place much reliance on comparative infection rates from different hospitals because of differences in methods of treatment and in prevalent strains of bacteria. These are by no means the only variables.

We have been studying wound infections at this hospital for the past two years and have carried out continuous monitoring of all infections for over a year. An initial difficulty was to define what constitutes a wound infection. When a wound discharges pus from which a pathogenic organism is grown the diagnosis is self-evident; but how are we to define the case with some redness about the stitch holes which might be due to infection or might be simply due to operative trauma and tight stitches? How do we assess the case where a wound exudes a bead of serum from which pathogens are cultured in small numbers but the wound shows no other clinical evidence of infection? In our study we are classifying wounds into six grades of presumed infection but for practical purposes these can be grouped into those which, while showing evidence of infection, do not delay the patient's progress in any way and those which retard the patient's recovery, however slightly. The latter form about a third of the total.

It is also necessary to be clear, in recording the proportion of cases becoming septic as a percentage of operations performed, to what total figure the percentage refers. If one simply takes as a starting figure the total number of operations in the theatre operations' book, excluding only those carried out for septic conditions such as abscesses, the infection rate may look very flattering. But it is also necessary to eliminate cases, such as manipulations under anaesthesia, in which no possibility of wound sepsis arises, tonsillectomies, in which the operative field is in any case not sterile and superadded infection may be impossible to define, and various other categories. The important figure is the proportion of "clean" cutting operations which become infected, excluding those which are at risk because the operation was through a previous, potentially infected, scar or involved opening a non-sterile hollow viscus such as the colon; this gives the true index of iatrogenic infection.

This military general hospital contains both modern cubicalized and open wards, and our preliminary figures support the contention of O'Riordan *et al.*¹ that open wards are an important predisposing factor in wound infection. Another point suggested by our figures is that while the infection rate in "clean" cases shows no correlation with the total number of surgical admissions or total number of operations per month there is a positive correlation with the number of septic cases admitted to the hospital—despite the fact that the great majority are admitted to a separate ward remote from the rest of the surgical section of the hospital and are operated on in a theatre which is not used for "clean" cases. It may yet be shown that to achieve maximum safety from infection in procedures, such as total hip joint replacement, where the risk of sepsis is particularly serious, it is necessary to admit such cases only to hospitals which never under any circumstances accept a septic case.

You mentioned the problem of late deep infections in total hip replacement and noted that half of these are due to *Staphylococcus aureus*. An appreciable number are now being found to be due to coagulase-negative *Staph. albus*, previously regarded as a

usually harmless skin commensal. This and many other anomalies will need prolonged and detailed study before we can sort out the really important factors in infection control from useless but time-honoured shibboleths of aseptic routine. We intend to continue our studies at Haslar over a period of years in the hope of throwing light on some of these problems.

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Fibrocystic Disease of Pancreas and Crohn's Disease

SIR,—The case we report here appears to be the first published report of the coexistence of fibrocystic disease of the pancreas and regional enteritis in a child.

The patient, a girl, was admitted when new born to the Hospital for Sick Children, Great Ormond Street, London, with signs of intestinal obstruction. Laparotomy revealed an ileal atresia with inspissated meconium proximal to the obstruction. A resection of small bowel was performed with end-to-side anastomosis and exteriorization of the blind end of distal ileum as an ileostomy. The postoperative course was uneventful and after closure of the ileostomy she was discharged for further attendance at the Harold Wood Hospital, Essex, where the diagnosis of cystic fibrosis of the pancreas was made, the sweat test showing a level of 137 mEq/l. of NaCl.

Seven years later, in June 1968, she attended Stobhill General Hospital, Glasgow, for follow-up examination. She was symptomatically well and was taking 3 g of pancreatin daily with supplemental vitamins. In 1970 she complained of rectal bleeding of some weeks duration. Examination showed a small anal fissure which was treated conservatively. In January 1971 she had lost weight and there was clubbing of the fingers. The anal fissure persisted and she also complained of frequent, loose bowel movements. The descending colon was thickened and tender. The sweat test showed a level of only 90 mEq/l. of NaCl. A faecal trypsin film test was positive in dilution of up to 1 in 6,¹ corroborating the diagnosis of fibrocystic disease of the pancreas. Arrangements were made to perform a defunctioning colostomy to try to divert the faecal stream to allow the fissure to heal. Laparotomy revealed a thickened, inflamed area of sigmoid colon which macroscopically suggested Crohn's disease. Therefore a local resection of colon was carried out and a double-barrelled colostomy fashioned.

The specimen consisted of 7 cm of thickened colon which had a congested, inflamed serosa. On opening the bowel an ulcerated area 3 cm in diameter was noted. The mucosa presented the "cobblestone" appearance of Crohn's disease. The mucosal ulceration was confirmed on microscopy, and one probable fissure was seen. There was widening of the submucosa with dilated lymphatic and blood vessels and an increase in lymphoid tissue. Hypertrophy of the muscle coat was present and the serosa was inflamed. Sarcoid-like granulomata were found in the submucosa, in the muscle layers, and in the serosa. They contained multinucleate giant cells, epithelioid cells, and chronic inflammatory cells. No caseation or necrosis was seen. No tubercle bacilli were seen in films stained by the Ziehl-Neelsen method. The features appeared consistent with Crohn's disease.

Within three weeks the anal symptoms settled and the fissure began to heal. Further investigation of the alimentary tract by a barium enema and a Gastrografin meal and follow-through examination failed to show any skip lesions. To date the haemoglobin level has re-

mained between 10 and 11 g/100 ml and the E.S.R. has varied between 16 and 45 mm in the first hour (Westergren). Serum immunoglobulin examination has shown a rise in IgA and IgG levels to 451 and 1,861 mg/100 ml respectively with a normal IgM level.

The sweat test levels and the low level of faecal trypsin in this child fulfil the accepted criteria for a diagnosis of fibrocystic disease of the pancreas. She is well within the age group in which Crohn's disease has been reported, the youngest recorded case being in a child aged 9 months.² Though it is dangerous to be dogmatic in relation to inflammatory disease of bowel, the histological features of this case are compatible with the diagnosis of Crohn's disease. The raised IgA level may be a feature of both conditions and is thought to represent local gut immunity phenomena.³⁻⁵ The case therefore shows the coexistence of fibrocystic disease of the pancreas and Crohn's disease of the large bowel. Modern therapy has resulted in longer survival of children with fibrocystic disease and it is to be expected that the development of other disease processes may occur to complicate their clinical course. Further management of the present case remains problematical. Evidence from adult series of perianal Crohn's disease suggests that faecal diversion alone may not provide long-lasting symptomatic relief.^{6,7}

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Pancreatitis and Basal Emphysema

SIR,—Deficiency of the enzyme α_1 -antitrypsin is known to be associated with a severe form of basal emphysema. It has been postulated that lung damage is due to the unopposed action of trypsin or other proteolytic enzymes circulating in the blood. It is impossible to prove this hypothesis in man, but it would receive some support if basal emphysema were to occur in patients with raised blood levels of digestive enzymes, as in chronic relapsing pancreatitis. We now report such a case in the hope that others will be encouraged to look for further examples.

A 60-year-old man, a lifelong non-smoker, was admitted to a nursing home in March 1970 with fever, shortness of breath, constipation, and severe upper abdominal pains of several days' duration. In recent months he had noticed dyspnoea when hurrying over rough ground, but otherwise he had previously been in good health, without respiratory symptoms. He was well enough to be discharged after one week, but on his return home he complained of an