THROMBO-EMBOLIC COR PULMONALE IN THE BANTU IN DURBAN*

S. KALLICHURUM, M.B., CH.B., M.D., *Department of Pathology, University of Natal, Durban*

The purpose of this communication is to record the frequency of pulmonary thrombo-embolic disease as a cause of cor pulmonale among Bantu patients in Durban. The data analysed were obtained from postmortem examinations as detailed below.

Although pulmonary thrombo-embolism is a common cause of acute cor pulmonale, it is a relatively infrequent aetiological factor in the production of chronic cor pulmonale. However, of the many possible causes of obliterative pulmonary hypertension it has been suggested[1] that pulmonary thrombo-embolism is probably the commonest. Cases with chronic pulmonary heart disease following on recurrent embolism as opposed to locally formed pulmonary arterial thrombosis have been described by Ljundahl,[2] Belt,[3] Carroll[4] and Petch.[5] While the occurrence of venous thrombo-embolism is not infrequent in the chronically bed-ridden, especially those with severe heart disease and with malignancy, it has also been found to occur in active, previously healthy young persons for reasons as yet unknown. Erhner *et al.*[6] thus recorded the finding of chronic cor pulmonale following thrombo-embolism in 3 previously healthy young men who showed the presence of peripheral venous thrombosis. Larger series of cases of thrombo-embolic cor pulmonale have more recently been reported by Owen *et al.*,[7] Thompson and Hamilton,[8] Goodwin *et al.*[9] and Wilhelmsen *et al.*[10]

MATERIAL AND METHODS

Material for the study consisted of 30 consecutive cases of cor pulmonale on which postmortem examinations were undertaken during the period April 1964 - February 1966. These included Bantu patients of both sexes of 10 years of age and over. Cases were investigated in the following way:

Necropsy Examination

At necropsy particular attention was paid to the state of the heart and lungs. The heart was examined for hypertrophy and dilatation of its chambers, and all abnormalities of the pericardium, myocardium and endocardium were recorded. Intramural thrombi were specially sought and their location noted.

The lungs were examined for pleural thickening and adhesions between the lung and thoracic cage, and diaphragm. One lung was sectioned and examined at necropsy for parenchymal and/or vascular disease, the type and extent of abnormality being recorded. The lung less damaged during removal was reserved for further investigation.

With few exceptions, in the earlier cases, the pelvic veins and those of the lower extremities, including calf veins, were examined for venous thrombosis.

Assessment of Ventricular Hypertrophy

Total heart weights were recorded as a routine in all cases. Ventricular hypertrophy was assessed by the following 2 methods:

Measurement of thickness of ventricular wall. The free walls of the right and left ventricles were measured at standard sites, the right ventricle being regarded as hypertrophic when this measurement exceeded 5 mm.,[11] and the left when the measurement was greater than 13 mm.[12] Before such measurements can be meaningful it is important to know whether or not dilatation of the chamber is present. A dilated chamber with these dimensions must be hypertrophied, whereas a contracted chamber which exceeds them is not necessarily hypertrophic. Because there is no accurate method for the assessment of degrees of dilatation, the results of such measurements must be treated with some reserve. When a lesser degree of hypertrophy of a ventricle is present, dilatation may mask this slight hypertrophy. In the course of this study it soon became apparent that the results obtained by this method, even though recommended and widely used by others, were often difficult to interpret. Because of these deficiencies this method was abandoned in favour of the following:

Separate ventricular weights. To achieve this the free wall and infundibulum of the right ventricle were dissected from the rest of the heart, and the weight was taken as representing that of the right ventricle. The free wall of the left ventricle, together with the septum, represented left ventricular weight. The method employed for the separation of the ventricles was that described by Keen,[13] except that the heart valves were not removed.

It was necessary to have control values for comparative purposes, and to this end 53 normal hearts from Bantu subjects, 20 years of age and over, were dissected and weighed in a similar manner. These were obtained from patients dying sudden traumatic deaths and were correlated with both height and body-weight. The normal weight of the free wall and infundibulum of the right ventricle was found to be 66·3 G (SD 7·7), and that of the septum and left ventricle to be 160·5 G (SD 22·49). The normal LV/RV ratio was assessed as 2·48 G (SD 0·21). The values obtained correlate well with those of a similar study by Fulton *et al.*[14]

This method is a time-consuming procedure, but the results obtained not only clearly showed the presence or absence of ventricular hypertrophy, but also indicated which of the 2 ventricles was exclusively or predominantly enlarged, irrespective of the degree of dilatation.

Histological Examination

This entailed a study of sections of relevant organs, stained by the ordinary haematoxylin and eosin method. Sections from the heart and lungs were also stained with Weigert's elastic stain for the purpose of studying structural changes involving the endocardium and lung vasculature, and with toluidine blue for endocardial changes. The findings relating to pulmonary vasculature are based on the criteria as laid down by Brenner[15] and as accepted by most modern workers in this field. Histological studies of the pulmonary arteries were performed on both the injected and non-injected lung. It

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soon became apparent, however, that the picture obtained in the injected lungs was erroneous, on account of the marked distension of such vessels with the radio-opaque medium which caused distension of the lumina and thinning of the walls. There was thus absolutely no correlation between the findings in the injected and non-injected lung.

