

CORRESPONDENCE

PROTECTIVE EFFECT OF THIOPENTONE ON INDUCED MYALGIAS

Sir,—Graig (1964) observed that thiopentone 4.7 mg kg^{-1} induced a time-dependent protective effect on suxamethonium-induced myalgia, the protection having almost disappeared 5 min after the administration of the thiopentone.

We wished to see whether the protective effect of thiopentone is also dose-dependent. Male patients ($n = 150$: ASA I) aged 30–60 yr, undergoing orthopaedic surgery, were randomized to three groups of 50. Premedication was accomplished with atropine 0.6 mg and diazepam 0.3 mg kg^{-1} i.m. Anaesthesia was induced in the first group with 2.5% thiopentone 2.619 mg kg^{-1} taken as UD_{95} (the unconsciousness dose in 95% of patients (Stella, Torri and Castiglioni, 1979)), while 1.8 UD_{95} was used in the second group and 2.6 UD_{95} in the third group. The induction of anaesthesia was carried out according to Clarke and others (1968). After the first dose of suxamethonium 1.5 mg kg^{-1} was injected 1 min after the induction of anaesthesia, a maximum of two or three doses of suxamethonium 50 mg were administered. No non-depolarizing agent was used and the trachea was intubated in all patients. Anaesthesia was maintained with neurolept-analgesia supplemented with enflurane or halothane. No analgesic was administered in the period after operation and the patients who could do it were allowed to move about freely after the operation. At observation times of 24, 48, 72, 96 and 120 h after the end of operation the frequency of myalgia, their distribution between upper limbs, neck, chest, abdomen and lower limbs and intensity were evaluated according to the method described by Fry (1975). The intensity of myalgia was statistically evaluated by the F test for the analysis of variance. Other statistical evaluations were by the chi-square test with Yates' correction, taking the first group as a control.

Thiopentone 2.6 UD_{95} significantly ($P < 0.05$) decreased the frequency of myalgia 24, 48 and 72 h after the end of the operation. Thiopentone 1.8 UD_{95} decreased the frequency of neck myalgia 96 h after the end of the operation ($P < 0.05$), while thiopentone 2.6 UD_{95} decreased neck and chest myalgia 72 h after the end of the operation ($P < 0.01$). The average intensity of the myalgia was not influenced by the doses of thiopentone. In all groups the mean intensities of myalgia reached the greatest values 48 h after the end of the operation and decreased progressively thereafter. The frequency of patients without myalgia was lower (18%) after thiopentone UD_{95} and greater (34%), although statistically not significant, after 2.6 UD_{95} .

The present results show that increasing doses of thiopentone tend to decrease the anatomical distribution and the daily frequency of myalgia and increase the number of patients not complaining of myalgia without interfering with the intensity of suxamethonium-induced myalgia. The dose-dependent effects of thiopentone are in agreement with a post-synaptic blocking action of thiopentone as suggested by the *in vitro* studies of Kraunak, Pleuvry and Rees (1977) and Torda and Gage (1977) and also with the hypothesis of Bali, Dundee and Assaf (1975) who claim that thiopentone may make the muscle cell membrane less prone to the trauma caused by suxamethonium-induced fasciculations.

G. MANANI G. P. GIRON
L. GALZIGNA
Padova, Italy

REFERENCES

- Bali, I. M., Dundee, J. W., and Assaf, R. A. E. (1975). Immediate changes in plasma potassium, sodium and chloride concentrations induced by suxamethonium. *Br. J. Anaesth.*, **47**, 393.
- Clarke, R. S., Dundee, J. W., Barrow, D. W., and McArdle, L. (1968). Clinical studies of induction agents. XXVI: The relative potencies of thiopentone, methohexitone and propanidid. *Br. J. Anaesth.*, **40**, 593.
- Fry, E. N. S. (1975). Reduction of post-suxamethonium muscle pains. *Br. J. Anaesth.*, **47**, 1342.
- Graig, H. J. L. (1964). The protective effect of thiopentone against muscular pain and stiffness which follows the use of suxamethonium chloride. *Br. J. Anaesth.*, **36**, 612.
- Kraunak, P., Pleuvry, B. J., and Rees, J. M. H. (1977). *In vitro* study of interaction between i.v. anaesthetics and neuromuscular blocking agents. *Br. J. Anaesth.*, **49**, 765.
- Stella, L., Torri, G., and Castiglioni, C. L. (1979). The relative potencies of thiopentone, ketamine, propanidid, alphaxalone and diazepam. A statistical study in man. *Br. J. Anaesth.*, **51**, 119.
- Torda, T. A., and Gage, P. W. (1977). Postsynaptic effects of i.v. anaesthetic agents at the neuromuscular junction. *Br. J. Anaesth.*, **49**, 771.

PULMONARY DEADSPACE DURING INDUCED HYPOTENSION

Sir,—We would like to comment on several aspects of the recent paper by Khambatta, Stone and Matteo (1982) on pulmonary deadspace during induced hypotension with sodium nitropruside. In the introduction they claim that this has not been studied previously, yet later in the paper they refer to precisely such measurements made in an earlier study (Wildsmith, Drummond and MacRae, 1975). Their claim for priority is unjust.

