as in this case. I think that the most marked mental changes which occur in cases of brain tumour occur in those in which the lesion is in the temporal lobe. In regard to Dr. Elvidge's question, the third phenomenon in the attacks of this patient was salivation and the fourth micropsia. One presumes that the spread was backward toward the visual association centres, and that therefore salivation resulted from a discharge in the brain posterior to the tumour. Although it is difficult to know the mode of advance of symptoms we seem to have evidence sometimes of a linear spread rather than a circular one.

BRONCHIAL STENOSIS IN PULMONARY TUBERCULOSIS

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BRONCHIAL stenosis complicating pulmonary tuberculosis and producing asthma-like symptoms is comparatively uncommon and even more rarely recognized. Eloesser[1] has recently called the attention of phthisiologists to the condition. McConkey[3] also has reported a case, and others are to be found in the literature. The two cases included here were recently observed on the Medical Services of the Saskatchewan Anti-Tuberculosis League.

Case 1

Mrs. P.W., stenographer, aged 24 years, was admitted on April 17, 1928, with a tuberculous lesion involving the middle two-thirds and the hilum of the left lung. She complained of cough, slight expectoration, and pain in the left chest. There was slight shortness of breath. The onset apparently had been three months previously with a "cold". She had had measles, mumps and scarlet fever; also influenza (1918) at 14. Adenoids had been removed at 15 years and the appendix at 18 years. There had been no asthmatic attacks. The family history was negative for tuberculosis and asthma.

On admission, physical examination revealed slight dullness over the left side of the chest and crepitations were heard over the same area. X-ray showed mottling about the root, extending outward and involving the middle two-thirds of the lung. There was a small area of fine, discrete densities at the tip of the second rib on the right side, which was interpreted as a healed lesion. Her temperature was normal. There was no difficulty in breathing or huskiness of voice. Tubercle bacilli were found in the sputum. The Wassermann reaction was negative. Left pneumothorax was instituted at once, and an excellent collapse obtained, although there was an adhesion in the apex. In June slight effusion developed, which subsided in September. Pneumothorax was continued. Fluid reappeared in September, 1932, which never entirely disappeared.

In January, 1932, examination by our oto-laryngologist was reported as follows: "Nose and sinuses normal except for excessive secretion from turbinates. Tonsils fairly healthy. Larynx normal." In May the patient was well enough to be allowed home on ten days' leave. She had no cough, and only one or two drachms of sputum daily (negative for several months). Slight shortness of breath on exertion was noticed at this time.

In April, 1933, the patient appeared to have come to a standstill and was very discouraged. The sputum was again positive. It was thought that after five years of such complete collapse the lung would never expand and that thoracoplasty would have to be performed eventually. Phrenicectomy was done as a preliminary measure. She had a rather severe gastric upset following the operation. In July the first complaint of shortness of breath and "wheezing" was entered on the charts. This was accompanied by pain in the right side. The second attack, lasting several days, was in August. The difficulty in respiration was mostly inspiratory. Adrenalin gave some relief, also ephedrine, which was used after the first day.

These attacks became more frequent and distressing. Three were rather severe ones in January, March, and April of 1934, the last continuing for almost the entire month. During attacks there was both inspiratory and expiratory stridor, the former predominating. The attacks usually terminated by the expulsion of considerable thick tenacious mucus. Between attacks she was comparatively comfortable, except for the "wheezing" respiration. There was a definite inspiratory stridor and expiration was prolonged. Induced alteration of the intra-thoracic pressures, either up or down from her usual levels, had no apparent effect. This was tried once during an attack with a similar lack of result. Atropine now gave more relief than anything else. Expectorant mixtures were tried, which noticeably facilitated expectoration. No attempt was made to desensitize with tuberculin. During the summer she became much worse, and died, August 17th, during a suffocative attack, in which she was cyanosed. The terminal event was a huge inspiration with inability to exhale. Artificial respiration was of no avail.

An autopsy was performed by Dr. G. R. Townsend, who reported as follows.

"On opening the thorax the heart was found to be retracted to the left. The left parietal pleura was greatly thickened. On cutting through it into the pleural space the left lung was found to be completely collapsed against the mediastinum and covered with thickened visceral pleura. There was a dense pleural adhesion from the apex of the lung to the upper part of the thoracic cage. The free pleural space contained about 100 c.c. of clear fluid. The right lung was large, voluminous and airy, and extended well over the midline. The thoracic organs were removed *en bloc*.

