distributions of blood pressure might be more susceptible to the hypertensive effects of oral contraceptives: studies in the United States found higher mean blood pressures in black compared with white adults, and in England in both factory workers[13] and civil servants[14] black men had higher mean blood pressures than white men. In our study, however, we found no differences between black and white women, though with the numbers available a 5 mm difference would have been significant. Few studies have examined the effect of different contraceptives on different ethnic groups. In 2676 black women attending a family planning clinic in Atlanta no significant differences in mean blood pressure were found between those using oral contraceptives and controls.[15 16] In our study no significant differences in systolic blood pressure were detectable, and diastolic pressures were virtually identical. Though several explanations are possible—among them selection bias—there is no evidence that black women are particularly susceptible to the hypertensive effects of oral contraceptives.

The differences in mean blood pressure documented here were small (around 5 mm Hg) but are of interest both in investigations of the aetiology and mechanisms of the rise in blood pressure and in terms of community impact. In a population in which a large proportion of healthy women are using oral contraceptives a small shift in the distribution of a risk factor may have a large impact on the community's overall burden of disease,[17] and there is no doubt that raised blood pressure is one of the major factors in morbidity and mortality in most societies. Though different effects of oral contraceptives cannot be considered in isolation, any oral contraceptive that can be shown to have a less adverse effect on blood pressure has implications for general prescribing policy. Within this context even the small but important differences in the progestogen contents of low-dose oestrogen pills may constitute differences in risk that, though insignificant to the individual, may be of considerable importance to the community.

We thank Dr P Thompson, specialist in family planning, North-west District, and staff of Raymede and Queens Park family planning clinics.

References

1 Fisch IR, Freedman SH, Myatt AV. Oral contraceptives, pregnancy and blood pressure. *JAMA* 1972;**222**:1507-10.
2 Royal College of General Practitioners. Oral contraception study. Effect on hypertension and benign breast disease of progestogen component in combined oral contraceptive. *Lancet* 1977;i:624.
3 Royal College of General Practitioners. Hypertension. In: *Oral contraception and health.* Tunbridge Wells: Pitman Medical, 1974:37-42.
4 Weir RJ, Briggs E, Mack A, Naismith L, Taylor L, Wilson E. Blood pressure in women taking oral contraceptives. *Br Med J* 1974;i:533-5.
5 Pritchard JA, Pritchard SA. Blood pressure response to estrogen-progestin oral contraceptive after pregnancy-induced hypertension. *Am J Obstet Gynecol* 1977;**129**:733-9.
6 Meade TW, Haines AP, North WRS, Chakrabarti R, Howard DJ, Stirling Y. Haemostatic, lipid and blood pressure profiles of women on oral contraceptives containing 50 micrograms or 30 micrograms of oestrogen. *Lancet* 1977;ii:948-51.
7 Briggs M, Briggs M. Oestrogen content of oral contraceptives. *Lancet* 1977;ii:1233.
8 Spellacy WN, Birk SAA. Effect of intrauterine devices, oral contraceptives, estrogen and progestogens on blood pressure. *Am J Obstet Gynecol* 1972;**112**:912-9.
9 Rose G. Standardisation of observers in blood pressure measurements. *Lancet* 1965;i:673-4.
10 Weir RJ. Reduced hypertensive risks of low dose contraceptive steroids. In: Yamori Y, ed. *Prophylactic approach to hypertensive diseases.* Vol 4. New York: Raven Press, 1979;15-20.
11 Smith RW. Hypertension and oral contraception. *Am J Obstet Gynecol* 1973;**113**:482-7.
12 Kotchen JM, Kotchen TA, Cottrill CM, Guthrie GP Jr, Somes G. Blood pressures of young mothers and their first children three to six years following hypertension during pregnancy. *J Chron Dis* 1979;**32**:653-9.
13 Sever PS, Peart WS, Meade TW, Davies IB, Gordon D. Ethnic differences in blood pressure with observations on noradrenaline and renin. *Clin Exp Hypertens* 1979;**1**:733-44.
14 Marmot MG, Rose G. Epidemiology of hypertension. In: Sleight P, Jones JV, eds. *Scientific foundations of cardiology.* London: Heinemann (in press).
15 Hall WD, Douglas MB, Blumenstein BA, Hatcher RA. Blood pressure and oral progestational agents. *Am J Obstet Gynecol* 1980;**136**:344-8.
16 Blumenstein BA, Douglas MB, Hall WD. Blood pressure changes and oral contraceptive use: a study of 2676 black women in the south eastern United States. *Am J Epidemiol* 1980;**112**:539-52.
17 Rose G. Strategy of prevention: lessons from cardiovascular disease. *Br Med J* 1981;**282**:1847-51.

