*Thorax* (1951), 6, 359

# THE ANATOMY AND APPLIED ANATOMY OF THE MEDIASTINAL FASCIA

BY

PAUL MARCHAND

*From the Thoracic Surgery Unit, Johannesburg Group of Hospitals*

(RECEIVED FOR PUBLICATION JULY 4, 1951)

It is common knowledge amongst thoracic surgeons that the pulmonary arteries and veins at the hilum of the lung are invested by fascial sheaths. The hilar bronchi, too, have tough fascial investments which are readily demonstrated during dissection pneumonectomy. Anatomical textbooks remain strangely silent upon the origin, and even upon the existence, of these fascial sheaths. *Gray's Anatomy* (1935) merely remarks that the fibrous pericardium is prolonged around the pulmonary arteries, " to be gradually lost upon the external coats of these vessels." It does not mention the existence of a definite sheath at the hilum of the lung related either to the pulmonary vessels or to the bronchus. It seems obvious that the fascial sheaths surrounding the pulmonary vessels are derived from the fibrous pericardium. There is no such ready explanation for the origin of the bronchial fascial sheath. The only possible source of this seems to be the pretracheal fascia, which is described as passing downwards from the neck into the superior mediastinum to fuse eventually with the fibrous pericardium. A review of the literature failed to provide any mention of lateral extensions of the pretracheal fascia around the bronchi.

In an effort to demonstrate the origin and distribution of these fascial planes 12 heart and lung specimens have been injected and dissected. The findings of this investigation, and a discussion of various clinical and pathological conditions in which these fascial planes appear to play a part, are included in this paper.

## MATERIALS AND TECHNIQUES

The technique adopted consists of injecting fluid under considerable pressure beneath a recognizable fascial layer so as to produce a space limited by the fascia under investigation. A distinctively coloured fluid was used so as to facilitate recognition whilst tracing the distribution of the fluid by dissection. The fluid used was rendered radio-opaque, and radiography of the injected specimens provided evidence of the spread and distribution of the injection mass.

Eight specimens of heart and lungs were removed from adults who had died from some extra-thoracic cause. Great care was exercised in separating the mediastinum from the vertebral bodies through a plane just anterior to the prevertebral fascia. The tongue, larynx, trachea, pharynx, and the whole oesophagus were removed as part of the specimen.

In five of these specimens two polythene catheters were introduced between the so-called pretracheal fascia and the trachea about half an inch below the thyroid isthmus. The fascia is readily recognizable in this position as a whitish membrane reflected off the thyroid gland. The polythene catheters were pushed down for a distance of about one inch on either side of the trachea. By means of a Y tube the catheters were connected to a canister containing a modification of Hill's bismuth oxychloride formula (Marchand, Gilroy, and Wilson, 1950). This suspension was diluted with an equal volume of saturated sodium iodide solution. The suspension is radio-opaque, and bright yellow. The lungs were fully inflated with air through the trachea. Regurgitation of the injection mass was prevented as far as possible by firmly tying the cervical tissues around the polythene catheters with braided silk. The canister was raised five feet above the level of the specimen, and left for 24 hours. The specimens were radiographed and finally dissected to determine the distribution of the yellow medium.

Three specimens were prepared by peribronchial and perivascular infiltration of the hilum with Hill's suspension, using a 20 ml. Record syringe and a wide-bore hypodermic needle. Before radiography the pulmonary vessels were perfused with physiological saline solution in order to remove any of the injection mass which might inadvertently have been introduced during the infiltration.

Four intact corpses were injected by introducing catheters into the pretracheal compartment just below the thyroid gland. The infiltration was performed by the canister method. The lungs were fully inflated with air before injection. The chests were radiographed before and after injection. The heart, the lungs, and the mediastinum were removed and dissected.

## Experimental Findings

In each of the five specimens treated by peritracheal subfascial injection it was noticed that the mediastinum became tensely distended, and the only loss of injection fluid occurred around inefficiently sealed off cannulae. Radiographs (Fig. 1) showed that the injection mass had entered the lung fields, causing hilar shadows. The degree to which the lung fields were penetrated varied greatly. In three of the specimens the shadows produced were minimal and confined entirely to the hilum. In the remaining two specimens the hilar shadows were large and extended out towards, but never reached, the periphery.