Pulmonary Arteriography

The method employed was that described by Short,[16] and the radio-opaque medium consisted of a barium sulphate suspension of 80% Micropaque and 3% gelatine. The ideal medium for this purpose is one which, while filling the smallest arterioles, does not cross the capillary bed. The Micropaque-gelatine medium satisfied these requirements (Fig. 1).

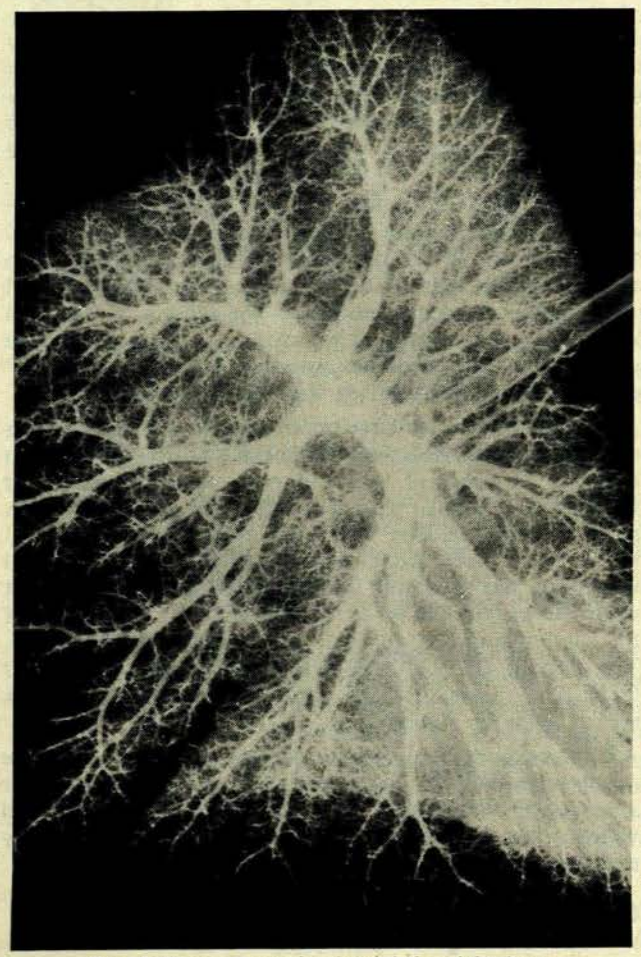

Fig. 1. Normal arteriogram of the right lung.

After removal at necropsy and before injection the lungs were refrigerated for 48 - 72 hours.

It was noted that maximum inflation preceding injection was of great value in ridding the lungs of excess fluid and loosely attached antemortem thrombi which escaped via the pulmonary vessels. The lungs were then deflated and the radio-opaque medium was injected at a pressure of 50 - 80 mm.Hg. Following injection the lung was again fully inflated and positioned on a 12 inch × 10 inch casette fitted with Fast Tungstate screens and Ilford Red Seal film. The film was exposed at 34 kv., 25 mA for 0·1 sec. at a distance of 48 in. for lateral views. A Watson mobile 100 mA unit was used and the films were developed manually in Planocop 1 : 1 for 1½ minutes at 74°F. The resultant film was reasonably satisfactory, but there was room for improvement in contrast to enhance detail. The later series was exposed to Ilfex non-screen film placed in a cardboard casette and inserted into a perspex film box which supported the lung. The Ilfex film was exposed at 64 kv., 25 mA for 0·1 sec. at a distance of 20 in. for lateral views. These were developed in Planocop 1 : 1 for 2¼ minutes at 74°F, the increased time compensating for increased thickness of the emulsion of non-screen films. These showed a marked improvement of definition as compared with the screen-film technique.

In every case histological sections from the injected lungs were examined to confirm full distension of the pulmonary arteries before conclusions were drawn from the arteriograms (Figs. 2 and 3). Satisfactory injection,

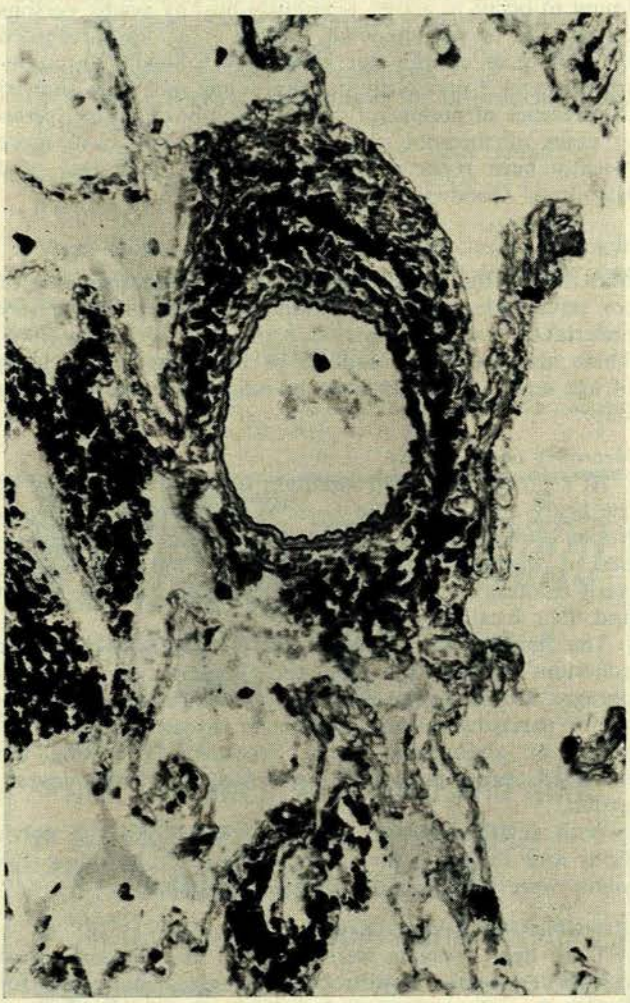

Fig. 2. Non-injected lung. Normal muscular pulmonary artery with deeply crenated elastic laminae (Weigert's elastic × 150).