"On dissecting down the trachea to its bifurcation a small amount of muco-pus was exposed lying in the lumen. Just above the bifurcation was an area of constriction, the lumen being about 1/12th of an inch in diameter. The lumen of the right main bronchus was found to be completely occluded by thick tenacious muco-pus which extended into the lumen of the three lobar bronchi and down into their divisions. In the wall of the right main bronchus were several areas of ulceration and caseation necrosis, one triangular in shape, measuring 1 and ¼ of an inch in length and

¼ of an inch across the base. The basal bronchi were dilated to some extent and filled with the same muco-pus. On cutting into the right lower lobe, muco-pus and frothy fluid exuded from the bronchi. Two or three circumscribed areas of caseation necrosis were exposed. The middle lobe on section appeared relatively healthy. In the upper part of the upper lobe on section there was exposed a large circumscribed area of caseation necrosis the size of a tangerine orange. The left main bronchus was greatly thickened and its lumen narrowed to the size of a match. This was, too, completely occluded by muco-pus. On opening into the collapsed left lung it was found to be nothing but a sac of thickened pleura containing necrotic tissue and this same muco-pus. The left diaphragm was found to be thinned out in its central portion and to contain mostly fibrous tissue enclosed by serous membrane. Peripherally, there were some areas of muscle tissue, but these, grossly, had a degenerative appearance."

Histological sections removed from the bifurcation of trachea and right main bronchus disclosed "Tuberculosis, showing giant cells, caseation, necrosis, etc."

CASE 2

Miss E.H., stenographer, aged 27 years, was first admitted on May 30, 1932, following the discovery of a minimal tuberculous lesion in the right apex. On May 13th, the left posterior cervical glands had been removed and found to be tuberculous. She had not been feeling well and had tired easily for about a year prior to this.

She had had small-pox, measles, mumps, scarlet fever and chicken-pox before her eighth year, and whooping-cough at ten years. There had been no asthmatic attacks. There was an indefinite history of exposure in 1928 to a boarder in her home who was later reported to have tuberculosis. The family history was negative for tuberculosis and asthma.

On admission, physical examination revealed only roughened breath sounds over the right apex. X-ray showed slight mottling above the clavicle, in the first two interspaces, and about the hilum on this side. Her temperature was normal. There was no difficulty in breathing or huskiness of voice. No tubercle bacilli were found in the sputum, which was very slight in amount, and the Wassermann reaction was negative. In July she was examined by an oto-laryngologist, following complaints of nasal obstruction with posterior discharge and a "lump" in the throat in the region of the larynx. The nose was reported as free from abnormality, but excessive secretion present. The lump in the throat was put down as "globus hystericus". The vocal cords and the interior of the larynx were normal. Her general condition improved, her weight increased from 102 to 107 lbs., and she was discharged in October of the same year to continue treatment at home. X-ray now showed the apical shadows to be discrete and suggesting considerable fibrosis.

On November 9th the patient reappeared at our clinic complaining of persistent cough and "wheezes and clicks", particularly on her right side. She was feeling well otherwise. Examination revealed rhonchi on both sides, but more on the right. X-ray showed no change from the condition on discharge. She was observed frequently after this. Cough and slight expectoration persisted. Her weight remained the same and she felt well. Crepitations were heard later over the right apex, and she was readmitted in January, 1933. The sputum was now repeatedly positive; the temperature, 99°. Examination on April 20th revealed well marked bronchial breathing over the right apex with numerous crepitations. X-ray showed a dense even haze above the right clavicle which had a sharply defined lower border, concave downward and curving to the hilum. This was interpreted as atelectasis involving at least part of the upper lobe and masking the areas of disease. No mottling was visible elsewhere. At this time there was no complaint of difficulty in breathing, but there were periodic attacks of "wheezing", suggestive but not typical of asthma. No huskiness of voice was noticed. She had had several attacks of right-sided pain around and below the clavicle, lasting two to three days. These were not accompanied by any constant symptoms and ended suddenly. In August some huskiness of voice was noticed. Laryngeal examination showed the cords to be normal, but the arytenoids were somewhat injected. There was no ulceration. Physical examination now revealed only roughened breath sounds over the right apex. X-ray showed clearing of the dense haze referred to, with only some hazy bands extending up from the hilum to the medial part of the apex.

