Cardiovascular collapse due to intravenous verapamil in two patients with persistent atrial tachycardia

J. B. Lawrenson, A. M. Okreglicki, R. N. Scott Millar

Intravenous verapamil was given to two haemodynamically stable patients with persistent atrial tachycardia, resulting in circulatory arrest requiring CPR in one and collapse with unrecordable blood pressure in the other. Both responded to resuscitation and tachycardia was subsequently controlled with propranolol in one and sotalol in the other. Factors contributing to the cardiovascular collapse included: (*i*) left ventricular dysfunction; and (*ii*) failure to convert the tachycardia to sinus rhythm.

It was concluded that verapamil may be dangerous in supraventricular tachycardia not due to atrioventricular (AV) junctional re-entry, despite normal blood pressure and perfusion, particularly if left ventricular dysfunction were present. If the diagnosis of AV junctional re-entry is in doubt, adenosine is preferable as it is less likely to cause haemodynamic collapse and will assist in making the diagnosis.

Intravenous verapamil, commonly used to treat paroxysmal supraventricular tachycardias, nearly resulted in the death of one patient with an ectopic atrial tachycardia and caused cardiovascular collapse in another. We describe the patients and discuss alternative management strategies.

Patients

Case 1

A 49-year-old man complained of palpitations for 5 days. He had been treated successfully elsewhere for palpitations for 16 years with propranolol, but had stopped taking this 1 year before. In the Emergency Unit, a paroxysmal supraventricular tachycardia was diagnosed. He was not shocked or distressed and his blood pressure was 110/80 mmHg. A mobile chest radiograph showed mild cardiomegaly and pulmonary congestion. After carotid sinus massage failed, verapamil 10 mg was given slowly

Cardiac Clinic, Department of Medicine, Groote Schuur Hospital and University of Cape Town

J. B. Lawrenson, F.C.P. (S.A.)

A. M. Okreglicki, F.C.P. (S.A.)

R. N. Scott Millar, F.C.P. (S.A.)

intravenously. The heart rate slowed to 80/min initially, but was followed shortly thereafter by extreme bradycardia with no palpable pulse; the bradycardia was noted on the monitor, but no electrocardiograph (ECG) was obtained at the time. It is uncertain whether the bradycardia was due to complete heart block or atrial arrest. The chest was thumped and CPR started. Atropine and calcium gluconate were given intravenously. He responded promptly to resuscitation and on regaining consciousness had a blood pressure of 65 mmHg. His heart rate was 80/min and irregular, but shortly thereafter the tachycardia recurred. Inotropic support was needed for 3 hours.

The initial ECG (Fig. 1) showed a tachycardia, rate 176/min with QRS duration of 0,08 seconds and axis of −45°. The P wave was superimposed on the T wave. Following the patient's recovery from circulatory collapse, the atrial rate was 166/min but 2:1 atrioventricular (AV) block had developed. A day later the ECG showed an atrial tachycardia with a 1:1 ventricular response at a rate of 160/min and a P-R interval of 0,16 seconds with a P wave axis of +45°. P waves were intermittently absent without influencing the timing of subsequent P waves; this suggested exit block from an ectopic atrial focus (Fig. 2). Incessant ectopic atrial tachycardia was therefore diagnosed. Echocardiography showed impaired left ventricular function (fractional shortening 15%).

After 1 day of observation during which the tachycardia persisted, intravenous atenolol (given over 5 minutes) resulted in slowing of the atrial and ventricular rates to 120/min. Oral propranolol (80 mg twice a day) resulted in the heart rate's slowing to 100/min with no change in the P wave pattern. Thyroid function was normal. Sinus rhythm returned subsequently and the arrhythmia was satisfactorily controlled for 8 months, during which the patient remained asymptomatic. Thereafter he returned to his home in India.

