senior officer in the hospital or group. The term "officer," which to medical men still has an odd ring to it, may denote a medical or a lay person: he might be the medical superintendent, or the group secretary, or the hospital secretary. He will take legal advice if necessary, investigate the complaint, and, if the patient remains dissatisfied, report it to the hospital board. The information booklet often sent to a patient before he goes into hospital will have an "appropriate paragraph" to explain the procedure to him, and outpatients will have it drawn to their attention on the back of the appointment card and by notices in the departments.

In the past few years reports of several deplorable episodes in hospitals have shocked everyone who has read them, and more numerous but lesser cases of conduct falling below the best standards doubtless go unreported. Partly because of this there are rumours in political circles of the appointment of a special ombudsman for the health service, or even a bevy of them to serve different regions of the country. The creation of machinery to hear complaints within the service is part of the same governmental response to pressure from the public—that is, in the last resort, voters. Politicians should remember that the prompt, fair, and thorough investigation of a complaint is an obligation that every professional person accepts as part of his work. The sister in charge of the ward, the doctor in charge of the patient, and the matron and hospital superintendent are already regarded as the normal recipients of complaints, and seldom have any abused their trust.

These disastrous episodes must be seen in the context of our hospitals as they are today. A high proportion are antiquated, nineteenth-century buildings where the staff who work in them simply have to make the best of conditions in which any modern industrialist or shop steward would throw up his hands in despair. In some areas staff is seriously short. Many of the resident doctors and nurses who work devotedly in these places are from overseas and therefore sometimes puzzled by the language or customs of the patients in their care. Time and again the real failure when disaster has struck has been shown to stem from the impossible conditions in which professional people are expected to care for patients. Until the root causes of inefficient service are corrected, the setting up of a publicized and elaborate complaints machinery can be little more than a placebo for the public and a source of irritation and anxiety to doctors and nurses in the hospital service. Though the Scottish report emphasizes that any steps taken to protect the patients' interests should not place an unreasonable burden on hospital staff or harm their interests, its proposals seem likely to arouse misgivings.

Geography of Primary Liver Cancer

The incidence of primary liver cancer differs widely throughout the world.[1] If the rate among males is used as an index the world may be divided into three main groups. In group 1 the rates are over 5 per 100,000 population. Countries with these high rates include Angola, South Africa, Nigeria, Uganda, Singapore, and Hawaii. The high rates in Singapore and Hawaii depend on the high Chinese and Japanese populations living in these areas. Group 2, comprises a few areas with an intermediate rate between 5 and 3·1. It includes Japan and Denmark. Countries in group 3 have a rate of 3·0 or less and contain the Americas, Europe (including Great Britain), Australia, and Northern India.

Wide gaps in our knowledge remain because information from many countries is scanty or non-existent. Moreover, figures from some underdeveloped countries may be based on reports from only one region, often an area where a medical school is situated. The Nigerian figures, for instance, are based on Ibadan, the Uganda ones on Makerere. Though knowledge is increasing, little may be known of such countries as a whole. Again statistics are often based on information from death certificates or necropsies, and where neither is forthcoming no figures are available. But much is being done by the International Agency for Research on Cancer, under the directorship of Dr. John Higginson and situated at Lyons in France, to make good the geographic differences in the reporting of cancer. This organization recently sponsored a conference on liver cancer in London, and among the conclusions was the great need for further world-wide studies of the incidence of the disease and its relationship to the general environment of the various populations at risk.

Primary liver cancers from all over the world seem to have a common histology. Bizarre columns of anaplastic cells, caricatures of normal liver cells, surround blood spaces simulating the normal hepatic sinusoids. The histology of the tumour cannot be related to the patient's country or to other diseases with which it may be associated. It does seem, however, that the tumours found at necropsy in Africans are much larger than those in Caucasians reported from Johannesburg, Cape Town, and Los Angeles.[2,3] The association with cirrhosis also differs, being closer for tumours reported from Europe and North America than from Africa.[4] Some of the differences may be due to different criteria for the diagnosis of cirrhosis. Where the cirrhosis is macronodular, a diagnosis may be possible only at necropsy and not in a small-needle biopsy specimen.[5] If the necropsy rate is low and only needle biopsy is done, the numbers of patients with primary liver cancer developing in a cirrhotic liver may be underestimated.

The clinical picture also differs in different parts of the world. In the West primary liver cancer is a disease of older people, often men and especially those with established cirrhosis. In Africa younger people are affected, the disease is more acute, often simulating liver abscess, the course is shorter, and evidence of previous cirrhosis absent.[3]

The geographical differences are reflected in the different proportions of patients having α_1-feto-protein in the serum. This fetal protein is a normal component of the serum proteins of the human fetus older than 6 weeks.[6,7] It reaches a maximal concentration between 12 and 16 weeks of fetal life, and within a few weeks of birth it disappears from the circulation and is then absent from the serum of normal children and adults. It can be easily detected by a semiquantitative Ouchterlony immunodiffusion method. Apart from its association with a few embryonal tumours its presence in serum is specific for primary liver cell cancer.[8,9] But its occurrence with primary liver tumours greatly differs in sera from various parts of the world. In Africa the percentage of sera containing this fetal protein is high. In West Africa, for instance, 79% of 81 patients were positive,[10] and in South Africa sera from 74% of 130 Bantu patients with primary liver cancer gave positive results.[11] But in Uganda the incidence was lower, the fetal protein being found only in 50% of patients with primary liver

