

The expense of silicone implants for impotence and incontinence has led to the concentration of the work in special centres. £800 may seem a lot of money for a two-thirds chance of curing incontinence, but it is a relatively small price for a dramatic change in the patient's way of life, his ability to work, his contribution to the community, and the relief from being socially ostracised by his anxiety that others may be aware of the uriferous smell. In the same way the price may be acceptable for the young man who has been rendered impotent either by trauma or by diabetes for it may provide him and his wife with an almost normal life. However, some age limit should perhaps be imposed on such a procedure. Operations on 10 patients between the age of 60 and 69 and one patient aged over 70 for impotence⁹ might be an unjustifiable financial imposition on our National Health Service.

¹ *British Medical Journal*, 1977, 1, 254.

² Furlow, W L, *Mayo Clinic Proceedings*, 1976, 51, 325.

³ Scott, F B, Bradley, W E, and Timm, G W, *Urology*, 1973, 2, 80.

⁴ Lash, H, Zimmerman, D C, and Loeffler, R A, *Plastic and Reconstructive Surgery*, 1964, 34, 75.

⁵ Loeffler, R A, Sayegh, E S, and Lash, H, *Plastic and Reconstructive Surgery*, 1964, 34, 71.

⁶ Pearman, R O, *Journal of Urology*, 1972, 107, 802.

⁷ Small, M P, Carrion, H M, Gordon, J A, *Urology*, 1975, 5, 479.

⁸ Small, M P, *Mayo Clinic Proceedings*, 1976, 51, 336.

⁹ Furlow, W L, *Mayo Clinic Proceedings*, 1976, 51, 341.

Animal ringworm

Ringworm fungus infections may be divided into two groups: those which infect only man (anthropophilic species) and those which primarily infect animals, when human infection is acquired from the animal sources (zoophilic species). The three common animal species are cattle ringworm (*Trichophyton verrucosum*), cat and dog ringworm (*Microsporum canis*), and small mammal ringworm (*T mentagrophytes* var *granulare*).

In Britain the most common animal ringworm infection in man is that due to cattle ringworm. It usually infects exposed areas but any part of the skin may be affected. Patches have a red, scaly, and active edge and tend to clear in the centre—as with any ringworm infection. In addition, follicular pustules are often present, and these are prominent, particularly in hairy areas. The whole lesion is red, raised, and purulent (kerion), closely mimicking pyococcal infection. Cattle ringworm develops more slowly than impetigo and boils, taking a few weeks to reach the maximum response. The hair over the site can be removed easily and later falls out. The condition usually takes three to six months to resolve unless the patient is treated with griseofulvin.

Cat and dog ringworm causes a ringed lesion with an active edge, and, though it is not so inflamed as that due to cattle ringworm, there may be some follicular pustules. The scalp is commonly affected, when there are circular patches of redness and scaling with short broken hairs. These hairs will fluoresce under Wood's light in a similar way to that seen with the species causing human ringworm (*M audouini*), which up to three decades ago was so common.

Small mammal ringworm also produces lesions with an active edge and ringed patches. These are usually in exposed sites, particularly on the face, forearms, and lower legs. Commonly there are one or two patches, but the condition may be widespread. The various animals concerned (ranging from hedgehogs and voles to rats and mice) usually have their own

specific variety of fungus, which can be identified in culture. An interesting epidemiological survey of animal ringworm infection among agricultural workers by Chmel *et al*¹ has recently been reported from Bratislava. Mammals were trapped in different natural localities, and over two years 1288 animals of 13 species were examined mycologically. Ringworm fungi were isolated from 57, and it was found that the animals harbouring the fungus most commonly were voles (5.6%). Field mice and shrews (3.6%) and house mice (3%) were also quite frequently infected. The study did not include hedgehogs—a common source of ringworm infection in Britain. Morris and English² have shown that 20% of British hedgehogs harbour the fungus of hedgehog ringworm, *T mentagrophytes* var *erinacei*. British voles are commonly infected with *M persicolor* and infection with *T mentagrophytes* is very rare.³ Neither of these studies, however, refers to tame small mammals, which are one of the commoner sources of small mammal ringworm in patients other than agricultural workers.

Discussing their epidemiological findings, Chmel *et al* point out that the importance of these natural foci of infection depends on the density of distribution of the mammals concerned, their migration, and their close contact with man. Those which frequent barns and granaries are most likely to pass on infection. They also mention that of 445 infected workers working with animals 72% were infected with *T verrucosum* and 28% with *T mentagrophytes* var *granulare*, while of the 137 infected workers working with crops *T mentagrophytes* var *granulare* occurred in 77% and *T verrucosum* in only 32%. The small mammal ringworm apparently affects the face in 22% of men and only 6% of women, while the lower legs were affected in 26% of women and only 4% of men, presumably because in Bratislava women do not wear trousers.

