Verapamil in the Management of Supraventricular Tachyarrhythmias Occurring after a Recent Myocardial Infarction

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SUMMARY In 16 patients, a sustained supraventricular tachyarrhythmia occurring less than 72 hours after the first symptom of an acute myocardial infarction was treated with titrated doses of verapamil. The drug was given intravenously in 1 mg increments every minute under continuous monitoring of electrocardiogram and blood pressure. Endpoints for the administration of verapamil were the following: reversion to sinus rhythm; a ventricular rate slower than 100/min; hypotension; a pulmonary capillary wedge pressure rising above 15 mm Hg; signs of intolerance; a maximum dose of 20 mg. Reversion to sinus rhythm was observed in 7/8 patients in atrial flutter and in 1/8 patients in atrial fibrillation; in all the ventricular rate could be slowed below 100/min without untoward effects.

DURING THE ACUTE phase of myocardial infarction in man, supraventricular tachyarrhythmias may be observed in 10% of all patients.1-2 Their onset is often heralded by frequent supraventricular extrasystoles, which may escape detection by conventional monitoring techniques. The mechanism of their genesis is still debated: atrial stretch caused by incipient ventricular failure, ischemia of the sinus node, atrial infarction, or pericardial effusion with mechanical stimulation of the atria.

A sustained supraventricular tachyarrhythmia may require emergency treatment when a very rapid ventricular response is associated with severe anginal pain or with a marked drop in blood pressure and in cardiac output. Even when it seems well tolerated, a rapid heart action exerts unfavorable effects on ischemic myocardium by increasing oxygen requirements. Supraventricular tachyarrhythmias may thus increase the extent of ischemia and the area of necrosis.

Verapamil has been shown5-8 to terminate many supraventricular tachyarrhythmias rapidly, or at least to slow the ventricular response by a prolongation of atrioventricular conduction time.9-11 The other cardiovascular actions of verapamil include a slight slowing of the sinus rate;9, 10, 12, 13 arterial vasodilation, particularly of the coronary arteries, with an associated drop in blood pressure;12, 13 negative inotropic properties are observed at doses higher than those usually given in clinical practice.9, 12, 14, 16 Profound hypotension and cardiac arrest have been reported when verapamil was administered to patients in severe heart failure or those treated simultaneously with beta-blocking agents;4, 16-20 resulting probably from the calcium antagonistic properties of verapamil.12, 18, 21-24 A calcium antagonist could be dangerous in the acute phase of myocardial infarction if these hearts are abnormally sensitive. Reported therapeutic trials4-8 included few patients with a recent myocardial infarction; no dangerous side effects were observed. We therefore investigated the safety and therapeutic effectiveness of titrated doses of verapamil administered intravenously to patients with a prolonged supraventricular tachyarrhythmia occurring shortly after an acute myocardial infarction.

Material and Methods

Patient Selection

The 16 patients included in this study were all treated less than 72 hours after the first symptom of their acute myocardial infarction. This diagnosis was based on at least two of the following criteria: a typical history, a characteristic electrocardiogram, and specific serum enzyme changes. Clinical data are included in tables 1 and 2. These patients were not preselected but treated by the author whenever a prolonged supraventricular tachyarrhythmia was observed.

Tachyarrhythmia

Verapamil was used only if the supraventricular tachyarrhythmia was sustained (at least 20 min), with a ventricular rate in excess of 100 beats/min; treatment was initiated if the rhythm disturbance did not disappear after carotid sinus massage, or if the patient was symptomatic (angina pectoris or hypotension). Administration of a beta-blocking drug during the 24 hours preceding the supraventricular tachyarrhythmia was an absolute contraindication for verapamil.

Monitoring

An electrocardiographic lead was displayed on a monitoring oscilloscope at the bedside and at the nursing desk, analyzed by computer and written out on a stripchart recorder at a paper speed of 5 or 25 mm/sec. Systemic blood pressure was measured every minute (cuff). If a Swan-Ganz catheter was in use, pulmonary artery pressures were measured every minute and mixed venous oxygen saturation at 5 min intervals.