X-ray examination in January, 1934, revealed recurrence of the atelectasis and mottling about the right root. Since the previous examination she had had two asthma-like attacks with laryngitis, and considerable bronchitis and difficulty in breathing. These were relieved by inhalations. Right pneumothorax was instituted at this time. It resulted in increase in the "asthmatic" symptoms and difficulty in breathing. Inhalations continued to give some relief. Films showed fair collapse, with a few adhesions in the apex. The dense haze in the apex had disappeared and the area was now comparatively clear. There was some new mottling near the periphery, opposite the hilum. The attacks of difficulty in breathing became more frequent, distressing and prolonged. Adrenalin did not relieve them. Inspiration was most markedly prolonged, and was rasping and difficult. In view of this pneumothorax was discontinued in June. Examination again by an oto-laryngologist revealed the cords to be normal in appearance and movements. Tracheal obstruction was looked for, but could not be seen.

Ephedrin was tried in the attacks and gave some relief. Atropine was tried on one occasion, with no apparent benefit. When the patient complained of inability to raise sputum she felt to be present an expectorant mixture was tried cautiously. It apparently facilitated expectoration a little. The difficulty in respiration became progressively worse, while ephedrin continued to give slight relief. In October her condition became serious. Breathing was very laboured and the accessory muscles of respiration came into play. Inspiration was extremely difficult and expiration somewhat rasping. She was obviously going down hill.

The patient died, October 29th, apparently of suffocation. The terminal event was inability to inhale. Just before death the respiratory sounds were markedly diminished over the right, and this side of the chest appeared fuller than the other. A post-mortem film showed absence of pneumothorax, mottling on the right in the first interspace, more dense in the second and third, and, again, about the root and along the lower vertebral trunks. There was a small localized area of mottling beneath the first rib on the left. There was no evidence of atelectasis.

Permission for a partial autopsy was obtained. On opening the chest, the right lung appeared enlarged and was adherent to the chest wall by numerous fibrinous adhesions and a few fibrous ones in the apex. The middle lobe was markedly enlarged and lighter in colour than the other two. The left was free, except for one fibrinous adhesion in the apex. There were no enlarged tracheo-bronchial glands. The trachea, heart and lungs were removed in one piece and the bronchial tree dissected from above.

The lining of the trachea was rough and corrugated in vertical folds. The cartilage rings could not be seen from the inside. Just above the bifurcation definite ulceration was present, involving the entire inner circumference, with constriction of the walls so that the lumen was not more than ⅛th of an inch in diameter. This ulceration continued into both main bronchi. Along the right there was a second constriction with the lumen even smaller. The right upper lobe bronchus was involved in