(*Accepted 24 June 1982*)

SHORT REPORTS

Avoidance of tracheostomy in sleep apnoea syndrome

Obstructive sleep apnoea (the Pickwickian syndrome) as a cause of daytime hypersomnolence, intellectual deterioration, and eventual respiratory failure with cor pulmonale and death is well recognised.[1] Tracheostomy may be life saving and is the treatment of choice in some centres, although other methods have been tried with limited success.[1] It is, however, a drastic treatment with medical and psychological implications and if possible should be avoided.

Case report

A 37-year-old company director presented with obesity and hypersomnolence. At the age of 9 years he had developed asthma, which had troubled him only intermittently. Eighteen months before presentation he had begun to experience disturbed sleep and hypersomnolence, and his weight had increased from 83 to 121 kg. He had consulted a neurologist and had since been taking amphetamines for a diagnosis of narcolepsy. His weight and symptoms had gradually increased. His main complaint was of irresistible somnolence, repeatedly falling asleep in the middle of important business meetings and while driving (crashing his car and injuring his daughter). He had never fallen asleep while standing up. His performance at work had deteriorated and the company was now in serious financial trouble. Associated features were morning headaches and loud snoring.

Examination disclosed no abnormality apart from drowsiness, obesity, a considerably deviated nasal septum partially blocking both nasal passages, and large tonsils. Spirometry showed moderate, reversible airways obstruction (forced expiratory volume in one second 2·1 l, vital capacity 4·8 l). Results of routine haematology, biochemistry, chest radiography, and electrocardiography were normal. Arterial oxygen tension was 11·5 kPa (86 mm Hg) and carbon dioxide tension 5·0 kPa (38 mm Hg). Overnight monitoring showed periods of obstructive apnoea during sleep producing swings of arterial oxygen saturation from 94% to 72%. Arousal occurred only occasionally at the nadir of the drops in arterial oxygen saturation.

Because of the relative mildness of his apnoea and his inability to lose weight protriptyline 60 mg/day was tried for one month but produced no benefit. Medroxyprogesterone acetate 50 mg nightly produced initial improvement followed by an appreciable decline. These drugs have been reported as beneficial in some cases of sleep apnoea syndrome.[1] Repeat overnight monitoring showed recurrent 40-second obstructive sleep apnoeas throughout the night, accompanied by arterial oxygen desaturation to 65%, arousal on each occasion, and heart rate oscillating between 85 and 125 beats/min.

Submucus resection and tonsillectomy were performed to reduce his upper airways resistance. He suffered obstructive apnoea at induction but intubation was accomplished without difficulty. No narcotics were used and the endotracheal tube was left in place for 18 hours postoperatively. Despite this he slept for long periods with intermittent nitrous oxide as the only analgesia. The next day he was fully alert; the hypersomnolence did not return.

Eighteen days later he was fully recovered. He had discovered business errors made in recent months, which he was trying to correct. Overnight monitoring showed no sleep apnoea, but snoring persisted. A tracing of arterial oxygen saturation showed small oscillations (< 2%). There were no recurrent arousals as before. In six weeks he lost 15 kg in weight; the snoring subsequently disappeared.

Comment

In normal sleepers the pharyngeal dilator muscles maintain lumen patency by opposing the negative intra-airway pressures during inspiration,[2] but in the adult sleep apnoea syndrome the respiratory nervous output to these pharyngeal openers is inadequate and airway collapse occurs, causing obstructive apnoea.[2 3] A pre-existing small pharyngeal lumen encourages this collapse, and weight loss with reduction of fat in the pharyngeal area may be helpful. In these patients weight loss is often extremely difficult, but after surgical cure weight is more easily shed,[1] perhaps due to increased physical activity.

