussed.

Patients and Clinical Methods

This report is a retrospective study of a series of patients who underwent high percutaneous cervical cordotomy performed since 1977. The patients who died following the procedure secondary to sleep apnea were identified for this report. The indication for the cordotomy in each case was chronic severe pain and in almost all of the cases, malignant disease was the cause of the chronic pain. Bilateral cordotomy was performed as a staged procedure if pain on the other side of the body was intractable following the original cordotomy.

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The cordotomies were performed under local anesthesia with the patient being moderately sedated but able to give appropriate responses. The technique used for the cordotomies was similar to that described by Tasker.[3] The percutaneous cordotomy electrode consisted of a 0.4 mm stainless steel wire insulated with teflon tubing. The tip of the wire projected 2 mm beyond the tubing. This electrode was introduced through an 18 gauge lumbar puncture needle. The Owl Universal RF System provided the electrical stimulation and the output for making a radio-frequency lesion.

With the patient in the supine position, the LP needle was introduced at the C1-2 level under fluoroscopic control. Spinothalamic tract localization was performed by repeat stimulation once the needle tip was in the spinal cord. Lesion size was determined by the duration and level of the current flow. Motor testing of the ipsilateral leg was performed during the lesion-making to prevent inadvertent injury to the corticospinal tract. Postoperative care consisted of analgesics as necessary and close monitoring of the vital signs. Because of the nature of the malignant disease, aggressive resuscitative efforts were not always performed. Although the risk of respiratory complications was realized, it was understood in certain cases that a peaceful death by sleep apnea was a possibility and at times welcomed by the relatives.

In those cases in which an autopsy was performed, the cervical spinal cord was taken for pathological examination. The spinal cord was fixed in formalin, serially sectioned and stained, and examined by light microscopy.

Case Histories

Patient 1: A 59-year-old female was admitted to hospital in 1977 with severe left shoulder pain secondary to carcinoma of the left breast. The carcinoma had previously been treated by a left modified mastectomy and radiation. Because of the severe shoulder pain, a right percutaneous cervical cordotomy was performed with good results. However, she was readmitted in 1979 with severe left flank pain. Further investigation revealed a second malignant tumour, a retroperitoneal leiomyosarcoma. Chemotherapy was instituted. The pain continued and in November, 1979, a second right percutaneous cervical cordotomy was performed, again with good results. One month later, however, she was readmitted with severe right sided pain extending from her right shoulder to her right flank.

In December, 1979, a left percutaneous cordotomy was performed resulting in analgesia below the C-4 level on the right side. Post cordotomy, she was free of pain and was up walking about the ward. During the night of the fifth postoperative day, she was found dead in bed without evidence of distress. An autopsy was performed.

Patient 2: A 55-year-old female was admitted to hospital in November, 1980, with severe left arm pain secondary to metastatic adenocarcinoma of the stomach. Investigations revealed that her liver and left supra-clavicular lymph nodes were involved by the metastatic adenocarcinoma and that her left phrenic nerve was not functioning. A right percutaneous cervical cordotomy was performed with good results. On the first post-operative day she was free of pain but it was noted that she was short of breath. That evening she was found dead in bed without any evidence of distress. An autopsy was performed.

Patient 3: A 66-year-old man was admitted to hospital in August, 1981, with intractable severe pain of the back and left leg. A metastatic work-up revealed metastases in the skull, lung and left femur. A right percutaneous cordotomy was performed with good analgesia below the C-3 level on the left side. Shortly after this procedure, pain emerged in the right shoulder, flank and leg. A left percutaneous cervical cordotomy was performed one week later. Good results were obtained below the C-4 level on the right side. Two days following surgery, he was mobile but suffered mild shortness of breath and mild confusion. That evening he was found dead in bed after being observed sleeping peacefully one hour earlier. An autopsy was performed.

Patient 4: A 55-year-old man was admitted to the hospital with intractable left leg pain secondary to metastatic carcinoma. No primary carcinoma could be identified. A right percutaneous cordotomy was performed with good results. Severe right leg and arm pain soon emerged and this led to a left percutaneous cordotomy being performed one week later. On the first postoperative day he slowly developed severe respiratory distress and was intubated and ventilated. He was gradually weaned from the ventilator and extubated successfully. However, nine days following the second cordotomy, he died in his sleep.