Case 2

A 30-year-old woman presented during the 24th week of her pregnancy with a 5-day history of dyspnoea on exertion and ankle swelling. On admission to the Maternity Unit, she was found to be in cardiac failure with a regular supraventricular tachycardia at 200/min (Fig. 3). Cardiac failure improved after a 4-litre diuresis in response to furosemide. The next day she was still in tachycardia (208/min), with blood pressure 110/80 mmHg and mild cardiac failure. The tachycardia was thought to be due to AV junctional re-entry, so verapamil 5 mg was given intravenously. Tachycardia persisted so a further 5 mg was given after 5 minutes. Five minutes later the heart rate slowed to 90/min (Fig. 4) and the blood pressure dropped to unrecordable levels. Intravenous fluid was infused rapidly and she was given calcium gluconate; this was followed by improvement of the systolic pressure to 80 mmHg. The heart rate rose to 120 - 130/min and it became clear that atrial tachycardia with variable AV block was present. Echocardiography showed a dilated left ventricle with a fractional shortening of 8%. After transfer to the Coronary Care Unit, a quadripolar electrode was inserted percutaneously into the right femoral vein and positioned in the right atrium. Arterial pressure was monitored directly via femoral artery cannula. Overdrive atrial pacing failed to terminate the tachycardia. Striadyne (adenosine triphosphate) 10 mg produced transient 2:1 AV block (ventricular rate 90/min) without a decrease in arterial
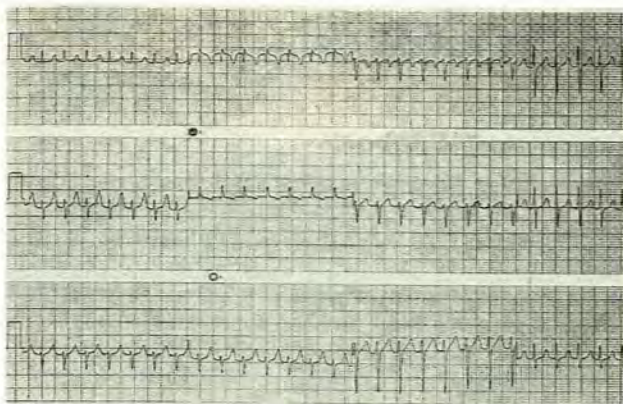
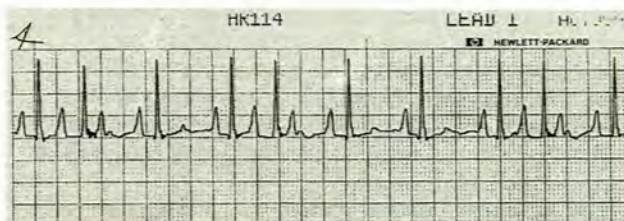Fig. 1. Twelve-lead ECG on admission (patient 1).

Fig. 2. Rhythm strip following recovery from verapamil (patient 1); atrial tachycardia, 169/min with varying AV block. The 5th, 10th and 12th P waves are absent without disturbing the atrial rhythm, suggesting intermittent exit block from an atrial focus.
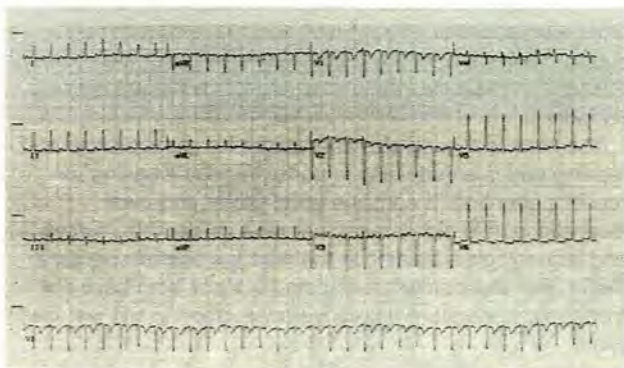

