THE TREATMENT OF PULMONARY EMBOLUS
BY STELLATE BLOCK

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In recent years the blocking of the stellate ganglion has become more universally used but its application has more practical aspects than formerly realized. Recently, we have used this treatment for the syndrome of severe pulmonary emboles. The Trendelenburg operative procedure for removal of the pulmonary embolus has proved impractical and rarely successful. The treatment of pulmonary embolus by stellate block is offered as an adjunct to present therapy to relieve the acute episode of this entity. The subsequent treatment by anticoagulants, vein ligation, and routine supportive therapy does not fall within the scope of this paper.

The stellate ganglion is frequently formed by the fusion of the inferior cervical and first thoracic sympathetic ganglion. It lies between the transverse process of the seventh cervical vertebra and the neck of the first rib on the medial side of the costocervical artery.

The technic of blocking the stellate ganglion varies with the individual performing it. Whether the anterior or posterior approach is used, with their variations, one must be careful to avoid the following:

1. Entering the pleura which may cause pneumothorax.
2. Injecting the anesthetic solution into a blood vessel which may result in circulatory or respiratory collapse or both. Convulsions may also result from inadvertent intravenous injection of the anesthetic agent.
3. Injecting the anesthetic solution into the spinal fluid or spinal cord which may produce temporary or permanent paralysis.
4. Anesthetizing the recurrent laryngeal nerve which will cause paralysis of the vocal cord on the injected side manifested by hoarseness and some difficulty in breathing.
5. Anesthetizing the phrenic nerve which causes paralysis of the diaphragm on the injected side.

Because of the last two complications mentioned, a stellate block should not be done bilaterally at the same time. A successful block results in a Horner’s syndrome which is characterized by ptosis, miosis, anhidrosis, injection of the sclera, and increased temperature.
of the face, neck, arm and chest wall of the injected side (1). We have used the anterolateral approach (2) and after producing a typical Horner’s syndrome with 2 to 6 cc. of 2 per cent metycaine, then 1 cc. of a long acting anesthetic in oil (novestoil) is injected for prolonged effect. With this technic, the Horner’s syndrome persists from two to thirty-six hours and the effect of the block from two to six days or longer.

The first case report is presented in detail for the many interesting aspects of this patient’s clinical course. A stellate block was done for two episodes of severe pulmonary embolus, with dramatic and immediate relief of chest pain, dyspnea, orthopnea, cyanosis, and with a probable reversal of a shock syndrome.

Case 1.—A 36-year-old colored man was admitted to the hospital on October 7, 1946, because of cough, shortness of breath and swelling of the feet and ankles.

Examination revealed that the patient was acutely ill and in marked respiratory distress. The respirations were 40 per minute, labored and he was unable to lie flat. The blood pressure was 100 mm. systolic and 45 mm. diastolic, and the pulse rate was 90 per minute and regular. There was a soft systolic mitral murmur, not transmitted. A systolic thrill was noted over the aortic area, and on auscultation, a harsh blowing systolic murmur was heard, followed by a less intense diastolic murmur. These murmurs were transmitted into the veins of the neck which were moderately distended. There were many moist inspiratory and expiratory rales at both lung bases. The liver edge was 6 cm. below the right costal margin and slightly tender. Pitting edema of both feet and ankles was present as well as clubbing of the fingers and toes. Laboratory examinations revealed sedimentation rate of 38 mm. per hour (corrected) and a positive Kahn but negative Wassermann. On roentgenologic examination, the heart was grossly enlarged downward and to the left and the aorta was diffusely dilated and tortuous. The hazy density throughout both lung fields was attributed to pulmonary edema. There was no fluid in the pleural cavity. The clinical impression was chronic rheumatic heart disease with aortic insufficiency and acute congestive heart failure. Syphilitic heart disease was also considered.

The patient was given digitalis, diuretics, and placed on supportive therapy. He made an excellent initial response but the cough and dyspnea persisted, and on the second hospital day auricular fibrillation developed. An electrocardiogram confirmed this diagnosis and also showed digitalis effect. The daily blood pressures averaged 115 mm. systolic and 50 mm. diastolic, with an average pulse pressure of 65. On the eighth hospital day quinidine was given for the fibrillation but was discontinued because of nausea, vomiting and diarrhea. It was again started on the tenth day without untoward effects. The cardiac rhythm became regular but on the fourteenth hospital day, the patient complained of a sudden onset of severe pain in the right lower chest. He had associated dyspnea, orthopnea, cyanosis, an elevation of temperature, marked apprehension, moderate sweating, and he expectorated a small amount of blood-tinged sputum. Later, a severe pain developed over the lower left chest as well. The chest pain, dyspnea and cough could not be controlled even with large doses of morphine. A stellate block was done on the right side with 2.5 cc. of 2 per cent metycaine and after a typical Horner’s syndrome appeared, 1 cc. of a long-acting anesthetic
agent in oil was injected. Within a few minutes a dramatic result followed. The patient experienced almost complete relief of pain in the right chest and later also in the left chest. His breathing became easier, his chest expansion greater, chest splinting was less, and he fell asleep for the first time since the onset of the pulmonary infarction. A roentgenogram confirmed the diagnosis of pulmonary infarction of the right chest.

