A PERFORATED ENDOTRACHEAL CONNECTION PIECE

BY

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THE Cardiff perforated connector (Picken, 1950) is a useful addition to the anaesthetist's armamentarium but has the disadvantages (a) that its use necessitates a double connection, and (b) that should the need to assist the respiration arise then the connector will have to be removed.

I have eliminated these disadvantages in a connection piece illustrated in figure 1. Basically this is a Rowbotham type connection piece with elongated stem in which the perforations have been made. A rotating inner drum with matching perforations allows partial or complete occlusion of the holes when necessary. The internal diameter of the stem is the same as in a standard Rowbotham piece.

I have used the connection in clinical practice for over twelve months and have found it very satisfactory both in children and adults.

W. W. Mapleson, Ph.D., the physicist of the Department of Anaesthetics, Welsh National School of Medicine, Cardiff, has kindly investigated the physical resistance offered by the instrument (perforations open) and his report is as follows:

<table>
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<th>Flow (litres/min)</th>
<th>Pressure drop across connector (mm H₂O)</th>
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<tr>
<td>10</td>
<td>1</td>
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<tr>
<td>20</td>
<td>3</td>
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<tr>
<td>30</td>
<td>7</td>
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<td>40</td>
<td>13</td>
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"In inspiration most if not all of the patient's requirements will, it is assumed, be supplied by the fresh gas supply so that the amount of air breathed in through the perforations will be so small as to give a negligible pressure drop across them. As regard expiratory resistance, even with as high a flow rate of fresh gases as 15 litres per minute the connector compares very favourably with any ordinary expiratory valve" (Mushin and Mapleson, 1954).

ACKNOWLEDGMENT

I wish to express my gratitude to Dr. Mapleson for his assistance and to Professor W. W. Mushin, for his advice.

The connection piece is made in different sizes and can be obtained from Medical and Industrial Equipment Ltd., New Cavendish Street, London, W.1.

REFERENCES

CORRESPONDENCE

SOME ASPECTS OF OBSTETRIC ANAESTHESIA

Sir,—I have read Dr. J. Selwyn Crawford’s article (Brit. J. Anaesth., 28, 146) with interest, but I fear a few points need further comment.

I cannot see how, from the results of his investigations, he arrives at the conclusions in his summary: "The maternal and foetal levels at time of delivery were approximately equal over a wide range of operation times (3-30 min), the placenta, apparently, offering no barrier to the passage of thiopentone."

The article does not state whether the blood samples were arterial or venous. Thiopentone levels in the arteries or the veins cannot be presumed to be equal in either the mother or the foetus. The first "round" of arterial blood after a rapid injection of 250 mg of thiopentone may be expected to have up to 250 \( \mu g \) of thiopentone per ml of blood, even allowing 1 litre for the central blood volume. This means that the chorio-decidual space will be subjected to this large concentration of thiopentone (see figure). When the thiopentone comes round to the peripheral veins, a large quantity will have been removed by the tissues, so that the venous level will give no indication of the concentration to which the placenta is being subjected.

Brown and Veall (1953) found that radio-active isotopes, if injected into the chorio-decidual pool, dropped to half strength in 25 seconds in normal pregnancy and in 75 seconds in eclampsia. This would suggest that the maternal arterial blood is held up for some time in this space in pregnancy. There is probably a very much longer delay late in the second stage of labour, especially if there is foetal distress. Thus the placenta may be subjected to a very high concentration of thiopentone while that in a peripheral artery may have already dropped to a low level (i.e. time lag).

Again, the blood vessel (umbilical vein) feeding the foetus with thiopentone cannot be expected to give a true representation of the thiopentone level of the foetal serum. E. J. O’MULLANE

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REFERENCE


Sir,—I would like to thank you and Dr. O’Mullane for the courteous way in which I have been given the opportunity of reading his letter prior to publication, thus enabling me to reply in the same issue.

After reading Dr. O’Mullane’s criticism, and the article to which he refers, I am happy to say that I find my faith in the conclusions expressed in the article unshaken. It should, of course, have been made plain that the samples of maternal blood were venous. As indicated, the first sample was taken two minutes after the administration of thiopentone, and the crucial sample "B" at periods ranging from three to thirty minutes after administration. I am rather pressed for time at the moment, and have not been able to look up the literature on the subject, but I imagine that after such a period the difference between the thiopentone concentration in the arterial and venous phases of one circulation "round" would be insignificant relative to the actual concentration involved. As far as I have been able to ascertain, all the previous workers in this field have used venous blood to obtain the maternal serum concentration for comparison with the foetal concentration. A similar argument applies to the foetal samples. Blood was obtained from all the contained arterial and venous vessels of the umbilical cord.

With regard to the applied significance of the work of Brown and Veall (1953), these workers
contended that, in normal pregnancy between about the 38th and the 40th weeks, the placental pool volume was approximately 250 ml blood. The maternal placental blood flow is in the order of 600 ml per minute (i.e. about one-tenth of the total cardiac output); it is reduced to about one-third of this figure in pre-eclampsia and in chronic hypertension. It is thus unlikely that the "very high concentration of thiopentone" in the placenta, feared by Dr. O'Mullane, will actually develop. Furthermore, Brown and Veall indicated that working with No. 24, even when this substance was injected directly into the placental pool, most of the isotope was distributed through the maternal circulation instead of crossing the placental barrier. Finally, I would point out that in almost all of our cases the maternal and foetal serum thiopentone levels were in actual fact equal, as were, in the smaller series, the bromide concentrations. Surely then, it is not too much to ask that the foetus be regarded as an "extra limb" as far as the distribution of thiopentone administered to the mother is concerned?

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REFERENCE

DUAL ACTION OF SUXAMETHONIUM CHLORIDE

Sir,—I should like to be among the first to congratulate Dr. H. J. Brennan on his excellent paper "Dual action of suxamethonium chloride" (Brit. J. Anaesth., 1956, 28, 159). However, the conception which is created—namely that the suxamethonium changes its mode of action—is misleading. I would, therefore, with your permission like to review some of the evidence to date.

In 1952, Dr. Richardson and I described a "dual type of response" occurring under certain conditions in man: at the same time Dr. Zaimis reported a similar finding in some species of animals. Together we developed the thesis that there was, in fact, a new and third type of neuromuscular block. Its principal features were that at first it showed signs of depolarization, but these rapidly changed until it became typical of a non-depolarization block, i.e. like d-tubocurarine. This type of block was named a "dual block" because it emphasized that two salient features were concerned and they occurred in a precise order.

The administration of decamethonium to cases of myasthenia gave rise to a dual block, yet this drug could be recovered unchanged from the urine of these patients and when injected into the chick caused a pure depolarization block (Churchill-Davidson and Richardson, 1953). This evidence suggests that in the dual response it is the motor endplate and not the depolarizing drug which changes its response. Dr. Brennan now brings forward evidence that under certain conditions suxamethonium can produce a similar type of block in normal subjects, but in his conclusion states that the drug changes its mode of action. From the fact that he uses our original terminology we must assume that he does not consider the response differs radically from that already described with decamethonium in myasthenia. I think, therefore, it is most important to emphasize that in our conception of the "dual block" it is the response of the motor endplate, and not the drug, which is the principal factor concerned.

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REFERENCES