understand that the marks on the syringe represent 2 or 4 units according to the strength of insulin used. The illustration given makes the problem look simpler than it really is; for example, with U40 insulin and a 1-ml syringe graduated in tenths 14 units is 0·35 ml, which is more difficult to calculate and less accurate to measure than 7 marks on a standard insulin syringe.

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## Intravenous drug administration for domiciliary emergencies

SIR,—The problem of giving drugs intravenously for domiciliary medical emergencies such as status epilepticus or status asthmaticus is one that general practitioners often face. Many factors contribute, including holding the needle in a vein while refilling a syringe, changing the syringe to give an alternative drug, or awaiting a response with a view to further therapy.

Most practitioners are aware of the butterfly type of needle for the continuous infusion of intravenous fluids into a baby's scalp vein or small adult vein. Some perhaps are not aware that the same firm (Abbot Laboratories Ltd, Queenborough, Kent) markets a similar needle with a short extension tubing and reseal injection site at the end suitable for the intermittent administration of drugs. Once this type of needle has been placed in the vein and fixed with a small length of tape to the skin the drug can be injected through the reseal site and the syringe changed at leisure for the administration of further drugs.

I have found this type of butterfiy needle to be of great value when treating medical emergencies in the home; I recommend it as a solution to some of the problems encountered in these situations.

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## Occupational exposure to inhaled anaesthetics

SIR,—We were interested in the excellent method for sampling environmental pollution described by Dr H T Davenport and others (20 November, p 1219) but we were disturbed by their recommendation of a scavenging system using pipeline vacuum as this method was specifically not recommended by the Department of Health and Social Security[1] and the associated problems were pointed out in the advice given by the Association of Anaesthetists.[2]

There are several problems which can arise from the use of pipeline vacuum for scavenging patients' expired air. Accidental obstruction of the holes provided to reduce the vacuum will lead to the full vacuum pressure being applied to the patient within a few seconds. Problems may arise as the vacuum plant will not be designed to handle high concentrations of volatile anaesthetic agents or the increased volume load resulting from the use of high flows for scavenging. A final criticism of the system they describe is that the anaesthetist does not have a visible indication that the scavenging system is working.

A system which is closer to the recommendations of both the Association of Anaesthetists and the DHSS has been described.[3] The essential component is a "safety block" which includes a positive pressure relief valve and a 2-l reservoir bag which acts as a compliance to reduce peak flows and also gives the anaesthetist a visible indication that the scavenging system is working. The "safety block" is mounted in a prominent position on the anaesthetic machine or ventilator and connected to either a ducted expiratory valve or to the outlet of the ventilator. The final disposal of the scavenged gas can be either into the extract side of the theatre air conditioning system or to the outside of the building. In the latter case wind effects may necessitate the use of an active system to assist the flow of scavenged gas. The effective pressure and flow generated by such an extractor can be monitored and adjusted by observing the reservoir bag on the safety block. If there is no other alternative a less satisfactory solution is to attach a large-volume concentric T piece to the safety block, in which case the pipeline vacuum can be used to dispose of the gas, using a flow slightly greater than the patient's minute volume.[5]

A safety block can be used to provide a universal interface with the final disposal system and will give a continuous visual indication of the functioning of the complete scavenging circuit.

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[1] Department of Health and Social Security Health Circular HC(76)38. London, DHSS, 1976.
[2] Association of Anaesthetists of Great Britain and Ireland, *Anaesthesia*, 1975, **30**, 697.
[3] Bethune, D W, Collis, J M, and Latimer, R D, *Anaesthesia*, 1976, **31**, 1254.
[4] Vaughan, R S, *et al*, *Anaesthesia*. In press.

## Biguanides and lactic acidosis

SIR,—We would agree with three important points made by Drs E A M Gale and R B Tattersall regarding biguanide therapy in diabetics (23 October, p 972).

Firstly, the incidence of phenformin-associated lactic acidosis is frightening. In South Australia 15 cases presented over two years to a hospital which serves approximately 500 000.[1] Assuming 1% of the population to be having treatment for diabetes, one-half of these to be on drug therapy, and one-quarter of the drug takers to be taking phenformin the incidence of *known* cases of lactic acidosis is 12 per 1000 phenformin treatment-years. These patients have a mortality of 62·5%.

Secondly, it is difficult to screen out patients with conditions which might make them susceptible to biguanide-induced lactic acidosis. The difficulty arises because diabetic clinics are busy; diabetics frequently have renal, hepatic, or cardiovascular disease; the degree of organ dysfunction which should contraindicate biguanide therapy is not known; changes in organ function can occur in the interval between monitoring; and intercurrent illness or other drug therapy rapidly alters the situation. Even biguanide clinics with drug monitoring facilities would not solve all these problems.

Thirdly, the place of phenformin as a hypoglycaemic agent is questionable. Drs Gale and Tattersall state that "if a biguanide is indicated metformin should be used since it appears less likely to precipitate lactic acidosis." This is soundly based on epidemiological and physiological evidence. In France, where three times as much metformin was consumed as phenformin, there were 68 cases of phenformin-associated and four of metformin-associated lactic acidosis reported to mid-1975. Assuming that these figures reflect the incidence of lactic acidosis and that the patients on the two drugs are similar, the relative risk with metformin is 1/50th of that with phenformin. This suggestion of a decreased risk is supported by our physiological studies in normal and diabetic subjects (see table).

*Lactate half life (min) in normals and diabetics on biguanides*

| | Normals[2] | Diabetics[3] |
|---|---|---|
| Control .. .. .. | 9 | — |
| Metformin .. .. | 15 | 39 |
| Phenformin .. .. | 19 | 68 |

In normal subjects both biguanides decrease lactate clearance (increase the lactate half life) but phenformin has 1¼ times the effect of metformin.[2] In maturity-onset diabetics taking phenformin and metformin with equivalent diabetic control lactate clearance on phenformin was half of that on metformin.[3] The mechanism of the hypoglycaemic effect of the biguanides is unclear and indeed metformin and phenformin may differ in the degree of their effects on different organs. For example, phenformin is concentrated in the liver (the major lactate-removing organ) whereas metformin is not.[4] This may partly explain the observed differences in effects on lactate metabolism.

We agree that lactic acidosis is a distressingly frequent complication of biguanide therapy and that it is difficult if not impossible to screen out patients at risk. We also agree that phenformin should be withdrawn and that metformin should be used if a biguanide is indicated.

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[1] Wise, P H, *et al*, *British Medical Journal*, 1976, **1**, 70.
[2] Phillips, P J, *et al*, *Australian and New Zealand Journal of Medicine*, 1976, **6**, 174.
[3] Phillips, P J, and Edwards, J B, *Abstracts IXth International Diabetes Federation*. Amsterdam, Excerpta Medica, 1976.
[4] Beckman, R, *Diabetologia*, 1969, **5**, 318.

## Postcoital contraception

SIR,—In reply to Dr D J Hill's opinions (25 December, p 1562) I feel that some points need clarification.

The notion of a "great difference" between preventing fertilisation of the ovum and the use of an arbortifacient after fertilisation depends upon the following factors: (1) knowledge of the mode of action of postcoital contraceptives; and (2) the distinction between biological and human life.

With regard to the intrauterine contraceptive device (IUCD) there are reports suggesting that it may not be abortifacient. The production of an endometrium unsuitable for implantation has been observed but not proved. Some workers propose that the IUCD