Dissection of the specimens showed that the injected fluid had diffused around the trachea and the oesophagus, both in the neck and in the superior mediastinum. Below the level of the tracheal bifurcation the fluid surrounded the intertracheal lymph nodes where they abut directly against the serous layer of the parietal pericardium (Fig. 8). In the two specimens in which the infiltration appeared to be most successful the fluid had penetrated between the serous and fibrous layers of the parietal pericardium around the front of the ascending aorta and in the region of the transverse and oblique sinuses behind. The injection fluid in all the specimens had diffused around the bronchi as far as the hilum of the lung. In the two specimens showing the large radiographic shadows, the fluid had extended in a strictly peribronchial situation as far as the second, and occasionally to the third, bronchial divisions.

Radiographs of the three specimens treated by direct hilar infiltration showed well-developed hilar shadows extending into the lung fields but never reaching the periphery (Fig. 2). Subsequent dissection of these specimens showed a generalized hilar infiltration, but the diffusion into the lung mass had occurred along strictly peribronchial and perivascular planes.

The findings in the injections of the four intact corpses were essentially the same as those of the first series of observations. Radiographs before and after injection (Figs. 3 and 4) illustrate the extent to which the lung fields have been encroached upon by the subfascial peritracheal injection performed at the base of the neck.

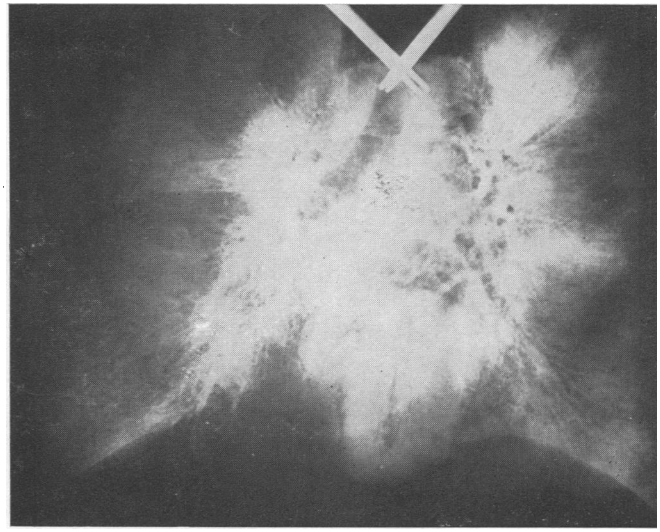

FIG. 1.—Radiograph of an inflated heart-lung specimen after peri-tracheal infiltration with a sodium iodide suspension.

