the too energetic insertion of the Boyle-Davis gag. They comment that when impaction of the epiglottis occurs, physical removal from its abnormal position is usually necessary, since obstruction is usually complete.

These cases have much in common with my own case, except that respiratory obstruction occurred early, before intubation had been attempted. The entity of a long, excessively mobile epiglottis, although rare, may prompt the anaesthetist to anticipate the possibility of impaction during anaesthesia.

J. G. K. THOMAS

Liverpool

REFERENCES


Pokrzywnicki, Stanley (1953). Impaction of the epiglottis during anaesthesia. Anaesthesia, 8, 47.

SEMANTICS AGAIN!

Sir,—From your comment "Semantics again!" with which you provide the letter of Z. Lett in the January issue of this journal on page 66, I conclude that you are irritated by semantics. But semantics can play a decisive role even in anaesthesia. I suggest you make an inquiry among British anaesthetists what meaning decisive role even in anaesthesia. I suggest you make an inquiry among British anaesthetists what meaning the terms VA, VB, and TV convey to them. You might be surprised to find that a good many of them think that fresh gas enters the lung-alveoli directly with each breath. But this is semantic confusion or "noise in the transmission of meaning" as Norman C. Staub (1963) points out.

The term "controlled respiration" obviously does not denote "to have complete control" over unit respiration. Has not this meaning been taken for granted by those who advocate "hyperventilation-anaesthesia"?

PAUL ZALUD

Czechoslovakia

REFERENCE


FACTORS AFFECTING THE TERMINATION OF CURARIZATION IN THE HUMAN SUBJECT

Sir,—In their paper on the termination of curarization, Bush and Baraka (Brit. J. Anaesth., 36, 356) refer to my comment (Bateman, 1963) on the use of suxamethonium for peritoneal closure towards the end of a long curarization, but they imply that I mentioned "a small dose" and go on to state that the response to such a dose would be unpredictable and therefore it would be more logical to use a supplementary dose of tubocurarine. They illustrate the point with tracings involving doses of 50 mg suxamethonium.

In fact, I did not mention the size of dose at all and would certainly agree with them that a mere 50 mg is very likely to be antagonistic to the existing curarization. If, however, 100 mg or more is used then the response is always of more paralysis irrespective of the stage of recovery from the original curarization. And yet, when this superimposed depolarizing block has worn off, it is my experience that the degree of residual curarization is found to be less than it would otherwise have been and correspondingly less neostigmine is necessary to effect complete reversal at the end of the operation. This "bonus" given when suxamethonium is used in this situation I do not recall having seen previously described, and I was therefore particularly interested to see its experimental confirmation in the tracings of Bush and Baraka.

I would, however, offer a further comment on their findings. Although they seek to demonstrate that respiratory alkalaeaemia antagonizes the action of tubocurarine under experimental conditions, it is my belief that, in clinical practice, vigorous hyperventilation and the concomitant alkalaeaemia have the effect of prolonging the curarization—at least as regards abdominal musculature. I have always visualized this as being due to the absence of any stimuli to the accessory muscles of respiration arising from an alkalaaemic respiratory centre.

These two points serve to emphasize, once again, the importance of being aware of the misleading differences that may appear between observations made in laboratory and clinical situations.

DONALD V. BATEMAN

Essex

REFERENCE


Dr. Bush has replied to the above letter as follows:

Sir,—Thank you for the opportunity to reply to Dr. Bateman's comments on our article (Brit. J. Anaesth., 36, 356). I must apologize for crediting (perhaps discrediting would be more appropriate) Dr. Bateman with a word that he did not include in his statement concerning the use of suxamethonium after a prolonged period of curarization but am glad that this has now been amplified. Since we investigated the effects of a dose of only 50 mg of suxamethonium, our remarks about the unpredictability of action of this drug in a partially curarized muscle must be applicable only when a dose of this magnitude is used. We have no data concerning the action of 100 mg or more of suxamethonium in this situation, but are interested that Dr. Bateman finds that the response is always one of more paralysis irrespective of the stage of recovery from the original curarization.

Dr. Bateman believes that pulmonary hyperventilation and the resultant respiratory alkalaeaemia prolong the "curarization" at least of the abdominal muscles due to lack of central stimuli and therefore suggests that there is a variance between clinical and experimental findings. I believe that this arises because of confusion over the term "curarization" and suggest that in this context muscle relaxation would be more appropriate. Curarization as I understand it refers to the action of tubocurarine at the myoneural junction which is what we tried to investigate, whilst muscle relaxation is a product of many components, both peripheral and central, which we did not investigate. I cannot see, therefore, any incompatibility between our findings and the clinical experiences of Dr. Bateman.

May I add that our recordings were obtained in the operating theatre on patients undergoing routine surgery.

GORDON H. BUSH

Liverpool