Drug Administration

Verapamil was drawn into a 20 ml syringe and brought to a volume of 20 ml with isotonic saline (final concentration: 1 mg/ml). The drug was administered in 1 mg increments given intravenously at 1 min intervals to a maximum total dose of 20 mg. After each increment, the indwelling catheter was flushed with 2 ml of saline. Other endpoints to the administration of verapamil were reversion to sinus rhythm or a ventricular rate slower than 100 beats/min. Neither systemic hypotension nor an elevated pulmonary capillary wedge pressure was regarded as a contraindication for ini-
titating verapamil therapy. However, if during the administration of verapamil, left ventricular failure seemed to become worse (systemic pressure dropping below 90 mm Hg or pulmonary capillary wedge pressure increasing beyond 15 mm Hg), no further increment of verapamil was given.

Precautions and Follow-up

Excessive bradycardia and hypotension were to be treated with an intravenous bolus of atropine (0.5–1.0 mg) or isopropyl noradrenaline (1–2 mg). If no side effect was observed but sinus rhythm was not restored, titrated rapid digitalization was carried out: ouabain was administered intravenously in 0.125 mg increments at hourly intervals if the ventricular rate remained slower than 100 beats/min, at 30 min intervals if the ventricular rate was in excess of 100 beats/min, until resumption of a sinus mechanism or to a maximum of 1.0 mg.

Results

Atrial Fibrillation

Eight patients were treated for atrial fibrillation with a rapid ventricular response (table 1). Only one patient reverted to sinus rhythm; in the others, the ventricular rate...
Table 2. Effects of Intravenous Verapamil in Atrial Flutter

<table>
<thead>
<tr>
<th>Pt/Age/Sex</th>
<th>Location</th>
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<th>After Verapamil</th>
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<tbody>
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<td>II</td>
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<td>II</td>
<td>150</td>
<td>120/90</td>
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<td>90</td>
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<tr>
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<td>A</td>
<td>II</td>
<td>150</td>
<td>65</td>
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<td>A</td>
<td>II</td>
<td>150</td>
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<td>III</td>
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<tr>
<td>8/48/M</td>
<td>A</td>
<td>II</td>
<td>150</td>
<td>100/65</td>
</tr>
</tbody>
</table>

*Reversion to sinus rhythm in two hours after ouabain 0.375 mg i.v.
†Possibly supraventricular tachycardia.
Abbreviations as in Table 1.

was slowed down to less than 100 beats/min (fig. 1) but
digitalization was necessary to achieve sinus rhythm. A drop
in systemic blood pressure was observed in six patients; none
developed shock or signs of left ventricular failure.

Atrial Flutter

Eight patients were treated for atrial flutter with 2:1
atrioventricular (A-V) block (table 2). Verapamil produced
sinus rhythm in 7 patients (figs. 2–4). One of these episodes
ended with multiple ventricular extrasystoles (fig. 5) before
resumption of a stable sinus mechanism; usually the flutter
waves became irregular shortly before termination of the
rhythm disturbance (fig. 2). In the last patient (fig. 4), sinus
rhythm was not achieved after verapamil (20 mg); however,
the ventricular rate was slower than 100 beats/min, and in-
travenous ouabain brought about a reversion to sinus
rhythm two hours later. Systemic blood pressure was un-
changed in five patients, decreased slightly in two patients,
and in one patient after reversion to sinus rhythm.

Hemodynamic Effects of Verapamil

In one patient with flutter and two with atrial fibrillation,
a Swan-Ganz catheter was still positioned in the pulmonary
artery; pulmonary capillary wedge pressure and mixed
venous oxygen saturation were measured before and after

Figure 3. (opposite). Atrial flutter with 2:1 A-V block reverting
to sinus rhythm without atrial ectopic activity after verapamil
10 × 1 mg i.v.

Figure 2. Atrial flutter with 2:1 A-V block unmasked by carotid sinus massage (upper strip). Verapamil 5 × 1 mg
unstabilizes the flutter mechanism and produces sinus rhythm with supraventricular extrasystoles (lower strip).