The patient remained comfortable until the twenty-second hospital day when he had a second pulmonary embolus. He requested block therapy for his pain. At this time, he appeared in extremis; he had marked pain, orthopnea, dyspnea, cyanosis, very shallow respiratory excursions, and profuse sweating. The pulse rate was irregular, the apical rate being 148 per minute. The pain was most intense about the right lower chest but again also extended over the left lower chest. A second stellate block was done immediately on the right side with 6 cc. of 2 per cent metyamine and after a typical Horner's syndrome appeared, 1 cc. of a long-acting anesthetic agent in oil was injected. He experienced almost immediate relief of his pain and dyspnea and was able to breathe and talk at the same time. There was no longer the look of apprehension and anxiety on his face; he appeared more comfortable and relaxed. A roentgenogram again confirmed the clinical findings of pulmonary embolic phenomena of the right lower lobe. The patient remained comfortable until the thirty-first hospital day at which time he had his third pulmonary embolus. A stellate block was not done at this time for he was semicomatose and remained so until his death.

At necropsy, an area was found between the posterior cusp of the aortic valve which was calcified and showed evidence of having probably been the seat of a thrombus because this area was hemorrhagic and contained what appeared to be thrombus material. The right lung showed evidence of recent red nodular-like areas on the external surface, which were deep red on section. Dissection of this lung revealed small pulmonary emboli present in the small branches of the pulmonary artery supplying the area of infarction. The left lung revealed old, well-defined infarcts and there was evidence of an old fibrinous pleurisy in the region of the left lower lobe posteriorly. The final diagnosis was rheumatic myocarditis with aortic stenosis and insufficiency, cardiac failure, multiple pulmonary infarction of the right lung and old multiple infarctions of the left lung. It was the opinion of the pathologist that only the new infarcts found in the right lung were of significance in this patient's terminal illness.

The second case report is briefly stated for the patient did not present the clinical picture of extremis. Marked relief from her pain, dyspnea, and apprehension resulted from stellate block therapy after opiates had failed to give her but slight relief.

Case 2.—A 39-year-old white woman was suddenly awakened at 1:30 a.m. on December 27, 1946, with a severe, excruciating pain in the right chest. She stated that the pain was so severe that she could not think. Change of position did not give her any relief. She could take only very shallow breaths because of the pain and a constricting sensation about the chest. She was given hypodermics at home which eased the pain only slightly. She was admitted at 6:00 p.m. the same day with the same symptoms as stated.

Physical examination revealed that the patient was well nourished and well
developed, in moderate respiratory distress. She appeared anxious and tired. There was marked splinting of the chest; the respiratory rate was 34 per minute and the respiratory excursions were diminished. Crepitant rales were heard over the right anterior chest. The blood pressure was 125 mm. systolic and 80 mm. diastolic, and the pulse rate was 88 per minute and regular. A roentgenogram confirmed the clinical diagnosis of pulmonary embolus; there was a sharply defined area of consolidation in the anterolateral aspect of the right middle lobe which involved all the pleural surfaces and appeared characteristic of an infarct. Laboratory examinations were within normal limits.

The patient had delivered a baby two months before her present illness but had been entirely symptom-free during this two month interval.

At 9:00 p.m. on December 27, 1946, a stellate block was done with 3 cc. of 2 per cent metycaine and after a typical Horner's syndrome appeared, 1 cc. of a long-acting anesthetic agent in oil was injected. After an interval of five minutes, the patient experienced marked relief of chest pain and was immediately able to breathe more freely and deeply. She no longer appeared anxious. She was given 1½ grain of nembutal and 1 grain of luminal and slept soundly all night. The next morning, December 28, 1946, she was completely comfortable at rest. Coughing, yawning or deep exaggerated inspirations, however, would precipitate a mild twinge of pain over the right lower chest. On the afternoon of this same date, under spinal anesthesia, the femoral veins were ligated for it was thought that the source of the embolus was from the veins of the lower legs. The patient has remained completely free from pain and is making an uneventful recovery.