¹ Chmel, L, Buchvald, J, and Valentova, M, *International Journal of Epidemiology*, 1976, 5, 291.

² Morris, P, and English, M P, *Sabouraudia*, 1969, 7, 122.

³ English, M P, and Southern, H N, *Sabouraudia*, 1967, 5, 302.

Insulin regimens for diabetic ketoacidosis

Recommendations on the use of insulin in treating diabetic ketoacidosis vary considerably as to the best route, dose, and timing. Recently the newer regimens of continuous intravenous infusion or regular hourly intramuscular injections of insulin have greatly simplified treatment. Previous uncertainties have been eliminated, because the treatment is now continuous and the dose of insulin the same for almost all patients.

The aim in giving insulin is to achieve an effective serum insulin concentration of 20 to 200 μ units/ml as quickly as possible.¹ Intravenous insulin does this most rapidly² and most reliably, but because the half life is short (roughly four minutes³) continuous infusion is needed to maintain an adequate serum concentration. Sönksen¹ first observed the effect of a small dose infusion in correcting ketoacidosis during insulin infusion studies. The technique is very simple, using either an infusion pump or the addition of insulin to the delivery chamber of a paediatric giving set⁴; no loading dose is needed. Intermittent bolus intravenous injections given hourly,⁵ or over 10-15 minutes every hour,⁶ are also effective,

but the blood glucose concentration falls more variably and slowly than during continuous insulin infusion. Intramuscular insulin is more slowly absorbed from the injection site: a loading dose should therefore be given, and thereafter (because of the longer half life by this route) hourly injections will maintain the correct serum concentration.^{7 8} Subcutaneous insulin is absorbed from the injection sites too slowly and erratically to be of value in treating very sick patients.²

Soluble insulin (or one of the neutral soluble insulins) should always be used for managing diabetic emergencies. The dose for correcting ketoacidosis is much smaller than was supposed,^{9 10} and insulin resistance is extremely rare. Most reports have described insulin infusion rates of between 2-4 and 12 units per hour^{4 11-15}, or 0.1 unit per kg per hour for children.^{6 16 17} These doses certainly achieve the required serum insulin concentrations and there is a remarkable consistency in the resulting blood glucose responses.^{11 12} Smaller amounts are less effective¹⁸ and inadequate for treating ketoacidotic patients. The recommended dose by intramuscular injection, which requires no special apparatus, is 5 units hourly preceded by a loading dose of 20 units.^{7 8}

Comparisons between these small dose insulin regimens and those using much larger amounts^{8 14} have shown that the blood glucose and ketone responses are very similar, with blood glucose falling by more than half of its initial concentration within four hours of starting treatment. Small doses have the advantage that hypokalaemia is less likely,^{7 8 14} though it may still occur if insufficient potassium is given. The rise in lactate concentrations observed after larger doses^{7 8 11} does not seem a problem with small-dose regimens, and late hypoglycaemia is also less common, though even in the past this was not an important complication.

The need for additives (albumin or polygeline) to the insulin solution to avoid excessive insulin loss by adsorption on to glassware and plastic has been much argued.¹⁹⁻²¹ This is unnecessary if excessively dilute insulin solutions (such as may result when small amounts are added to drip bottles) are avoided. At a concentration of 1 unit/ml of soluble insulin in saline, which is suitable for use in an infusion pump, the loss is negligible: in a comparison of glucose and ketone responses with and without added albumin no difference was detected.^{11 22} Even at an insulin concentration of 1 unit in 10 ml saline the loss is still unimportant.²⁰ The addition of albumin or polygeline or even small amounts of the patient's own blood²¹ makes a simple treatment more complicated. It is better to use insulin at an adequate concentration without any additive.

These new insulin regimens are, then, simple, safe, and effective. The same dose may be used for all patients whether they have been previously treated with insulin, are ketoacidotic or not, and regardless of the initial blood glucose concentration. The presence of infection causes a small decrease in the rate of fall of blood glucose concentrations,^{4 7 11} and patients without ketosis are rather more sensitive to insulin. Frequent biochemical analyses must still be performed, however, both to detect the patient who is unresponsive and to control the amount of potassium which needs to be given. But above all it is the simplicity and reliability of these new treatments which commend them to doctors, so that the reluctance of some authorities in the USA²³ to recommend them is surprising²⁴.

