Responsibility for effective provision of the service includes all doctors. Who is not a manager?

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# Bronchioloalveolar carcinoma

Bronchioloalveolar carcinoma accounts for less than 3% of all primary lung cancers but has attracted disproportionate interest because of its intriguing clinical behaviour and unusual pathological appearance. Indeed, pathologists have been so exercised by this tumour that some have given it 36 different names while others claim that it does not exist at all.[1]

Tumour cells of many different origins may adopt a similar histological pattern in the distal airway, and the term bronchioloalveolar carcinoma is confined to those originating in bronchioles or alveoli; it excludes metastasising adenocarcinomas from the bronchus or distant sites.[2] The tumour may arise from ciliated, mucinous, or clara cells in the bronchiolar epithelium or from type II pneumocytes in the alveolus. The cells all grow as a single layer along the walls of the airspaces, and the unusual environment of the alveolus—with a plentiful supply of food, oxygen, water, and space—probably encourages many different cells to grow in the same way.

The cause of bronchioalveolar carcinoma is equally uncertain and interesting. Smoking and gender appear to be unimportant, while pre-existing lung damage—either local scarring or diffuse pulmonary fibrosis—is definitely associated. The strange case report of a man who developed the carcinoma after habitually going to bed with his mouth full of olive oil led to suggestions of a link with the inhalation of oil,[3] although such a history is rare. The pathological similarity between bronchioloalveolar carcinoma and the viral disease of sheep jaagsiekte suggests an infectious cause, but epidemiological evidence does not support this hypothesis.

Clinically bronchioloalveolar carcinoma has two distinct forms.[4,5] More common is an unremarkable peripheral lung tumour that may be diagnosed by needle biopsy: metastases are unusual, and five year survival after surgery is 70%. The second form is more distinctive and presents radiographically as consolidation affecting one or more separate lobes or segments. About 10% of these patients have bronchorrhoea, and spread is assumed to be airborne, although a multifocal origin cannot be ruled out. Regional and distant metastases occur less commonly than in other lung cancers, and death may therefore be from respiratory failure as more and more of the lung becomes occupied by the tumour. Because this type of bronchioloalveolar carcinoma is widespread surgery is ineffective, and neither radiotherapy nor chemotherapy help. Nevertheless, the tumour may grow slowly, and some patients live for three years after the diagnosis. Both forms of the carcinoma (as well as the metastatic adenocarcinoma) may progress to widespread pulmonary nodules, and at this stage it is uncertain whether spread occurs by blood or airways.

Recently attempts have been made to classify bronchioloalveolar carcinoma cytologically into mucinous (secretory), non-mucinous (non-secretory), and undifferentiated groups.[6,7] The mucinous variety tends to associate with multifocal disease, and the non-mucinous with peripheral nodules, while distant metastases are more common in the undifferentiated group; but these correlations are imprecise. Since bronchioloalveolar carcinoma represents many tumours with similar histological appearance clinicopathological correlations will remain difficult until the cell of origin can be identified more reliably.

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# Haematology, ethnography, and thrombosis

The activities of the coagulation and fibrinolytic systems and the reactivity of platelets vary as widely as the haematological indices measured by a "full blood count." Such differences are of interest to those who wish to understand thrombosis: if those at high risk of thrombosis could be identified by examining factors that promote or control fibrin and platelet deposition steps might be taken to prevent the thrombosis.

Identifying relations between blood values and thrombosis has not proved easy. In certain individuals thrombosis is linked to a congenital deficiency of certain blood factors—for example, antithrombin III, protein C, or plasminogen activator—but such deficiency states are rare and in most people the reason for a thrombosis is unknown. The main reason for our slowness in getting to grips with the problem may be that blood values change dramatically after a thrombosis.

The prospective approach is most likely to yield information, and several studies of patients at high risk of venous thromboembolism—for example, surgical patients—have suggested a relation between haematological values and thrombosis. For example, evidence of "hypercoagulability" (short activated partial thromboplastin time, higher factor VIII activity, higher concentrations of fibrinogen and fibrin degradation products, and prolonged euglobin lysis time) was found preoperatively in plasma from surgical patients who went on to develop venous thromboembolism.[1,2]

The information that we have for arterial thrombosis is derived from a few very large studies in which haematological