work. This result may well have delayed the introduction of flooding. Nevertheless, undoubtedly behaviourism has provided a framework for the development of behaviour therapy, which has been a framework for the application of common sense. The link may be tenuous—a controversial theory of psychosexual development provided a framework for the development of individual psychotherapy—but the need for these frameworks must surely tell us something about human psychology.

[1] Beech, H R, in *A Textbook of Human Psychology*, eds H J Eysenck and G D Wilson. Lancaster, Medical and Technical Publishing, 1976.
[2] Pavlov, I P, *Conditioned Reflexes*. London, Oxford University Press, 1927.
[3] Watson, J B, and Rayner, R, *Journal of Experimental Psychology*, 1920, **3**, 1.
[4] Marks, I M, *American Journal of Psychiatry*, 1976, **133**, 253.
[5] Siligman, M E P, in *The Psychology of Depression: Contemporary Theory and Research*, eds R J Friedman and M M Katz. New York, John Wiley, 1974.
[6] Yates, A J, *Theory and Practice in Behaviour Therapy*. New York, John Wiley, 1975.
[7] Lazarus, A A, *Psychological Reports*, 1971, **28**, 349.
[8] Masserman, J H, *Behaviour and Neurosis*. Chicago, University of Chicago Press, 1943.

Hairy legs

Methods tried to remove hair[1] from the legs have included use of the arsenic compound orpiment, hot leeches, ants' eggs, and the blood of yellow frogs. One at least of these, arcana, a syrup of sugar and water laid on the hairs and stripped off when dry, is still used for the legs in some parts of the Middle East—together with another traditional method, the plucking of hairs by a rapidly moving loop of twisted thread.[2]

Hair may be removed[3 4] at the surface (depilation) by shaving, chemical degradation, and abrasion; or from the roots (epilation) by wax—the counterpart of sugar syrup—and electrolysis. Shaving is simple and cheap. Any subsequent dryness can be kept to a minimum by the use of bland creams. Shaving does not make the hair regrow faster or thicker, but has to be done frequently, and the regrowing hair has a bristly truncated end. The popular depilatory creams, sprays, and lotions are generally based on thioglycollates with calcium hydroxide or some other alkali; this combination has largely supplanted the sulphides of barium, strontium, and calcium and is less smelly. It is left on for five to 15 minutes and is then washed off. The regrowing hair is less bristly than after shaving, and, though there may be slight irritation of the skin, allergy is rare. Abrasion, though somewhat hard on the skin, can remove a light growth. Both pumice stone and sandpaper mitts to slip over the hands are available.

Waxes are usually applied warm, though a few may be used cold. The wax sets, and at a crucial moment (which is a matter of nice judgment) is pulled off with its embedded hairs. Areas of up to a few centimetres across are dealt with in succession. Some hairs may break below the surface and inflammation supervene as they grow, but this is unusual with wax—though Dilaimy[2] found that it was common with the sugar syrup and thread methods. Much depends on the operator's skill, and a salon treatment may be a good investment. The regrowing hair feels natural and allergic reactions are rare. The process has to be repeated after a few weeks. Electrolygists assert that wax distorts the follicle, making later electrolysis difficult.

Permanent removal of hairs by electrolysis (galvanic or with short-wave diathermy) is formidably slow and expensive, even on facial hair; it may be surprising that it is in demand for hair on the legs. The procedure is safe if carried out by a well-trained operator, but even with frequent treatment would take at least two years.[5] That it should be contemplated owes much to current mores, in which exposure to the sun of a body exiguously clad is thought to have snob value. Removal of hair from the genitocrural area ("the bikini line") may be requested, and other methods are difficult there. A new method of epilation, in which the current passes down the hair as opposed to coming from a needle inserted in the follicle, has yet to be assessed as regards safety and efficacy. Self-operated electrolysis gadgets are not to be advised.

Most women will have no difficulty in choosing a method acceptable to their individual liking and need. Racial and cultural factors affect the type of hair and attitudes towards it. Views may be changing, however: already some younger women are leaving body hair alone. The reason may be current fashion, indolence, a respect for untouched nature, women' lib, or some other individual attitude. Who can say what the custom will be ten years from now?

