of the paper was never clearly stated. It is clear enough to me that it meant to call into question the usefulness of widespread fetal monitoring and operative delivery. Dr J Selwyn Crawford gives his opinion that the writers "have done a grave disservice to current obstetric practice." I believe the reverse is the case.

Anyone who questions the omnipotence of the scientific god is likely to provoke a loud and immoderate reaction from the believers, but this phase will pass. Meanwhile, those of us who have genuine doubts based on experience of the clinical aspects of "fetal distress" should stand up and be counted. It is those who are performing large numbers of unnecessary operative deliveries who have done, and are doing, "the grave disservice" and who are devaluing obstetric practice in the eyes of the public.

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Caucasian or Europid?

SIR,—Dr Freedman establishes a case for Europid so strong as almost to preempt debate. Born a Scot, taught early that "we're a' Jock Tamson's bairns," former officer of an old and prestigious surgical club with the inspiring motto "Without frontiers," and having failed in much travel to perceive any clear difference in the characteristics of my fellow men on either side of many geographic frontiers, I am naturally suspicious of ethnic classifications while aware of their necessity. Caucasian is romantic, euphonious, mysterious, and misleading. Europid is brief, clear cut, and as precise as one can expect a designatory epithet in this wide field to be. If this debate ends in a vote mine (despite regrets at which I hint) would inevitably go to Europid, allowing Caucasian to withdraw into the nostalgic mists of linguistic and sociological pre-history.

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SIR,—Dr Bernard Freedman's paper (3 March, p 696) will not stimulate much debate—only applause. In case, however, there are critics who believe that any usage, no matter how slipshod or inaccurate, that has persisted for long enough becomes "accepted" and therefore acceptable I want to record my support for Dr Freedman. I have long skirmished against "Caucasian" as an obnoxious transatlantic invader, and I am delighted to see a weightier armament brought to bear. Some years ago I was able to persuade a young pathologist who had been to America to give up using the word on necropsy reports. I hope that Dr Freedman's article will persuade others to do likewise.

It is difficult for neologisms—except bad ones—to gain acceptance. The suggestion that "Europid" be substituted for "Caucasian" is a good one and deserves encouragement. It is correctly derived and sufficiently anglicised to avoid the implication of some sort of spuriousness that would be (wrongly) attached to its etymological equivalent "Europoid."

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Cardiac arrest following naloxone

SIR,—As Dr Francis M Cuss and others pointed out (4 February, p 363), their patient was suffering from alcoholic cardiomyopathy among other conditions all associated with cardiac arrhythmias. In addition, the metabolic state of the patient (known to be a crucial factor in arrhythmogenesis) was not given in any detail.

The papers cited in support of the hypothesis of a causal link between naloxone and arrhythmias describe postanaesthetic problems in patients with serious underlying cardiac disease, complicated anaesthetic regimens (with potentially arrhythmogenic drugs), or both¹; two healthy patients who died after anaesthesia reversed by naloxone but without evidence of cardiac rhythms during the acute event²; and a drug addict with the adult respiratory distress syndrome where nalorphine, but not the subsequent dose of naloxone, was closely followed by ventricular fibrillation.³

At the London National Poisons Information Service we have never been informed of such a suspected adverse reaction despite being contacted in about 800 cases of opiate poisoning each year, where our advice includes the use of naloxone. Moreover, no adverse reactions have been reported after using naloxone in treating alcoholic coma.⁴

So we hope that the title and conclusion of this report do not deter doctors from using naloxone, which is a lifesaving drug with one of the highest therapeutic ratios of drugs used in common medical emergencies.

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¹ Pallasch TO, Gill C. Naloxone associated morbidity and mortality. *Oral Surg* 1981;52:602-3.

² Andree RA. Sudden death following naloxone administration. *Anaesthesia and Analgesia* 1980;59:782-4.

³ Lawrence JR, Lee FR. Ventricular fibrillation after narcotic withdrawal. *Lancet* 1975;ii:717.

⁴ Dole VP, Fishman J, Goldfrank L, Khanna J, MacGivern RF. Arousal of ethanol-intoxicated comatose patients with naloxone. *Alcoholism: Clinical and Experimental Research* 1982;6:275-9.