according to Short,[36] meant a thinning of the arterial wall in relation to the size of the lumen, and smoothing-out of the normally deeply crenated elastic coat. Filling defects observed on arteriograms were investigated by gross dissection and histological examination.

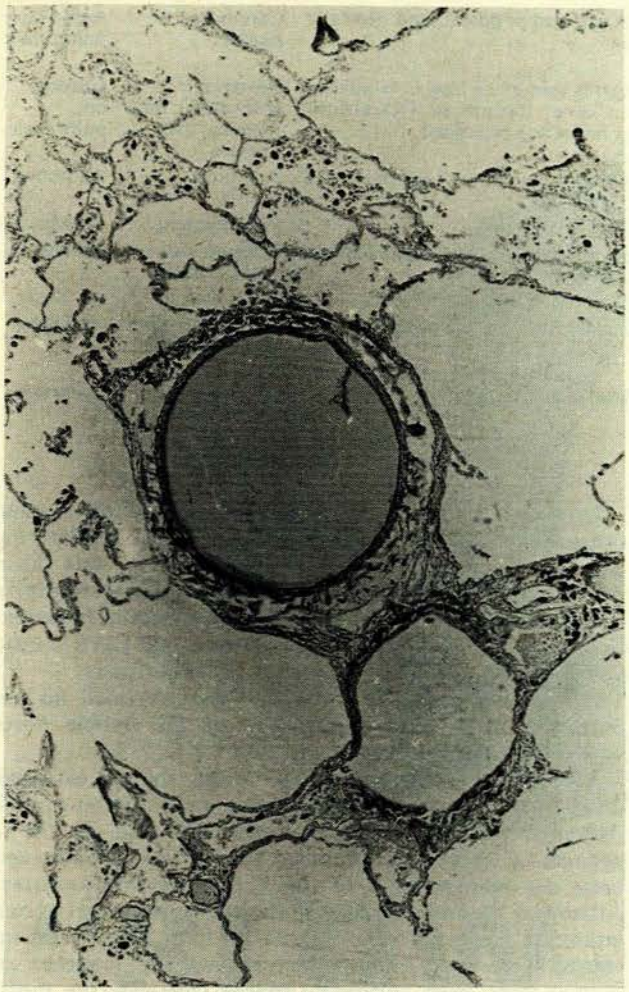

Fig. 3. Injected lung. Muscular pulmonary artery showing attenuation of its wall and stretching of the elastic laminae (Weigert's elastic × 60).

Pulmonary arteriography proved to be of immense value in the study of right heart failure in establishing the level and extent of loss of vascular territory. Arteriograms were particularly helpful in assessing the number, size and distribution of pulmonary thrombo-emboli and in giving some indication as to the degree of reduction of pulmonary arterial capacity resulting from embolization.

Whole Lung Sections

These were prepared from the lungs on which arteriography was performed, and the method used was that described by Gough and Wentworth.[37] Whole lung sections proved to be of great value in the study of emphysema as regards the anatomic distribution and extent of parenchymal destruction. The collapse of emphysematous spaces in fresh necropsy lungs distorts the anatomic location of emphysema in relation to lobules and masks the true extent of parenchymal damage. Fixation of the lung in the expanded position is essential also for the recognition of the more serious centrilobular form of emphysema.

ANALYSIS OF MATERIALS

Table I summarizes the findings in relation to the aetiological type of cor pulmonale in the 30 cases studied.

TABLE I. AETIOLOGICAL TYPES OF COR PULMONALE IN 30 CONSECUTIVE BANTU NECROPSIES

Cause of cor pulmonale	*No. of cases*	*Percentage*
Fibrosing lung disease	13	43·4
Thrombo-embolism	9	30·0
Emphysema and chronic bronchitis	6	20·0
Pulmonary bilharziasis	1	3·3
Primary pulmonary hypertension	1	3·3
Total	30	100·0

Although clinical distinction is made between the acute, subacute and chronic forms of thrombo-embolic cor pulmonale, it is not easy to distinguish between the latter 2 forms at necropsy, on account of the similar pathological features present in both. Clinical separation into subacute and chronic types is based largely on the duration of heart failure, and such evidence was not always available from case histories. Hence all instances of thrombo-embolic cor pulmonale with evidence of pure or predominant right ventricular hypertrophy and chronic passive venous congestion of the organs will be referred to as cor pulmonale from chronic recurrent pulmonary thrombo-embolism. The diagnosis of acute cor pulmonale was made at necropsy in those cases showing recent pulmonary thrombo-embolism, marked dilatation of the right side of the heart without exclusive or preponderant hypertrophy of the right ventricle, and signs of acute congestion of the viscera.

