Psoriasis in the Operating Theatre

Many patients with psoriasis and other skin diseases carry pathogenic bacteria, especially *Staphylococcus aureus*, on their skins.1-5 The incidence of infection is much higher among patients in hospital than in outpatients,6 but patients may pick up infection soon after admission to hospital,4 and once acquired the infection is not easily lost. Patients harbouring staphylococci carry the organisms on normal skin as well as on the psoriatic plaques5 and become a real danger as a source of infection. This was well demonstrated by R. W. Payne,6 who recorded an outbreak of staphylococcal wound sepsis traced to an anaesthetist who had psoriasis. He had suffered from psoriasis for eight years and was a known nasal carrier of staphylococci, but it was not until after he had been admitted to hospital for treatment that the outbreak of infection occurred. The organisms are disseminated into the atmosphere on the scales.6 In spite of heavy contamination patients with psoriasis normally show no clinical evidence of infection,6 but the infection may become overt if treatment with occlusive dressings is used.

Y. L. Lynfield and colleagues7 examine the importance of this fact in relation to surgical operations on patients with psoriasis. Since about 1% of the population of the U.S.A. and up to 2% in Britain suffer from psoriasis, this is an important problem. They show that the skin, including areas actually affected with psoriasis, can be sterilized by the use of routine methods of skin preparation. The standard presurgical skin preparation in use in their hospital consisted of alternate wiping with 0·05% iodine in 70% alcohol and plain 70% alcohol, three times each. At operation there is no need to avoid the plaques of psoriasis, as wounds through these heal as well as wounds through the unaffected skin. There is also no need to fear that the psoriasis will be aggravated by the surgical cleaning. The earlier after admission to hospital that operation can be arranged the better, as the patient is less liable to be carrying pathogenic bacteria on admission than he is after being in hospital for a short time. It is unwise therefore to delay operation in the hope that the skin lesions will be cleared by inpatient treatment. Once the patient is known to have become a skin carrier of pathogenic organisms he should be discharged as soon as possible to prevent cross-infection to other patients in the ward. The patient, however, should not be deprived of routine surgical procedures simply because he has psoriasis.


Leucocytes in the Faeces

A microscopic examination of the faeces is important in the diagnosis of many diarrhoeal disorders. The significance of parasites and ova in the stools is obvious in amoebic and hemorrhagic dysentery, while the presence of undigested meat fibres is noted in some cases of the malabsorption syndrome. It is less often realized that an assessment of the leucocyte content can also be useful. Specialists in tropical medicine have long learnt to associate white cells in the faeces with bacillary dysentery, but in many instances the intestinal pathogens have not been adequately identified.1-3 The recent investigations of J. C. Harris and his colleagues in Baltimore are therefore particularly useful.4

They studied the faecal leucocyte content of 169 persons, of whom 114 were volunteers who ingested known intestinal pathogens ranging from *Shigella* and *Salmonella typhi* organisms to various viruses. The other 55 were patients with naturally occurring diarrhoeal disease of various types. In all cases wet preparations of mucus or stool were stained with methylene blue and examined directly for white cells. They found that all shigella infections gave rise to a heavy out-pouring of leucocytes in the faeces. Nine of the eleven cases of naturally acquired non-typhoid salmonella infections showed a moderate number of faecal leucocytes, as did also all eight volunteers with typhoid fever and the four volunteers who had ingested an invasive strain of *Escherichia coli*. The two patients with idiopathic ulcerative colitis and the one who had an allergic diarrhoea after the ingestion of a new cereal also had white cells in their stools. In all these cases the predominant cell type was the polymorphonuclear leucocyte, except in typhoid fever and allergic diarrhoea, when mononuclear leucocytes preponderated.

There was a notable absence of faecal leucocytes in 65 healthy controls, and also in volunteers with cholera and viral and non-invasive toxigenic *Escherichia coli* diarrhoea. Acute non-specific diarrhoea associated with systemic disease, the diarrhoea of "irritable colon," and that associated with antibiotic therapy did not give rise to white cells in the faeces. Likewise, two typhoid carriers and two carriers of other salmonella organisms were free of faecal leucocytes. It was found that the stools of volunteers with shigella infections often contained leucocytes when the organisms could not be recovered from them. The onset of diarrhoea coincided closely with the appearance of faecal leucocytes, which often disappeared at the same time as the symptoms abated. In the volunteers with typhoid fever the faecal white cells preceded