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MANAGEMENT OF PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA UNDER GENERAL ANESTHESIA

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Although many articles regarding the different ways of terminating Paroxysmal Supraventricular Tachycardia have been reported, we can rarely find a report of how to treat Paroxysmal Supraventricular Tachycardia in a patient under general anesthesia.Following is the presenta tion of two patients who developed this tachyarhythmia under general anesthesia and were successfully treated by use of pressure amines.

REPORT OF TWO CASES

Patient I: A 30-year-old woman, ASA II, was admitted to the hospital for elective ligating of patent ductus arteriosus, Her electrocardiogram was within normal limit preoperatively.

One hour before anesthesia was started, pethidine 50 mg,and valium,IO mg,were given intramuscularly.Induction of anesthesia was accomplished with valium IO mg,halothane

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and nitrous oxide. Trachea was intubated after 60 mg of succinylcholine I.V.,.Anesthesia was maintained with nitrous oxide,oxygen, and halothane Pancuronium was used as a muscle relaxant. Surgery proceeded uneventfully. However, as soon as the chest was opened,the patient developed Paroxysmal Supraventricular Tachycardia with the heart rate increasing from I00 beats/min. to 175 beats/min. and blood pressure dropped from $\frac{130}{50}$ torr to $\frac{50}{40}$ torr(Fig I Part A&B). Surgical manipulation was stopped at this time. Carotid massage did not reverse tachycardia, however, when it was combined with methoxamine(20 mg, IV, in divided doses),heart rate decreased from 175 beats/min. to 125 beats/min. and systolic blood pressure increased from 50 torr to 140 torr(Fig I part B&C).Arterial blood gases were stable during this period. A few minutes after 50 mg pethidine was given intravenously, manipulation of the chest was started again. Normal sinus rhythm was maintained and the recovery period was uneventfull. Her ECG was within normal limit postoperatively and she did not develop tachyarhythmia during the rest of her stay in the hospital.

Patient II: A 40-year-old, 80 Kg woman was scheduled for abdominal hysterectomy for ca in-situ. She denied any serious medical problem in the past. Her pre-op laboratory data were within normal limits. The patient was premedicated with valium I0 mg,pethidine 50 mg and atropine 0.5 mg which were all given intramuscularly an hour prior to anesthesia was started. Anesthesia was induced with sodium thipental 400 mg, and intubation was accomplished with 60 mg of succinylcholine given intravenously. For maintenance of anesthesia halothane I percent in 4 l of nitrous oxide and 2 l of oxygen per minute

were given via the endotracheal tube and 40 mg of d-tubocurarine was used as a muscle relaxant during surgery. Shortly after change of the position to Trendelenburg, to provide a better exposure to the surgeon,pulse rate decreased from 80/min. to 40/min and blood pressure was no longer obtainable. At this point, it was noticed that the patient was disconnected from the anesthesia machine. She was ventilated with 100 percent oxygen. Following I mg atropine which was given intravenously to treat bradycardia, pulse rate increased from 40/min. to 200/min.and the blood pressure was still not obtainable, The ECG indicated a I:I ratio of arterial to ventricular conduction. Fortunately, the pulse rate and blood pressure returned to normal by IV injection of 20 mg of methoxamine. The rest of anesthesia course was uneventful. Repeated ECG taken postoperatively did not show any abnormalities and she was discharged 12 days after admission in a satisfactory condition.

DISCUSSION

Paroxysmal Supraventricular Tachycardia results from the rapid discharge of an abnormal focus in the atria at a rate between 140 and 220/min. Conduction of the supraventricular node is usually normal and therefore each atrial conduction is followed by a ventricular conduction. Paroxysmal Supraventricular Tachycardia usually starts and ends suddenly.

During the attack of Paroxysmal Supraventricular Tachycardia acceleration of the heart rate and marked shortening in the filling periods of ventricles, causes in decline in cardiac output. This decline is more serious in

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a- The ECG indicated a I:I ratio of atrial to ventricular conduction.

an abnormal heart.[2]

As far as the treatment is concerned, the attacks of Paroxysmal Supraventricular Tachycardia in an awake patient may be terminated by carotid sinus pressure, valsalva manoeuver, or sedatives, and even IV injection of edrophonium chloride or pressure amines[3-4] in more severe cases.However, if the immediate discisive measures are not undertaken, Paroxysmal Supraventricular Tachycardia in a patient whose cardiovascular stability is changed by general anesthetics, might cause serious problems.

Although Paroxysmal Supraventricular Tachycardia was probably produced by manipulation of the chest under light anesthesia in the first case reported here,increasing the depth of anesthesia at the presence of systolic blood pressure of 40 torr seemed to be too dangerous.So carotid massage which was effective when it was accomplished by pressure amine, methoxamine, was used to terminate this tachycardia. In the second case, when the injection of atropine switched bradycardia, which was induced by hypoxia, to Paroxysmal Supraventricular Tachycardia,time could no longer be lost to treat tachyarhythmia with physical stimulation of the vagus nerve alone. Therefore, dysrhythmia was successfully terminated by immediate IV injection of methoxamine, an α-receptor stimulant.