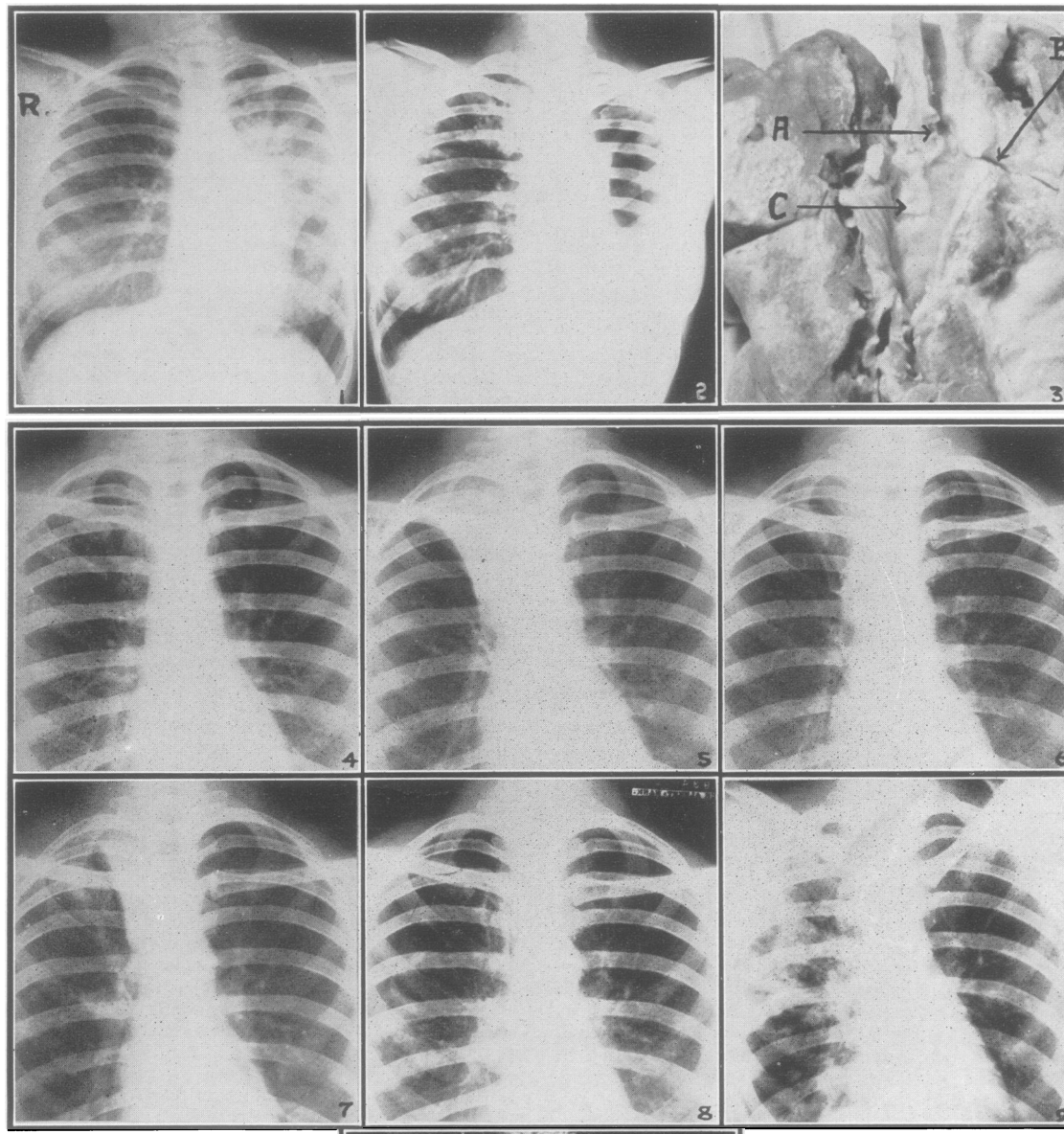

FIG. 1.—*Case 1.* On admission, April, 1928.

FIG. 2.—*Case 1.* May, 1933. Hydro-pneumothorax. None of the many films showed any significant difference from this.

FIG. 3.—*Case 1.* Hardened specimen. A—Stenosis of trachea (block removed for microscopic examination). B—Stenosis of left main bronchus. C—Area of ulceration and caseation necrosis in wall of right main bronchus.

FIG. 4.—*Case 2.* On admission, May, 1932.

FIG. 5.—*Case 2.* April, 1933. Atelectasis of right upper lobe.

FIG. 6.—*Case 2.* August, 1933. Almost complete aëration of atelectatic lobe.

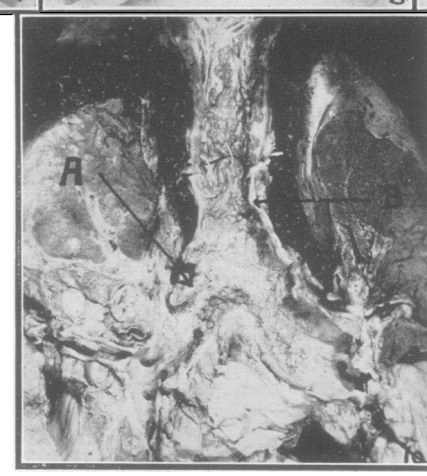

FIG. 7.—*Case 2.* January, 1934. Recurrence of atelectasis.

FIG. 8.—*Case 2.* July, 1934. Pneumothorax established. Atelectatic lobe completely aërated.

FIG. 9.—*Case 2.* Post-mortem film, August, 1934. Spread throughout right lung.