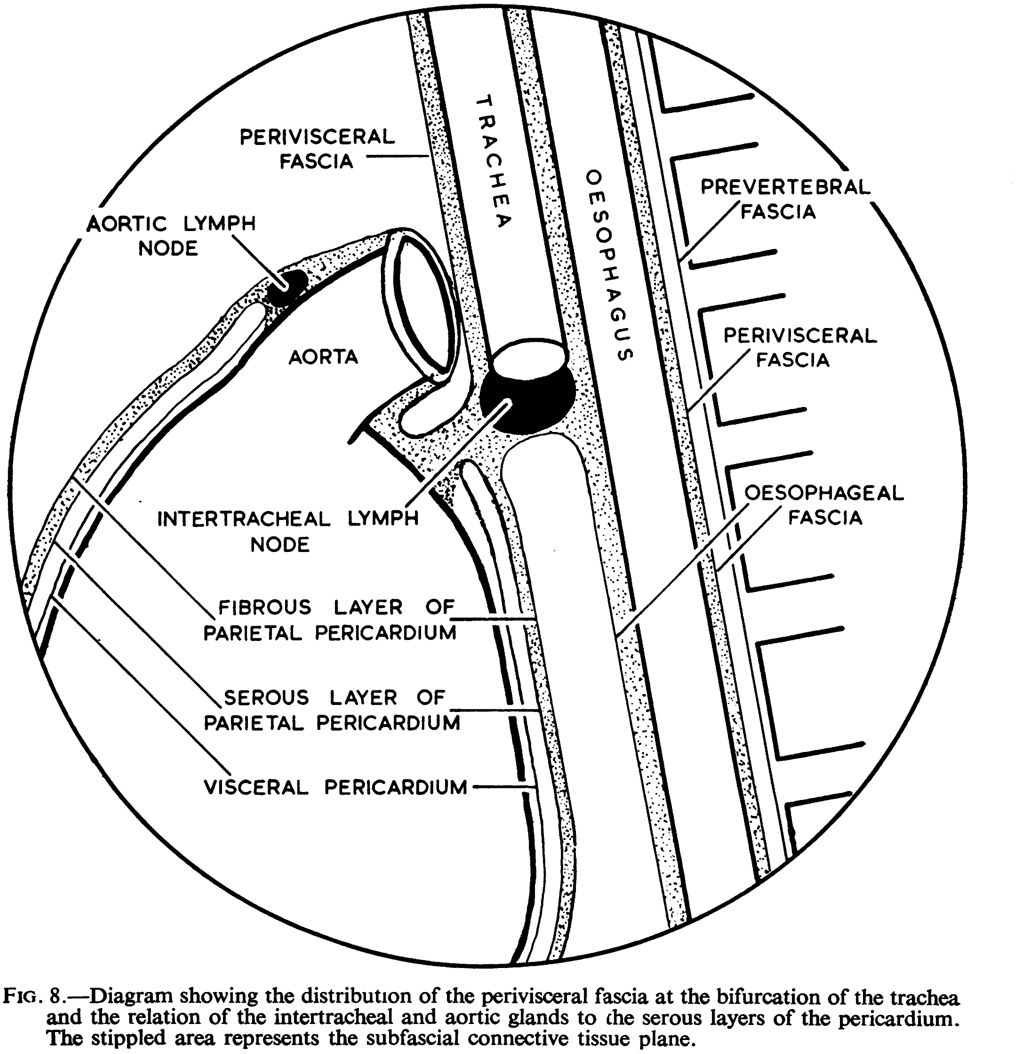


FIG. 8.—Diagram showing the distribution of the perivisceral fascia at the bifurcation of the trachea and the relation of the intertracheal and aortic glands to the serous layers of the pericardium. The stippled area represents the subfascial connective tissue plane.

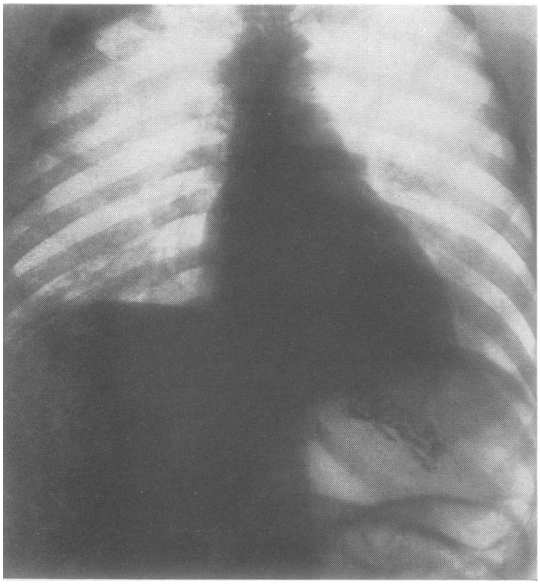

FIG. 3.—Control radiograph of the thorax of a corpse before peritracheal infiltration with sodium iodide suspension.

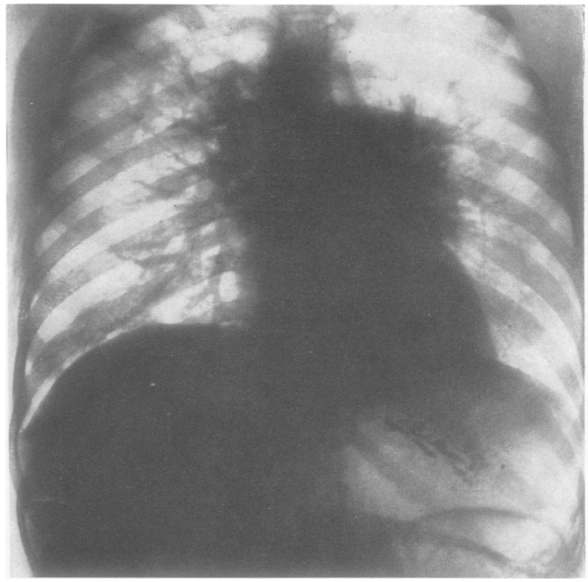

FIG. 4.—Radiograph of the thorax of a corpse after peritracheal infiltration with sodium iodide suspension (same body as in Fig. 3).

