CORRESPONDENCE

JOHANNESBURG A–D CIRCUIT SWITCH

Sir,—Humphrey, Downing and Brock-Utne (1980) are less than accurate in describing the destiny of expiration switched to inspiration in the Johannesburg Circuit. They vaguely refer to experimental evidence gathered “while studying a similar system”—evidence which simply does not tally with known pharmacokinetic principles.

With a fresh gas inflow of 70 ml kg\(^{-1}\) min\(^{-1}\) in a patient weighing 65 kg, the time constant is circuit volume (certainly no more than 6 litre) plus a large functional residual capacity of 3 litre, divided by the 4.5 litre min\(^{-1}\) gas flow; at most 2 min for 63% clearance and 6 min for 95% clearance. There is no possibility that washout could amount to “more than 10 min” in the given circumstances. I have just seen the Johannesburg Switch performing well in Holland and it would be a pity if its clinical use is discredited unfairly.

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REFERENCES

PLASMA CHOLINESTERASE AND MALIGNANT HYPERTHERMIA

Sir,—A surprisingly high frequency of the fluoride-resistant gene controlling plasma cholinesterase has been found in patients who survived malignant hyperthermia (Whittaker, Spencer and Searle, 1977) and in patients proved by in vitro tests of a muscle biopsy to be MH-susceptible (Ellis, Cain et al., 1978).

We have determined activity and genotype of plasma cholinesterase in 26 consecutive patients from 13 different families in Denmark, referred to the Danish Malignant Hyperthermia Investigation Unit for investigation of susceptibility to MH.

One patient had survived a severe attack of MH; six patients were referred because of spasm of the masseter muscle after succinylcholine and 19 patients were first degree relatives of patients who had died of MH. Susceptibility to MH was determined by in vitro investigation of a muscle biopsy according to the method described by Ellis, Harriman and colleagues (1978). Fifteen patients were found to be MH-susceptible and 11 patients were normal. Activity and genotype of plasma cholinesterase was determined as described by Viby-Mogensen and Hanel (1977), assessing the dibucaine, fluoride, chloride, scoline and urea numbers. The results are shown in table I. Normal cholinesterase activity and genotype (E\(^+\)E\(^+\)) was found in 24 patients. One patient who was MH-positive was either normal or heterozygous for the silent gene (E\(^+\)E\(^-\) or E\(^+\)E\(^+\)) and one patient who was MH-negative was heterozygous for the atypical gene (E\(^+\)E\(^+\)). In none of the patients was the fluoride-resistant gene found. (We did not look for the C\(^+\) variant of cholinesterase, but as none of the patients had higher than normal values of cholinesterase activity, it may be assumed that they were all normal (Harris et al., 1963).)

The difference between our results and those of Whittaker, Spencer and Searle (1977) and Ellis, Cain and others (1978) is a matter of speculation. There is apparently a difference in frequency of the fluoride-resistant gene between the two populations, the frequency being 0.5% in England (Whittaker, Spencer and Searle, 1977) and only 0.23% in Denmark (Hanel, Viby-Mogensen and Schaffalitzky de Muckadell, 1978) but this would not explain the higher frequencies of the gene reported. Ellis investigated 25 patients but did not state the number of families to which they belonged. A high frequency of abnormal genes found in one or two large families might explain the high frequency found in the total population investigated. However, Whittaker found this combination of genes in at least three and possibly five families. If we assume the MH-trait to be found in 1 of 14 000 persons and the frequency of the fluoride-resistant gene to be 0.23%, we should expect to find this combination of genes in one of six million persons, provided that no association exists between the genes.

In conclusion, our results do not indicate any association between the genes controlling susceptibility to MH and the fluoride-resistant gene controlling plasma cholinesterase in the Danish population.

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REFERENCES


Sir.—We would agree with Forster and others (1980) that midazolam 0.15 mg kg⁻¹ is insufficient to induce anaesthesia reliably in unpremedicated patients. However, they imply that 0.2 mg kg⁻¹ is satisfactory in 100% of such patients by referring to a study in which only 10 patients received that dose (Reves, Corssen and Holcomb, 1978). Neither the appropriate induction dose nor the degree of individual variation in response to this agent is yet established (Dundee, 1979). We have found 0.225 mg kg⁻¹ to be inadequate for induction of anaesthesia in unpremedicated patients and consider a dose of 0.3 mg kg⁻¹ to be a more realistic dose.

Furthermore, loss of the eyelash reflex may not be a reliable sign of induction of anaesthesia with midazolam. Conner and others (1978) found that loss of lid reflex was an unreliable sign of induction for patients in whom tracheal intubation was planned. Many of their patients made purposeful arm and hand movements after an apparently successful induction. We have frequently noted that unpremedicated patients who have received midazolam still respond to oral commands despite the loss of this reflex.

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REFERENCES