full extent in a few days, though scattered new lesions may continue to appear for some weeks. The lesions are brown or yellowish-red, thickened scaly plaques, sharply margined. Either the napkin area or the scalp may be affected first. In the napkin area large plaques appear mainly on the convexities. Lesions of the scalp may be diffuse or in numerous circumscribed plaques, but in either case they have a clearly defined border. Smaller plaques often develop on the limbs and trunk and sometimes on the face. Though its appearance is distressing to the mother, the child rarely shows signs of itching or other discomfort. The eruption clears in a few weeks.

Controversy has centred on three aspects of the disease. Is it a form of psoriasis? Is it caused or initiated by candidiasis? Is the increase in incidence, if indeed it has occurred, to be attributed to the use of topical steroids?

The possibility that napkin psoriasis is a form of true psoriasis has been discussed for some years. S. la C. Andersen and K. Thomsen,² of Denmark, have recently followed up for an average of 5.4 years 60 children who had suffered from napkin psoriasis. The average duration of the disease had been 1.8 months. During the follow-up period two of the children developed psoriasis, more than would have been expected in a normal population. Moreover, a family history of psoriasis was elicited in 26% of the affected infants, a figure which is far in excess of the 4 to 5% of normal persons who give such a history. The evidence will remain inconclusive until a longer follow-up and more extensive genetic studies can be carried out, but it seems probable that napkin psoriasis is in fact an acute infantile form of true psoriasis.

Girdwood Fergusson and his colleagues³ in Glasgow reported in 1966 that the psoriasiform eruption was an "ide" or allergic reaction to candidiasis of the napkin area. J. M. Beare and colleagues⁴ studied 86 infants with seborrhoeic dermatitis and an equal number of normal controls matched for age and sex. Eight sites were sampled in each infant. *Candida* was isolated from one or more sites in 69% of those with seborrhoeic dermatitis but in only 38% of those without skin disease. P. N. Dixon and colleagues,⁵ of Bristol, isolated *Candida albicans* from the napkin area of 41% of 117 infants with napkin eruptions but from only 1 of 68 infants with clinically normal skin. Twenty-seven of the affected infants had napkin psoriasis. But candida was not isolated significantly more frequently from this than from other types of napkin eruption. In a well-designed investigation in a warm climate L. F. Montes and colleagues⁶ isolated candida from the napkin area of 5 of 25 infants with normal skin but in 27 of 35 infants with napkin dermatitis. Abnormal skin in the infant is readily colonized by *Candida albicans*, and some of these infants develop clinical candidiasis, but there is no evidence that candida initiates ordinary napkin eruptions and even less that it initiates napkin psoriasis.

In the absence of reliable clinical or mycological data for comparison it is impossible to establish whether napkin psoriasis or indeed any other form of napkin eruption is commoner now than it was 10 or 20 years ago. On theoretical grounds it seems probable that plastic pants produce physical conditions of increased warmth and humidity which favour maceration of the skin and the growth of bacteria and yeasts. Inflammatory changes, in a person genetically predisposed to psoriasis, can provoke the development of psoriasis as a so-called isomorphic reaction. The often unnecessary use of topical steroids under such conditions may be expected to increase the incidence of candida

carried on the skin and of clinical candidiasis. Is it not yet proved that steroids increase the incidence of napkin psoriasis, nor is the possibility excluded.

¹ Meara, R. H., *Transactions of St. John's Hospital Dermatological Society*, 1968, 54, 92.

² Andersen, S. la C., and Thomsen, K., *British Journal of Dermatology*, 1971, 84, 316.

³ Fergusson, A. G., Fraser, N. G., and Grant, P. W., *British Journal of Dermatology*, 1966, 78, 289.

⁴ Beare, J. M., Cheeseman, E. A., and Mackenzie, D. W. R., *British Journal of Dermatology*, 1968, 80, 675.

⁵ Dixon, P. N., Warin, R. P., and English, M. P., *British Medical Journal*, 1969, 2, 23.

⁶ Montes, L. F., Pittillo, R. F., Hunt, D., Narkates, A. J., and Dillon, H. C., *Archives of Dermatology*, 1971, 103, 400.