## INTERPRETATION OF FINDINGS

The pretracheal fascia is usually described as a layer of cervical fascia which lies in front of the trachea and is prolonged downwards into the mediastinum to become attached to the pericardium. This description appears inaccurate and incomplete when viewed in the light of the above findings. It is not suggested that the extent of the fascia is entirely limited by the distribution of the injection mass in the recorded experiments. The fluid only collected in those areas where the subfascial connective tissue is sufficiently lax to constitute a potential space. Further penetration of the fluid was limited by the firm attachment of the fascia to an underlying structure. It was possible to follow the further fascial extensions around the bronchi and over the pericardium by sharp dissection. The following description of the mediastinal fascia and its extensions is based upon the findings of the injection experiments and those obtained by subsequent dissection.

The key to the understanding of the distribution of the fascia lies in the realization that neither in the neck, nor in the mediastinum, is the fascia solely pretracheal, but that it forms a complete investing layer to both trachea and oesophagus. It is best described as perivisceral. In the neck the anterior part of this perivisceral fascia is thin and fibrous, but becomes thicker and fibro-fatty in nature as it passes into the thorax. Laterally and dorsally the fascia is thinner, of delicate texture, and is continuous above with the buccopharyngeal aponeurosis. The posterior aspect of the perivisceral fascia is closely but loosely applied to the tough prevertebral fascia. A plane of cleavage can readily be established between the prevertebral fascia and the posterior aspect of the perivisceral fascia. It is through this plane that the cervical and mediastinal structures are generally removed at necropsy.

The perivisceral fascia surrounding the trachea and the oesophagus is prolonged into the mediastinum. Below the carina the anterior aspect of the fascia sweeps on to the upper posterior surface of the pericardium to become continuous with the fibrous layer of the parietal pericardium. Around the origin of the aorta anteriorly the fibrous and serous layers of the parietal pericardium are separated by loose connective tissue, which may be quite considerable in amount, and which may be markedly infiltrated with fat. Separation of the two layers in this situation can be readily accomplished by blunt dissection. Over the ventricles, however, the two layers become so firmly adherent that separation is only possible by sharp dissection. The great vessels, with the exception of the inferior vena cava, are invested in their extrapericardial course by fascial sheaths derived from the fibrous layer of the parietal pericardium (Fig. 6). The fascial sheaths of the pulmonary arteries and veins are prolonged around these vessels into the substance of the lung. Separation of the fascia from the vessel wall is fairly readily affected at the hilum, but becomes more difficult once these vessels have begun to branch, until finally the fascia seems to blend with the outer coat of the vessels.

The perivisceral fascia is prolonged laterally as a fibrous investing layer of the left and right bronchi (Fig. 5). The fascia is readily separated from the extrapulmonary bronchi, and it is within this plane that the bronchial artery, lymphatic vessels, and nodes are situated. The fascial covering of the bronchus becomes thinner and more adherent to the bronchus after the hilum has been entered. It becomes progressively more difficult to separate fascia from bronchus the further the lung is penetrated. After the third or fourth bronchial division, separation, even by sharp dissection, becomes very difficult on account of the delicate structure of the smaller bronchi and the firm, fibrous adherence between fascia and the bronchial wall. The coloured medium was not found beyond this level, when the fascia appears to have fused with the outer bronchial wall. It is possible that the fascia is prolonged around the bronchioles, and perhaps forms the alveolar septa.

Situated within the plane between bronchus and peribronchial fascia within the lung lie the radicles of the bronchial artery and vein, the lymphatic vessels, and the interbronchial lymph nodes.

Below the carina the perivisceral fascia continues as an investing layer of the oesophagus to blend eventually with the outer coat of the stomach (Fig. 6).

## The Applied Anatomy of the Mediastinal Fascia

The prolongation of the perivisceral fascia around the bronchi into the lung substance constitutes a highway connecting the lung to the mediastinum. Not only does this plane contain the bronchial arteries, lymphatic vessels, and lymph nodes, but it forms a potential space which can be occupied by air or exudate. These facts would seem to be of considerable importance in the following pathological conditions.

*Mediastinal Emphysema.*—The Thoracic Surgery Unit at Baragwanath Hospital treats many cases of stab wounds of the chest. On several occasions we have noted the presence of mediastinal emphysema following stab wounds of the chest well clear of the mediastinum and unaccompanied by collapse of the lung. A likely explanation seems to be that the stab has penetrated the lung and has injured a bronchus. The absence of a pneumothorax is due to established pleural adhesions.