Occlusion of a pulmonary artery initiates a chain of events that is well known. The resultant severe chest pain, dyspnea, orthopnea, cyanosis and shock may cause sudden death. The same clinical picture may occur, however, when a moderate-sized branch of the pulmonary artery is occluded or when multiple small emboli occlude the lesser branches of the pulmonary artery. The pathologic physiology explaining the mechanism of the pulmonary embolus syndrome is not known. Certain salient facts are of interest in attempting to understand these mechanisms. Surgical ligation of the pulmonary artery does not produce chest pain, dyspnea, orthopnea, cyanosis, shock, or death. Therefore, factors other than the embolus per se must come into play in order to explain the severe symptoms associated with a pulmonary infarction. One, then, can only speculate as to the real cause or chain of events that leads to death in these patients. The possible physiologic patterns in this mechanism may be as follows:

1. The fact that pain impulses are transmitted by way of the sympathetic nervous system has recently been accepted by many as a definite entity. It is probable that the pulmonary embolus causes reflex vasospasm (3) of the pulmonary vessels of not only the lung affected but of the vessels of the opposite lung as well. This spasm initiates painful impulses that are mediated by the sympathetic fibers innervating these vessels. These painful impulses plus vasospasm
could account for the severe chest pain, dyspnea, orthopnea, chest splinting and resultant shock seen in patients having a severe pulmonary embolic syndrome.

2. In addition to reflex vascular spasm of the pulmonary vessels, there may also be a regional sympathetic spasm of the coronary vessels as well, regardless of the pulmonary vessel involved, whether it be right or left. This premise would also explain the bilateral chest pain that may occur and the frequent cardiac irregularities found in many of these cases.

It is hoped that in the future we will be able to form some definite answer to the true mechanism involved in this problem of explaining the cause of the pulmonary embolus syndrome. We hope to do this by means of venous pressures, electrocardiograms, serial blood pressures, serial roentgenograms, and chest measurements of patients so stricken.

The blocking of the stellate ganglion interrupts the painful irritative impulses arising from the sympathetic nerves innervating the pulmonary vessels. This apparently breaks up the vicious cycle (pulmonary and/or coronary vascular spasm phenomena), by blocking the painful nerve impulses as stated and permits vasodilatation of the pulmonary vessels. There is immediate relief of pain, dyspnea, orthopnea, and cyanosis; a greater chest expansion results and the state of shock is apparently reversed. So far as we know, the caliber of the bronchioles is unchanged by stellate block therapy. Repetition of the block is indicated if the cycle recurs.

**Summary**

The anatomy, surgical and anesthetic approach, case reports, pathologic physiology and method of stellate block, with their possible complications, have been mentioned in relation to the treatment of pulmonary embolism. The possible mechanisms explaining the pulmonary embolus syndrome have been presented. This syndrome has been attacked through interruption of the sympathetic nervous system by the blocking of the stellate ganglion. This treatment obfuscates painful impulses transmitted by the sympathetic nerves accompanying the pulmonary vessels and also results in vasodilatation of these same vessels. Clinically, there is immediate relief of pain, dyspnea, orthopnea, and cyanosis. Apprehension and anxiety are almost entirely relieved and there seems to be a reversal of the shock picture that usually plays a part in the pulmonary embolus syndrome. It is thought that this treatment should be used in patients who have severe pulmonary infarction but it should be used early in an attempt to bring about a favorable outcome. Certainly, the breaking of a cycle that produces profound pain and shock may alter our morbidity and mortality in these cases.
CONCLUSION

A stellate block was done on three occasions in two patients who had episodes of pulmonary emboli, with dramatic and almost immediate relief. The first patient obtained relief of bilateral chest pain on two separate occasions although the stellate block was done only on the side of the infarction. He also had relief of severe dyspnea and orthopnea and the block therapy probably reversed the state of shock. The second patient was not in extremis, as was the first, but she, too, experienced relief of pain, chest constriction, and dyspnea that was not afforded her by opiates. It is thought that no dogmatic conclusions can be drawn from the few patients treated by stellate block therapy, but its use is recommended in patients who have the syndrome of severe pulmonary embolus. The treatment is advocated for the acute episode. Caution ordinarily observed in elective stellate block procedures should not delay the immediate performance of the block in these patients. A sterile tray for the procedure should be available at all times.

REFERENCES


The following officers of the Section of Anesthesia of the New York State Medical Society were elected for the coming year:

Chairman: Dr. Rose M. Lenahan, Buffalo, N. Y.
Vice-Chairman: Dr. Harold F. Bishop, Valhalla, N. Y.
Secretary: Dr. Paul M. Wood, New York City, N. Y.
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