FIG. 10.—*Case 2.* Hardened specimen. A—Stenosis of right main bronchus. B—Stenosis of trachea. Lumen at both constrictions does not appear as small as was actually the case. The area of ulceration extends downwards from the tracheal constriction, across the left main bronchus, down the right, and into the right upper lobe bronchus.

a similar ulcerative process. The lower bronchi on both sides were occluded with strands of ropy purulent mucus. Pea-sized and smaller tubercles were present in the upper part of the right upper lobe, in the tip of the middle lobe, and in the posterior lower part of the lower lobe. This lower tip was atelectatic. Some of these tubercles were caseous. There was also a large tubercle in the left apex near the lateral wall.

Histological examination confirmed the diagnosis of tuberculous ulceration of trachea and main bronchi. A specimen from the distended, light-coloured, middle lobe was emphysematous.

Eloesser's[1, 2] thorough clinical, radiological, bronchoscopic and pathological studies have thrown a great deal of light on these obstructive conditions occurring in tuberculosis. He follows the classification of v. Schroetter in grouping them as intra-mural, mural and extra-mural.

The intra-mural form is the rarest. It may occur in children when a peri-bronchial or peri-tracheal lymph node may suppurate and ulcerate through the bronchial wall, part of the node sloughing into the lumen. Mural processes are more common. They may be *ulcerative*, a caseous ulcer; *hyperplastic*, a submucous infiltrate, producing sufficient thickening to obstruct the lumen, or submucous tuberculous granulomata with hyperplasia of the submucous lymph follicles; and *shrinking*, in which fibrous scars may kink and distort the bronchi sufficiently to cause obstruction. Extra-mural processes include enlarged hilar lymph nodes and peri-bronchial sclerosis. The latter condition is also seen in syphilis. A more or less diffuse sclerosis of the smaller bronchi and bronchioles is also included.

Eloesser describes in detail tuberculous cases of various types. Two were of obstruction to the trachea. One was in an adult in whom the x-ray revealed a constriction about the middle of the trachea, with dilatation above and below. Autopsy revealed tuberculous ulceration (bacilli found) with much fibrous tissue at the site of stricture. There was emphysema of one lung and atelectasis of the other, with no evidence of pulmonary tuberculosis. The other case was in a child in whom a large solitary tubercle obstructed the trachea just below the thyroid cartilage.

Unilateral obstruction of a main bronchus or a single lobe bronchus occurs more frequently in tuberculosis. Eloesser gives particulars of several cases in which the obstructing stenosis was seen with the bronchoscope. These cases have considerable similarity to the two reported here. In some of them more extensive atelectasis was present.

McConkey's[3] case had obstruction at the lower end of trachea due to a fibro-caseous ulcerative lesion implicating the cartilaginous rings. The right main bronchus and some of its major bronchi were almost occluded with caseous material. Smaller bronchi were also thickened and occluded. There had been attacks of so-called asthma and laryngitis, with loss of voice and pain on swallowing. The respiratory difficulty here was chiefly on expiration. Coryllos,[4] in an article on atelectasis, cites two cases (Nos. 3 and 5) of proved bronchial obstruction in tuberculosis. In one a fibrinous plug was found at autopsy obstructing the right main bronchus; in the other, a cicatricial stenosis of a lower lobe bronchus was found bronchoscopically. He does not mention, however, any accompanying disturbance of respiration. Myerson[5] reports a case of pulmonary abscess with tuberculous thickening of the mucosa and ulceration of the bifurcation and right main bronchus, diagnosed by bronchoscopic biopsy. The patient had an attack of dyspnœa, inability to cough, chest pain, and a chill the night before admission. Packard[6] describes a case of old fibroid tuberculosis which came to autopsy in which obstruction was due to the main bronchus being "thickened and its lumen greatly compressed, its mucosal surface redundant and deeply injected". There had been an attack of dyspnœa, a feeling of weight and constriction, and productive cough, changed to non-productive two years before death. General health had been "fairly good" in the interval, with the massive collapse persisting.

The symptoms and findings in tuberculous obstruction are in general the same as in that due to other causes. In partial obstruction of the trachea or bifurcation affecting both bronchi simultaneously there is often extreme dyspnœa with a loud inspiratory stridor. The patient may complain of extreme suffocation. The trachea moves up and down with respiration. The chest tends to be barrel-shaped in spite of the retracted intercostal spaces. Complete obstruction of a main brochus produces less severe dyspnœa. It is not usually paroxysmal. There is often pain or a pulling sensation on the affected side. There may be no symptoms after the condition is established. Examination reveals contraction and immobility of the affected side, dullness, absence or marked diminution of the breath sounds, and shifting of the trachea and heart toward the side of the obstruction. The diaphragm is raised and relatively immobile.