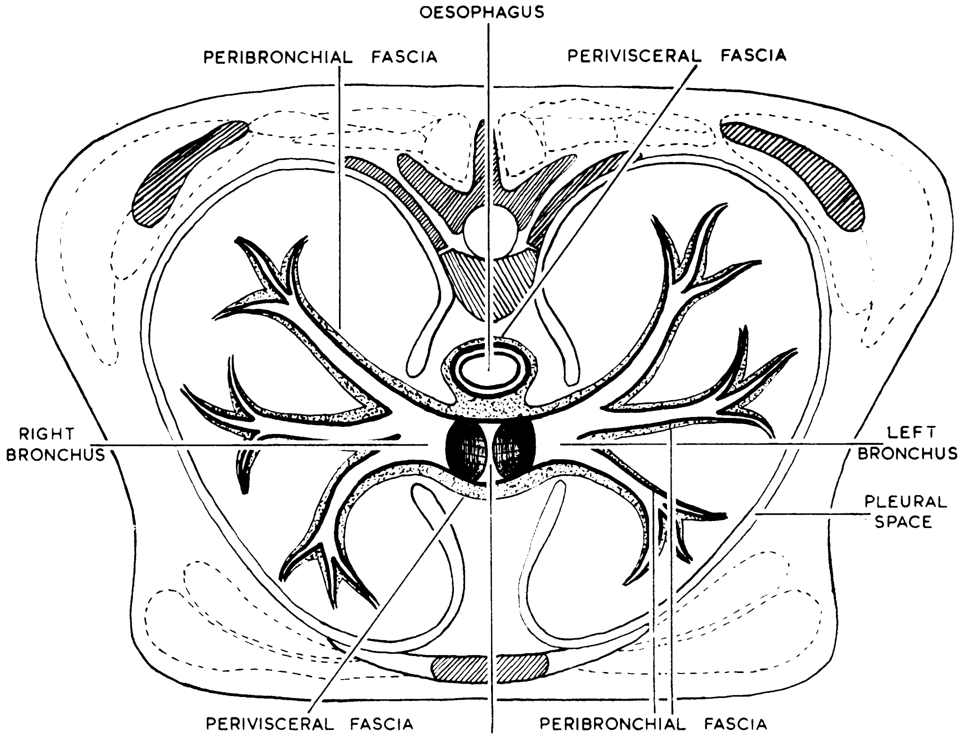


FIG. 5.—Transverse section through the thorax. Diagram showing the manner in which the perivisceral fascia is prolonged around the right and left bronchi into the lung substance to form the peribronchial fascia. The stippled area represents the subfascial plane, occupied by connective tissue and in which lie the bronchial vessels and the pulmonary lymphatics.

Following the injury air is forced into the subfascial peribronchial and perivascular planes, along which it tracks towards the mediastinum.

The peribronchial and perivascular planes supply an explanation for those rare cases of mediastinal emphysema which occur during labour. It has been presumed in these cases that the great increase in intrabronchial pressure occasioned by straining against the closed glottis has resulted in a slight tear, or an actual rupture of a bronchus (Hamman, 1945), or of an alveolus (Macklin and Macklin, 1944). In the case of a tear of the bronchus itself air would be forced through the bronchial defect directly into the subfascial peribronchial plane, and so back through the hilum into the mediastinum. Macklin and Macklin ascribe the occurrence of mediastinal emphysema during labour to a direct rupture of an alveolus into the perivascular sheaths, along which the air passes into the mediastinum.

Similarly, spontaneous mediastinal emphysema, or mediastinal emphysema accompanying a spontaneous pneumothorax, may be due to rupture of an alveolus or an emphysematous bulla into the peribronchial or perivascular fascial planes. Macklin and Macklin (1944), after overdistending the alveoli of cat's lungs, were able to demonstrate air bubbles beneath the perivascular sheaths. With continued insufflation they were able to trace the subfascial air around the vessels at the hilum

