Temporary "Spontaneous" Paralysis of the Diaphragm*

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The published articles on spontaneous paralysis of the diaphragm, particularly in the English literature, are few indeed. This is remarkable because the condition may, at times, give rise to manifestations every bit as dramatic as coronary occlusion. Frequently, however, the symptoms are minimal or absent altogether.

Unilateral elevations of the diaphragm may be a normal finding. In most persons the hemidiaphragms are not at the same level and their relative position may change with normal functional processes, e.g. food intake, etc. If the upward displacement is marked and accompanied by impairment of function, it is due to one of the following causes:

1) It may simply be an upward displacement by "push" or "pull," i.e. either by retracting intrathoracic processes or by space-consuming intra-abdominal conditions. These displacements, as a rule, are not as marked as those caused by the other conditions listed, and the pathognomonic mechanism is self-evident.

2) Affections of the muscle itself will produce an elevation of the diaphragm. Attention was drawn to this myositis by Joannides, in 1946, who first described a primary diaphragmitis which he named "Hedbloom's Syndrome," and reported 12 cases presenting this symptom-complex. Another 12 cases were reported by Meyler and Huizinga in 1950, and in their patients the involvement was secondary to inflammatory processes in the abdomen and thorax.

3) Most frequently, elevations of the diaphragm are caused by lesions of the phrenic nerve. There is abundant literature on the anatomy and physiology of phrenic innervation, and the subject has been studied extensively since Schroeder described the effects of artificial interruption of the nerve in 1902. Curiously enough, up to Schroeder's investigations, paralysis of the phrenic nerve was considered a rare and exceedingly serious, if not fatal, condition. The few cases which were reported leave ample doubt as to the correctness of the diagnosis. The dismal outlook attributed to the disease was due to the fact that it was usually a complication of a serious primary affliction.

In recent times most phrenic nerve paralyses are due to mechanical interruptions, either surgical or traumatic. Therapeutic severance of the nerve is still a valuable procedure in the treatment of tuberculosis and accounts for the overwhelming majority of cases encountered. Uninten-

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tional damage to the phrenic nerve during operative procedures around the neck and in the thorax also furnished a fair number of cases.

Traumatic paralyses of the phrenic nerve do not appear to be as rare as was thought. Frequently they are due to birth-trauma and are accompanied by paralysis of the brachial plexus (Erb’s palsy).\(^{4-6}\) They would, without questions, be discovered more frequently if every newborn with brachial paralysis were x-rayed to ascertain the motility of the diaphragm. The obvious paralysis of the extremity in conjunction with the relative absence of symptoms in one-sided diaphragmatic paralysis commonly accounts for the phrenic lesion being overlooked. In rare instances, a lesion of the phrenic nerve will be found where there is no damage to the brachial plexus,\(^{7-9}\) and occasionally both nervi phrenici may be involved.

While there are ample descriptions of these surgical-traumatic lesions “Medical,” or as it is commonly called “Spontaneous” diaphragmatic paralysis is mentioned only in a very few investigations and case reports, and most of these are in foreign medical writings.\(^{10-19}\)

The phrenic nerve contains motor and sensory fibers in the proportion of about two to one.\(^{20}\) It arises chiefly from the fourth cervical nerve, but receives a branch from the third and another from the fifth. It descends across the front of the Scalenus anterior and beneath the Sternocleidomastoides and passes in front of the first part of the subclavian artery, between it and the subcavian vein. Within the thorax, it descends nearly vertically in front of the root of the lung, and then between the pericardium and the mediastinal pleura to the diaphragm. The right nerve is situated more deeply, and is shorter and more vertical in direction than the left; it lies to the right of the innominate vein and superior vena cava. The left nerve is rather longer than the right, from the inclination of the heart to the left side, and from the diaphragm being lower on this than on the right side. In the superior mediastinal cavity it lies between the left common carotid and left subclavian arteries, and crosses superficially to the vagus on the left side of the arch of the aorta.\(^{21}\)

A paralysis of the nerve may be due:

1. To destruction of the nuclei.
2. “Neurotoxic” infections or poisons.
3. Encroachment on the nerve itself during its course, either by invasion or by compression.

The symptoms of phrenic paralysis may be completely absent, especially if the encroachment is slow or if the lesions are central. In those cases a paralyzed diaphragm will not be detected unless x-ray examination is made. This may account for the relatively large number of cases described in pulmonary tuberculosis, where routine radiography has been done for many years. In other cases where the paralysis is secondary, the symptoms, though present, are submerged in the generally much more impressive primary disease, and are apt to be overlooked by doctor and patient. If symptoms are manifested they tend to be fairly characteristic.

