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A Double-lumen Right Atrial Catheter for Open Heart Surgery

To the Editor:—Monitoring of right atrial pressure (RAP) using a central venous catheter (CVC) is desirable in patients undergoing open heart surgery. The need to use a CVC simultaneously for pressure monitoring and drug or fluid infusion frequently arises. Since the insertion of a second CVC can be time-consuming and carries with it increased risks,1–5 we developed a double-lumen catheter that by itself will allow continuous pressure monitoring with simultaneous administration of drug infusion or blood sampling.

A tapered, radiopaque 7F double-lumen catheter was developed that is 20 cm long with a 5-cm proximal port.* The distal lumen may be used for blood sampling or for drug infusion while the smaller proximal lumen may be used for pressure monitoring. The catheter is inserted by Seldinger technique, using a standard 0.035-inch guide wire inserted through the larger distal lumen. The catheter serves as its own dilator and no sheath introducer system is necessary for its insertion. Both lumens are filled with a heparinized saline solution in order to prevent clotting.

The catheter has been employed without complications in ten patients undergoing open heart surgery. The right internal jugular vein has been used in all instances. We have been able to monitor RAP accurately and continuously without interference even during blood sampling or drug infusion. It is felt that this method is reliable, convenient, and useful when RAP monitoring and simultaneous drug infusion or blood sampling are required.

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Thiobarbiturates Induce Arrhythmias in Dogs

To the Editor:—We would like to comment on the recent paper by Smith and Dresel1 in which they describe the possible origin of epinephrine-induced arrhythmias in halothane-anesthetized dogs. It is well-recognized that thiobarbiturates often induce ventricular bigeminy which is coupled to the preceding sinus beat in dogs.2 While it has been reported that thiamylal sodium causes these arrhythmias more frequently than thiopental sodium, both agents will cause ventricular bigeminy when given at anesthetic dosage.2 The clinical importance of this arrhythmia in the dog is in dispute at the present time. The cause has been related to increased arterial pressure, the concentration, and dose of thiobarbiturate administered.2,3 In addition, the

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mechanism responsible for the ventricular bigeminy is most likely due to an imbalance between parasympathetic and sympathetic activities with increases in both heart rate and blood pressure.\textsuperscript{4}

The dose of thiopental sodium (30 mg/kg) used in this study\textsuperscript{1} is relatively higher than the commonly used induction dosage of 15–20 mg/kg, iv prior to maintenance of inhalational anesthesia. Further, in these vagotomized dogs, it is quite possible thiopental sodium caused the observed bigeminy. This, in combination with halothane anesthesia and infusion of epinephrine, may have resulted in an increase in both heart rate and blood pressure in this study, although it was not mentioned.\textsuperscript{1}

Thus, while the ventricular bigeminy reported in this study may have in fact originated from the interventricular septum, it is not possible to unequivocally state that in dogs the bigeminy was due to the combination of halothane anesthesia and epinephrine infusion. To rule out the possible effects of thiopental sodium, one must induce and maintain anesthesia with halothane alone.

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\textbf{In reply:—} We thank Drs. Seeler and Thurmon for their comment and apologize to them and other readers for not correcting a proofreading error. The dose of thiopental used in our experiments was in fact 20 mg/kg rather than 30 mg/kg. This decreases the probability of the arrhythmia being due to the combination of thiopental–epinephrine alone. We have observed arrhythmias including bigeminy immediately after injection of thiobarbiturates, but these are usually of short duration and were never present at the time halothane administration was begun. Thiobarbiturate-induced arrhythmias first were reported by Gruber.\textsuperscript{1} Both blood pressure and hypercapnia were found to be important to their genesis (Gruber \textit{et al.},\textsuperscript{2} Woods \textit{et al.},\textsuperscript{3} and Johnstone\textsuperscript{4}).

There is clearly an interaction between thiopental and sensitizing anesthetics. We established this in the case of cyclopropane\textsuperscript{5} and have seen recent confirmation and extension for the case of halothane.\textsuperscript{6} The doses of epinephrine required to cause various types of arrhythmia are increased in the absence of thiopental, but each type may be demonstrated. Potentiation of very long duration is seen on injection of 5 mg/kg, iv, into cyclopropane-anesthetized dogs, or brief potentiation may be caused by injection of less than 2 mg into the circumflex coronary artery.\textsuperscript{5}

We therefore believe that the arrhythmia we observed was indeed due to thiopental-halothane-epinephrine.

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