it is commonly associated with man-to-man transmission, particularly within hospitals. It has also been carried on several occasions by sick patients from West Africa to Europe and North America. Though secondary cases are common, tertiary spread is rare. Lassa has been isolated in Nigeria, Liberia, and Sierra Leone, and serological surveys suggest that it also occurs in Guinea and the Central African Empire.

Among hospital patients the death rate is about 36%, but in the general community it may be as low as 3%-5%.

All three viruses cause remarkably similar symptoms. The onset of illness is insidious, with chills, malaise, headache, myalgia, and nausea followed by fever, conjunctival infection and suffusion, and exanthem and oedema of the face, neck, and upper thorax. Petechiae and lymphadenopathy are common. After a few days the features get appreciably worse, with the development of hypotension, oliguria, and haemorrhages which may lead to death. An exanthem is often seen early, but a constant finding with Lassa alone is a pronounced pharyngitis. Subclinical infections with Junin and Machupu are rare but are quite common with Lassa. Recent unpublished findings by J B McCormick, working in an endemic area of Sierra Leone, have shown that roughly half of febrile patients attending hospital have Lassa infection though few develop serious disease.

Because the distribution of the three viruses is geographically limited few problems arise with diagnosis. All grow readily in cell culture, and the most rapid means of serological diagnosis is the fluorescent antibody technique. Complement-fixing antibodies appear late, and this test is much less specific. Some difficulty in diagnosis was experienced in Argentina when an outbreak of Junin was complicated by a simultaneous outbreak of lymphocytic choriomeningitis.

Complement fixation tests did not readily distinguish between these viruses, and similar difficulties could occur in Africa between Lassa and LCM. A further complication has occurred recently in South Africa, where five identical arenavirus strains were isolated from rodents collected in Mozambique. With the fluorescent antibody test the strains are almost indistinguishable from Lassa, and no satisfactory neutralisation system has yet been developed. Fortunately no human disease has been associated with this new strain, but Africa has had a nasty habit recently of turning up new and dangerous virus diseases such as Lassa, Marburg, and Ebola, and we must hope that this is not going to be another.

Psoriasis and stress

Most people believe that skin diseases are caused by worry, stress, or "nerves"; but the scientific evidence is often found to be nebulous. The whole subject has recently been reviewed by Whitlock. Probably there is only one skin disease, dermatitis artefacta, that is entirely nervous in origin. In others there are multiple causes, of which stress is one—and one of variable importance. The term neurodermatitis has been used by so many people in such different ways that it should be abandoned. Even where there is a clear link between emotional problems and a skin disease such as eczema or psoriasis it may be difficult to decide whether the mind influences the skin disease or vice versa; usually both are true. The true nature of psoriasis is still elusive, but advances are being made rapidly. A genetic tendency has been convincingly shown by twin studies, though less than 40% of patients with psoriasis have a family history. Many of the clinical manifestations may be explained by the rapid cell turnover in the epidermis; and much also is known of the changes in the cell cycle and its controls, including progestaglandins, cyclic AMP, and cyclic GMP. But what lies behind these remains a mystery.

Clinically, the factors aggravating psoriasis include trauma (the Körner phenomenon), infection (especially streptococcal throat infection), and the rather variable effects of pregnancy and climate. Injudicious treatment, especially with strong topical or systemic corticosteroids, is an increasingly important precipitant of exacerbations.

All clinicians would agree that stress can be an important factor both in initial attacks of psoriasis and in subsequent exacerbations, but the incidence of stress quoted in clinical reports varies from 10% to 90%. Work in the past two decades has discredited the idea of a particular type of personality associated with psoriasis and of a particular type of stress, and some recent papers have tended to play down the importance of stress of any kind. A recent study by Seville suggested that stress was a factor in 44% of an unselected group of inpatients—only 10% of a control group similarly interviewed admitting to comparable problems. In a much larger postal survey of patients with psoriasis in the United States the incidence of stress was 40%. Minor stresses, however, are commonplace and many patients will believe what they think they should believe—even pityriasis rosea has been attributed to stress by a considerable portion of patients asked the appropriate leading questions.

Not perhaps surprisingly, the prognosis for clearing and continued remission was considerably better in Seville's study where the disease could be linked with a specific stress than where the exacerbation was apparently spontaneous. Probably this also applies to exacerbations induced by streptococcal sore throats and other clear-cut causes. The prognosis in the stress-induced exacerbations was even better when the patient had insight into the causal relationship between the stress and the psoriasis.

Psoriasis is sometimes claimed to cause itching only in emotionally disturbed patients—or even not to cause itching at all, which is far from true. Such claims not only have not been confirmed but do a grave disservice to patients.

What is the message for the management of patients with psoriasis? Some patients learn to live with or ignore their psoriasis, preferring to leave it untreated; but they should not be forced into this attitude when they first come for help. They are seldom sufficiently disturbed to feel in need of psychiatric help. Group therapy has its advocates, but perhaps more important, the doctor should have time and patience to discuss the problems besetting the sufferer. This is more valuable than the wholesale prescription of tranquillisers and antidepressants—lithium indeed has recently been shown to aggravate psoriasis in some patients. Simple psychotherapy and explanations, however, should not replace conventional treatment with local preparations despite the

real limitations these have. Even when an exacerbation has clearly been caused by a stress, removing the cause is usually too late to arrest the condition; the bolting horse requires physical methods of restraint or else has to be left to run itself out.