Of the 9 patients in whom thrombo-embolism was incriminated as the cause of cor pulmonale, 5 were females and 4 were males, and their ages ranged from 19 to 70 years, 6 being in the group aged 40 years and over, and the remaining 3 being 19, 33, and 35 years of age, respectively.

Acute thrombo-embolic cor pulmonale was encountered on 2 occasions, while the remaining 7 cases showed the more chronic form of right heart failure resulting from recurrent episodes of pulmonary thrombo-embolism. Table II summarizes the relevant necropsy findings in these 9 cases.

As observed in Table II the total heart weights in cases of thrombo-embolic cor pulmonale, even those with recurrent episodes of pulmonary thrombo-embolism, showed little increase, being below 400 G in all except one instance (case 7). In the first 4 cases ventricular hypertrophy was assessed by measuring the greatest thickness of the free wall of the right and the left ventricles (Table III). These results show the right ventricle to be hypertrophic in the 2 cases where cor pulmonale followed on recurrent pulmonary thrombo-embolic disease, and this correlates with the impression gained by naked-eye examination of these hearts.

TABLE II. NECROPSY FINDINGS IN 9 CASES OF COR PULMONALE FROM PULMONARY THROMBO-EMBOLISM IN BANTU

Case No.	Sex	Age	Total heart weight (G)	Lung findings	Possible site of emboli	Other disease	Cause of death
1	F	50	355	Widespread bilateral arterial occlusion from recent thrombo-embolism. No infarction	Not known. Lower limb and pelvic veins not examined	Nil apparent	Acute cor pulmonale
2	M	70	350	Both main branches and a few smaller branches occluded by recent thrombo-emboli. No infarction	Left calf and popliteal, and prostatic veins	Carcinoma of bladder	Acute cor pulmonale
3	M	70	350	Widespread bilateral arterial thrombo-embolism, both recent and organizing. No infarction	Hepatic vein extending into inferior vena cava; thrombosis (R) atrium. Leg veins not examined	Posteriorly placed (R) lobe amoebic liver abscess	Chronic cor pulmonale
4	M	35	380	As above	Inferior vena cava. Leg veins and (R) heart nil	As above	As above
5	F	65	370	As above; infarction present	Bilateral calf veins	Nil apparent	As above
6	M	40	250	As above; infarction present	Bilateral calf, popliteal and femoral veins	Malnutrition and cachexia	As above
7	F	50	440	As above; infarction present	Bilateral calf and pelvic veins	Nil apparent	As above
8	F	19	350	As above, infarction present. In addition, apical TB (not extensive)	Bilateral calf and left popliteal veins	Nil other than localized apical TB	As above
9	F	33	350	Widespread bilateral arterial thrombo-embolism, both recent and organizing. Infarction present	Bilateral calf veins	Amoebic liver abscess plus cerebral cysticercosis	As above

Hypertrophy of the left ventricle is evident by measurement in 3 cases. While this could be explained on the basis of systemic hypertension in case 2, no such explanation was apparent in the other 2 cases. On macroscopic examination of the specimen, however, left ventricular hypertrophy was not suspected.

TABLE III. MEASUREMENT OF VENTRICULAR WALL THICKNESS IN 4 CASES WITH PULMONARY THROMBO-EMBOLIC COR PULMONALE

Case	Sex	Age	Total heart weight (G)	RV thickness (mm.)	LV thickness (mm.)	Cause of death
1	F	50	355	4	12	Acute cor pulmonale
2	M	70	350	3	20	Acute cor pulmonale
3	M	70	350	7	14	Chronic cor pulmonale
4	M	35	380	7	15	Chronic cor pulmonale

In the remaining 5 cases, hypertrophy was assessed by weighing the ventricles separately. Ventricular weights and LV/RV ratios in these further cases of recurrent pulmonary thrombo-embolism are shown in Table IV. An

TABLE IV. SEPARATE VENTRICULAR WEIGHTS AND LV/RV RATIO IN 5 CASES WITH PULMONARY THROMBO-EMBOLIC COR PULMONALE

Case	Sex	Age	Total heart weight (G)	RV weight (G)	LV weight (G)	LV/RV	Cause of death
5	F	65	370	98	145	1·48	Chronic cor pulmonale
6	M	40	250	61	92	1·51	Chronic cor pulmonale
7	F	50	440	105	213	2·02	Chronic cor pulmonale
8	F	19	360	101	146	1·46	Chronic cor pulmonale
9	F	33	350	101	126	1·26	Chronic cor pulmonale

obvious increase in right ventricular weight (normal mean 66·3 G, SD 7·7) was evident in 4 of the 5 cases shown. In the one patient (case 6) where this was not apparent, both total heart weight and left ventricular weight were well below normal but the body weighed only 39·1 kg. In view of these findings and a LV/RV ratio of 1·51 (normal 2·48), the right ventricle could in this case also be considered predominantly, if not exclusively, enlarged although weighing 61 G.

Left ventricular weights were within or below normal range (160·5 G, SD 22·49) in all except case 7. Here the only blood pressure reading (130/90 mm.Hg) was taken during failure but both kidneys showed features suggestive of chronic focal pyelonephritis, so that systemic hypertension could not be excluded. Nevertheless, the LV/RV ratio was below the normal value, as in all other cases.

