Their recommendations are far-reaching, but the basis of their philosophy is that so far as possible mentally handicapped people should be accommodated in small groups in homes or hostels, closely related to urban residential areas. In all fairness they print counter-arguments put forward by medical staff on the spot, who point out with every justification that at present hostels are not available to allow mentally handicapped people to live outside hospital and, moreover, that such hostels and their occupants might not be accepted by the general public in “normal” residential areas. To pursue such a policy in the teeth of public opposition could be to court disaster, or at least to risk the danger of translating a geographic isolation into a social one.

The report goes on to argue that “in the short term it was probable that a large number of such patients [higher grade] would remain on hospital sites.” And, “since higher grade patients predominate there at the present time, we felt that the relief of overcrowding should be given priority in terms of their needs.” To this end it considers a variety of alternatives.

In all, this is a sensible and humane document which, though paying strict attention to the terms of its brief, recognizes the fact that in the final analysis plans are useless without pairs of hands to operate them. And there’s the rub. As the staff situation deteriorates, so does morale. The authors of the report wisely point out, “the very improvement of working conditions may relieve this shortage of staff,” and, it could be added, give a fillip to morale. The sooner its recommendations are carried out, the sooner may we see some amelioration of the present unhappy state of affairs.

Cultivation of Osler’s Nodes

An unfamiliar method of diagnosing an obscure infection is suggested by the observations of J. E. Puklin and his colleagues on a man of 42 who proved eventually to be suffering from pulmonary actinomycosis. He gave a history of six weeks’ fever, cough, and severe weight loss. There were signs, physical and radiological, of an infiltrative lesion at the left base with a small effusion.Thoracentesis failed, and sputum examination was negative. The contents of a pustule on his hand were reported to have grown an enterococcus, whereupon enterococcal endocarditis was diagnosed (supported by the existence of a cardiac murmur), and treatment was begun with 20 mega units of penicillin and 2 g streptomycin daily. Meanwhile two Osler’s nodes had appeared on the palmar surface of one hand, and material obtained from one of these by injecting saline and withdrawing it yielded a growth of Actinomyces israelii and a Fusobacterium. The treatment already begun on the strength of a totally different diagnosis was appropriate to the new one (though the streptomycin probably contributed little), and its continuance led to steady and complete recovery.

Pulmonary actinomycosis can usually be diagnosed by anyone with an eye open for actinomycotic granules by examining pleural exudate or sputum. This is an examination in which success or failure may be determined at what should be the first step, careful naked eye inspection. Opportunities of diagnosing it in the way described here may not often occur, because haematogenous spread is most unusual in this disease except via the portal system from the ileo-caecal region to the liver; direct extension from the original focus is all that usually occurs. Nevertheless aspiration and cultivation from Osler’s nodes, should they appear, should certainly be considered in this and other conditions. In bacterial endocarditis these nodes are caused by small infected emboli from vegetations: the fact that they are usually sterile is all of a piece with the behaviour of infarcts elsewhere in this disease, which are almost never septic. The body develops a high degree of immunity to the organism causing a subacute endocarditis and can destroy it rapidly anywhere but in the avascular infected valve itself. Thus the possibility of recovering Streptococcus viridans from an Osler’s node may be small unless it is aspired very soon after its first appearance.

Haemorrhagic Ulceration of Gut

Focal haemorrhagic extravasations into the gastric and intestinal mucosa are seen fairly often in necroses on patients dying of heart failure, shock, and acute infection. The epithelium undergoes necrosis to produce superficial ulceration. Similar lesions in the small intestine and colon tend to become confluent and produce larger areas of haemorrhagic necrosis. The necrotic mucous membrane may become partly detached and form a pseudomembrane over the bowel wall. This condition is called necrotizing, or pseudomembranous, enterocolitis, and it causes ill-defined abdominal pain and gastrointestinal haemorrhage. The most severe cases are rapidly fatal. Histologically in all these lesions there are extensive mucosal haemorrhages and a non-inflammatory necrosis of much of the mucous membrane. The underlying submucosa shows vasodilatation.

Lesions of this type in the stomach and duodenum probably form part of a spectrum that includes the well-known “acute,” or “stress,” ulcer that is a complication of shock. Unlike the common peptic ulcer it has no chronic inflammatory component, but it may penetrate the wall of the viscus and cause perforation as well as haemorrhage. In the intestine the lesions can be distinguished from those due to mesenterial arterial and venous thrombosis by their patchy mucosal distribution. In addition the larger vessels are patent.

These haemorrhagic conditions appear to be due to vascular insufficiency. Both in heart failure and in shock there is a reduced flow of gastrointestinal blood and venous stasis, which together predispose to haemorrhagic necrosis. Sludging of the red cells may further impede the local circulation. Necrotizing enterocolitis is associated with the staphylococcal bowel infection which may follow the use of broad-spectrum antibiotics after gastrointestinal surgery. The two conditions are probably separate, but the staphylococcal infection may predispose to the necrotizing lesion by the dehydration and shock it engenders.

Thrombotic occlusion of the related capillaries and venules is also a factor leading to the mucosal ischaemia.