Fig. 3. Twelve-lead ECG on admission (patient 2); atrial tachycardia 197/min. The abnormal P waves can be seen distorting the T waves in leads I, II, AVL, V1, V2 and V3. The P-R interval is about 180 m/second.
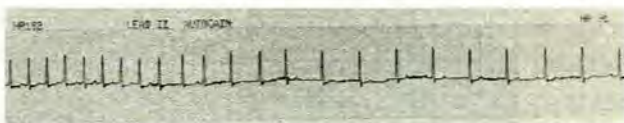

Fig. 4. Monitor strip after verapamil (patient 2). The ventricular rate slows from 182/min to 91 as a result of 2:1 AV block.

pressure. Intravenous esmolol 50 mg slowed the atrial and ventricular rates to 165/min without hypotension. The patient was treated with esmolol infusion, which was subsequently changed to oral metoprolol. Diuretics were continued and captopril and digoxin treatment initiated. Because the atrial rate remained 140 - 160/min, propafenone was tried, but

had no effect. Sotalol was then used in increasing doses until the atrial and ventricular rates slowed to 100 - 110/min. She was maintained on oral sotalol 320 mg twice daily. Ventricular function gradually improved and diuretics and captopril were stopped after 4 weeks. Sotalol was continued throughout the pregnancy with good control of heart rate, but the P waves remained abnormal. A healthy male infant was delivered 19 weeks after presentation. At the first follow-up visit, sinus rhythm had returned and sotalol was discontinued. She has remained well for 10 months, but left ventricular function remains mildly reduced (FS 24%, ejection fraction 47%).

Discussion

Intravenous verapamil can be expected to terminate up to 90% of paroxysmal supraventricular tachycardias due to re-entry in which the AV node forms part of the circuit (AV nodal re-entry tachycardias and AV re-entry tachycardias).[1,2] It is usually not effective in terminating arrhythmias within the atria (atrial fibrillation, atrial flutter or atrial tachycardia), but slows the ventricular rate by increasing the degree of block within the AV node.[1,2] Ventricular slowing may be beneficial; if sinus rhythm is restored, haemodynamic improvement usually results. However, if there is no effect on ventricular rate, for example if verapamil is given to a patient with ventricular tachycardia, haemodynamic collapse may occur.[3] This is presumably the result of a combination of peripheral arterial vasodilatation and myocardial depression. The sudden decrease in peripheral resistance, together with a further decrease in cardiac output, cause marked hypotension.

Circulatory arrest in patient 1 was due to extreme bradycardia, either because of transient complete heart block or atrial arrest with a slow escape rhythm. Unfortunately, an ECG strip was not recorded until partial recovery had occurred (Fig. 1). A continuous ECG should have been recorded from the start of the verapamil injection. However, even after the ventricular rate recovered to 80/min, blood pressure remained low despite intravenous calcium, and he required inotropic support for several hours. This suggests that marked myocardial depression occurred. The verapamil had been given despite the cardiomegaly and pulmonary congestion on chest radiograph because the blood pressure was 110/80 and peripheral perfusion was considered to be normal. The patient was subsequently shown to have reduced left ventricular function on echocardiogram.

The decision by the doctors to use intravenous verapamil in the cases reported here was understandable. The ECG in each case was misinterpreted as paroxysmal AV junctional re-entry tachycardia because the P waves were obscured by the T waves. There were no clinical signs of shock in either patient and the blood pressure was satisfactory before verapamil. In retrospect, however, the decision to use verapamil in the presence of even mild heart failure was unwise, particularly when the arrhythmia was unlikely to revert to sinus rhythm with the drug.