Histological examination of the heart revealed no abnormality other than hypertrophy of the muscle fibres where this was suspected.

The lungs at necropsy, in all 9 cases, showed extensive involvement of the pulmonary tree by thrombo-emboli. Although lung infarction was not observed in the 2 patients in whom death resulted from acute thrombo-embolic cor pulmonale, 5 of the 7 patients with recurrent pulmonary thrombo-embolism showed evidence of recent infarction. In every case arteries of both lungs were involved, and in 8, multiple intrapulmonary branches of varying sizes were affected. Table V illustrates the distri-

TABLE V. LOCALIZATION OF THROMBO-EMBOLI IN PULMONARY ARTERIES IN 9 CASES

	Type of vessel affected	No. of cases involved
A.	Elastic arteries	
	Main trunk	0
	Left and right main branches	1
	Secondary branches	4
	Tertiary branches	8
B.	Muscular arteries	9
C.	Arterioles	5 (sparsely distributed in 4 cases)

bution of the thrombo-emboli in pulmonary arteries. In all 9 cases, elastic arteries were affected at one or other level. Similarly, muscular arteries were involved in all instances. Arteriolar involvement was demonstrated by histological examination in 5 patients and was found to be extensive in 1 case (case 3).

Arteriography was performed on the one lung of 4 patients with thrombo-embolic cor pulmonale showing evidence of right ventricular hypertrophy and failure. In all 4 instances the arteriogram showed large filling defects caused by obstruction of either secondary or tertiary branches of the pulmonary artery (Figs. 4 - 6). Areas or segments not affected by thrombo-emboli showed a normal arterial and arteriolar pattern. These arteriograms, together with the knowledge that the contralateral lung was also involved, must suggest a considerable reduction in the total pulmonary arterial bed.

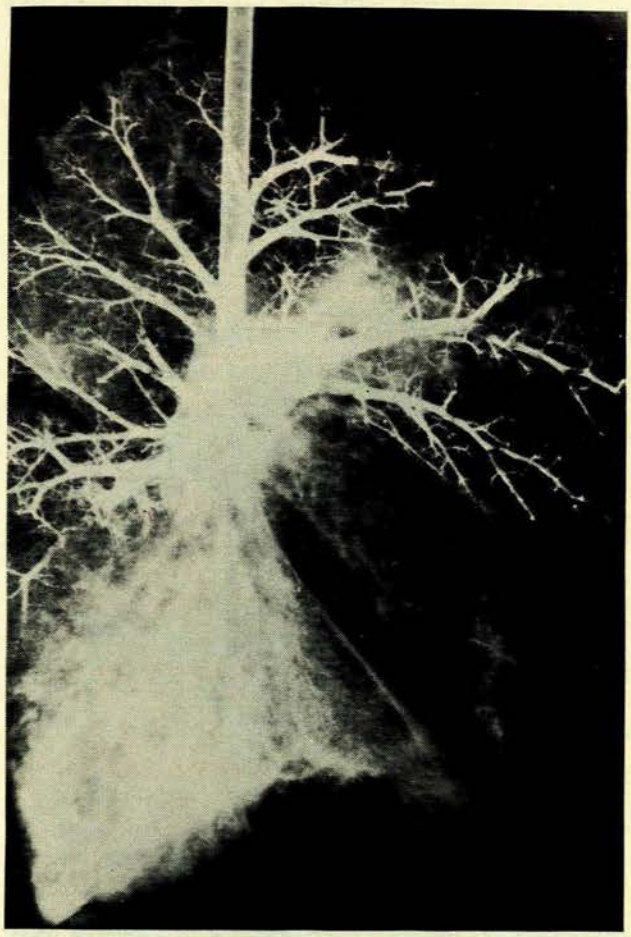

Fig. 4. Recurrent pulmonary thrombo-embolism, cor pulmonale. Left pulmonary arteriogram. Filling defects from thrombotic occlusion of large arteries. Some filling of finer vessels related to unaffected segmental arteries. Infarction of lower half of lower lobe.

Recent haemorrhagic infarction of the lung was present in 5 of the 9 cases of thrombo-embolic cor pulmonale investigated in this series (Figs. 4 and 6). All 5 were examples of recurrent pulmonary thrombo-embolism. Evidence of healed infarction was noted in one of the 5, plus another case. Thus 6 of the 7 patients with cor pulmonale from recurrent episodes of pulmonary thrombo-embolism can be said to have suffered lung infarction at some stage of the disease. Abscess formation from secondary infection of the infarcted areas was observed in one patient. On further examination of these lungs after arteriography, thrombo-embolic obstructions were noted distal to those stopping flow of the radio-opaque medium, and these also varied as regards degree of organization.