¹ Sönksen, P H, *et al*, *Lancet*, 1972, **2**, 155.

² Guerra, S M O, and Kitabchi, A E, *Journal of Clinical Endocrinology and Metabolism*, 1976, **42**, 869.

³ Sönksen, P H, *et al*, *Clinical Science and Molecular Medicine*, 1973, **45**, 633.

⁴ Campbell, L V, *et al*, *Medical Journal of Australia*, 1976, **2**, 519.

⁵ Clumbeck, N, *et al*, *Lancet*, 1975, **2**, 416.

⁶ Malleon, P N, *Archives of Diseases in Childhood*, 1976, **51**, 373.

⁷ Alberti, K G M M, Hockaday, T D R, and Turner, R C, *Lancet*, 1973, **2**, 515.

⁸ Alberti, K G M M, in *10th Symposium on Advanced Medicine*, ed J G G Ledingham, p 68. London, Pitman Medical, 1974.

⁹ Felig, P, *New England Journal of Medicine*, 1974, **290**, 1360.

¹⁰ Genuth, S M, *Journal of the American Medical Association*, 1973, **223**, 1348.

¹¹ Page, M Mc B, *et al*, *British Medical Journal*, 1974, **2**, 687.

¹² Kidson, W, *et al*, *British Medical Journal*, 1974, **2**, 691.

¹³ Semple, P F, White, C, and Manderson, W G, *British Medical Journal*, 1974, **2**, 694.

¹⁴ Kitabchi, A E, Ayyagari, V, and Guerra, S M O, *Annals of Internal Medicine*, 1976, **84**, 633.

¹⁵ Soler, N G, *et al*, *Lancet*, 1975, **2**, 1221.

¹⁶ Kaufman, I A, Keller, M A, and Nyhan, W L, *Journal of Pediatrics*, 1975, **87**, 846.

¹⁷ Moseley, J, *British Medical Journal*, 1975, **1**, 59.

¹⁸ Schade, D S, and Eaton, R P, *Diabetologia*, 1976, **12**, 417.

¹⁹ Kraegen, E W, *et al*, *British Medical Journal*, 1975, **3**, 464.

²⁰ Weisenfeld, S, *et al*, *Diabetes*, 1968, **17**, 766.

²¹ Sönksen, P H, *British Medical Journal*, 1976, **1**, 151.

²² Oakley, W G, Pyke, D A, and Taylor, K W, *British Medical Journal*, 1976, **1**, 279.

²³ Madison, L L, *New England Journal of Medicine*, 1976, **294**, 393.

²⁴ Page, M Mc B, *et al*, *New England Journal of Medicine*, 1976, **294**, 1183.

Ascaris infection

Ascaris lumbricoides has a world-wide distribution with a peak age-specific prevalence in the tropics in children between 3 and 8 years. Infection is acquired by ingestion of eggs contaminating food or drink. During the migrating phase the larval stages may give rise to a pneumonitis 4-16 days after infection, with fever, cough, expectoration, eosinophilia, and pulmonary infiltration; larvae may be found in the sputum. How important this larval migratory stage may be as a cause of morbidity and mortality in tropical areas is difficult to assess.¹ Established infection, however, gives rise to a variety of clear-cut symptoms and signs. The presence of the adult worms (about 20 cm long) in the small intestine results in gastrointestinal discomfort and vomiting, but the most important result of a heavy ascaris infection is small bowel obstruction. This complication usually occurs in children but may occasionally present in adults²; as many as 1000 worms weighing a total of 4 kg have been extracted from a single patient.³

Migration of the adult worm may occur to almost anywhere in the body, but especially in and about the gastrointestinal tract. Complications may include volvulus and gangrene of the bowel, intussusception, appendicitis, intestinal perforation and peritonitis, pancreatitis, pyogenic cholangitis, acute cholecystitis, obstructive jaundice, and perforation of the bile ducts. Adult worms have also been described in the eustachian tube, the paranasal sinuses, and even within the cavity of the heart itself. Both granulomatous peritonitis and hepatitis have been ascribed to the presence of ascaris ova.

The diagnosis of ascaris infection usually depends on finding the characteristic eggs in the stools. Occasionally the diagnosis may be established by chance by radiological examination of the abdomen, with or without barium. Plain films show the worms as linear or round opacities surrounded by gas, while in a barium study the worms usually appear as tubular filling defects surrounded by contrast material.

By choice the treatment of intestinal obstruction due to ascaris worms should be conservative, with intravenous fluids and nasogastric suction followed by the appropriate anthelmintic⁴ when the acute symptoms and signs have subsided. Other complications are treated by conventional means.