[1] *Administrative Practice of Hospital Boards in Scotland*, Scottish Health Services Council. Edinburgh, H.M.S.O. 1966.
[2] *Suggestions and Complaints in Hospitals*, Scottish Home and Health Department. Edinburgh H.M.S.O. 1969.

cancer.[12] The incidence in the Western world may be lower still. In the United States, only 8 of 28 (28%) patients with the disease were found to have the fetal protein,[13] and in another study 40% of sera gave positive results.[14] In London only 5 out of 17 Britons with primary liver cancer showed the fetal protein in their serum, whereas 5 out of 8 from West Africa and 4 out of 10 from other countries were positive.[15] Yet the histological features of the tumours from various parts of the world were similar. The level of fetal protein was poorly correlated with liver size, and serial estimations showed little rise in the amount of it as time passed.[11] More study is required of serum fetal proteins in the various forms of hepatic tumours encountered in different parts of the world. The introduction of more sensitive methods of assay may increase the percentage of patients giving positive results, and quantitation may allow more accurate correlation of concentration with size of tumour, histological type, rate of growth, and clinical course.

These geographic differences might be linked with aetiological factors. In the tropics in recent years it has been suggested that primary liver cancer may be associated with poisoning by various mycotoxins, particularly aflatoxin B. This carcinogen was first described in 1960, when it was responsible for an outbreak of jaundice in turkeys and ducklings in Britain and on the Continent.[16] The common causal factor was traced to the animals having fed on ground-nut meal prepared from nuts shipped from Brazil. The active compound was extracted from the nuts and found to be an aflatoxin produced by a contaminating mould, *Aspergillus flavus*. This and similar toxic moulds can readily contaminate food such as ground-nuts or grain when they are stored in tropical conditions, and aflatoxin has in fact been detected in 40% of Ugandan foods.[17] Moreover, Ugandan tribes with the lowest socio-economic status, and so the greatest chance of eating mouldy foods, have the highest incidence of liver-cell carcinoma.[17] Clearly many more studies must be made of the food consumed by populations which seem particularly at risk of developing this tumour. Simple screening tests performed on urine samples for aflatoxin and other mycotoxins, and capable of field use, are urgently required.

Aflatoxin is highly carcinogenic to certain species, particularly to the rainbow trout, to the mouse, and, on a long-term basis, to the guinea pig. But species vary in their susceptibility.[18] For instance, it does not appear to be carcinogenic to the monkey.[19] But this is no certain indication that tumours will not develop in primates, or indeed in man, under different conditions and particularly with long exposure. Primary liver cancer in the tropics is marked by a high incidence of fetal protein in the serum. This is not so in the aflatoxin-induced experimental hepatic tumours. This may be taken as some, though only circumstantial, evidence that aflatoxin is not the whole answer to the problem of primary liver cancer in the tropics.

The relationship of primary liver cancer to a preceding attack of acute virus hepatitis would presumably be through an intervening post-hepatitis cirrhosis. Follow-up studies of recent large epidemics of virus hepatitis indicate that in fact cirrhosis is an extremely rare complication of this common infectious disease.[20] Moreover, much of the cirrhosis of the tropics is unrelated to hepatitis. But any relationship between primary hepatic carcinoma and virus hepatitis remains uncertain. Recently it has been recognized that patients with acute virus hepatitis often have an antigen in their serum (Australia antigen or hepatitis-associated antigen[21 22]), and in some instances this persists.[23] The frequency of hepatitis-associated viruses in patients with liver-cell carcinoma has not been determined, and knowledge of the world-wide incidence of the antigen in various populations is incomplete. Data on these questions are immediate research objectives and should be obtained in the next few years.

The primary cholangio-cellular carcinoma encountered in the Far East and particularly well described from Hong Kong[24] also has geographical variations. It is associated with infestation of the bile ducts by the Chinese liver fluke, *Clonorchis sinensis*. The tumour apparently arises in direct relation to flukes residing in the small intrahepatic bile ducts. A metaplasia of the mucous glands precedes the cholangio-cellular carcinoma. Further work is needed to determine the possible nature of any carcinogen contained in the fluke.

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Paralysed Hemidiaphragm and Shingles

A paralysed hemidiaphragm may be suspected when a radiograph of the chest shows the leaf to be abnormally raised. Confirmation of the paralysis is obtained by fluoroscopy. The paralysed side moves paradoxically during the act of sniffing—that is, it moves passively upwards as the opposite leaf descends during contraction and displaces the abdominal contents. The cause of the paralysis is usually obvious. It may be, for example, bronchial carcinoma with extension to the mediastinum; trauma, including surgical operation; or an infection such as poliomyelitis. Occasionally phrenic paresis is discovered without apparent cause, possibly in a patient who complains of breathlessness or who has undergone routine mass miniature radiography.

The virus of herpes zoster typically attacks a single spinal