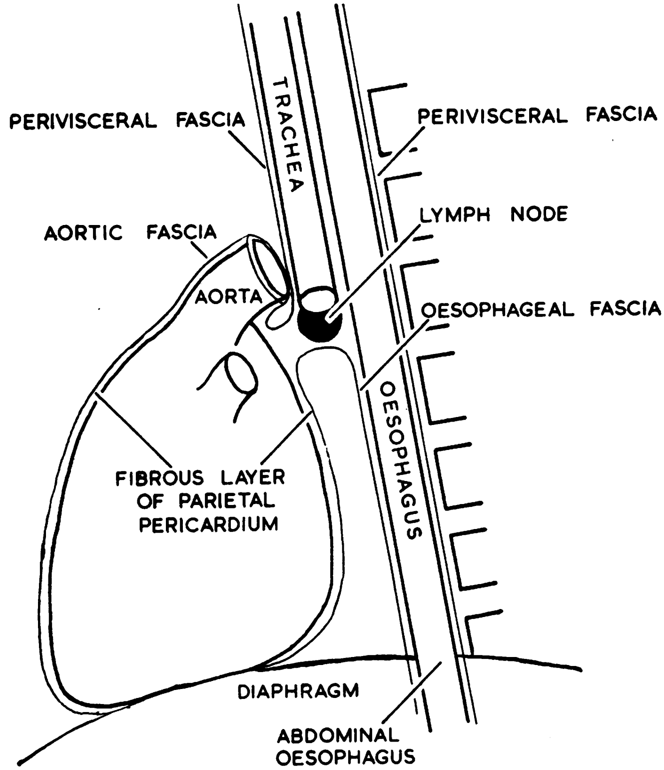


FIG. 6.—Diagram showing the extensions of the perivisceral fascia in relation to the pericardium and oesophagus. The serous layers of the pericardium are not included in this diagram.

and in the mediastinum. They concluded that they had caused a rupture of the marginal alveoli into the adjacent perivascular plane. Kelman (1919), in similar overdistension experiments, described air in both peribronchial and perivascular distributions, but maintained that air entered the mediastinum by travelling beneath the visceral pleura.

*The " Batswing Shadow."*—Roubier and Plauchu (1934) described four cases of hypertensive heart failure presenting radiographic shadows spreading out from the hilar region. The batswing shadow, angel's wing, or butterfly shadow are names which have come to be descriptively applied to these appearances. The shadow may be limited to a small area adjacent to the hilum, or may occupy the larger portion of the lung field. When fully developed it is a large, wing-shaped opacity, the central part merging with the hilar shadow. The peripheral area, particularly at the apex and the base, is translucent, being separated from the shadow by a well-defined edge. The wing often presents a waist, the site of which corresponds with the peripheral edge of the interlobar fissure (Hodson, 1950). The shadow seems to be uninfluenced by gravity and is usually bilateral. All recent authors are agreed that the appearances are associated with left ventricular failure, and are a manifestation of subacute oedema of the lungs.

Hodson (1950) finds difficulty in accepting the explanation that the radiographic appearances are due to a local outpouring of protein-laden fluid into the alveoli, because of the central distribution of the shadow combined with the clear subpleural zones. Kerley (1950), in an editorial, considers that the distribution must have an anatomical basis.

One of the most striking results of the present investigation has been the resemblance between the radiographic shadows of the injected lungs and a batswing shadow (Fig. 7). The resemblance is most marked in those specimens treated by peribronchial and perivascular hilar infiltration (Fig. 2). An exudate accumulating

FIG. 7.—Radiograph of the chest of a patient aged 45 suffering from chronic nephritis and showing signs of left ventricular failure. Note the similarity in shape and distribution between this batswing shadow and the shadows shown in Figs. 1, 2, and 4.

FIG. 2.—Radiograph of an inflated heart-lung specimen after peribronchial and perivascular hilar infiltration with a sodium iodide suspension. The appearances closely simulate those of the batswing shadow, illustrated in Fig. 7, found in some cases of subacute pulmonary oedema.