Increased upstream resistance during inspiration will further lower intrapharyngeal pressure and increase the chances of collapse.[4 5] We suspect in this patient that the considerably deviated nasal septum, and possibly the tonsils, acted as added upstream resistance. The reduction of this resistance was enough to prevent actual apnoea, although not the snoring. His subsequent weight loss further reduced the tendency to pharyngeal collapse and abolished the snoring. This case suggests that tracheostomy and other complex operations for sleep apnoea may not be necessary.

[1] Guilleminault C, Hoed J, Mitler MM. Clinical overview of the sleep apnea syndromes. In: Guilleminault C, Dement WC, eds. *Sleep apnea syndromes*. New York: Alan R Liss Inc, 1978:1-12.

[2] Hill MW, Guilleminault C, Simmons FB. Fibreoptic and EMG studies in hypersomnia-sleep apnea syndrome. In: Guilleminault C, Dement WC, eds. *Sleep apnea syndromes*. New York: Alan R Liss Inc, 1978:249-58.

[3] Borowiecki B, Pollak CP, Weitzman ED, Rakoff W, Imperato J. Fibreoptic study of pharyngeal airway during sleep in patients with hypersomnia obstructive sleep apnea syndrome. *Laryngoscope* 1978;**88**:1310-3.

[4] Sukerman S, Healy GB. Sleep apnea syndrome associated with upper airway obstruction. *Laryngoscope* 1979;**89**:878-85.

[5] Anch AM, Remmers JE. Nasopharyngeal resistance in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1981;**123**, suppl:185.

(*Accepted 11 May 1982*)

Department of Respiratory Medicine, Hammersmith Hospital, London W12 0HS

J R STRADLING, BSC, MRCP, registrar

Suicidal insulin overdose managed by excision of insulin injection site

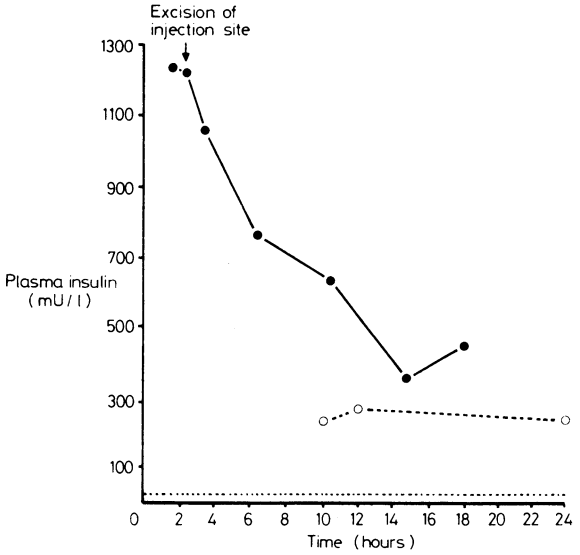
Deliberate suicide or "parasuicide" (self-poisoning) with insulin in diabetics is uncommon in any individual physician's experience but recent reports suggest that it might be more common than previously appreciated.[1-3] The management of hypoglycaemia in such patients may be difficult, especially if a long-acting or depot insulin has been injected and also if the ingestion of excessive alcohol accompanies the overdose. We report the management of a case of suicidal insulin overdose in which the insulin injection site was excised completely in an attempt to remove the depot insulin.

Case report

A 24-year-old man had had insulin-dependent diabetes for eight years which since diagnosis had been controlled with soluble and protamine zinc insulin. He referred himself to hospital about 45 minutes after deliberately injecting subcutaneously into the lateral aspect of left thigh the contents of two 2 ml British Standard insulin syringes (BS 1619), one containing insulin injection BP (soluble) and the other protamine zinc insulin injection BP. The site of injection looked erythematous and macular. He admitted pulling the plunger to the very top of the syringe, beyond the 2 ml graduation mark, and was reckoned to have injected 200 units of soluble and 200 units of protamine zinc insulin injection. He had recently lost his job, a police case was pending on an assault charge, his wife had left him, and he was an alcoholic. He had quickly regretted his suicidal action, however, and rapidly sought medical advice. He had eaten no food for 12 hours but in the four hours before referral had drunk six pints (3·4 l) of lager.

