DEATHS UNDER STOVaine: A CRITICISM

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IN the British Medical Journal, April 22, 1950, two deaths immediately following the injection of 2 ml. of Stovaine were reported (Walker and Mathews, 1950) from the department of Obstetrics and Gynaecology, University College, Ibadan, Nigeria, West Africa. It is quite obvious that the deaths were directly due to the anaesthetic. In both cases death followed immediately on the administration of 2 ml. of amylocaine hydrochloride and before the operation had begun. Various measures for resuscitation were carried out but unsuccessfully. In the order given they were: (1) ephedrine grain $\frac{1}{4}$ (50 mg.); (2) adrenaline, 8 min.; (3) an intra-cardiac injection of 1 ml. of nikethamide, and also anal stimulation. “Despite these measures and artificial respiration there was no response.” If that was the order in which these measures were carried out, of course there would be no response. A spinal anaesthetic has no direct effect on the heart so why stimulate it? It does paralyse respiration if it goes too high, and it is quite obvious that the Stovaine did go too high. Nothing is said about the position of the table, but if the patient were in the Trendelenburg position and if the Stovaine solution were of the hyperbaric variety these two factors would account for the extreme rapidity of the deaths.

The proper treatment for cases of this sort is to have a skilled anaesthetist to give the anaesthetic, for it is more important who gives the anaesthetic than what is given. Nevertheless, with this proviso in mind, it may serve some
useful purpose to discuss the relative merits of the drugs used and the exact procedure to be adopted if, as may happen to anyone, the life worthiness of the patient has been overestimated. This estimation of the life worthiness of a patient is the most difficult problem that any of us ever meet and one on which physician, surgeon and anaesthetist alike at times fall down.

Stovaine has a number of undesirable qualities; it is irritant and shows a much greater tendency to cause fainting and collapse, possibly due to the fact that it acts on the motor fibres before the afferents. It is not used as much as formerly. What of nupercaine and amethocaine? Professor Sebrechts, a very great authority on spinal anaesthesia as the result of long experience with cocaine, Novocaine, Stovaine, Tutocaine, Tropococaine, and nupercaine, prefers light nupercaine. He does not like heavy nupercaine; he thinks that it causes too many headaches. This is what he says about light nupercaine (Sebrechts, 1934).

1. "The factor which characterizes it and gives it an entirely special place in the series of anaesthetic agents is the intensity and duration of the blockage of the motor fibres."

2. "This [paralysis of all the intercostal nerves] is a condition frequently seen when one uses Percaine, which, as we have said, seems to have a special affinity for the motor fibres."

3. "In such cases the patient exhibits that slight degree of cyanosis which is so characteristic of Percaine anaesthesia."

4. "The suppression of the inspiratory movements of the thorax immediately causes the stoppage of the aspiration of the venous blood, and the action of the heart weakens. This is how the diminution of the respiratory amplitude is a cause of hypotension."

5. "The anaesthetic agent which blocks the anterior roots
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of a high segment of the medulla [probably meaning spinal cord?] puts out of action not only the motor fibres, but also those of the vaso-constrictor orthosympathetic nerves which accelerate the heart and promote adrenalin secretion.”

I submit that every one of these statements is a good reason for not giving nupercaine and particularly the light solution, at any rate to cases for Caesarean section.

The evidence before us to-day is to the effect that a spinal anaesthetic can cause the death of a reasonably healthy patient only by paralysing respiration. It is perhaps doubtful if all authorities would go so far, but what is not in doubt at all is that all agree that the paralysis or even the weakening of respiration is a serious event in the course of a spinal anaesthesia, one that demands immediate counter-measures. It is a fair thing, then, to take as the criterion or test of a technique of spinal anaesthesia, the effect of that technique on respiration. If we apply this test to nupercaine what do we find? An anaesthetic that occupies an entirely special place amongst anaesthetics by reason of “the intensity and duration of its blockage of the motor fibres”, in the course of the use of which we frequently find paralysis of all the intercostal muscles, which paralysis, “so frequently seen when one uses Percaine,” is the cause of that diminution of amplitude of respiration which the professor cites as the cause of hypertension; an anaesthetic whose hallmark is “a slight degree of cyanosis”. Please note that these are the statements of an enthusiastic user of nupercaine. But if the weakening and paralysis of respiration is the chief danger which we wish particularly to avoid, more especially when respiration is already embarrassed, then nupercaine is peculiarly unsuitable.

Amethocaine on the other hand has none of these disadvantages. Its action is not characterized by “a slight
degree of cyanosis”; the brunt of its power falls on posterior roots and because of this it does not paralyse the upper vaso-constrictors, only the lower intercostal muscles; it has therefore a very trifling effect on respiration or on the blood-pressure. These characteristics would seem to indicate that it is the more suitable anaesthetic for Caesarean sections. The one advantage that nupercaine has over amethocaine is that it lasts longer; that advantage is irrelevant in the matter of Caesarean sections.

In the correspondence which followed, O'Driscoll (1950) suggests that the excellent results published by Rufus Thomas (1942) were due to his use of heavy nupercaine. I would suggest that they were obtained in spite of its use, though, for obvious reasons, a heavy solution is better than a light one. Knowles (1950) suggested that the cause of the collapse was due to the Stovaine having got into the blood-stream. The chances are that if the Stovaine had got into the blood by mistake both patients would still be alive. 300 mg. procaine injected intravenously in the course of ten minutes influences neither the blood-pressure nor respiration. If procaine is injected into the jugular vein of a cat at the rate of 7.5 mg. per minute, it takes 24 minutes or 180 mg. to paralyse the cat’s respiration, whereas 10 mg. injected into the cisterna magna will paralyse the respiration in 1½ minutes. Professor Sebrechts says, “The injection into a vein of 20 cc. of Percain solution 1:1,500 without adrenalin causes no disquieting symptoms whatever.” If the intravenous injection of these quantities of procaine and nupercaine cause no disquieting symptoms whatever, it is unlikely that the injection of 2 ml. of Stovaine would have had such a dramatic effect.

As regards restorative measures, first comes artificial respiration, an intratracheal tube with oxygen inflation of
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the lungs at once, and 10 degrees Trendelenburg; only subsequent to these have you time for intravenous injections of ephedrine, etc. Timely and efficient artificial respiration will always save a cat and prolong its life till the effect of the anaesthetic has passed off. In the operating theatre it is often applied too late and not efficiently. There are cases so ill with advanced diabetes or uræmia, that any interference will precipitate a fatal issue but, if artificial respiration is applied in time and efficiently, even an advanced uræmic can be saved for the time being.

REFERENCES