Termination of Ventricular Tachycardia after Methoxamine

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ABSTRACT
A case of ventricular tachycardia which reverted with intravenous methoxamine is presented. The methoxamine was used to increase blood pressure during anaesthesia for direct current cardioversion. While alpha adrenergic agonists have been used previously to increase vagal tone via a baroreceptor mechanism to revert supraventricular tachycardias, this is the first case where a sustained ventricular tachycardia reverted with an alpha adrenergic agonist. The mechanisms likely to have caused the reversion are discussed. (Critical Care and Resuscitation 2001; 3: 259-261)

Key words: Methoxamine, ventricular tachycardia, alpha adrenergic agonist, cardioversion

Methoxamine is an alpha adrenergic agonist used predominantly for the treatment of hypotension during general and regional anaesthesia. It causes peripheral vasoconstriction and a rise in blood pressure. It may cause bradycardia, thought to be secondary to baroreceptor reflex activation.

A case of sustained ventricular tachycardia is reported which reverted to sinus rhythm by intravenous methoxamine. The possible mechanism for this cardioversion is discussed.

CASE REPORT
A 33 year old man was admitted to casualty with a two hour history of palpitations. His electrocardiogram showed ventricular tachycardia at a rate of 185 beats per minute (Figure 1). He had been admitted previously with ventricular tachycardia requiring direct current (DC) cardioversion. His investigations after his first episode of ventricular tachycardia included a resting electrocardiogram showing anterior T wave inversion, a cardiac MRI within normal limits and a normal echocardiogram. Electrophysiological studies showed inducible ventricular tachycardia originating from a focus in the right ventricular outflow tract and he was started on atenolol 50 mg daily.

At the time of the current admission he was slightly breathless, with a blood pressure of 85/40 mmHg but no syncopal symptoms. He was fasted and DC cardioversion was arranged. For the procedure, he was pre-oxygenated and anaesthesia was induced with 120 mg of propofol intravenously. In view of his low blood pressure he was given 2 mg of methoxamine intravenously after induction. Within 30 seconds, his electrocardiogram reverted to sinus rhythm at a rate of 75 beats per minute (Figure 2) with a blood pressure of 135/80 mmHg. He regained consciousness and remained stable in sinus rhythm for the next 48 hours when he was discharged home.

DISCUSSION
The patient’s ventricular tachycardia reverted within 30 seconds of the administration of intravenous propofol and methoxamine. As he had previously received propofol anaesthesia for DC cardioversion with no change in rhythm, it seems likely that methoxamine was the cause of the return to sinus rhythm.

Methoxamine is an alpha adrenergic agonist used predominantly in the treatment of hypotension during

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anaesthesia. It causes peripheral vasoconstriction which usually results in a rise in arterial blood pressure. Heart rate may decrease after methoxamine administration and this is thought to be due to activation of the baroreceptor reflex. Supraventricular tachycardias can often be treated by vagotonic manoeuvres such as ocular pressure or cold water applied to the face. Activation of the baroreceptor reflex by carotid sinus massage may also slow or cardiovert supraventricular tachycardias. Ventricular tachycardias do not typically respond to these manoeuvres and this has been used to differentiate ventricular and supraventricular tachycardias.

Methoxamine has been used to treat supraventricular tachycardias with baroreceptor activation being the suggested mechanism.\(^1\) One case of non sustained ventricular tachycardia aborted by methoxamine has been reported,\(^2\) but this is the first reported case of sustained ventricular tachycardia being terminated by methoxamine. The mechanism of action is unclear but may also involve baroreceptor activation. Cases of
phenylephrine (also an alpha adrenergic agonist) terminating ventricular tachycardias have been reported,\cite{3,4} and increased vagal drive is thought to be the mechanism as combined carotid sinus massage and high dose edrophonium have also been reported to terminate ventricular tachycardias.\cite{4}

However, this phenomenon is difficult explain, as the vagus acts predominantly on the sino atrial and atrioventricular nodes. It is possible that ventricular tachycardia requires a much higher degree of vagal activation for termination making it largely, but not completely, refractory to usual manoeuvres. Further research is needed to investigate the effects of vagal stimulation on ventricular tachycardias.

The use of methoxamine alone (or any alpha adrenergic agonist) for the treatment of ventricular tachycardia cannot be recommended. However, the use of alpha agonists to treat hypotension prior to DC cardioversion may have the added benefit of cardioverting either supraventricular or ventricular tachycardias.

Received: 18 August 2001
Accepted: 8 October 2001

REFERENCES