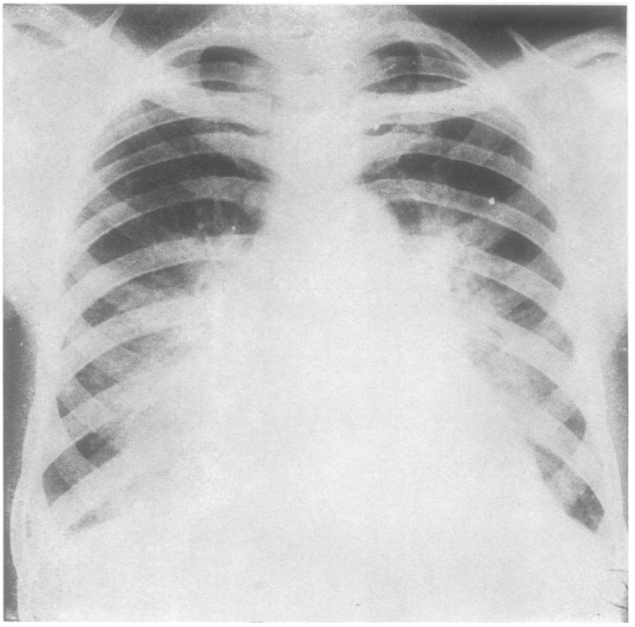

FIG. 7

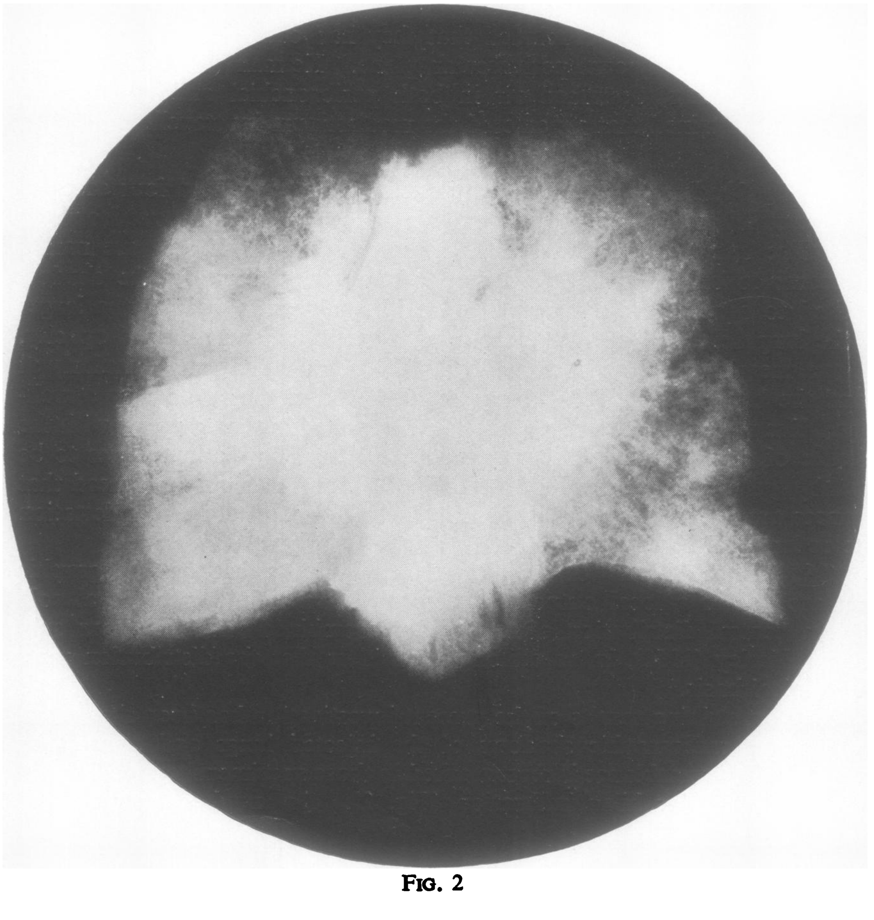

FIG. 2

within the peribronchial and perivascular planes, accompanied by oedema of the fascia itself, provides a sound anatomical explanation for a batswing shadow. The peripheral translucency is explained upon the absence of perivascular and peribronchial planes in that situation where the vessels and bronchi are of small calibre, and the lung tissue consists predominantly of alveoli. The same considerations explain the waist of the shadow at the site of the interlobar fissures. Such a shadow would be unaffected by gravity (Hodson, 1950). Doniach (1947) has described oedema in the interstitial tissues in all his cases subjected to histological examination. The presence of albuminous exudate in the alveoli, which he also describes, constitutes no objection to this hypothesis, as pulmonary oedema, of which the batswing shadow is only an occasional manifestation, is characterized by intra-alveolar exudate.

The exudate could originate from the pleurohilar and true bronchial veins and their capillaries (Marchand, Gilroy, and Wilson, 1950), which lie within the peribronchial planes. They are thin-walled, delicate vessels in free communication with the pulmonary venous system and subject, therefore, to the same hydrostatic strains in left ventricular failure. Anatomically these veins correspond identically with the limits of distribution of the batswing shadow, their capillary network being situated in the region of the terminal bronchiole, and their course lying within the subfascial planes.