Patient 5: A 74-year-old female was admitted to the hospital September, 1978, for treatment of severe left chest pain secondary to metastatic carcinoma of the breast. Although no lung metastases were seen on chest x-rays, a left pleural effusion was present. A right percutaneous cordotomy was performed with good results. Right chest pain soon developed and one week following the first cordotomy a left cervical cordotomy was performed. Initially, this procedure was well tolerated with good analgesia but she developed respiratory failure two days postoperatively, and she required mechanical ventilation for several days. Her respiratory status improved and she was extubated successfully. On the seventh postoperative day she was found dead in bed.

Patient 6: A 66-year-old man was admitted to the hospital with severe left flank pain and low back pain secondary to metastatic transitional cell carcinomas of the left ureter. A right percutaneous cordotomy was performed with good analgesia below C-4 on the left side. Right flank pain soon emerged and a left percutaneous cordotomy was performed one week later. Good analgesia was obtained. Sleep apnea was observed on the 3rd and 5th postoperative days and respiratory failure developed on the 6th day. He was ventilated for 48 hours but then was easily weaned. Although the level of analgesia remained satisfactory, he remained short of breath and was found dead in bed three weeks later.

Results

All of the percutaneous cervical cordotomies were performed since 1977. A total of 144 cordotomies were performed on 112 patients. Eighty unilateral cordotomies and 32 bilateral cordotomies were performed. Chronic intractable pain was the indication for the operation in each case. The chronic pain was secondary to malignant disease in all patients except 4. Bilateral procedures were only performed for patients with malignancies and were performed as a staged procedure. The second cordotomy was delayed by an average of 16 days (4-60 days).

The incidence of the respiratory complication, sleep apnea, following percutaneous cervical cordotomy was 5% in the 112 patients (Table 1). The mortality rate following bilateral cordotomy was 16% and following unilateral cordotomy it was 1%. Three patients developed severe respiratory insufficiency following cordotomy and were mechanically ventilated for a short time. Their recovery was only temporary and their deaths were attributed to sleep apnea. The six patients whose deaths were attributed to sleep apnea died peacefully during their sleep without any signs of distress. These patients died 8 days (mean) following the final cordotomy.

Autopsies were obtained on three of the patients who died of sleep apnea. The spinal cord was dissected from the spinal canal and cut into serial sections at the C1-2 level. In case 1 three surgical lesions were found when the cervical spinal cord was sectioned at the C1-2 level (Figure 1). The most recent lesion, approximately one week old, consisted of a large area of necrosis located in the anterior two-thirds of the left anterolateral column. This necrosis also extended into the left ventral horn. Two older surgical lesions were found in the right anterolateral column of the spinal cord. One of these lesions, in the lateral

column, consisted of healing tissue, while the more anterior lesion was composed of gliotic scar. Bands of necrosis were also found in the left dorsal column and the age of these lesions were comparable to the age of the healing lesion in the right lateral column.

The spinal cord in case 2 revealed a well defined area of recent necrosis in the anterior third of the right anterolateral column of the spinal cord at the C1-2 level (Figure 2). The necrosis also extended medially into the anterior part of the ventral gray matter.

The spinal cord in case 3, revealed two well demarcated areas of necrosis in the anterolateral columns at the C1-2 level (Figure 3). The most recent lesion, approximately three days old, consisted of necrosis in the anterior one-third of the right anterolateral column. An older area of necrosis (1 week old) occupied approximately two-thirds of the left anterolateral column. This old lesion also extended into the ventral gray matter.

Discussion

The etiology of post-cordotomy respiratory failure and sleep apnea has not been satisfactorily described in the current literature. A number of pathways in the cervical spinal cord have been implicated but the importance of a lesion in these tracts continues to be debated. We anticipated that examination of the spinal cord pathology would help determine which of the many tracts in the cervical spinal cord may be responsible for sleep apnea following cordotomy.