There will be a more or less sudden pain in the shoulder on the affected side. This pain occasionally may resemble the “anginal” pain-distribution,
though it is usually more localized at the tip of the shoulder on the affected side, along the upper border of the trapezius muscle and in the supraclavicular area. At the same time, an internal pain may be described anywhere within the affected side of the chest, and there is shortness of breath which is aggravated by acute anxiety.20

If paralysis of the hemidiaphragm is suspected, confirmation may be easily obtained by fluoroscopic examination. The involved diaphragm is elevated and immovable. These findings, however, are not sufficient for a diagnosis of interrupted diaphragmatic innervation since they may also be seen in diaphragmitis, subphrenic abscesses and other conditions. More pathognomonic are the so-called “paradoxical movements,” i.e. opposite movements to those of the healthy side with deep respiration and still easier detected with sudden changes of intrathoracic pressure as in coughing and snifﬁng.

Infectious diseases may cause phrenic paralysis either by invasion of the central nervous system (e.g. poliomyelitis14) or by the production of neurotoxins (e.g. diphtheria11). Similarly, numerous organic and inorganic16 nerve-toxins have been described as causing interruption of diaphragmatic innervation. Most commonly, however, the nerve is compressed, invaded or destroyed in its course through the thoracic cavity by neoplastic10,14,18,19 in inﬂammatory processes.11,15,17,18 An unexplained paralyzed phrenic nerve, today, is most likely to be considered evidence of intrathoracic malignancy, although until only a few years ago it would have been most frequently thought to be due to tuberculosis. This was probably due to the fact that patients with tuberculosis were more frequently and regularly examined with x-rays than any other group of people. The best study of phrenic paralysis in tuberculosis was written by Gimeno Ondovilla,17 who published his series of ﬁndings. Most of his patients, like most of the other published reports, had permanent paralyses, although one showed a “potentially reversible process,” i.e. if this patient had lived, function might have reappeared. Temporary, “spontaneous” phrenic paralysis is extremely rare and it appears worthwhile reporting a case observed in this hospital.

Case Report

H.B., a 55 year old white male, who was well until the beginning of 1947, when he complained of anorexia, weight loss and unproductive, hacking cough. He was admitted to the Veterans Administration Hospital, Oakland, California, in March 1947, and pulmonary tuberculosis was diagnosed on the basis of acid fast bacilli in his sputa and lesions in both apices and the right infraclavicular region seen on x-ray ﬁlms. These were read as exudative lesions and the lordotic view showed what appeared to be a small cavity in the left apex. He was transferred to Veterans Administration Hospital, Livermore, California, in September 1947. Pneumoperitoneum was induced in October 1947, and he was discharged to out-patient treatment, his pneumoperitoneum being refilled once a week. In July 1948, his sputa had remained negative for acid fast bacilli for more than six months. The x-ray ﬁlm ﬁndings at that time were interpreted as bilateral, ﬁbroid minimal disease. From June 1948, until February 1949, he felt well, had no complaints and led a moderately active life. Intensive questioning failed to reveal any exposure to
neuro-toxic substances of any sort. On February 5, 1949, he returned for a regular refill of his pneumoperitoneum, and at this visit he stated that for the preceding several days he had noticed slight shortness of breath. He attributed this shortness of breath to excessive air in the abdominal cavity and requested that less air be given. This request was complied with. Two days later he developed a sudden sharp pain in the left chest. The pain was localized in the intra-scapular area and did not show any radiation. It occurred around three o'clock in the morning and woke him from a sound sleep. It was aggravated by respiratory movements and increased upon deep inspiration. It slowly and gradually subsided over the next 48 hours. On February 21, he was well on awakening and started to get up to go to the bathroom when he suddenly felt a severe pain, again in his left chest, and a marked shortness of breath. He returned to bed as quickly as he could and his symptoms eased somewhat, but when he tried to get up, the pain recurred. He consulted a local physician who advised immediate hospitalization. The patient was admitted around noon of the same day. Upon admission the patient appeared to be in rather acute distress, although the only significant finding was a tachycardia of 118, with a normal sinus rhythm. Examination of the lungs showed only some minimal change of breath sounds in both apices. Extensive work-up showed essentially normal laboratory findings save for a slightly elevated sedimentation rate (21 mm./hr.), and acid fast bacilli in several sputum specimens examined. An electrocardiogram was normal. Posterior-anterior x-ray films of the chest were interpreted as showing fibrotic and infiltrative lesions in both apices without any cavity being found. Pneumoperitoneum, displacing both diaphragmatic leaves upward about equally, was present. The patient was put on bedrest and no specific therapy was instituted. He appeared to be improving, had no complaints. No further refills of his pneumoperitoneum were given.