Figure 4. Computer display of heart rate (upper graph) and dis-

ergence, i.e., ΔR-R (lower graph) in a patient with atrial flutter and

2:1 A-V block. Verapamil 20 × 1 mg i.v. was given slightly before 3
p.m., resulting in a 4–5:1 A-V block (increased dispersion) and a
ventricular rate of 90 beats/min. Ouabain 0.375 mg i.v. ad-
ministered over two hours produced normal sinus rhythm (very low
dispersion), 90 beats/min, at 4:20 p.m.
therapy (tables 1 and 2). These are insufficient data to make firm conclusions about the hemodynamic effects of verapamil in patients presenting with supraventricular tachyarrhythmias during the acute phase of their myocardial infarction.

Complications and Survival

Patients 3 and 6 with flutter (table 2) developed severe left ventricular failure with class IV cardiogenic shock, 5 and 7 hours, respectively, after their supraventricular tachyarrhythmia. The electrocardiogram and serial serum enzyme changes indicated sufficiently extensive myocardial damage to explain the onset of cardiogenic shock. Intra-aortic balloon pumping without surgery was effective in both patients; one patient is a long-term survivor, the other died one week after weaning from circulatory assistance. Another patient with flutter (2) died 36 hours after verapamil therapy from a recurrent myocardial infarction. In total, 14/16 patients were hospital survivors.

Discussion

Intravenous verapamil can bring supraventricular tachyarrhythmias under control.9-11 However, few patients in the reports of this property of the drug had sustained a recent myocardial infarction. In Heng's series,7 nine patients had an acute myocardial infarction; all reacted favorably to verapamil, although three of them showed some systemic hypotension. Milne6 reports that the condition of one patient with a recent infarction deteriorated because of atrial fibrillation with a rapid ventricular response; he improved after administration of 10 mg of verapamil i.v. Our results confirm the efficacy of verapamil even when the rhythm disturbance presents in a patient recovering from an acute myocardial infarction.

After intravenous administration, verapamil acts very rapidly on atrioventricular conduction, as illustrated in figures 1 and 4. A reduction in the rate of the ventricles can be brought about almost immediately, by a bolus injection of 10 mg of verapamil.4 We prefer to administer 1 mg increments of verapamil at one minute intervals in order to detect early symptoms of drug intolerance in those patients who are recovering from a recent myocardial infarction.

Is verapamil a safe drug to use under these circumstances? The known pharmacological properties of verapamil suggest the possibility of very dangerous complications. Both in animals and in man, verapamil has been found to depress myocardial contractility; the drop in systemic pressure often
found after verapamil is due to generalized vasodilatation rather than to decreased ventricular contractility. Newly, an intravenous dose of 20 mg is well tolerated, but in the presence of recent myocardial necrosis the heart might be more easily depressed by the drug. Indeed, severe shock has been reported after verapamil was administered to patients in failure for reasons other than the arrhythmia, particularly when they had previously received beta-blocking agents. When pump function is critically impaired, any pharmacological depression may be fatal. We observed in one patient in shock who was not responding favorably to intra-aortic balloon pumping: a bolus injection (5 mg) of verapamil, given to slow a sinus tachycardia of 130 beats/min, resulted in sinus arrest and ventricular standstill within 30 seconds; resuscitation attempts were unsuccessful. However, when verapamil was titrated carefully as in the present study, no dangerous hemodynamic deterioration was observed.

In patients with impaired function of the sinus node or of atrioventricular conduction, verapamil may produce sinus arrest, heart block, and even cardiac standstill. This type of complication was not observed in any patient of the present series.

Intravenous verapamil effectively reduces the ventricular rate in patients with a recent myocardial infarction complicated by a sustained supraventricular tachyarrhythmia. The drug may be administered safely if the dosage is titrated to the needs and to the tolerance of the individual patient. The electrocardiogram and systemic blood pressure should be controlled at frequent intervals. The total dosage of verapamil should be the minimum required to achieve either of two therapeutic goals: a return to sinus rhythm, or a decrease in ventricular rate to less than 100 beats/min.

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