On admission the patient was conscious, had no focal neurological signs, but was sweating, and hypoglycaemia was confirmed with a plasma glucose concentration of 1·4 mmol/l (25·2 mg/100 ml). A solution of 50% dextrose (20 ml) was given intravenously and an intravenous infusion of 20% dextrose, 0·5 l two-hourly, begun. A venous sample was removed one hour after admission and the plasma stored at −20°C for insulin determination at time 0, followed by further plasma samples at 0·5 h, 1·5 h, 4·5 h, 8·5 h, 13·0 h, and 16·5 h. Just before the 0·5 h sample the site of injection was infiltrated with 1% lignocaine hydrochloride and a block of tissue about 5 cm long by 2 cm wide by 2 cm deep excised, removing skin and subcutaneous fat down to muscle, and the wound easily sutured with subsequent uneventful healing. After six hours the 20% dextrose infusion rate was reduce to 0·5 l four-hourly for eight hours and thereafter 5% dextrose infused four-hourly for 24 hours. His usual insulin regimen was then reinstituted. During the 24-30 hours after admission the plasma glucose concentrations were checked hourly for the first four hours, two-hourly for the next four hours, then four-hourly for 24 hours (BM-Test-Glycemie 20-800) and values between 7 and 22 mmol/l (126 and 396 mg/100 ml) obtained. The patient remained conscious and made a full recovery.

Insulin concentrations were measured by double-antibody radioimmunoassay using plasma diluted greater than 16-fold. At these dilutions the endogenous insulin-binding activity in the plasma was shown to be insignificant and did not invalidate the insulin assay. Insulin concentrations at 0, 0·5, 1·5, 4·5, 8·5, 13·0, and 16·5 hours were 1240, 1220, 1061, 764, 643, 352, and 452 mU/l, respectively (see figure). The immediate effect of excision was to reduce the plasma insulin concentration from 1220 to 1061 mU/l. Plasma insulin, however, declined more rapidly after excision (half time for disappearance about 10 hours) than before (half time about 18 hours). Both these half times for disappearance were considerably longer than in normal subjects and might have been related to the buffering effect of insulin antibodies, which were shown to be present in moderate concentration by a second antibody co-precipitation technique (IgG insulin-binding capacity 18·4 μg/l).[4] This binding value was very close to the median for a population of patients maintained with soluble and protamine zinc insulins.[5] Six weeks later plasma insulin concentrations were 239, 283, and 243 mU/l 10, 12, and 24 hours after normal insulin replacement with 12 units soluble and 68 units protamine zinc insulin (figure). The constancy of plasma insulin concentrations presumably again reflected the buffering effect of insulin antibodies.



Plasma insulin concentrations after insulin overdose (● ——— ●). Zero time refers to time of injection. Injection site excised about 2·25 h after injection. Plasma insulin concentrations after normal insulin replacement six weeks later also shown (○ — — — — ○). Zero time refers to time of injection of normal insulin-replacement dose. Lower horizontal interrupted line indicates upper limit of fasting insulin concentrations in normal non-diabetic subjects.

Comment

Suicidal insulin overdoses are perhaps more common than previously appreciated. Four out of 204 severe hypoglycaemic episodes in one year in a casualty department were intentional overdoses and, though the patients survived, two of the patients subsequently committed suicide by other means.[1] Over two years (1978-9) 18 cases of deliberate self-poisoning with insulin were reported to the National Poisons Information Service.[2] Four of the diabetics died and three others sustained irreversible brain damage. In 1979, 17 (4%) of the 448 deaths of diabetics under 50 years of age in Britain were caused by hypoglycaemia and, though it was not stated how many were suicidal or self-poisoning episodes, most of the patients were noted to have personality or psychiatric problems and three were known alcoholics.[3]

The treatment of patients who have taken excessive doses of insulin