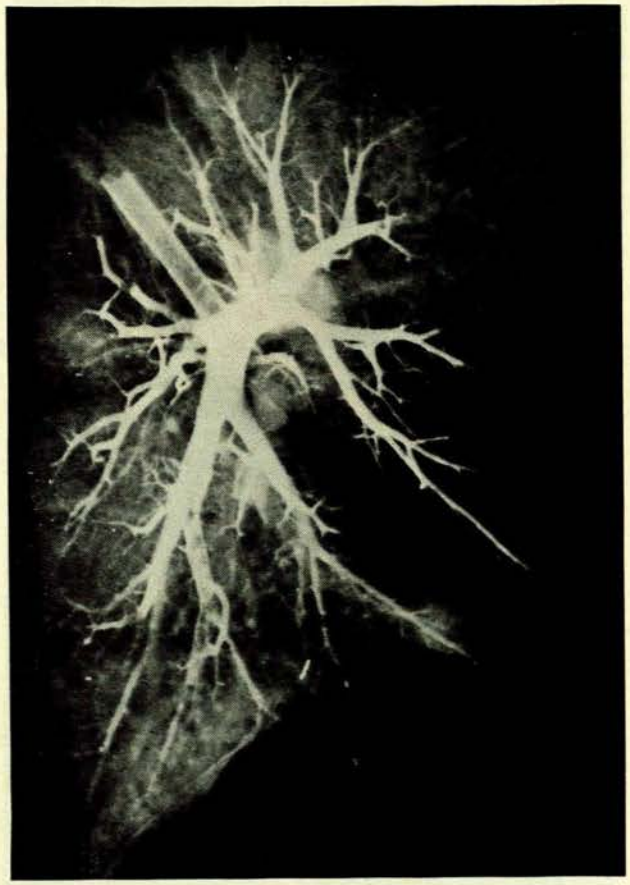

Fig. 5. Recurrent pulmonary thrombo-embolism, cor pulmonale. Left pulmonary arteriogram. There is generalized depletion of the finer branches due to occlusion of segmental and lobular arteries.

Parenchymal pulmonary disease other than that caused by thrombo-embolism was observed in one case and was tuberculous in aetiology (Table II). Whole lung sections, prepared in 5 of the 7 cases of recurrent pulmonary thrombo-embolism with right ventricular hypertrophy and failure, failed to demonstrate diffuse emphysema. Bullous emphysema was seen in 2 instances.

Histological examination in all cases confirmed the presence of antemortem thrombi (Fig. 7), and showed that besides a variation in size, the thrombo-emboli also varied widely as regards age. In the 2 patients in whom death resulted from acute cor pulmonale the lesions were mainly recent in origin and pulmonary infarction was absent.

In all 7 cases of chronic recurrent pulmonary thrombo-embolism both recent and organizing lesions were present, and thrombi within any particular age-group were multi-

ple. While degrees of organization varied, completely organized lesions were infrequent in this group. It was therefore apparent from histological examination that while several thrombo-embolic episodes must have occurred in these 7 patients, the course was relatively rapid.

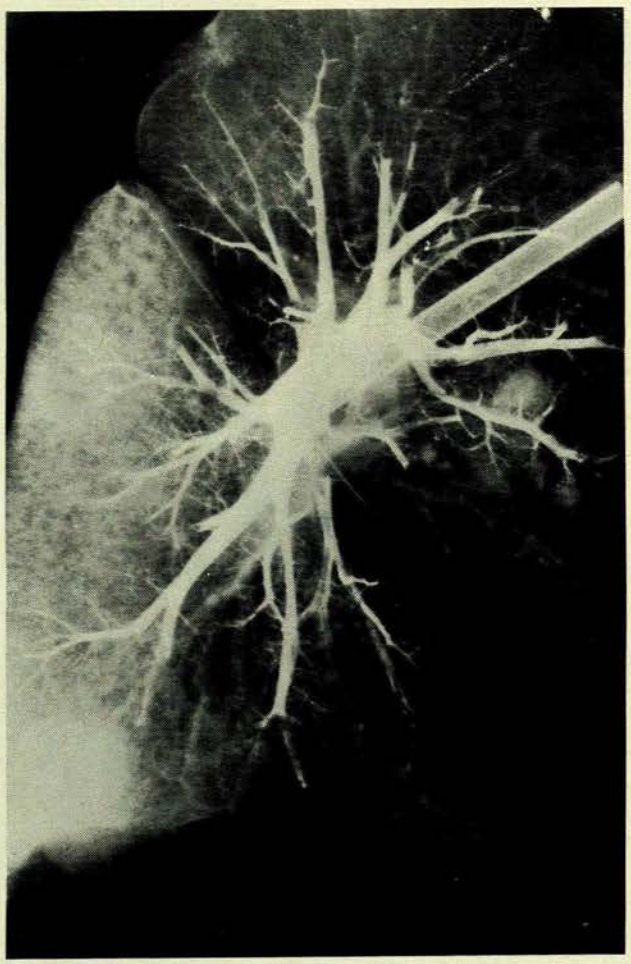

Fig. 6. Recurrent pulmonary thrombo-embolism, cor pulmonale. Left pulmonary arteriogram. Almost generalized depletion of finer branches due to occlusion of segmental and/or lobular branches. Large infarct of the lower lobe.

A source for embolization was present in 8 of the 9 patients in this series, of whom 6 had thrombi in the veins draining the lower extremities. In those with venous thrombosis of the lower limbs the deep calf veins were most frequently involved. In the only instance where the source of embolism was unknown the pelvic veins and those of the lower limbs had not been examined at necropsy.

