

hormones accounts for the weak thyroid stimulating activity of very large quantities of HCG.

Undoubtedly, then, hyperthyroidism in association with hydatidiform mole results from the very high levels of HCG found in this condition, and this association is probably more frequent than has been generally recognised. A detailed and systematic study of patients with trophoblastic tumours would provide further data on the true frequency and clinical importance of this association. The frequency of clinical hyperthyroidism in women with moles is uncertain. Higgins *et al*<sup>11</sup> found clinical evidence of hyperthyroidism in nine of their 14 patients and biochemical hyperthyroidism in one of the remainder. Galton and her colleagues,<sup>12</sup> however, found increased thyroid function in all 11 patients studied, but clinical hyperthyroidism was not seen in any of this group. These differences may be attributed to patient selection or to variable sensitivity to this thyroid stimulator. Clearly this is an association which must be suspected in all cases, but particularly<sup>11</sup> when serum levels of HCG exceed 300 IU/ml.

<sup>1</sup> Hoffenberg, R, *British Medical Journal*, 1974, 3, 452.

<sup>2</sup> Tisné, L, Barzelatto, J, and Stevenson, C, *Boletín de la Sociedad Chilena de Obstetricia y Ginecología*, 1955, 20, 246.

<sup>3</sup> Steigbigel, N H, *et al*, *New England Journal of Medicine*, 1964, 271, 345.

<sup>4</sup> Winand, R, *et al*, *Journal of Clinical Endocrinology*, 1969, 29, 1369.

<sup>5</sup> Odell, W D, *et al*, *Journal of Clinical Endocrinology*, 1963, 23, 658.

<sup>6</sup> Hershman, J M, and Higgins, H P, *New England Journal of Medicine*, 1971, 284, 573.

<sup>7</sup> Kock, H, *et al*, *Journal of Clinical Endocrinology*, 1966, 26, 1128.

<sup>8</sup> Cohen, J D, and Utiger, R D, *Journal of Clinical Endocrinology*, 1970, 30, 423.

<sup>9</sup> Kenimer, J G, Hershman, J M, and Higgins, H P, *Journal of Clinical Endocrinology*, 1975, 40, 482.

<sup>10</sup> Odell, W D, Wilber, J F, and Paul, W E, *Journal of Clinical Endocrinology*, 1965, 25, 1179.

<sup>11</sup> Higgins, H P, *et al*, *Annals of Internal Medicine*, 1975, 83, 307.

<sup>12</sup> Galton, V A, *et al*, *Journal of Clinical Investigation*, 1971, 50, 1345.

## An unnecessary risk to children

In England and Wales alone during the three years 1971-3 there were 102 deaths and 10 783 injuries among children under the age of 14 years who had been travelling in the front passenger seat of cars and light vans. The annual death rate was relatively constant—being 37 in 1971, 28 in 1972, and 37 in 1973.

The front passenger seat of a car is known to be the most dangerous position in the vehicle. Deaths and injuries from road accidents are specially distressing in children, yet the front passenger seat is a favourite with youngsters, who are frequently indulged by well-meaning parents who seem unaware that they are exposing them to an unnecessary risk. Small children are often to be seen actually standing on the front seat squab or on the floor, gripping the fascia panel below the windscreen, being thus completely vulnerable to a deceleration impact. Furthermore, it is equally common to see other adult passengers, especially the mother, sitting in the back seat, so completely reversing the logical safety procedure. To make a considerable risk worse, seat belts are rarely used by children; and, even when they are, the adult fixation-point and their general unsuitable position probably make their use far less efficient for a small child than for an adult.

Nevertheless, of the 102 child deaths only eight occurred in children using a seat belt. Of the injuries, 885 occurred in children using seat belts and 9898 in cases where no belt was

used. In the absence of any figures showing how many uninjured children did or did not wear seat belts the figures for death and injury can only be a rough guide, but they do strongly suggest that, though the use of an adult safety belt by a child may not be mechanically ideal, many deaths and injuries in England and Wales are attributable to failure to require children to wear seat belts. This is all the more tragic since it could be prevented by discouraging or even prohibiting placing a child in this most dangerous of positions. Children prefer travelling in the front seat, but it is one where a child must be at a considerably greater risk from disadvantages owing to immaturity (they have not the adult anticipation of an accident) and physical size (bracing against the floor and fascia is less effective).