*Tuberculous Pericarditis.*—Tuberculous pericarditis is a very common disease amongst the South African Bantu. An almost invariable accompaniment of the condition is gross tuberculous caseation of the mediastinal lymph glands. The intertracheal, aortic, and superior vena cava lymph nodes are particularly closely related to the pericardium (Fig. 8). The peritracheal subfascial injection experiments have shown that fluid injected beneath the fascia in the neck accumulates around these glands and insinuates itself between the fibrous and serous layers of the parietal pericardium in these positions. It seems highly possible that caseous pus from these infected glands can track along the same planes to appear between the layers of the parietal pericardium, there to initiate the tuberculous pericarditis. It is possible to obtain considerable confirmatory evidence of this at necropsy. Recently, at necropsy on a case of acute tuberculous pericarditis, the aortic gland was found grossly caseous, and it was possible to trace the infection from this gland to the adjacent pericardium. Localization of the process in the early stages may well vary with the gland affected. In the case of the intertracheal gland being the site of origin, the tuberculous pericarditis would start in the region of the transverse sinus behind. Once the infection has involved the pericardial sac there are no anatomical barriers to the spread of infection, and the disease rapidly disseminates throughout the pericardium.

## Discussion

Much of what has been written here about tuberculous pericarditis and mediastinal emphysema is not new, and much is conjecture. An effort has, however, been made to link established clinical and pathological observations with anatomical fact. The similarity between the batswing shadow and the radiographic appearances of the infiltrated specimens is so striking as to provide strong support for the contention that the batswing shadow is the result of oedema of the connective tissue framework of the lung. Actual proof of this is very difficult to obtain, as the shadow is an evanescent radiological finding, present on one examination, absent the next.

Very few people die within a short time of finding a radiographic picture of a batswing shadow of their lungs. In these few cases it should be possible to obtain some confirmation, or otherwise, by sectioning the hilar structures to note whether there is indeed an oedema of the peribronchial and perivascular connective tissue. The fact that so large and alarming a shadow is often associated with minimal constitutional effects and physical signs (Nessa and Rigler, 1941) seems to confirm that the site of the shadow lies in the supporting tissues of the lung and not within the vital blood vessels and alveoli.

## Summary

Twelve heart and lung specimens have been treated by subfascial, peritracheal, and hilar infiltration in order to demonstrate the extensions of the mediastinal fascia.

A fascia surrounding both trachea and oesophagus, which includes the so-called pretracheal fascia and the extensions of this fascia into the pericardium and around the bronchi into the lungs, has been described.

Mediastinal emphysema due to pulmonary causes has been discussed in the light of these anatomical findings.

An anatomical basis for the appearance and distribution of the radiological batswing shadow of subacute pulmonary oedema has been presented.

It is suggested that some cases of tuberculous pericarditis may arise as a result of tuberculous pus from a caseous mediastinal gland tracking along anatomical fascial pathways into the pericardium.

I wish to express my appreciation for the encouragement and assistance which my chief, Mr. L. Fatti, Senior Thoracic Surgeon to the Johannesburg Hospitals, has given me. Dr. T. Gillman, of the Anatomy Department, Witwatersrand University Medical School, has guided me in the preparation of this paper. Drs. J. C. Gilroy and V. H. Wilson, of the Department of Medicine, Baragwanath Hospital, have at all times been helpful during the investigation and in the preparation of this paper.

## Bibliography

Doniach, I. (1947). *Amer. J. Roentgenol.*, **58**, 620.
Gray, H. (1935). *Anatomy*, 26th ed., ed. by T. B. Johnston, p. 678, London.
Hamman, L. (1945). *J. Amer. med. Ass.*, **128**, 1.
Hodson, C. J. (1950). *J. Fac. Radiol.*, **1**, 176.
Kelman, S. R. (1919). *Arch. intern. Med.*, **24**, 332.
Kerley, P. (1950). *J. Fac. Radiol.*, **1**, 143.
Macklin, M. T., and Macklin, C. C. (1944). *Medicine, Baltimore*, **23**, 281.
Marchand, P., Gilroy, J. C., and Wilson, V. H. (1950). *Thorax*, **5**, 207.
Nessa, C. B., and Rigler, L. G. (1941). *Radiology*, **37**, 35.
Roubier, C., and Plauchu, M. (1934). *Arch. méd.-chir. Appar. resp.*, **9**, 189.