A preliminary report from D. H. Carver and D. S. Y. Seto[1] indicates that the causative agent of serum hepatitis may have been isolated by cell culture techniques. This is not the first time that reports of the isolation of hepatitis virus have been made, but this one does look encouraging. Studies by R. Ward and his colleagues[2] have shown that specially modified immune globulin added to the blood before transfusion reduced the incidence and severity of post-transfusion hepatitis. S. Krugman and his colleagues[3] have now shown that an antibody response to the Australia antigen can be detected in patients with serum hepatitis if a sufficiently sensitive serological test is used. If this is confirmed, the development of specific prophylactic measures becomes a possibility. Now a report from Krugman and colleagues[4] suggests that they have achieved active immunization.

[1] Carver, D. H., and Seto, D. S. Y., "Production of haemadsorption-negative areas by sera containing Australia antigen," Conference on Viral Hepatitis at the Centre Internationale de l'Enfance, Paris, 14-18 June 1971. To be published in *American Journal of Diseases of Children.*
[2] Ward, R., Katz, R., and Rodriquez, J., "Post-transfusion hepatitis; effect of modified gamma globulin added to blood transfusion," Conference on Viral Hepatitis, Paris, 1971.
[3] Krugman, S., Giles, J. P., and Hammond, J., "Viral Hepatitis, type B(MS-2 strain): studies on active immunization," Conference on Viral Hepatitis, Paris, 1971.
[4] Krugman, S., Giles, J. P., and Hammond, *Journal of the American Medical Association*, 1971, **217**, 41.

Violence in Hospitals

The tragic events at Farleigh Hospital[1 2] occasioned the latest in a long list of alarming reports about the ill treatment of patients in our mental hospitals, particularly those caring predominantly for the mentally subnormal. In all institutions the misconduct of a very few can redound to the discredit of all. The innocent can understandably feel aggrieved at this injustice to the point where the desire to protect their own reputation may lead them to resign from the institution they have loyally served. If they decide to stay, they are justified in demanding to know from their superiors and their employers what went wrong and how such tragedies can be avoided in the future.

It was this situation which provoked the nurses at Farleigh to address a petition to the Secretary of State.[3] In the petition simple questions are asked which in the main are concerned with the problem of violence—violence of a patient towards himself, violence towards his fellow patients, and, not least in importance, violence of a patient towards the staff. It seems likely that one of the recommendations in the report on the disaster was inspired by these vital questions. It reads: "A code of conduct for nurses in the handling of violent and difficult patients should be considered by representatives of the psychiatric and nursing professions."

Now the National Association for Mental Health has issued a pamphlet[4] which may be seen as a response to that recommendation, though it is not explicitly stated to be so. Unfortunately it is not an altogether satisfactory document. Perhaps too much was attempted in too little space. In only four pages of large print there are 13 items in its "guidelines." A brief prefatorial statement remarks, for example, that "incidents of violence are not a common occurrence." On what evidence is this somewhat facile statement made? Have any attempts been made to quantify episodes of violence in our hospitals comparable with work recently done in Sweden?[5] There, incidentally, in a cross-sectional study of its mental hospital population on 6 April 1965 it was shown that about 8% of the patients were classified as dangerous. Again, the pamphlet refers to "The use of physical methods of control, such as restraint or segregation." Does restraint mean the descent of a squad of muscular male nurses, or the use of old-fashioned instruments such a straitwaistcoats, muffs, leg-locks, and the like? What is segregation? Does it mean simply ostracism, or restriction to a locked single room, or even confinement in a padded cell? These are not pernickety details: they are of central importance to the whole issue.

Perhaps the most disconcerting statement is that which discusses the admission of a patient "who may require a high level of security because of his violent or unpredictable behaviour." If the hospital concerned is one of the special hospitals, there is no alternative to admission if a court so directs. But in a conventional hospital it is preposterous that this situation should ever arise. That it does is of course undeniable, and it reflects sadly on the present inadequacy of accommmodation for the care of known dangerous patients, or, for that matter, patients who as an expression of their mental disorder become dangerous. Repeated protests have been made in these columns about this grievous situation,[6-8] and the same protest can only be made again.

Despite its defects this pamphlet does serve to highlight the fact that our mental nurses must accept as an occupational hazard the risk of being at the receiving end of violent attacks. It is hoped that more detailed and authoritative advice will be forthcoming in response to the appeal from the Farleigh nurses, which must reflect the anxiety of all nurses who do their best in an honourable and exacting service.

[1] Farleigh Hospital, Committee of Inquiry, *Report*, Cmnd. 4557. London, H.M.S.O., 1971.
[2] *British Medical Journal*, 1971, **2**, 119.
[3] *British Medical Journal*, 1971, **2**, 180.
[4] National Association for Mental Health, *Guidelines for the Care of Patients who exhibit Violent Behaviour in Mental and Subnormality Hospitals. A Consultative Document.* London, National Association for Mental Health, 1971.
[5] Ekblom, B., *Acts of Violence by Patients in Mental Hospitals.* Stockholm, Svenska Bokforlaget, 1970.
[6] *British Medical Journal*, 1967, **1**, 317.
[7] *British Medical Journal*, 1969, **3**, 426.
[8] *British Medical Journal*, 1970, **3**, 537.

Napkin Psoriasis

Probably few infants pass through their first two years without at least some brief attacks of erythema in the napkin area. So many social and psychological factors influence a mother's decision to consult her doctor and the latter's decision to refer the child to a dermatologist that hospital statistics of incidence are difficult to evaluate. Conclusions drawn by comparing such statistics from different countries are meaningless. The lack of uniformity in the nomenclature of the various patterns of napkin eruptions makes it equally difficult to establish whether any particular type is increasing in frequency.

Napkin psoriasis is no new disease. Many authors have preferred to call it psoriasiform seborrhoeic dermatitis.[1] It affects infants between 1 week and 9 months old (usually 4 to 12 weeks), develops rapidly, and often reaches its

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 marginated. 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 colleagues[3] 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 colleagues[4] 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 colleagues[6] 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. McGowan[1] 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. Morgan[2] and J. E. Howard[3] 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 collagues, 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 Wills[5] 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. Nordin[6] 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.