Partial obstruction usually results in dilatation and emphysema of the affected side with the characteristic findings. Intermittent obstruction may be very puzzling, as the findings of emphysema and of collapse may reverse themselves from time to time. Eloesser states that these intermittent obstructions of a main bronchus are more often non-tuberculous. Coryllos[4] writes that partial obstruction causing emphysema is frequent in tuberculosis and sometimes "gives rise to true asthmatic attacks". Stenosis of a single lobe bronchus, producing atelectasis, may cause no symptoms of itself. Localized retraction and immobility may be observed. Dullness may sometimes be elicited. Absence or diminution of breath sounds is not constant. The x-ray is more definite, as the picture of an atelectatic lobe is characteristic. The radiological appearance of atelectasis of the different lobes is quite definite and well known. That of an upper lobe is described and shown in our Case 2. Collapse of the middle lobe gives a dense, sharply defined shadow, roughly triangular in shape, with the base of the triangle resting on the right border of the heart and the

apex toward the chest wall. The upper side is usually horizontal. Atelectasis of a lower lobe gives a similar dense shadow filling the cardiophrenic angle and the upper border of which extends from the hilum to the costo-phrenic angle or to the chest wall a short distance higher up. In addition, depending on the lobe involved, the trachea may be drawn over but not the heart, and *vice versa*.

In obstructions below the bifurcation in tuberculosis the clinical picture usually has added to it the toxicity resulting from damming back of purulent secretions. Intermittency of symptoms was characteristic of our two cases. The stenosis is probably constant and the intermittency of symptoms due to recurrent blocking of the narrowed lumen with caseous debris or thick mucus. Intermittency of the atelectasis was noted in our Case 2. It had no relation to the dyspnœic exacerbations. After the first x-ray demonstrations of atelectasis a film was secured showing almost complete aeration of the affected lobe. Later, atelectasis was definite again, only to disappear after the induction of pneumothorax. The disappearance of atelectasis after pneumothorax in this case was probably due to "unkinking" of the affected bronchus.

Eloesser states that the diffuse sclerosis of the smaller bronchi and bronchioles is not on such a firm foundation as the other types described. This type is sometimes seen in the tuberculosis grafted on the silicosis of quartz miners, and in others. There is no intermittency of the symptoms. It is a diffuse tuberculous emphysema, due, in Eloesser's opinion, to a diffuse stenosing bronchiolitis. The dyspnœa may be severe, but is inspiratory, in contradistinction to true asthma, and there is no stridor. The findings are those of emphysema; resonance is increased, breath sounds diminished, inspiration prolonged, and rhonchi and râles are heard throughout the chest. X-ray sometimes shows peri-hilar increase in density. These cases have been described as tuberculous asthma, due to tuberculous allergy. However, most students of allergy are of the opinion that an asthma due to sensitivity to tuberculo-proteins does not exist.* There may possibly be a direct toxic action on the bronchial musculature by the products of the tubercle bacillus.

Cases of pulmonary tuberculosis with asthmatic dyspnœa have been observed in which relief of symptoms has followed desensitizing treatment with tuberculin. However, this cannot be accepted as proof of their allergic nature, as numerous cases of asthma, some of them tuberculin-negative, have been treated with tuberculin with beneficial results.[7, 8]

Diagnosis of these obstructions to the large or smaller parts of the bronchial tree may be made on the clinical picture described. The onset of asthma-like "wheezing" may be the initial event in the developing train of symptoms. X-ray is of great help and injection of opaque oil may reveal the site of obstruction, but its use is not without risk in tuberculosis. In many cases the radiological findings are inconclusive, particularly regarding an upper lobe bronchus, and bronchoscopy remains the only way to confirm the diagnosis ante mortem. The lumen of the trachea, the bifurcation, the main bronchi and some distance along the single lobe bronchi, including those of the upper lobes, can be visualized by this procedure. Clerf[9] states that pulmonary tuberculosis is no bar to bronchoscopy when otherwise indicated.