By the middle of March the patient felt well and was semi-ambulatory. Repeated follow-up examinations by physical, fluoroscopic and electrocardiographic means did not produce any new findings.

On March 23, 1949, the patient developed a low grade temperature between 99.6 and 101.0 degrees F. There were no localized complaints nor any new findings. On March 28, around 10:30 p.m., while resting quietly in bed, the patient suddenly developed a severe pain in the right side which was localized anteriorly and radiated into the region of the right shoulder tip, the lateral aspect of the supraclavicular and trapezius regions. This pain was accompanied by severe dyspnea, grunting, and polypnea of 43 respirations per minute. His face was flushed, his pulse rapid (125 per minute), and he appeared apprehensive and fretful. Fluoroscopic examination of his chest was done immediately and revealed pneumoperitoneum to be still present though no refill had been given for six weeks prior to this episode, the right diaphragm being elevated four centimeters higher than the left one. No active movement of the right diaphragm was visible but paradoxical movements could be observed on sniffing. His temperature went to above 103 degrees F. orally and the patient was obviously and severely ill. He was put to bed and treated with sedation and intermittent oxygen. During the following four days his temperature remained elevated. Physical condition and fluoroscopic and roentgenographic findings remained unchanged. His sputa became slightly blood tinged and upon physical examination there was a decided lag of respiration on the right side with decreased resonance especially over the lower part. Numerous moist rales and areas of bronchial breathing in the paravertebral region at the base were found. All these symptoms slowly and gradually subsided within one week. Sputum examination for malignant cells during that time gave negative results. After the acute symptoms subsided bronchoscopy was performed. A normal bronchial tree was found on the left side. At the level of the orifice of the right upper lobe several irregular cartilage-hard nodules were seen protruding into the lumen from the lateral wall. Similar, but less marked, changes were seen on the wall slightly below the orifice of the upper lobe. Several small biopsies were taken
from these regions because on bronchoscopic examination they suggested carcinoma. However, pathological examination of these bite-specimens revealed only inflammatory non-specific lesions. The mucous membrane was intact and only small round cells were seen in moderate numbers. The patient continued to improve subjectively, and objectively the findings of compression in the lower lobe had disappeared by the 15th of April. On April 20, the patient was fluoroscoped again and a normally movable diaphragm was found. This was confirmed by a roentgenogram. From then on there was a steady improvement of all subjective and objective signs and symptoms. The patient remained well; there were no acid fast bacilli in his sputa. The x-ray films remained unchanged and during a two year observation period no recurrence of the paralysis was discovered. The patient was discharged from hospital care in January 1950, but now (September 1951), is still under out-patient observation at this hospital.

SUMMARY

This patient with an established diagnosis of pulmonary tuberculosis first developed what in retrospect appears to have been attacks of bilateral phrenic neuritis, and then phrenic paralysis, lasting less than three weeks. Although it is impossible to prove a definite diagnosis, the pre-existing tuberculosis and the absence of other significant findings, together with the bronchoscopic observations suggest a tuberculous process, probably of the lymph-nodes compressing the phrenic nerve, as the most likely cause.

A review of the causes of unilateral diaphragmatic elevation is presented in connection with this report.

RESUMEN

Este enfermo con un diagnóstico establecido de tuberculosis pulmonar presentó lo que retrospectivamente parece haber sido ataque bilateral de neuritis frénica y después parálisis frénica, con menos de tres semanas de duración. Aunque es imposible demostrar un diagnóstico preciso, la tuberculosis preexistente y la ausencia de otros signos importantes, así como la observación broncoscópica, sugieren que se trataba de un proceso tuberculoso y que la causa más probable fue la compresión producida por los ganglios linfáticos.

Una revisión de las causas de elevación diafragmática unilateral se presenta en relación con este trabajo.

RESUME

Il s'agit d'un malade atteint de tuberculose pulmonaire. Il présente ultérieurement des manifestations qui, rétrospectivement, semblent avoir été une atteinte de névrite bilatérale du nerf phrénique, suivie de paralysie phrénique ayant duré moins de trois semaines. Il est impossible dans ce cas d'apporter un diagnostic certain; cependant l'auteur pense qu'il s'agit d'un processus tuberculeux probablement ganglionnaire, ayant comprimé le nerf phrénique. En faveur de cette hypothèse, il note la tuberculose pré-existante, l'absence de toute autre manifestation pathologique et les consta-
tations bronchoscopiques.

Un exposé des causes habituelles de l'élévation unilatérale du diaphragme est présenté à l'occasion de cette communication.
REFERENCES