viscera" on a more specific basis, while it was Bourgeois *et al.*[4] who identified the specific toxin and produced a plausible experimental model. On the other hand the mere combination of fatty changes in viscera with encephalopathy was described much earlier (in 1929 to be precise) by Brain, Hunter, and Turnbull[5] and therefore should be called Brain's syndrome, or Turnbull's syndrome as the late Lord Brain himself suggested.[6]

Thus the confusion that Dr. Kang wishes to avoid can be averted by the use of two terms: (*a*) Brain-Turnbull syndrome to describe fatty degeneration of viscera with encephalopathy of undetermined cause and (*b*) Reye-Bourgeois syndrome to describe fatty degeneration of viscera and toxic encephalopathy due to a specific toxin.—I am, etc.,

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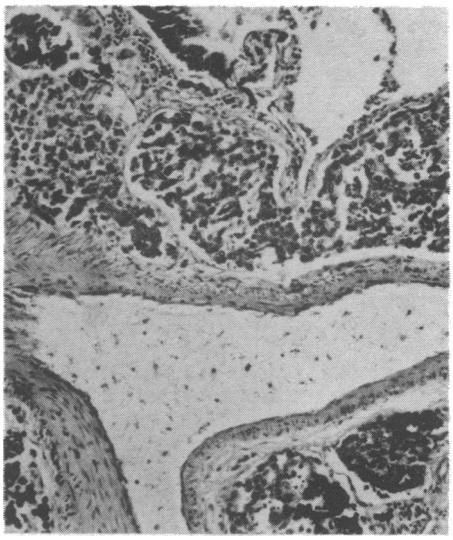
Carcinoid Pulmonary Embolism and Cor Pulmonale

SIR,—Multiple pulmonary metastatic emboli are a cause of acute or subacute cor pulmonale in different carcinomatous diseases.[1-3] We have found no report in the literature of metastasizing malignant carcinoid causing this symptom.

A 70-year-old woman was admitted to hospital with right lower abdominal pain and weight loss. There was neither history nor clinical finding of cardiac or pulmonary disease. A mobile, non-tender mass was palpated in the right iliac fossa. A prolonged blood sedimentation rate was the only pathological laboratory finding. Barium enema revealed a space-occupying lesion in the ileocaecal region. On laparatomy a hard mass was found in this region with extensive lymph node involvement along the mesenteric blood vessels. A right hemicolectomy with lymph node dissection was performed. Histological examination showed a malignant carcinoid of the caecum with metastases in the lymph nodes. From the third to the ninth postoperative day, when death occurred, there were recurrent episodes of respiratory distress characterized by extreme dyspnoea, cyanosis, and tachycardia compatible with recurrent showers of pulmonary emboli. Consecutive chest radiograms confirmed this diagnosis. The electrocardiogram showed right axis deviation which was not present previously. There was no peripheral thrombophlebitis that could explain the source of the emboli. The patient died during one of these attacks. At necropsy macroscopical examination showed metastases on the visceral pleura with many white nodules 1-3 mm in diameter in both lungs. Similar nodules were found in the mediastinal lymph nodes and ovaries. No liver metastases were found. Histological examination of the lungs showed perivascular and peribronchiolar lymphatic infiltration with tumour cells and organizing thrombi containing tumour cells in arterioles (see fig.).



Lung showing periarterial lymphatics distended by tumour cells. Haematoxylin and eosin x 100.

Carcinoid of the caecum is rather rare among carcinoids of the alimentary tract. Usually extra-appendiceal carcinoids are considered to be of low-grade malignancy. This did not seem to be so in our patient, in whom there was extensive local invasion by the tumour after only six months of history. Following the operation there was rapid lymphatic and haematogenous spread. This case is also unique in respect of the sites of spread of the tumour. We have found only three reported cases of lung metastases of alimentary tract carcinoid.[4-6] There were no signs of carcinoid syndrome, nor were there the characteristic cardiac findings of pulmonary stenosis. This case was characterized by a clinical picture of acute and subacute cor pulmonale caused by showers of pulmonary emboli. This entity was first described by Brill and Robertson in 1937.[3] The pathogenesis of cor pulmonale in cases of metastasizing tumours is explained either by invasion of lymphatic vessels by tumour cells compressing the alveoli and bronchioles or by compression of blood vessels by perivascular lymphatics filled with tumour cells. Another possibility is that multiple carcinomatous emboli obliterate pulmonary arterioles. The findings in our case seem to point to the recurrent pulmonary carcinomatous emboli as the cause of the clinical picture of acute and subacute cor pulmonale.—We are, etc.,

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Urinary F.D.P. Excretion in Glomerulonephritis

SIR,—I am glad that Dr. P. Naish and his colleagues (23 March, p. 544) have put into perspective the relation between urinary fibrinogen degradation products (F.D.P.) and non-selectivity of proteinuria. As I deduced from animal work,[1] the presence of high-molecular-weight fibrin products in the urine must mean membrane damage. Moreover, the graphs in their paper are similar to the one my colleagues and I showed[2] when we described what we then thought was a simple technique for estimating urinary F.D.P. In retrospect the discrepancy which we mentioned at that time stems from the fact that we were in fact relating F.D.P. to total proteinuria, since we have since found that protamine sulphate precipitates all proteins but so alters their antigenicity that they are not readily identifiable.

Unfortunately the main conclusion that biopsy fluorescence for fibrin will become the main criterion for anticoagulation could lead to confusion. High urinary F.D.P. excretion indicates "extra"-capillary fibrin deposition. This means that there is gross fibrinogen leakage so that crescent formation is stimulated, and in turn the crescent strangles the glomerulus. As an isolated finding this is surely not an indication for anticoagulation. Already Maggiore[3] has noted that heparin does not influence urinary F.D.P. excretion and has called this "exudative" loss of fibrin.

The theoretical principle is that anticoagulation is indicated for "intra"-capillary fibrin deposition which carries the threat of capillary occlusion. This is a dynamic event starting with immune complex damage to platelets,[4] but apart from the fact that fluorescence may detect fibrin monomer complexes, if fibrin is actually seen blocking capillaries, then local fibrinolysis of the vascular endothelium has already been lost and the damage done. Only further research will establish which functional tests will give early indication of the intravascular coagulation of immune complex disorders. We may well end up with the staggering conclusion that the more practical test is the E.S.R. In the meantime I would recommend consideration of platelet function tests,[5] including measurement of platelet factor 4, the radiofibrinogen catabolism study,[6] or the detection of plasma fibrin monomer complexes by chromatography.

Rapidly declining renal function is still the indication for consideration of anticoagulation, though this is a late stage. It should be a matter of concern that few hospitals have the service for safe monitoring of patients on heparin.—I am, etc.,

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Intermittent Calf Compression in Prevention of Deep Venous Thrombosis

SIR,—I was most impressed by the efficacy of preoperative intermittent calf compression in the prevention of postoperative deep vein thrombosis when this is diagnosed by the ^{125}I-labelled fibrinogen test as reported by Dr. V. C. Roberts and Mr. L. T. Cotton (2 March, p. 358). It is easy to accept that treatment given only during an operation might prevent immediate thrombosis, which is clearly demonstrated in their fig. 2. This figure suggests a further interesting conclusion. From the data it seems that treatment for up to a mean of 117 minutes only—that is, during the operation on day 0—