We reported VD/VT values before, during and after induced hypotension with sodium nitropruside in 26 patients (14 breathing spontaneously and 12 artificially ventilated) anaesthetized with halothane. Our study showed a small, but statistically significant, increase (from 49% to 54%) in VD/VT in artificially ventilated patients when hypotension was induced. They have reported much lower values for VD/VT (33%) with no change when hypotension was induced in six artificially ventilated patients, also anaesthetized with halothane.

They suggest that our values for VD/VT were greater than theirs because our patients probably had pulmonary disease. That was not the case, and we would point out that our values are of the same order as those quoted elsewhere (Nunn, 1977) for supine patients anaesthetized with halothane and where no correction is made for apparatus deadspace. Khambatta, Stone and Matteo (1982) give no indication of whether they corrected for apparatus deadspace or for the compressible volume of the ventilator tubing. Further, they neglected to consider a number of factors that influence VD/VT and which might explain some of the differences between the two studies.

First, their patients were younger and would be expected to have smaller VD/VT ratios (Cooper, 1967). Second, they adjusted $Paco_2$ to lower values than ours, and this would be expected to

produce lower ratios (Trimble et al., 1971). They also adjusted ventilation by changing frequency, whereas all our patients were ventilated at the same frequency. As a last comment on this point, we do not feel that they have given enough consideration to the effects of the prone position in which their patients were studied. Little is known about the effects of this particular position during anaesthesia, but regional distribution of ventilation differs considerably between supine and prone positions in conscious subjects (Cortese et al., 1976). Their conclusions can only be relevant to the prone position.

With regard to any possible change in V_D/V_T on induction of hypotension, we would accept that their policy of fluid loading would prevent any alteration resulting from changes in cardiac output or, perhaps more relevantly, pulmonary artery pressure. However, they do not consider the direct relaxant effect of sodium nitroprusside on tracheobronchial muscle (Kreye et al., 1975), which would lead to an increase in V_D/V_T , assuming that some bronchial tone were present.

J. A. W. WILD SMITH
G. B. DRUMMOND
W. R. MACRAE
Edinburgh

REFERENCES

- Cooper, E. A. (1967). Physiological deadspace in passive ventilation. *Anaesthesia*, 22, 199.
- Cortese, D. A., Rodaste, J. R., Rehder, K., and Hyatt, R. E. (1976). Effect of posture on the single breath oxygen test in normal subjects. *J. Appl. Physiol.*, 41, 474.
- Khambatta, H. J., Stone, J. G., and Matteo, R. S. (1982). Effect of sodium nitroprusside induced hypotension on pulmonary deadspace. *Br. J. Anaesth.*, 54, 1197.
- Kreye, V. A. W., Baron, G. D., Luth, J. B., and Schmidt-Gayk, H. (1975). Mode of action of sodium nitroprusside on vascular smooth muscle. *Naunyn-Schmiedeberg's Arch. Pharmacol.*, 288, 381.
- Nunn, J. F. (1977). *Applied Respiratory Physiology*, 2nd Edn. p. 229. London: Butterworths.
- Trimble, C., Smith, D. E., Rosenthal, M. M., and Fosburg, R. G. (1971). Pathophysiologic role of hypocarbia in post-

traumatic pulmonary insufficiency. *Am. J. Surg.*, 122, 633.

Wildsmith, J. A. W., Drummond, G. B., and MacRae, W. R. (1975). Blood-gas changes during induced hypotension with sodium nitroprusside. *Br. J. Anaesth.*, 47, 1205.

Sir,—We would like to thank Drs Wildsmith, Drummond and MacRae for their comments and interest shown in our work. Many investigators in the past, from Eckenhoff and his associates (1963) to Wildsmith, Drummond and MacRae (1975), have concluded that an increase in pulmonary deadspace occurs with deliberate hypotension using a whole gamut of agents from ganglionic blocking drugs to direct acting vasodilators. However, we have demonstrated that deliberate hypotension, *per se*, does not necessarily cause an increase in pulmonary deadspace (Khambatta, Stone and Matteo, 1982).

We believe that the apparent discrepancy between our results and those of others has a basis in cardiopulmonary pathophysiology. It has been repeatedly shown that, whenever cardiac output and pulmonary artery pressure decrease, pulmonary deadspace increases. In our study, these were maintained by adequate hydration. Thus, we conclude that the maintenance of cardiac output and pulmonary artery pressure during induced hypotension will prevent the increase in pulmonary deadspace.

H. J. KHAMBATTA
J. G. STONE
R. S. MATTEO
New York

REFERENCES

- Eckenhoff, J. E., Enderby, G. E. H., Larson, A., Eldridge, A., and Judenine, D. E. (1963). Pulmonary gas exchange during deliberate hypotension. *Br. J. Anaesth.*, 35, 750.
- Khambatta, H. J., Stone, J. G., and Matteo, R. S., (1982). Effect of sodium nitroprusside-induced hypotension on pulmonary deadspace. *Br. J. Anaesth.*, 54, 1197.
- Wildsmith, J. A. W., Drummond, G. B., and MacRae, W. R. (1975). Blood-gas change during induced hypotension with sodium nitroprusside. *Br. J. Anaesth.*, 47, 1205.