Plasma Chloride Levels in Hyperparathyroidism

In 1964 M. R. Wills and G. K. McGowan¹ made the interesting suggestion that plasma chloride levels above 102 mEq/litre and hypercalcaemia might be a reliable method of diagnosing primary hyperparathyroidism and differentiating it from the many other causes of a raised level of plasma calcium. These authors reported 33 patients with proved primary hyperparathyroidism. All had plasma chloride levels of not less than 102 mEq/litre, whereas in 27 of 28 patients with hypercalcaemia from a variety of other causes the plasma chloride was consistently below this level. A review of available literature at the time by Wills and McGowan failed, however, to show such a good separation on the basis of plasma chloride values, though the number of case reports was small and the method of collection of samples was often unsatisfactory. W. C. Thomas, T. B. Connor, and H. G. Morgan² and J. E. Howard³ had found that the levels of plasma bicarbonate tended to be low or normal in patients with primary hyperparathyroidism and raised in other cases with hypercalcaemia. These changes could be interpreted as the reciprocal of the changes observed in plasma chloride.

How good a predictive test have these changes in plasma electrolytes been shown to be? Apart from sporadic case reports, few series have been reported. L. N. Pyrah and his colleagues, at Leeds,⁴ examined retrospectively 30 cases of proved primary hyperparathyroidism and showed that 20 (67%) had plasma chloride values above 102 mEq/litre. The mean postoperative plasma chloride level of 99.5 mEq/litre was significantly lower than the mean preoperative value (103.1 mEq/litre) in these 20 patients, but how long after surgical operation these values were measured is uncertain. More recently Wills⁵ has reported a further group of 16 patients with primary hyperparathyroidism, 14 of whom had plasma chloride levels above 102 mEq/litre and an associated metabolic acidosis, and 13 patients with other causes of hypercalcaemia, none of whom had such a high level of plasma chloride.

It may be postulated that these electrolyte changes can be explained on the basis of the known renal tubular effects of parathyroid hormone. B. E. C. Nordin⁶ showed in man that the injection of parathyroid extract is followed by a rise in bicarbonate excretion and a rise in urinary pH. The hormone is thought to inhibit the sodium for hydrogen ion exchange in the renal tubules by a direct interference with the ability of the kidney to maintain a hydrogen ion gradient between the body fluids and the tubular urine.

This effect of parathyroid hormone on hydrogen ion gradient presumably accounts for the metabolic acidosis in patients with primary hyperparathyroidism.

Similarly, the low plasma chloride found in patients with hypercalcaemia from other causes may be due to renal tubular alkalosis and it is relevant that G. Richet and colleagues⁷ found a rapid fall in urinary pH in 16 adults given calcium by intravenous injection. Furthermore, a metabolic alkalosis has been detected in patients with hypoparathyroidism, where the absence of parathyroid hormone may lead to failure of tubular reabsorption of hydrogen ions. Patients with hypercalcaemia from causes other than hyperparathyroidism presumably have the secretion of their parathyroid hormone suppressed, this contributing to the metabolic alkalosis and low plasma chloride.

It has been realized for several years that a variety of tumours, either carcinomata or sarcomata, may produce the clinical picture of hyperparathyroidism.⁸ Immunological studies suggest that a polypeptide similar to parathyroid hormone is produced, though its chemical structure has not been elucidated. Five patients with this syndrome had a metabolic alkalosis and a plasma chloride below 102 mEq/litre in contrast to patients with primary hyperparathyroidism described above.⁵ On this evidence it would appear that the biological activity of the hormone produced by the tumour differs from that of parathyroid hormone.

Many biochemical tests have been proposed to distinguish the hypercalcaemia of primary hyperparathyroidism from other causes of hypercalcaemia. Cortisone suppression tests and indices of phosphate excretion by the kidney each have their advocates. The simple but accurate measurement of plasma chloride levels, together with an assessment of the patient's acid-base status, would appear to be worth studying as a diagnostic procedure.

¹ Wills, M. R., and McGowan, G. K., *British Medical Journal*, 1964, 1, 1153.

² Thomas, W. C., Connor, T. B., and Morgan, H. G., *Journal of Laboratory and Clinical Medicine*, 1958, 52, 11.