The unusual association of amoebic liver abscess and thrombosis of the inferior vena cava was observed in 2 patients. In both cases the liver abscess was placed posteriorly and a communication between the abscess and the inferior vena cava was established at necropsy. One of the 2 cases included here (case 3) has been reported previously.[18] Intramural thrombosis of the right heart was observed in one case, but a further source for embolization was found in the hepatic vein and inferior vena cava in this patient. In all suspected cases of venous thrombosis, histology confirmed the presence of antemortem thrombi. Both fresh and organizing thrombi were found, and all instances of venous thrombosis of the lower limbs were classified as examples of phlebothrombosis. In 4 patients no cause for peripheral venous thrombosis was apparent at necropsy. Sufficient disease from other causes was present in 5 subjects, including 2 of the 3 patients under 40 years of age. In one patient a localized apical tuberculous lesion was present, but this was considered an unlikely factor in producing peripheral venous thrombosis.

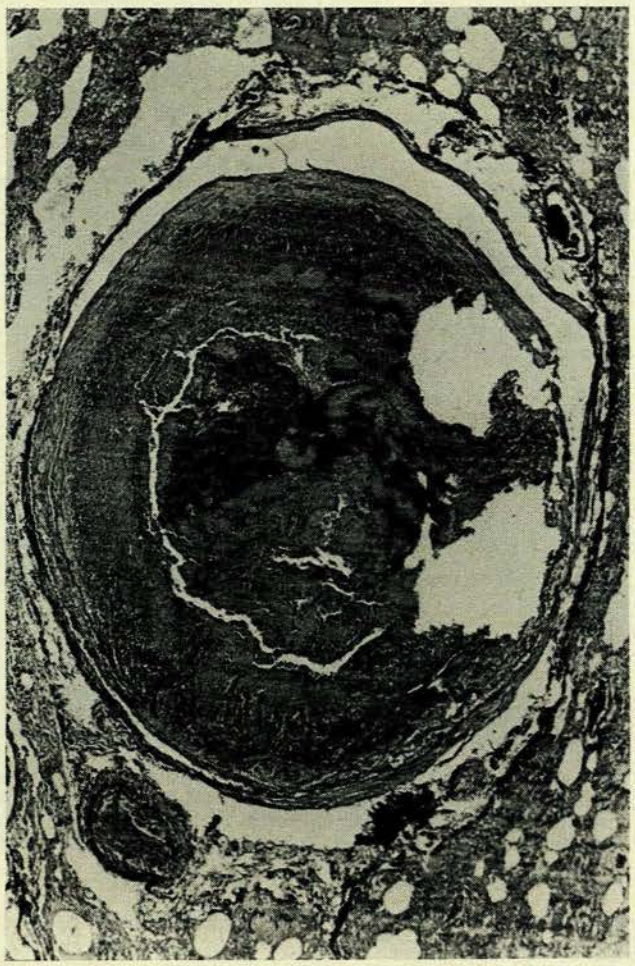

Fig. 7. Acute thrombo-embolic cor pulmonale. Fresh antemortem thrombi in an elastic artery and a small muscular artery (lung section, H & E × 25).

DISCUSSION

In contrast to the generally accepted views regarding peripheral venous thrombosis and pulmonary embolism in Bantu subjects, the findings here show pulmonary thrombo embolic disease with right ventricular strain and failure to be a relatively common cause of cor pulmonale in this population. In 30 consecutive cases of cor pulmonale investigated in this series, there were 2 instances of acute

right ventricular failure and both were thrombo-embolic in aetiology. Of the remaining 28 cases of the more chronic forms of cor pulmonale, 7 (25%) were thrombo-embolic in origin, making this disease the second most common cause (after fibrosing lung diseases) of cor pulmonale in the Bantu at necropsy. This finding supports the view of Turner[19] that thrombo-embolic cor pulmonale is probably not as rare in the Bantu as is generally believed. Although the chronic form of thrombo-embolic cor pulmonale was encountered in this study more frequently than the acute variety, it is possible that this may not be a true reflection of the incidence of these types, since acute cor pulmonale may easily be missed at necropsy unless the pathologist is alert to its possible presence.

In patients with the more chronic form of cor pulmonale, while the great variation shown in the age of emboli in individual cases implies the occurrence of repeated episodes, the infrequency, and often absence, of completely organized thrombo-emboli nevertheless suggest a somewhat rapid course, measured in months rather than in years. It is possible, therefore, that this group of patients (with the exception of case 3, where completely organized lesions were common) represents chiefly the subacute variety of thrombo-embolic cor pulmonale. A more rapid fatal course is further supported by the minor increase in the total heart weights of these patients. As shown, such weights were commonly below 400 G. These patients therefore differ from those reported by Owen *et al.*,[7] in whom marked right ventricular hypertrophy and well-organized pulmonary lesions were frequent, suggesting a longer course, measured probably in years rather than in weeks or months. In the 12 cases of thrombo-embolic cor pulmonale reported by the above authors, the total heart weights were above 400 G in 7 cases and over 500 G in 4 cases.

The main pathological type of pulmonary thrombo-embolic disease observed in this series is one in which fairly large pulmonary arteries (secondary and tertiary branches), as opposed to those of microscopic size (muscular pulmonary arteries and arterioles), are affected. The fact that such large vessels were often involved probably explains the frequency with which infarction was observed in this group of cases. Both Owen *et al.*[7] and Thompson and Hamilton[8] commented on the infrequent occurrence of extensive infarction in their cases. It is possible that in those cases where the disease extends over a longer period, with longer intervals between embolic showers, there is time for an adequate collateral circulation to become established, whereas with rapidly repeated episodes of pulmonary thrombo-embolism this is not possible and infarction will follow.