The deliberate section of the spinothalamic tract of the spinal cord for relief of pain was first suggested by Schuller in 1910.[4] But it was not until one year later that Martin performed the first dorsal cordotomy in man for the treatment of organic pain.[5] Foerster and Gagel were the first to perform an open cervical cordotomy in 1929.[6] A percutaneous method for cervical cordotomy was first introduced by Mullan and associates in 1963.[7] His initial method utilized a radioactive needle which was inserted into the spine at the C1-2 level and was positioned antrolaterally in the cervical arachnoid space adjacent to the ventral quadrant of the spinal cord. Although some success was claimed with this procedure, Mullan's method was modified by introducing a wire electrode into the parenchyma of the spinal cord and a direct current source was utilized for making the lesion in the ventral quadrant.[8] The results of this method were unpredictable. However Rosomoff and associates proposed the present technique of using a radiofrequency current to produce the lesion within the spinothalamic tract.[9] This RF current allowed for a more rapid and more controlled lesion to be made in the spinothalamic tract.

Although great success has been reported with percutaneous cervical cordotomy, significant complications and fatalities have occurred.[10,11] Ipsilateral hemiparesis occurs temporarily in approximately 5% of cordotomies and can last up to 14 days. Permanent hemiparesis has been reported in 3% of patients. Ataxia is a common complication being reported in about 20% of patients following cordotomy. Bladder dysfunction is found in approximately 10% of patients undergoing a unilateral procedure and is more common following bilateral cordotomies. Post cordotomy dysesthesia syndromes and paresthesia have also been reported.[11]

Respiratory failure is the most serious and life-threatening complication of percutaneous cordotomy. Foerster reported respiratory problems following open cordotomy as early as the 1930's.[1] It was anticipated that the smaller more refined lesion of the percutaneous cordotomy technique under local anesthesia would decrease the incidence of respiratory complications. Unfortunately, this has not turned out to be the case. They occur most commonly following bilateral procedures and are often only transient. The reason for the respiratory complications is that the spinothalamic tract is intermingled with fibers which mediate respiratory and autonomic information.[12] Descending reticulospinal axons from the medulla which mediate respiratory motor control and are destined for secondary phrenic, intercostal and abdominal motor neurons in the ventral horn, descend in tracts that are adjacent to the ventral gray matter and are just behind or intermingled with the spinothalamic tract (Figure 4).

Table 1: Summary of clinical data for 6 patients dying of sleep apnea following cordotomy

Case and Gender	Age	Diagnosis	First Cordotomy	Second Cordotomy	Respiratory Complication	Interval to Death
1 Female	59	Carcinoma of Breast Leiomyo-sarcoma	Rt 1977 Repeat Rt Nov. 1979	Lt Dec. 31,1979	Sleep Apnea	5 days
2 Female	55	Carcinoma of stomach	Rt Dec. 1 1980		Shortness of breath Sleep Apnea	1 day
3 Male	66	Carcinoma of colon	Rt Aug. 17 1981	Lt Aug. 24 1981	Shortness of breath Sleep Apnea	2 days
4 Male	55	Metastatic Carcinoma	Rt Sept. 14, 1981	Lt Sept. 24,1981	Respiratory Failure Sleep Apnea	9 days
5 Female	74	Carcinoma of Breast	Rt Sept. 14, 1978	Lt Sept. 21, 1978	Respiratory Failure Sleep Apnea	7 days
6 Male	66	Carcinoma of Ureter	Rt March 8, 1979	Lt March 15, 1979	Respiratory Failure Sleep Apnea	22 days

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These fibers are loosely arranged into two bundles. The anterior group delivers motor fibers to the phrenic motoneurons and the more posterior group controls the intercostal muscles.[13] This close association of the reticulospinal tract and the spinothalamic tract makes it difficult to perform an effective lesion in the spinothalamic tract without producing some respiratory dysfunction. Ascending spinoreticular fibres which transmit information for ventilatory control are also intermingled in the spinothalamic tract.

Lesions involving the afferent and/or efferent respiratory pathways of the spinal cord have been implicated as the cause of respiratory complications following cordotomy.[14,15,16] Belmusto measured an intraoperative reduction of respiratory tidal volume immediately following the sectioning of the ventral quadrant of the spinal cord and concluded that the respiratory impairment was caused by an interruption of the descending respiratory pathways (reticulospinal fibers).[17] Nathan, in 1963, after monitoring intraoperative bronchospirometry recordings and also studying postmortem specimens of the spinal cords, also suggested that these motor pathways which descend in the anterolateral column of the spinal cord would be interrupted by the cordotomy.[18] He localized these fibers to the lateral surface of the ventral horn medial to the spinothalamic tract.