Cardiovascular collapse in the second patient may have been facilitated by hypovolaemia due to diuretic treatment of her previous pulmonary oedema. The partial response to fluid infusion is compatible with this. Adenosine (given the same day) slowed the heart rate to the same extent as the verapamil but did not lower the blood pressure, presumably

because it did not cause significant myocardial depression or vasodilatation. Despite reduced left ventricular function, both patients tolerated beta-blockers, which also slowed the atrial rate. The response to beta-blockade suggests that the tachycardias were due to an automatic focus rather than re-entry. Sinus rhythm returned after delivery of the baby in the second patient, which suggests that the atrial tachycardia was precipitated by pregnancy.[4]

Verapamil is widely used and generally assumed to be safe in patients with haemodynamically stable supraventricular tachycardia, but should be avoided in patients presenting with broad-complex tachycardia.[3,5] Circulatory collapse has been reported in a patient who had marked cardiomegaly and atrial flutter with 2:1 AV block and who was given verapamil 10 mg intravenously.[6] However, our two cases serve to emphasise the potential danger of verapamil in any tachycardia which does not revert to sinus rhythm with the drug, particularly in the presence of left ventricular dysfunction. While pre-treatment with intravenous calcium may reduce the likelihood of hypotension without impairing the action of verapamil on the AV node,[7] it is better to use an ultra-short-acting agent such as adenosine[8] so that any ill-effects are likely to be short-lived. This is particularly so when the diagnosis is in doubt or when it is incorrect, as it was in our patients. Atrial tachycardia may be difficult to distinguish from some forms of AV junctional re-entry tachycardias on ECG criteria alone. Adenosine may help to distinguish between these possibilities by either terminating the tachycardia (AV junctional re-entry) or inducing temporary AV block (atrial tachycardia). The lack of adverse effect of adenosine in our second patient suggests that this approach may be safe even in those patients who cannot tolerate verapamil.

Adenosine is, unfortunately, not yet freely available in South Africa. It is widely used as the drug of choice in terminating paroxysmal supraventricular tachycardia in the UK and continental Europe. Adenosine's advantage over verapamil is its extremely short half-life (10 seconds). Unpleasant side-effects (e.g. chest discomfort) are therefore short-lived. Hypotension, if it occurs, is transient. Arrhythmias after adenosine are common, but are short-lived. They include sinus arrest, AV block, ventricular arrhythmias and transient acceleration of the ventricular rate in atrial flutter. Adenosine appears to be safe in infants in whom verapamil is contraindicated. It should not be given to asthmatics, however.

We believe that adenosine should be more widely available as an alternative to verapamil.

REFERENCES

1. Rinkenberger RL, Prystowsky EN, Heger JJ, Trout PJ, Jackman WM, Zipes DP. Effects of intravenous and chronic oral verapamil administration in patients with supraventricular tachyarrhythmias. *Circulation* 1980; **62**: 996-1010.
2. Waxman HL, Myerburg RJ, Appel R, Sung RJ. Verapamil for control of ventricular rate in paroxysmal supraventricular tachycardia and atrial fibrillation or flutter: A double-blind randomised cross-over study. *Ann Intern Med* 1981; **94**: 1-6.
3. Buxton AE, Marchlinski FE, Doherty JE, Flores B, Josephson ME. Hazards of intravenous verapamil for sustained ventricular tachycardia. *Am J Cardiol* 1987; **59**: 1107-1110.
4. Doig JC, McComb JM, Reid DS. Incessant atrial tachycardia accelerated by pregnancy. *Br Heart J* 1992; **67**: 266-268.
5. Garrat C, Antoniou A, Ward D, Camm AJ. Misuse of verapamil in pre-excited atrial fibrillation. *Lancet* 1989; **1**: 367-369.
6. Opie LH. Drugs and the heart: III. Calcium antagonists. *Lancet* 1980; **1**: 806-810.
7. Weiss AT, Lewis BS, Halon DA, Hasin Y, Gotsman MS. The use of calcium with verapamil in the management of supraventricular tachyarrhythmias. *Int J Cardiol* 1983; **4**: 275-280.
8. Camm AJ, Garret CJ. Adenosine and supraventricular tachycardia. *N Engl J Med* 1991; **325**: 1621-1629.