More attention should surely be drawn to this dangerous habit. Government publicity about its hazards could possibly be incorporated into the propaganda now being given to encourage wearing seat belts by adults. Several Continental countries, such as France, have recognised the risk and have introduced legislation prohibiting the practice. The high incidence of death and injuries from accidents to children in the front of cars seems hitherto to have received virtually no publicity: the case for action is compelling.

## $\beta$ -blockers in the treatment of chronic simple glaucoma

Chronic simple (open angle) glaucoma has presented an enigma over the years, and there have been several different approaches to its treatment. Pilocarpine, the mainstay of treatment for so long, has some drawbacks. It is successful in lowering the intraocular pressure, but the contraction of the pupil interferes with vision, especially in patients with central lens opacities, and the spasm of the ciliary muscle causes transient myopia and disturbance of accommodation. In consequence, other fields of pharmacology have been explored.

Adrenergic drugs are effective in reducing intraocular pressure,  $\alpha$ -agonists by increasing the outflow of aqueous humour from the eye and  $\beta$ -agonists by reducing its secretion. Adrenaline has established a place in open angle glaucoma therapy, either alone or in combination with guanethidine, which enhances its effect. The  $\beta$ -agonists isoprenaline and salbutamol have also been used to reduce the intraocular pressure, but the response declines with repeated doses and this tachyphylaxis usually occurs within a few weeks. Paradoxically the  $\beta$ -blocking agents also cause a fall in intraocular pressure when administered either locally or systemically. Their exact mode of action is unknown; it may be due to a mechanism other than that of  $\beta$ -blockade, such as a membrane-stabilising effect or an intrinsic  $\beta$ -mimetic action.

This ocular hypotensive effect was first reported with propranolol.<sup>1</sup> Practolol, pindolol, and atenolol have all been found to have a similar action. Propranolol, as drops, acts as a local anaesthetic, which precludes its use as a long-term topical agent. Pindolol does not suffer from this drawback and has been used topically to treat open angle glaucoma for up to one month. On short-term trial<sup>2</sup> the average drop in intraocular pressure was 7 mm Hg, which makes the effect comparable with that of adrenaline. In general, this group of drugs appear to be free of serious toxic effects—with the exception of practolol, which causes mucocutaneous reactions and dry eyes in a

small proportion of cases and must therefore be used with extreme caution.

One group of patients who form an obvious target for this type of treatment are those with both chronic simple glaucoma and systemic hypertension. If the blood pressure is suddenly reduced in these patients without attention to the intraocular pressure they may suffer a sudden deterioration in their fields of vision from embarrassment of the circulation in the choroidal vessels supplying the optic disc and nerve. When  $\beta$ -blockers are taken by mouth they reduce both systemic blood and intraocular pressures and so should be the drug of choice in these patients.

The  $\beta$ -blocking agents have yet to be proved effective in lowering intraocular pressure on a long-term basis. They form an interesting group which may one day find an established place in treating chronic simple glaucoma, but much work has still to be done.

<sup>1</sup> Phillips, C I, Howitt, G, and Rowlands, D J, *British Journal of Ophthalmology*, 1967, 51, 222.

<sup>2</sup> Bonomi, L, and Steindler, P, *British Journal of Ophthalmology*, 1975, 59, 301.

## Reservoir ileostomies

A complete proctocolectomy for granulomatous disease of the large bowel which has failed to respond to medical treatment or has led to severe local or systemic complications relieves what is often a life-threatening condition. Yet with it the operation brings the inevitability of a permanent ileostomy, which, despite the existence of modern appliances, still represents considerable disability. The patient's fear of an ileostomy may mean delay in accepting operation or the use of less radical surgery, with preservation of the rectal stump and anal sphincter and the risk of recurrence of the disease in this residuum of the large bowel.

