Responisbility for effective provision of the service includes all doctors. Who is not a manager?  
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1 National Health Service Training Authority. Better management, better health. Bristol:  
National Health Service Training Authority, 1986. (Chairman: J Done.)  
2 National Health Service Training Authority. Developing the role of doctors in the management  
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Bromhiloalveolar carcinoma  

Bromhiloalveolar 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, pathologist  
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  
bromhiloalveolar carcinoma is confined to those originating  
in bronchioles or alveoli; it excludes metastasising adeno-  
carcinomas from the bronchus or distant sites.2 The tumour  
may arise from ciliated, mucinous, or Clara cells in the  
bromchial 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 bromhiloalveolar 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 bromhiloalveolar carcinoma and the  
viral disease of sheep jaagsiekte suggests an infectious  
cause, but epidemiological evidence does not support this  
theory.  

Clinically bromhiloalveolar 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 radio-  
graphically as consolidation affecting one or more separate  
lobes or segments. About 10% of these patients have  
bromchonbronchoa, 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 bromhiloalveolar carcinoma is  
widely spread 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 bromhilo-  
alveolar 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 bromhiloalveolar carcinoma represents many  
tumours with similar histological appearance clinicopatho-  
logical correlations will remain difficult until the cell of origin  
can be identified more reliably.  

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Arméd Forces Institute of Pathology 1980:127-147.  
2 Schraufnagel D, Puleo A, Port JAF, et al. Differentiating bronchiholo-alveolar carcinoma from  
3 Maseen PPV, Lawes JH, van den Tweel JG. Bronchhlo-alveolar carcinoma after inhalation of  

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, since 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.12  

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