Sir,—Thank you for the opportunity to reply to Dr Chasapakis’s letter.

In our paper (Doenicke et al., 1970) we described the course of ChE activity after administration of aprotinin in 17 cases (healthy persons). After the initial ChE activity was determined in 6 of these cases aprotinin was injected i.v. and thereafter the ChE activity in vivo was assayed, using fresh blood samples. In 4 cases, 3 min after injection of aprotinin, a larger quantity of blood was withdrawn and the course of ChE activity determined in the serum of this quantity while it was stored in a water bath at 37°C.

In cases aprotinin (its quantity equal to 25,000 or 100,000 KIU in 3000 ml serum) was added to serum (in a water bath at 37°C) and its influence on ChE activity observed. The in-vivo/in-vitro arrangement (2nd group, 4 cases) and the in-vitro arrangement (3rd group, 7 cases) was used in order to eliminate the eventual influence of distribution and breakdown of aprotinin in the organism.

The results obtained out of all 17 tests corresponded to a loss of ChE activity between 5 and 16 per cent. The lowest activity level was reached 6–20 min after administration of aprotinin and initial values were regained after about 30 min.

We did not find the widely spread results that Dr Chasapakis and associates found when we used the method of Kalow and Lindsay (1955) to determine the ChE activity (standard deviation 1.6 per cent). But using the method of Caraway as Chasapakis did we got untypical results of ChE activity that could not be reproduced under standardized conditions (standard deviation 15.6 per cent). We discussed in detail in our paper the reasons why we consider the method of Caraway useless for studying the influence of aprotinin on ChE activity. Dr Chasapakis did not contradict this argument till now. So we don’t think that Dr Chasapakis’s letter is a response to our paper.

As described in our paper, we did not observe any influence of aprotinin on the action of suxamethonium. For theoretical reasons we consider it possible in cases in which there is a pathologically low degree of ChE activity. But then suxamethonium can cause prolonged apnoea in any case.

All the other points in Dr Chasapakis’s letter are discussed in our paper, too, and must not be repeated because there are no new aspects.

Another answer to the investigations of Chasapakis was given by Ambrus and associates (1970). They said that they have great reservations about the significance of the ChE changes in Chasapakis’s work.

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REFERENCES


CORRESPONDENCE

229

INFLUENCE OF APROTININ (TRASYLOL) ON THE ACTION OF SUXAMETHONIUM

Sir,—Prof. Doenicke and his associates, in their paper (Brit. J. Anaesth. (1970), 42, 948), found that aprotinin inhibited plasma cholinesterase activity by 5–16 per cent and not by about 25 per cent as we found in our study (Chasapakis et al., 1968).

The difference between the two sets of findings is, in our opinion, not only due to the different method used for the determination of ChE activity, but mainly because they measured the ChE activity in 14 cases only (6 healthy subjects and 8 patients during anaesthesia). We measured ChE activity in 28 patients, not a very large number either, and as we pointed out there was a big variation in the degree of fall in the enzyme levels from patient to patient in our study. In 4 patients of 28, the fall exceeded 50 per cent but in one the enzyme was lowered by 4.5 per cent only.

When the inhibiting effect of aprotinin on the ChE activity is so different from patient to patient, then determination of the effects of the drug in 14 cases permits one to express reservations about the validity of the results of Prof. Doenicke and colleagues. Nevertheless, they agree with us that aprotinin has an inhibiting effect on ChE activity, as we were the first to discover.

Concerning the possible influence of aprotinin on suxamethonium: due to its inhibitory effect on ChE activity, and hence prolonged apnoea, they state that they did not find any prolongation of apnoea when aprotinin was given and hence prolonged apnoea, they state that they did not find any prolongation of apnoea when aprotinin was given and hence prolonged apnoea, they state that they did not find any prolongation of apnoea when aprotinin was given.

And we ended our second paper (Chasapakis et al., 1968), but he did not include our answer (Chasapakis, 1969).

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REFERENCES


