REFLECTIONS ON PREHOSPITAL CARE

A dramatic drop in blood pressure following prehospital GTN administration

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Emerg Med J 2007;**24**:225–226. doi: 10.1136/emj.2006.042432

A male in his sixties with no history of cardiac chest pain awoke with chest pain following an afternoon sleep. The patient did not self medicate. The patient's observations were within normal limits, he was administered oxygen via a face mask and glyceryl trinitrate (GTN). Several minutes after the GTN the patient experienced a sudden drop in blood pressure and heart rate, this was rectified by atropine sulphate and a fluid challenge. There was no further deterioration in the patient's condition during transport to hospital. There are very few documented case like this in the prehospital scientific literature. The cause appears to be the Bezold-Jarish reflex, stimulation of the ventricular walls which in turn decreases sympathetic outflow from the vasomotor centre. Prehospital care providers who are managing any patient with a syncopal episode that fails to recover within a reasonable time frame should consider the Bezold-Jarish reflex as the cause and manage the patient accordingly.

CASE REPORT

A man in his sixties awoke from sleep with central chest heaviness and discomfort radiating to the left arm and back with a pain score of 4/10. There was no associated shortness of breath or nausea. The patient did not self medicate to relieve the discomfort. The patient's only past history was hypertension treated with acetylsalicylic acid (Aspirin).

On examination the patient was conscious and orientated to time and place with a regular pulse rate of 92 per minute, respiratory rate 22 per minute, blood pressure (BP) 180 mm Hg systolic by palpation, normal sinus rhythm at a rate of 92 per minute, skin was pink and warm, and chest auscultation revealed clear air entry to the bases.

The patient denied the use of Viagra and similar drugs in the preceding days when his wife was present in the room and when she was absent.

The patient was administered oxygen at 8l/min via a Hudson face mask, 0.3 mg of sublingual glyceryl trinitrate (GTN) (he had not had GTN previously) and 300 mg chewable Aspirin whilst semi-recumbent in bed. After approximately 3 mins the patient was pain free.

After several minutes the patient stated "I feel unwell", complained of nausea and looked pale. The cardiac monitor showed a junctional rhythm of 45/min, his blood pressure had dropped to 70 mm Hg systolic.

The patient was laid flat, the GTN tablet was found to be fully dissolved. After 0.6 mg of atropine sulphate intravenously the pulse rose to 80 per minute, the cardiac monitor showed junctional tachycardia, and blood pressure was 80 mm Hg systolic. As the patient had a dry chest, a fluid challenge of 250 ml Hartmann's Solution was administered, increasing the

blood pressure to 105 mm Hg systolic. The nausea resolved once the blood pressure increased.

On arrival at hospital the patient's blood pressure was 120 mm Hg systolic by palpation with other physiologic parameters within normal limits.

DISCUSSION

The potent vasodilatory effect of nitrates has been known for many years. Prehospital GTN for the management of cardiac type chest pain/discomfort in Victoria, Australia, has been available since 1991, with no sudden dramatic drop in BP as described here documented in the Australian scientific literature. However, anecdotal evidence from Victorian paramedics suggests that drops in BP of 40 to 60 mm Hg have occurred previously. Kloner *et al* found that 33% of patients had a decrease of blood pressure >30 mmHg after 0.4 mg of GTN spray.¹

Two previous studies have identified a small number of patients who suffered significant hypotension and bradycardia following administration of nitrates.^{2,3} Engelberg *et al* identified six patients who dropped their BP by 100 mm Hg; however these patients had an initial blood pressure >200 mm Hg prior to nitrite administration. They identified one patient who had a severe bradycardia and hypotension following nitrates.³ Evidence suggests that some patients have a delay in the phase shift between heart rate and blood pressure changes that is associated with the baroreflex response following GTN administration.⁴ The response is commonly known as the Bezold-Jarish reflex. This involves stimulation of the cardioinhibitory receptors in the walls of the ventricles which cause a decrease in the sympathetic outflow from the vasomotor centre and hence cause bradycardia and peripheral vasodilatation leading to hypotension.⁵ These patients are not easily identifiable by paramedics in the prehospital setting, however they should be alert to this rare phenomenon following any sudden drop in BP associated with bradycardia.

The patient was asked about Viagra or like drug use prior to the administration of GTN as research has demonstrated that a combination of GTN and phosphodiesterase inhibitor drugs (the group Viagra belongs to) within a 24–48 h timeframe can cause a significant drop in blood pressure.¹ It is possible that males will not confirm the use of Viagra and like drugs in the presence of their partners, therefore the patient was also asked when his partner was absent from the room.

IMPLICATIONS FOR PRACTICE

This incident demonstrates that even though GTN is commonly used in the general and prehospital management of cardiac chest pain it is a potent vasodilator that has the potential to dramatically worsen the patient's condition even when administered as prescribed. Any patient with a syncopal episode (not just those given GTN) that fails to recover within a reasonable time frame may be the victim of the Bezold-Jarish

reflex and should be managed with atropine. Prehospital care providers should be aware of these effects and be prepared to manage them accordingly.

Competing interests: None

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Accepted 27 November 2006

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