[1] Maxwell-Hudson, C, *Kaleidoscope of Beauty*. London, Octagon Press, 1968.
[2] Dilaimy, M, *Archives of Dermatology*, 1976, **112**, 507.
[3] *Lancet*, 1967, **1**, 488.
[4] Ridley, C M, *British Journal of Dermatology*, 1969, **81**, 146.
[5] *The Permanent Removal of Superfluous Hair by Electrolysis*. Blackburn, The Institute of Electrolysis.

Pulmonary complications of measles

In measles the rash is a cardinal feature, but it is possible to have measles without a rash. Disease in the respiratory tract, however, is invariable, and this is true not only of wild measles, where cough or croup precede the rash, but also of the mild reactions occasionally encountered with modern attenuated measles vaccines. Moreover, secondary pulmonary infection in measles is well known and is a recognised cause of death in measles.

What is the nature of these pulmonary changes? Measles is acquired by droplet spread, and the virus gains access to the respiratory passages by direct inoculation of the mucous membranes. Controversy still surrounds the possibility that the conjunctivae are a portal of entry, though there is experimental evidence supporting this possibility.[1] At all events the virus reaches the mucosa of the respiratory tract either by droplet inoculation or by passage down the nasolachrymal sac to reach the nose.

In the respiratory epithelium the characteristic response is the formation of multinucleate giant cells. These giant cells have been described in many tissues and secretions, such as the pharynx, nasal mucosa, skin, appendix, sputum, and urine.[2-4] Indeed, the presence of giant cells in scrapings of nasal mucosa or in the urine can give laboratory support to the diagnosis of measles before the onset of the rash in much the same way as Koplik spots support the early clinical diagnosis.

Like Koplik spots giant cells are transient. They are part of the pathological evolution of the illness; they come and they go. In very few cases they persist, and in these patients the fine crepitations and high fever characteristic of early measles continue, with the development of viral pneumonitis. The onset of giant cell pneumonia at this stage of measles is probably rare. Another form of giant cell pneumonia is a

chronic respiratory illness in patients with underlying disease such as mucoviscidosis, leukaemia, and Letterer-Siewe disease, and in these patients the respiratory complications are frequently the cause of death.

Patients with altered immunity may have unpredictable responses to measles virus. Thus they may have a rash but fail to produce antibody in serum, or on exposure to measles they fail to develop a rash. At least one child, a leukaemic, has died after receiving attenuated measles vaccine, with the development of giant cell pneumonia.[5] Recently Pullen and colleagues[6] have reported atypical measles in three leukaemic children on immunosuppressive treatment; two developed encephalopathy and died, the third survived a giant cell pneumonia. The risks to leukaemic children of exposure to measles virus are clear; children with leukaemia and measles patients should not be admitted to the same general ward.

It is possible then to postulate that the common respiratory symptoms in measles are transient symptoms of a host-virus relationship characteristically producing giant cells, the natural evolution of which is the rejection of giant cells with eventual complete resolution. A few patients with altered immune states progress to a chronic respiratory illness, and necropsy in these cases has shown numerous multinuclear giant cells and the presence of measles virus.[7] A small minority of patients die of giant cell pneumonia in the acute stage of measles, but the reasons for the occurrence of the complication at this stage of the illness are conjectural.

Secondary pulmonary infection is responsible for about half the mortality in measles. Millar[8] reported a mortality of 2 per 1000 cases in Britain. Larger series of cases from developing countries with a much higher mortality rate suggest that respiratory complications also account for about half the deaths.[9 10] Clinically, secondary respiratory infection is usually encountered when the patient is well on the way to recovery. Seven to 10 days after the onset of the rash fever returns, leucopenia is replaced by a neutrophilia, and the chest *x*-ray film may show consolidation. One of the common bacterial pathogens, pneumococci, *H influenzae*, streptococci, and staphylococci, is usually found and antibiotic therapy started.