Impaired control of ventilation by a lesion in the afferent ascending fibers (spinoreticular fibers) has also been suggested as a cause for respiratory dysfunction following lesions in the cervical lateral column.[6,14,19] Kreiger, in 1974, reported data on 10 patients who developed sleep-induced apnea following bilateral percutaneous cervical cordotomies.[14] Pulmonary function testing was interpreted as showing that both motor function and control of ventilation were disrupted by the cordotomy. He found that respiratory changes were characterized by an attenuated CO_2 response with or without a decrease in vital capacity. He felt that the occurrence of apnea during sleep, the reversal of apnea by arousal and the absence of persistent motor changes strongly suggested that the spinoreticular tract was present in the anterolateral column of the spinal cord and

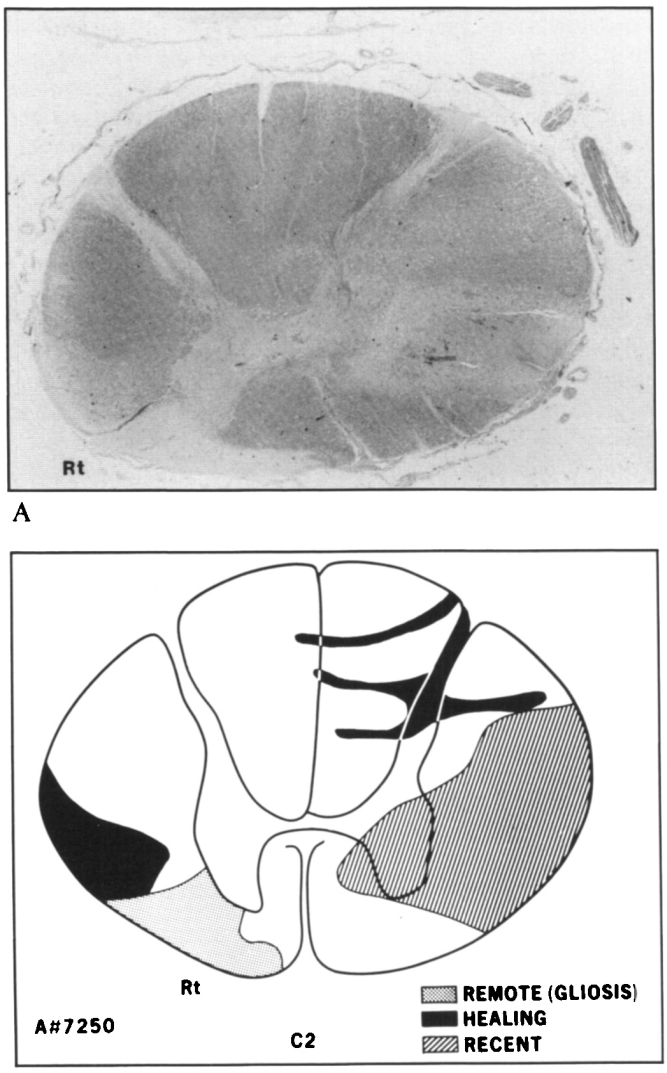

Figure 1A) — Section of spinal cord at the C1-2 level showing three cordotomy lesions performed in 1977 (remote), Nov. 1979 (healing) and Dec. 1979 (recent). B) Diagrammatic illustration of autopsy specimen of the spinal cord at the C1-2 level.

