ments were obtained in the absence of surgical stimulation. However, other investigations have demonstrated an increase in SVR and systemic arterial pressure during surgery, although anaesthesia was deepened with 67% nitrous oxide [1, 2] or with fentanyl 10 μg kg⁻¹ [3]. A further increase in SVR when comparing controlled with spontaneous ventilation has also been observed [1, 2]. Since the decrease in SVR may be counteracted during positive pressure ventilation or by sympathetic reflex responses during surgery, the value of propofol for controlled hypotensive techniques might be questionable.

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SUXAMETHONIUM-SENSITIVE GENOTYPES

Sir,—I read with interest the report in your journal from Whittaker and Britten in which they analysed the results obtained by the Cholinesterase Research Unit during the first 2.5 years following its transfer from Exeter to London [1]. However, their conclusion that almost 25% of referrals have an E⁺E⁺ genotype (103 out of 430) is utterly beyond belief. E⁺E⁺ can be ascribed to patients only following family studies in which either parents or children are shown to be E⁺E⁺, E⁺E⁻ or E⁻E⁻. It is rare that this situation occurs.

Whittaker and Britten suggest that, for each patient found to be E⁺E⁺, there are five who are E⁻E⁻. The sensitivity to suxamethonium of these two genotypes would be expected to be similar and, accordingly, the frequency with which they present to a cholinesterase unit should reflect their occurrence in the general population. The incidence of E⁺E⁺ in this country approximates to 1 in 25 [2]; if Whittaker and Britten are to be believed, that of E⁻E⁻ will be 1 in 5 and, consequently, that of E⁺E⁻ in 1 in 100. Since the observed incidence of E⁺E⁺ is less than 1 in 100,000 [2], such a proposition is plainly absurd.

I suspect that the authors of this article have attributed an E⁺E⁺ genotype to everyone with enzyme inhibitor numbers characteristic of the usual phenotype, but who have a low cholinesterase activity. If this is so they are seriously mistaken. In a hospital population most such patients are E⁺E⁻, with a reduced cholinesterase activity secondary to their illness. Some are E⁺E⁻ or E⁻E⁺, while only a small proportion are E⁺E⁺.

If the reports of the Cholinesterase Research Unit are to be taken seriously, great care is required in the interpretation of their laboratory findings. Arbitrary ascription of a genotype such as E⁺E⁺ without supportive evidence from family studies will result in incorrect advice being given to patients. Some will be labelled “suxamethonium sensitive” when they are not, causing them unnecessary worry which subsequently will be difficult to allay. In addition, such a policy risks engendering doubt in the minds of professional colleagues as to the validity of other aspects of the work of the Cholinesterase Research Unit.

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