Because a source for embolization was found in all except the one case where a search for this was unfortunately omitted, it is reasonable to assume that the pathogenesis is embolic as opposed to multiple autochthomous pulmonary arterial thromboses.

Goodwin *et al.*[9] have suggested that large pulmonary emboli probably come from the veins draining the lower limbs, and that the smaller pelvic radicles give rise to emboli of microscopic size which lodge in the muscular arteries and arterioles. Hunter *et al.*[20] have commented on the surprisingly large size of the thrombi situated in the deep veins of the legs, and these authors also reported that major pulmonary arteries could sometimes be obstructed by emboli originating in the calf veins. In the present series, although emboli in large pulmonary arteries were found to have originated in the deep calf veins, smaller muscular pulmonary arteries (100 - 1,000 μ in diameter) were frequently involved in addition to the larger vessels. This correlates with the findings of Owen *et al.*,[7] the pathological picture differing, as mentioned, only as regards the rapidity with which recurrent episodes of embolization occurred and the time lapse between obstruction and the supervention of cor pulmonale.

A predisposing cause of venous thrombosis in the form of severe illness from disease other than that involving the cardiovascular system was present in just over half the cases in this series. In 4 patients no such factor was apparent at necropsy, and the pathogenesis of peripheral venous thrombosis in these cases cannot be explained. While advancing age, relative inactivity and lack of muscle tone, as suggested by McCartney,[21] may be important factors contributing towards the development of venous thrombosis, the reason for its occurrence in healthy young individuals remains obscure.

Perhaps the most interesting point with regard to other diseases associated with venous thrombosis and pulmonary embolism, with subsequent cor pulmonale, in this series, is the relationship to hepatic amoebiasis. In both patients (cases 3 and 4) described here, necropsy revealed the presence of an amoebic liver abscess, posteriorly situated, and associated with thrombosis of the inferior vena cava. In case 4 the initial site of thrombosis formation was in the inferior vena cava and the abscess communicated with this vessel through a small perforation. The primary site of thrombosis in case 3 was the right hepatic vein, the process extending from here to involve the inferior vena cava. The final outcome in both cases was recurrent obstruction to the pulmonary arterial tree by emboli, with subsequent right ventricular hypertrophy and failure.

Three cases of amoebic liver abscess with inferior vena-caval thrombosis (including case 3 in this series) have previously been reported.[18] In 2 of these, extensive pulmonary thrombo-embolic disease and subsequent cor pulmonale were noted. Case 4 in this series is a further example encountered more recently.

Although thrombosis of the inferior vena cava may have been massive, the emboli were multiple and involved the smaller vessels episodically. It is reasonable to assume that thrombi in such infective circumstances are friable and fragment easily. This may also be so, however, in instances of phlebothrombosis involving the peripheral veins.[3]

With reference to cardiac enlargement in the group of cases described in this series, dilatation without hypertrophy was present in the 2 cases of acute thrombo-embolic cor pulmonale. In case 2 it is possible that dilatation may have masked lesser degrees of hypertrophy due not to pulmonary thrombo-embolism but to associated systemic hypertension with left ventricular hypertrophy.

In the more chronic forms, hypertrophy of the right ventricle was present according to either of the 2 methods used to assess this. In the evaluation of the left ventricle, however, difficulties were encountered when wall thickness

was used as a method of measuring hypertrophy. The results obtained were contrary to those found on macroscopic examination of the gross specimen, and erred always on the side of hypertrophy. Differential ventricular weights, however, undertaken in 5 cases, showed no increase in left ventricular weight above the normal values obtained for Bantu subjects. Thus, while chronic failure of one ventricle may lead to failure of the other, and while the occurrence of pulmonary shunts may increase the diastolic burden of the left ventricle, the results obtained in this study do not reveal evidence of chronic left ventricular strain. Minimal to moderate enlargement of the heart with exclusive right ventricular hypertrophy appears to be the rule, exceptions occurring when cardiovascular disease from other causes coexists.

The finding of pulmonary thrombo-embolic disease as an important aetiological factor in the development of cor pulmonale in Bantu subjects is in strong contrast to all previous opinion regarding venous thrombo-embolism in this race group. While such disparity may be explained by the protean clinical and pathological manifestations of the disease, the question naturally arises: is venous thrombo-embolism on the increase? While this may be so in western societies, the position in regard to the Bantu is difficult to assess on account of the lack of previous studies of this nature. However, because of this rather surprising result it seemed essential to obtain some estimate of the general necropsy incidence of peripheral venous thrombosis and pulmonary embolism in the Bantu, purely from the point of view of confirming these findings. Such a study was undertaken concurrently, and the results obtained will be discussed in a subsequent paper.

SUMMARY

Thrombo-embolic cor pulmonale was found to be the second commonest cause of cor pulmonale among Bantu subjects in Durban. While this was the only cause of acute cor pulmonale, it was found to account for 25% of all more chronic forms of cor pulmonale among Bantu.

The aetiological role of amoebic liver abscess in the development of pulmonary thrombo-embolism and subsequent cor pulmonale is described. The pathological type of pulmonary thrombo-embolism encountered here and the associated pathological state of the heart are discussed.

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