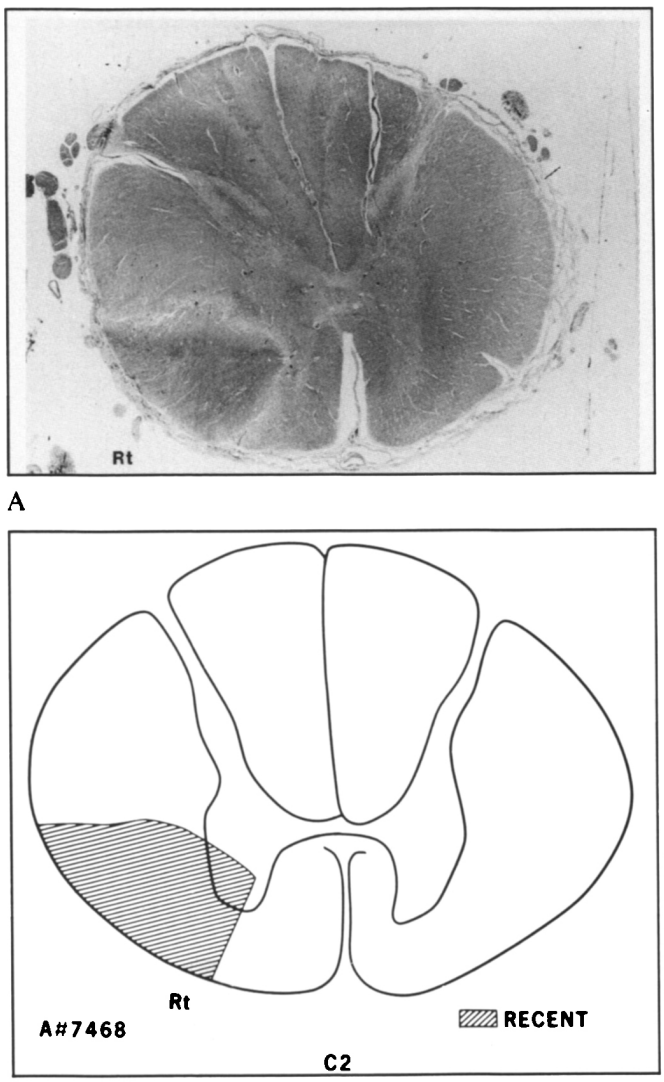

Figure 2A) — Section of spinal cord at the C1-2 level showing a large area of necrosis in the right anterolateral quadrant produced by a recent right cordotomy. B) Diagrammatic illustration of autopsy specimen of the spinal cord at the C1-2 level.

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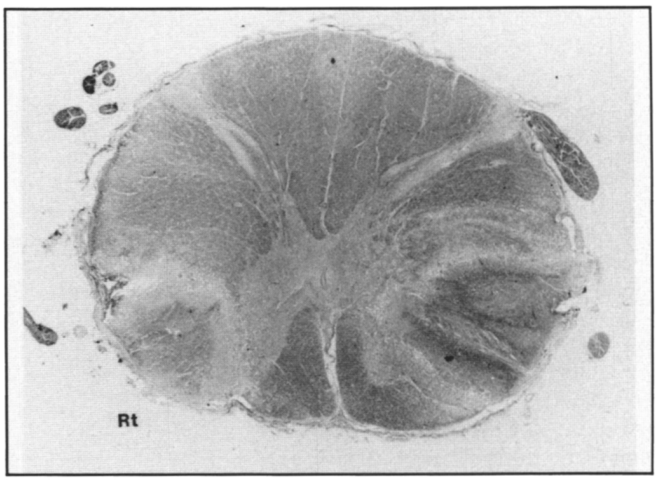
may be inadvertently injured by cervical cordotomy. Rosomoff and associates have also reported studies of respiratory function in patients undergoing percutaneous cervical cordotomy and have also concluded that the interruption of the ascending spinoreticular fibers contribute to the sleep apnea syndrome.[11]

Another possible cause of respiratory dysfunction following cordotomy is the destruction of respiratory neurons that may be located in the ventral horns at C1-2. Akoi and associates have found inspiratory unit activity in neurons at C1-2 in cats and has proposed that these neurons play an important role in generating phrenic motoneuron discharges.[20]

The sleep apnea syndrome can be difficult to recognize and often there are no warning symptoms before the patient dies during sleep. The incidence of this complication following cordotomy has been reported to be approximately 3-5%,[11,21] It is delayed in onset but may be predicted by testing patients for their response to breathing CO_2. The patient may complain of lethargy, generalized weakness, shortness of breath, and may develop a number of autonomic dysfunctions such as hypotension, hyponatremia and sphincter disturbance. Factors that depress the reticular formation activity such as sleep, barbiturates and narcotics potentiate this syndrome.[11] If the sleep apnea patient is watched closely during the postcordotomy period, it will be observed that he hypoventilates when he is awake and characteristically becomes apneic during sleep. If the patient is awakened, his regular breathing resumes but hypoventilation may persist and apnea remains a problem with the resumption of sleep. This condition is usually reversible but supportive ventilation may be required during the sleep apnea periods.