What of secondary viral infection as a possible cause of respiratory complication following measles? A recent paper by Warner and Marshall[11] describes four children who developed severe lung disease after measles, and the authors considered that adenovirus infection was responsible for this complication, which resulted in one death. The difficulty with this sort of study on a few patients is to prove a causal relationship between two conditions which may happen to coexist in the same patient. Adenoviruses are commonly isolated from tonsils and other tissues without apparently causing any disease, yet they can on occasions produce severe chronic respiratory illness[12 13] of the type described by Warner and Marshall in their patients after measles. One of their patients had a leucocytosis suggesting bacterial rather than viral secondary infection, though bacteriological cultures were not helpful. While on clinical and virological evidence these four children almost certainly suffered from measles, antibody studies relating both to measles virus and to adenovirus may have been altered by corticosteroids used in treatment.

Present views on respiratory complications in measles may be summarised as follows. Initially in wild measles respiratory symptoms are a characteristic part of the disease and are assumed to be due to a viral pneumonitis. In a small minority of patients this progresses to severe giant cell pneumonia and may cause death. In some developing countries this may be a relatively common pattern. Giant cell pneumonia is more characteristically a chronic illness in patients who are immunosuppressed by disease, such as leukaemia, or by cytotoxic drugs. Secondary bacterial infection is relatively common and important to diagnose, for antibiotics may be curative. Secondary viral infection has hardly been explored as a factor in the complications, but Warner and Marshall, while not proving that adenoviruses are responsible, at least remind us to keep an open mind on the aetiology of one of the respiratory complications of measles.

1 Papp, K, *Revue d'Immunologie et de Thérapie Antimicrobienne*, 1956, **20,** 27.
2 Stryker, W A, *American Journal of Diseases of Children*, 1940, **59,** 468.
3 Reiman, H A, *Medicine*, 1947, **26,** 167.
4 Davidsohn, I, and Mora, J, *Archives of Pathology*, 1932, **14,** 757.
5 Mitus, *et al*, *American Journal of Diseases of Children*, 1962, **103,** 413.
6 Pullen, C R, *et al*, *British Medical Journal*, 1976, **1,** 1562.
7 Enders, J F, *et al*, *New England Journal of Medicine*, 1959, **261,** 875.
8 Miller, D L, *British Medical Journal*, 1964, **2,** 75.
9 Morley, D, *British Medical Journal*, 1969, **1,** 297.
10 Bwibo, N O, *Tropical and Geographical Medicine*, 1970, **22,** 167.
11 Warner, J O, and Marshall, W C, *British Journal of Diseases of the Chest*, 1976, **70,** 89.

Hepatitis in clinical laboratories

For more than 25 years there have been sporadic reports,[1-3] mainly from the USA, of clinical hepatitis apparently acquired by laboratory workers from specimens. Nevertheless, the disease did not appear to be a serious occupational hazard in Britain until laboratory workers became affected by extensive outbreaks of viral hepatitis in haemodialysis units.[4] Two questions then required answers: how great was the risk? how could it be reduced?

Some diseases are almost completely notified—the risk of tuberculosis, for example, among laboratory workers may be estimated by direct comparison between incidence rates in the group and in the whole population.[5] In contrast, similar comparisons for hepatitis are unreliable: notifications of infective jaundice are incomplete; cases of anicteric hepatitis are excluded; and no differentiation is made between hepatitis A and B. The results of a postal survey of experience in about 250 British laboratories between 1970 and 1974 have provided valuable data.[6 7] No attempt was made to compare the incidence of hepatitis with that of the general population, but the study showed that attack rates were highest among technicians, and lowest among porters and domestic and secretarial staff; the overall incidence of clinical hepatitis was 143 per 100 000 person years in 1973-4. The annual number of cases remained steady throughout. None of the 73 cases reported in the five years was fatal, and only a quarter required admission to hospital.

More than half of these cases were of hepatitis B—in contrast with a survey in London, which showed that only 20% of cases among adults in the normal population are hepatitis B.[8] This suggests that much occupationally acquired hepatitis among laboratory workers is type B. Serological studies[9 10] among hospital staff in the USA have shown that workers in close contact with blood have the highest rates of infection with hepatitis B and have confirmed that of all laboratory workers technicians are at the highest risk. Symptomless infections are commoner than clinical hepatitis.

On the available evidence, then, hepatitis is an occupational hazard for laboratory workers; but in Britain the annual