Autoantibodies against human insulin

SIR,—The analogy between reovirus induced autoimmunity in mice and polyendocrine disease in man is intriguing,¹ but Dr Terence J Wilkin and Ms Nicholson (4 February, p 349) produce only slender evidence that "autoantibodies against human insulin" occur spontaneously in man.

Of the nine sera they describe, only four (from cases 1, 2, 3, and 5) contained relevant organ specific autoantibodies and showed preferential binding to human insulin with significant inhibition by that same material in their solid phase assay system. Serum from case 9, on the other hand, showed threefold greater binding to bovine than to pork or human insulin and sera from cases 6, 7, and 8 bound to all three ligands, raising the possibility that some of these antibodies may have been induced by exogenous insulin.² C peptide antibodies can provide a useful marker of proinsulin contaminated insulin administration³ provided that they are documented by their binding to C peptide presented within

the intact proinsulin molecule⁴ as opposed to free C peptide, which adopts a different conformation unrecognised by these antibodies.

Solid phase assays that immobilise antigens on plastic surfaces usually work well for large protein molecules, although the use of a second irrelevant protein—for example, bovine serum albumin or gelatin⁵—and the inclusion of detergent⁶ are often necessary to prevent components of sera or antiglobulins binding to residual sites on the plastic surface.⁷ Insulin may present special problems in view of its small size,⁸ and the differences in hydrophobicity of its species variants could be a factor in determining the binding characteristics of some sera to insulin coated polystyrene.

Thus the data presented by Dr Wilkin and Ms Nicholson require confirmation with an immunochemical fluid phase assay⁷ or a solid phase antibody class capture technique in which antiglobulin is immobilised on the plastic surface before incubation with test serum, with labelled insulin added in a final step.⁹

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¹ Reeves WG. The immunology of β -cell failure. A case for immune response genes? *Immunology Today* 1983;4:207-8.

² Reeves WG, Kelly U. Insulin antibodies induced by bovine insulin therapy. *Clin Exp Immunol* 1982;50:163-70.

³ Reeves WG. Immunology of diabetes and insulin therapy. In: Thompson RA, ed. *Recent advances in clinical immunology*. Edinburgh: Churchill Livingstone, 1983:183-220.

⁴ Reeves WG, Douglas CA. C-peptide antibodies induced by bovine insulin therapy. *Clin Exp Immunol* 1982;50:171-7.

⁵ Parsons GH, Jr. Antibody-coated plastic tubes in radioimmunoassay. In: Langone JJ, ed. *Methods in enzymology*. Vol 73. *Immunochemical techniques*. New York: Academic Press, 1981:224-39.

⁶ Nilsson R, Sjogren HO. Antigen-independent binding of rat immunoglobulins in a radioimmunoassay. Solutions to an unusual background problem. *J Immunol Methods* 1984;66:17-25.

⁷ Reeves WG. Insulin antibody determination: theoretical and practical considerations. *Diabetologia* 1983;24:399-403.

⁸ Butler JE. Adsorption of proteins on polystyrene. In: Langone JJ, ed. *Methods in enzymology*. Vol 73. *Immunochemical techniques*. New York: Academic Press, 1981:501-5.

⁹ Chantler S, Diment JA. Current status of specific IgM antibody assays. In: Voller A, ed. *Immunoassays for the 80s*. Lancaster: MTP Press, 1981.

*.*Dr Wilkin replies below.—ED, *BMJ*.

SIR,—I share both Dr Reeves's caution in interpretation and his concern for independent confirmation. Notwithstanding his second paragraph, however, Dr Reeves has tested personally all nine insulin autoantibody positive sera for bovine C peptide antibodies and found them, as we did, uniformly negative. Commercial (proinsulin contaminated) insulins are therefore unlikely to have caused these antibodies.

I recognise the problems of non-specific reactions of proteins on plastic and the need for careful control. The reactions we described were nevertheless antigen specific (no reaction against glucagon), antibody specific (no reaction with normal human serum or IgG fraction), and immunospecific (no signal with other than human γ globulin directed conjugates). All reactions could be inhibited in a dose dependent fashion but only by the species variant of insulin to which the serum bound. These data appear to satisfy fundamental criteria for immunological reactivity, but I remain open minded.

Confirmation of insulin autoantibodies by