In this series of cordotomy patients, 6 deaths were attributed to the sleep apnea syndrome. The postmortem specimens of spinal cord on three of the patients demonstrated extensive lesions within the anterolateral column of the spinal cord at the C1-2 level. The radiofrequency lesions of the cordotomy needle produced large areas of necrosis in the anterolateral column, often extending into the ventral gray matter. Our pathological specimens suggest that both the efferent motor fibers (reticulospinal fibers) and the afferent fibers (spinoreticular fibers) can be injured by a cordotomy lesion. Both the efferent fibers which are located medially to the ventral horn and the afferent fibers which are placed more laterally within the spinothalamic tract appeared damaged in the spinal cords of sleep apnea victims (Figure 4). Our clinical and pathological observations have led us to agree with others that the sleep apnea syndrome may be secondary to both dysfunction of the control of ventilation and the efferent motor pathways of the respiratory system.

There is no evidence that a smaller, more accurate lesion in the anterolateral column will decrease the incidence of respiratory complications.[15] Therefore, respiratory failure will likely continue to plague this simple and effective procedure for pain control. Patients requiring bilateral cordotomies are at risk for sleep apnea as well as patients who require a unilateral cordotomy but have pre-existing lung disease. Bilateral cordotomies should be staged; the second procedure being delayed by at least a week. A high thoracic cordotomy may be performed as the second procedure if the pain problem is in the trunk or leg, thus avoiding the risk of sleep apnea. Patients at risk should be monitored in the intensive care unit in the postoperative period using spirometry, arterial blood gases and vital signs as guides for therapy. Symptoms such as shortness of breath, confusion



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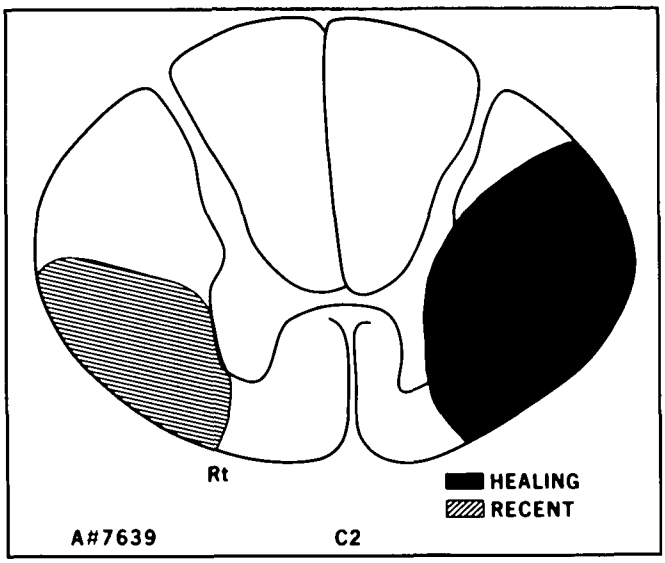

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Figure 3A) — Section of spinal cord at the C1-2 level showing a recent cordotomy lesion in the right anterolateral quadrant and a healing lesion in the left lateral column. B) Diagrammatic illustration of autopsy specimen of the spinal cord at the C1-2 level.

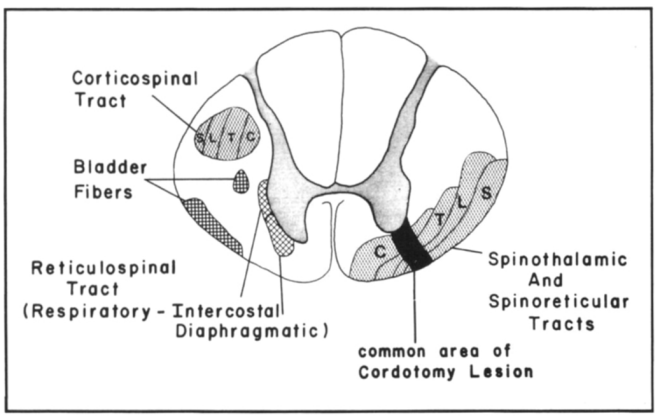

Figure 4 — Cross-Sectional anatomy of the spinal cord at the C1-2 level showing ascending and descending spinal pathways. (The spinoreticular tract is located within the spinothalamic tract.) The common lesion area represents an area which was involved in the significant cordotomies.

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and lethargy may herald the onset of sleep apnea. Apnea alarms are beneficial during the risk period and pharmacological depressants are to be avoided. Ventilatory support may be necessary if respiratory failure or sleep apnea occur, but because this condition is self-limiting, the patient usually can be weaned easily.

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