5 Farber, E M, and Nall, M L, Dermatologia, 1974, 148, 1.
6 British Medical Journal, 1971, 1, 998.
8 Savin, J A, Transactions of the St John's Hospital Dermatological Society, 1970, 56, 139.
12 Coles, R B, Transactions of the St John's Hospital Dermatological Society, 1967, 53, 82.

Gastrointestinal bleeding in acute respiratory failure

Acute upper gastrointestinal bleeding occurs in between 5% and 30% of critically ill patients with severe burns,1,2 head injuries,3 and postoperative respiratory failure.4,5 Recently attention has been drawn4 to the high incidence of this type of bleeding in the adult respiratory distress syndrome. The latter is an incompletely understood disorder characterised by diffuse damage to the pulmonary capillary endothelium and the alveoli leading to pulmonary oedema and arterial hypoxaemia. Though most commonly associated with severe trauma and sepsis, the syndrome is sometimes found in other conditions which cause non-cardiogenic pulmonary oedema, such as acute viral pneumonia and aspiration of gastric contents.6,8

In one series reported from Kansas City gastrointestinal haemorrhage occurred in 11 of 13 patients with the adult respiratory distress syndrome in contrast to four of 44 patients with acute exacerbations of chronic obstructive bronchitis who required assisted ventilation.

What causes the gastrointestinal haemorrhage? It may be due to reactivation of chronic peptic ulcer, a oesophagitis related to recumbency,10 or a bleeding diathesis,11 but in most cases is caused by the stress-induced gastric erosions,8 usually in the body and fundus of the stomach, which are seen in most critically ill patients.3,5 These erosions probably result from mucosal ischaemia with disruption of the gastric mucosal barrier and increased back-diffusion of acid.1 Paralytic ileus, with reflux of duodenal contents into the stomach, may occur in any severe illness, and experimentally reflux has been shown to increase both the incidence and the severity of stress erosions.1

Some additional factors may also be relevant to the high incidence of gastrointestinal haemorrhage in the adult respiratory distress syndrome. Arterial hypoxia would potentiate any cellular hypoxia in the gastric mucosa; and the low arterial carbon dioxide tension, which often occurs in the early stages of the syndrome, may, through vasoconstriction, further impair oxygen delivery to these cells. Once ulceration is established any coagulation defects will increase the risk of haemorrhage. Thrombocytopenia—frequently present in the syndrome—was a risk factor in the Kansas patients,6 including three of the four with chronic obstructive bronchitis who bled. Platelet aggregation and deposition of platelets on damaged pulmonary capillaries are probably factors in the pathogenesis of this type of respiratory distress syndrome2 and may partly explain the thrombocytopenia, though in severe acute respiratory failure (including this syndrome) platelets may be sequestered in the liver and spleen rather than in the lungs.12 Local fibrinolysis in the gastric mucosa may promote gastric haemorrhage in both acute and chronic ulceration13 and disseminated intravascular coagulation, a recognised complication of the syndrome, may also cause bleeding. Corticosteroids are commonly used to treat the adult respiratory distress syndrome, but do not appear to increase the risk of gastrointestinal haemorrhage: indeed, they may reduce the incidence of stress-induced erosions in various types of shock.14

The high morbidity and mortality from haemorrhage from stress-induced gastric erosions6–8 indicate that prophylactic treatment should be attempted. Treatment with antacids can reduce the incidence of gastrointestinal bleeding in patients with burns,6 trauma, sepsis, and acute respiratory failure.6,15,18 Antacids seem to limit or prevent stress erosions by increasing intragastric pH, but they need to be given often enough to maintain gastric pH above 5·0 if bleeding is to be prevented.16,17 The H₂-receptor blockers can arrest haemorrhage from acute erosions.18 In a controlled study of patients with fulminant hepatic failure intravenous infusion of cimetidine prevented haemorrhage from acute erosions by maintaining gastric pH above 5·0, whereas four-hourly antacid treatment, which kept gastric pH above 5·0 in only 35% of patients, failed to prevent haemorrhage.17 Fibrinolytic inhibitors might reduce the need for blood transfusion and operation in gastrointestinal haemorrhage from erosions, but in the adult respiratory distress syndrome fibrin deposition in alveoli is already occurring, and pulmonary thromboemboli are commonplace,7,8 so that further therapeutic inhibition of fibrinolysis seems unwise. The prophylactic value of propantheline bromide has not been established clinically, though in experimental stress it reduces the incidence of acute gastric mucosal lesions, blood loss, and acid secretion.19

At present, the simplest prophylactic approach is to give adequate doses of antacids. Cimetidine may prove equally effective; but we need a prospective trial to determine its value in patients with acute respiratory failure.

5 Locas, C E, et al, Archives of Surgery, 1971, 102, 266.
6 Harris, S K, Bone, R C, and Ruth, W E, Chest, 1977, 72, 301.
9 Williams, M H, Medicine (Baltimore), 1966, 45, 317.
18 MacDonald, A S, Steele, B J, and Bottomley, M G, Lancet, 1976, 1, 68.