In an attempt to overcome this disability, since 1969 Nils Kock<sup>1</sup> of Gothenburg has been trying to develop a continent intra-abdominal reservoir fashioned from the terminal ileum. With his present technique<sup>2</sup> a pouch is fashioned from the distal 40 to 50 cm of ileum, and the end is formed into an intussuscepted nipple some 5 cm long. The stoma is brought out almost flush with the skin. In successful cases the patient need wear no appliance but empties the pouch with a catheter several times a day. In Kock's 37 patients with exit conduits with valves, perfect functional results were obtained in every case.

In reviewing<sup>3</sup> this subject in 1974 we emphasised that the technique still required evaluation, and a detailed and thoughtful paper of Goligher's current experience with 26 patients in Leeds is therefore timely.<sup>4</sup> Two, or possibly three, of the patients were considered to have some form of Crohn's disease while the rest had ulcerative colitis. Thirteen had the pouch prepared at the time of total colectomy, carried out during a period of complete remission, and 13 were conversions from previous conventional ileostomies. All patients were in excellent health at surgery. In 17 patients a conduit with valve was fashioned, as in the most recent of Kock's techniques, and indeed at Leeds the intussusception valve seemed vital: none of the patients without a valve achieved full continence. There were no operative deaths, but no fewer than eight of the 26 patients had quite serious complications, including one example of necrosis of the entire reservoir, three faecal fistulae, three cases of obstruction, and one of volvulus of the reservoir. Five patients had to have the reservoir excised. Of the reservoirs

made with a nipple valve, nine achieved almost perfect results.

Total colectomy presents a tremendous burden to the patient, and inevitably a wave of enthusiasm will follow the introduction of any new operation that aims to ameliorate the disability of permanent ileostomy. Yet the Kock reservoir procedure, which many surgeons are now attempting, must still be regarded with caution. It should certainly be reserved for patients in good general condition and avoided in the seriously ill and those taking steroid drugs, since the greater hazards of the procedure compared with conventional ileostomy may tip the scales against the patients with fulminating disease. The operation should also be avoided in Crohn's disease, because of the risk of subsequent extension to the reservoir bowel. The patient must face the risks of a wide variety of complications and the prospects of further surgery. These are high enough even in skilled hands, and must prove greater still when carried out by less experienced surgeons. For the time being, perhaps, use of the technique should be confined to those patients with a poorly functioning ileostomy in whom conventional methods of control have proved of no avail; and in these circumstances the patient might best be referred to a surgeon with at least some experience of this new operation.

<sup>1</sup> Kock, N G, *Archives of Surgery*, 1969, 99, 223.

<sup>2</sup> Kock, N G, *Progress in Surgery*, 1973, 12, 180.

<sup>3</sup> *British Medical Journal*, 1974, 3, 592.

<sup>4</sup> Goligher, J C, and Lintott, D, *British Journal of Surgery*, 1975, 62, 893.

## Control of smallpox

When the World Health Organisation scheme for smallpox eradication was started in 1967 the disease was thought to be endemic in 30 countries of the world. By the end of 1975 transmission was probably limited to Bangladesh and Ethiopia. The likelihood of smallpox being imported into Britain has been greatly reduced, but the possibility remains until world eradication has been achieved. At the same time the proportion of the British population who have ever been vaccinated has steadily decreased; so there must be a plan of action which can be started without delay should smallpox appear in this country. There have in fact been 310 notifications of variola major in Britain with 57 deaths since 1950.

Such a plan rests on half a dozen clinical observations. A patient is infectious from about the beginning of symptoms until all crusted lesions have disappeared; the incubation period very rarely lies outside 7-16 days and is most commonly 12 days; the virus can remain viable outside the host on inanimate objects such as clothing and bedding; the closer the contact with an infectious person the more likely is transmission; the protection offered by vaccination diminishes rapidly as the interval since exposure lengthens and is virtually absent after three days; and, finally, human antivaccinia immunoglobulin offers immediate passive protection but is in short supply, and there is evidence that the antiviral agent methisazone may confer some temporary protection also.

The clinical diagnosis of smallpox may be difficult, especially in vaccinated persons; electron microscopy can identify a poxvirus and can identify the herpes virus of chickenpox, but it cannot differentiate between the various poxviruses—smallpox, vaccinia, cowpox, and monkeypox. Cultivation in hens' eggs can separate the various poxviruses, and final proof may be obtained by testing the cultivated virus against specific antisera, but all that takes some days.