Treatment of these stenotic complications is as yet mostly symptomatic. Adrenalin was no help in our cases. Ephedrin and atropine gave relief most consistently. Opiates to control cough were helpful. Oxygen by nasal catheter may be of some use. In cases of extreme cyanosis depletion by bleeding has been recommended. Heliotherapy and x-ray treatment are useful in reducing enlarged mediastinal glands. Expectorant mixtures, if used at all, should be tried with caution, as a suffocative attack might be precipitated. They were tried in both our cases with noticeable effect in one, and slight, if any, in the other. Eloesser states that in unilateral stenosis placing the patient on the good side in the hope of securing postural drainage is likely to provoke an attack, while putting the affected side at rest by placing the patient on it will often tide over an exacerbation.

Collapse therapy is of doubtful advantage. If it results in closure of a partially obstructed major bronchus with the damming back of septic secretions it will do harm. On the other hand, in the non-toxic patient whose obstruction is due

* Personal communication from Dr. R. M. Lichtenstein, of the Chicago Municipal Tuberculosis Sanitarium, and the Allergy Clinic, Rush Medical College, Chicago.

to sclerosis and shrinking, and where symptoms are mainly the result of the mechanical derangement of the thoracic organs, it may do good. In the case of obstruction to a single lobe bronchus collapse may also dam back purulent material or occasionally straighten sclerosed and kinked bronchi, clearing the obstruction. This latter probably occurred in our Case 2. Rest, mental and physical, and life in a warm, dry, equable climate without wind or dust, and at a low altitude is desirable. At present there is not much else to suggest for these patients. Bronchoscopy in the future may have something to offer. Removal of the caseous debris of an ulcerating gland and aspiration of the cavity may be done, and may be all that is necessary. The idea of repeated dilatation of the constrictions would not appear to hold much promise for permanent benefit,* while removal of debris and tenacious mucus should give temporary relief. Other methods of attack would seem as yet a subject for speculation and a challenge to medical ingenuity, but it is to be hoped that further study in our tuberculosis institutions will result in the development of new procedures and something useful in the way of treatment of these distressing complications.

From what has been presented it appears probable that many of the so-called "tuberculous asthmas" are in reality cases of complicating bronchial stenosis. And further, it would appear that bronchoscopy should have a place in the armamentarium of phthisiologists, as it already has in the hands of others specially interested in the various non-tuberculous disorders of the lungs or bronchi.

* Since this article was submitted for publication Werner (*Am. Rev. Tuberc.*, 1935, **31:** 44) has reported a case in which repeated bronchoscopic dilatation of the constriction was followed by considerable improvement.

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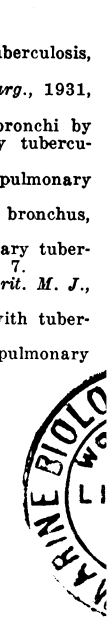

EARLY PROTECTED WEIGHT-BEARING IN THE TREATMENT OF FRACTURES OF THE FOOT, ANKLE AND LEG*

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IN the treatment of fractures of the long bones and of fractures about the joints certain *desiderata* are to be hoped for. In so far as possible, anatomical reposition of the bones should be accomplished. This necessitates, among other things, early reduction following the injury. In order that immediate interference may be carried out a well constituted fracture team is a necessity; in order that the procedure may be adequate, special training, including, *inter alia,* a knowledge of the mechanism responsible for the production of typical fractures, is imperative. Even more important, however, than replacement of the bone fragments in a normal position is the return to activity of the limb as a whole within as short a period as possible.

At the present time, among those surgeons who are particularly interested in the treatment of injuries of the extremities there is an argument with reference to the better method of permitting function, particularly in the case of the lower extremity. I believe I am correct in stating that the great majority of surgeons interested in fractures are of the opinion that the simple fixation of the injured limb in plaster of Paris, and the discharge of the patient from hospital, in order that he may move about with the help of crutches and with the limb dangling, is not followed by sufficiently favourable results to justify its employment as a method of choice. Of those who believe that early re-establishment of function is necessary, some, as for instance Robert H. Kennedy,[1] of the Beekman Street Hospital in New York City, advocate the employment of a fixation apparatus which is demount-

* From the Montreal General Hospital.