³ Howard, J. E., *Transactions and Studies of the College of Physicians of Philadelphia*, 1962, 52, 11.

⁴ Pyrah, L. N., Hodgkinson, A., and Anderson, C. K., *British Journal of Surgery*, 1966, 53, 245.

⁵ Wills, M. R., *Journal of Clinical Pathology*, 1971, 24, 219.

⁶ Nordin, B. E. C., *Clinical Science*, 1960, 19, 311.

⁷ Richet, G., Ardaillou, R., Amiel, C., and Lecestre, M., *Journal d'Urologie et de Nephrologie*, 1963, 69, 373.

⁸ Fry, L., *British Medical Journal*, 1962, 1, 301.

Prognosis in Industrial Dermatitis

Accurate information about the prognosis for workers with industrial dermatitis is scanty. In 1958 F. F. Hellier¹ reported his findings on 124 patients with industrial dermatitis four or more years after he had examined them for a legal report, and recently M. L. Johnson and H. T. H. Wilson² have followed up 87 out of 100 workers with oil dermatitis whom they had seen for a similar reason. Both these groups of patients were selected from the more severe and persistent sufferers and may not represent the true picture as seen by the works doctor in the factory. It is difficult to see how all mild cases could be followed up for any time owing to the constant movement of labour. Johnson and Wilson divide their patients into six groups, among which are those with oil dermatitis, oil dermatitis

with constitutional eczema, constitutional eczema, and oil folliculitis. The points in favour of a true oil dermatitis were absence of a history of previous skin trouble, contact with oil, the distribution of the rash on exposed areas, improvement on removal from the work, and relapse on re-exposure. Even with these criteria it is clear that if 20 men are doing the same job and only one gets dermatitis he must to some extent be predisposed to it. A major problem in assessing such cases at a tribunal is to determine the relative importance of the internal and external factors. The authors appreciate this by placing ten cases in the mixed group; 17 of their patients had purely constitutional eczema. They state, correctly, that a rash should not be considered constitutional merely because of its patchy distribution if it is otherwise acceptable as dermatitis. Experience has shown that a true exogenous dermatitis, particularly of some standing, may take on a constitutional pattern, and that an accurate history is just as important as how the rash is distributed. Oil folliculitis is a much less disabling condition than true oil dermatitis and affected only four of the men they examined. But an inspection of workers in the factory would show that it is common, though it does not prevent them from continuing to work in contact with oil.

The term oil covers a wide range of substances from heavy lubricating oils to soluble oils with their various additives, but Johnson and Wilson did not find any differences in their effects on the prognosis and so have included them in one group. Nor did they distinguish between dermatitis due to the primary irritant effect of oil and true sensitization. However, this can influence the prognosis. Hellier showed that in sensitization dermatitis removal from the offending irritant was followed by clearance of the rash in 32% of his cases, whereas when the outbreak was due to a primary irritant only 14% escaped further trouble. Johnson and Wilson found that of 46 patients with oil dermatitis who changed their job 26 (57%) lost the rash, but in 5 out of 6 patients with only mild disease the rash cleared up while they continued at work. This may be explained by the fact that many workers develop a mild rash soon after starting work, but it disappears as the skin becomes "hardened." Alternatively, they may have taken precautionary measures to cut down the exposure, a point made by Hellier in discussing dermatitis due to primary irritants, when he suggested that such men should be encouraged to continue at work with their old firm with suitable precautions rather than try to find work elsewhere. Johnson and Wilson found that the chance of ultimate recovery was less in those who continued at work for over a year with the rash than for those exposed for a shorter time. Those cases diagnosed as constitutional eczema had the worst prognosis, as might be expected, whether they changed their job or not.

It has usually been considered that the prognosis is worse in older workers than younger, but Johnson and Wilson could not confirm this, though their figures are admittedly rather small. They also found that the payment or not of "compensation," which it is assumed includes injury benefit, did not affect the prognosis. Possibly it is delay in the settlement of a claim rather than the actual decision which is important, though this was not shown in the small numbers with which they dealt.

¹ Hellier, F. F., *British Medical Journal*, 1958, 1, 196.

² Johnson, M. L., and Wilson, H. T. H., *British Journal of Industrial Medicine*, 1971, 28, 122.