Pressure amines terminated the Paroxysmal Supraventricular Tachycardia in above cases by initiating the carotid sinus reflex and thus increasing the vagal tone and prolonging the conduction in the A-V nodes.[4-5] Slowing the heart rate results in ventricular dilation and so increased fiber length at end-diastole. This increased force conduction due to Frank-Starling mechanism compensates for the need

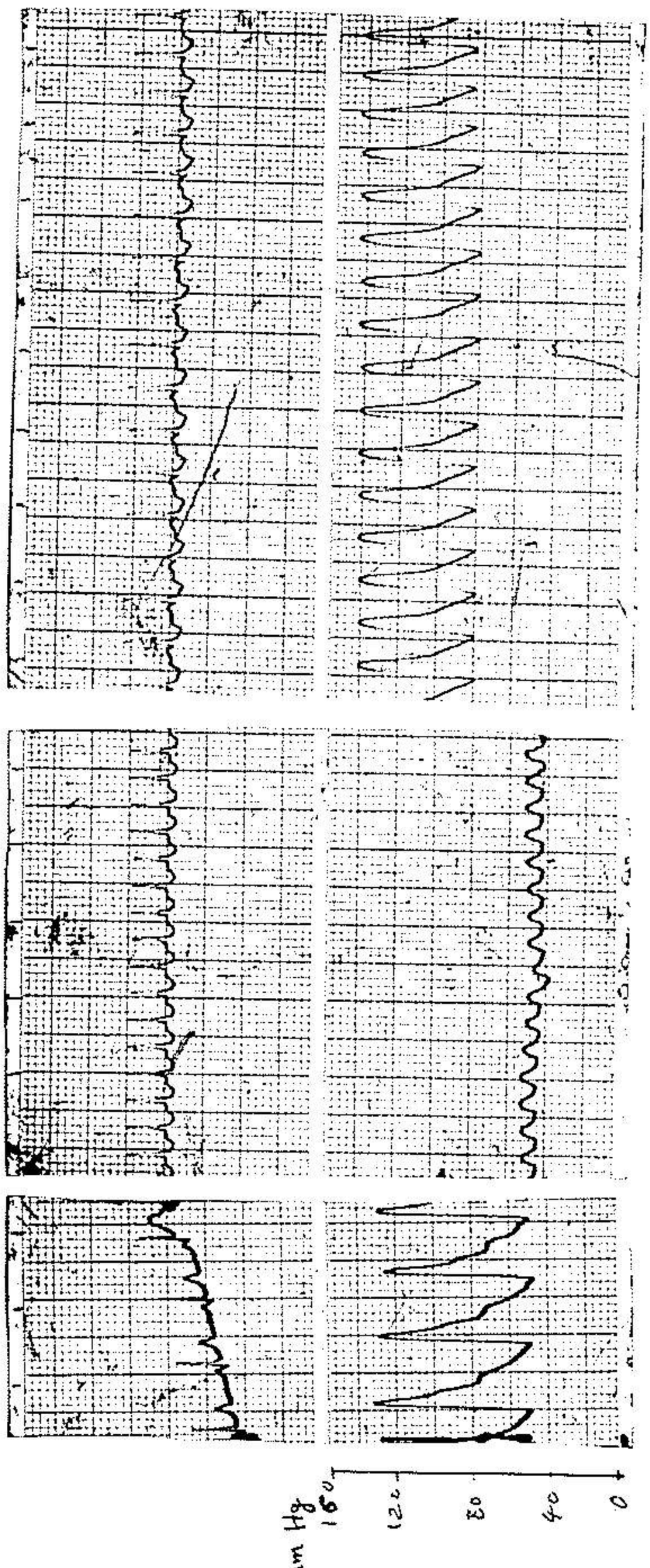

Fig. I, Lead II BCG tracings obtained under anesthesia (A) the sinus rhythm present before chest was opened, (B) sudden supraventricular tachycardia after the chest was opened, and (C) the reversion to sinus rhythm immediatley after vasopressure given intravenously. Lower tracings demonstrate blood pressure changes before, during and after paroxysmal supraventricular tachycardia,

Paper speed is I0 mm/sec.

Paper speed is I0 mm/sec.

for increased wall tension to maintain pressure.

Edrophonium chloride has been reccommended as a drug of choice in terminating the Paroxysmal Supraventricular Tachycardia. However to avoid the reverse effect of non-depolarizing drugs being used during surgery,edrophonium chloride was not used in above cases.

It can be concluded that methoxamine might be the drug of choice in terminating the Paroxysmal Supraventricular Tachycardia which occures in a patient who has an unstable cardiovascular function due to the use of anesthetic agents. However, further investigation is needed to determine this point.

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