Concealed Ventricular Parasystole Uncovered in the Form of Ventricular Escapes of Variable Coupling

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SUMMARY Exit block from a parasystolic focus is recognized when automatic discharges fail to become manifest during the excitatory phase of the ventricular cycle. In the present study, an apparently complete exit block and a persistently concealed ventricular parasystole (VP) resulted from an exit refractory period (ExRP) longer than the sinus cycle length. Slowing of the heart rate caused the concealed VP to become apparent in the form of ventricular escapes of variable coupling and as an idioventricular rhythm that failed to show initial "warming up." These features reflect the fact that the automatic focus is protected from activity of the sinus impulses, which, however, can induce a prolonged ExRP. Seven cases of concealed VP are discussed. In two, concealment occurred spontaneously during follow-up of a typical VP; in three, it was provoked by conduction-depressing drugs; and in two, ventricular escapes of varying coupling were shown to represent the manifestation of a concealed and previously unrecognized VP. In two cases, isoproterenol caused reappearance of typical VP, probably through a shortening of the ExRP. While isoproterenol may be useful for uncovering a concealed VP, conduction-depressing drugs may be used to provoke or increase exit block. Total extinction of the VP seemed to occur in two patients during follow-up studies.

DURING the follow-up of 49 patients with ventricular parasystole (VP), we observed a sustained disappearance of the arrhythmia, occurring spontaneously in two cases and under the effect of antiarhythmic drugs in three. When the VP was no longer manifest, a slowing of the heart rate caused the arrhythmia to reappear under the form of ventricular escapes (VEs) of variable coupling, or as an idioventricular rhythm in which the first escape interval could be either shorter or longer than the regular interectopic interval (IEI). We also studied two patients in whom VEs of varying coupling were shown to represent the manifestation of a concealed and previously unrecognized VP. These seven cases are reported in order to discuss (1) the existence and diagnostic criteria of concealed VP, (2) the way to uncover this arrhythmia and prove its parasystolic mechanism, (3) the type of exit block underlying this form of VP, and (4) the natural history of VP.

Case Reports (table 1)

Case 1

A 12-year-old boy showed typical VP during a follow-up of 14 months (fig. 1A). After 3 weeks of treatment with amiodarone, a long tracing showed that the VP was no longer present, and at that time the four strips shown in figure 1B were recorded during carotid sinus massage. A single VE or an escape rhythm was shown to occur, with a QRS pattern similar to the ectopic beats of figure 1A (checked in different leads). The VE interval ranged from 2640-1350 msec, and was substantially different from the cycle length of the idioventricular rhythm. An isoproterenol infusion then caused reappearance of the manifest VP and markedly shortened the IEI, from 1830 to 1220-1240 msec (fig. 1C). When amiodarone was discontinued, the initially typical VP returned, and three repeat studies with the same drug showed similar results.

We postulate that the absence of parasystolic beats during antiarrhythmic treatment was due to an apparently complete exit block related to a state of refractoriness generated by each sinus impulse, and that the effective refractory period responsible for such exit block (ExRP) was longer than the spontaneous cycle length. Accordingly, the parasystolic discharges became manifest only when sinoatrial impulses were suppressed by vagal stimulation. If the concealed discharges are traced from the regular IEI (fig. 1B), the timing of the first VE depends not on the preceding sinus beat but on the preceding concealed discharge, and therefore, the VE interval can be either longer or shorter than the IEI. This suggests that the ectopic focus was protected and that a parasystolic mechanism was at play. The fact that the idioventricular rhythm did not show "warming-up" is an additional evidence of protection. However, although sinus impulses could not discharge the ectopic focus, we must assume that they could penetrate partially and create an ExRP in or around the parasystolic
center. Restoration of the manifest VP by isoproterenol was attributed to a shortening of the ExRP from an estimate of more than 1030 msec, to shorter than 730 msec (fig. 1).

Case 2

A typical VP was seen to occur transiently, in a single ECG, during the follow-up of a patient with sick sinus syndrome. A few days later, a long tracing revealed total absence of ectopic beats, but VE and an idioventricular rhythm appeared during vagal stimulation (fig. 2A). The VE interval was 1880–3840 msec, while the cycle length of the escape rhythm was 2130–2150 msec. This was interpreted as evidence of a concealed VP that was attributed, as in case 1, to an

![Image](http://circ.ahajournals.org/content/64/1/200.full)

**FIGURE 1. Case 1. Manifest ventricular parasystole (VP) concealed by amiodarone and restored with isoproterenol. Below each tracing the lower horizontal line represents the ectopic ventricular focus. Small vertical arrows indicate manifest discharges and short interrupted bars represent ineffective and concealed discharges of the parasystolic center. The values just to the left of each manifest and concealed discharge (smaller numbers) indicate, respectively, the manifest and concealed coupling to the preceding sinus beat. The values between the upper horizontal lines (larger numbers) indicate the interectopic intervals of the manifest discharges; the longer intervals are also given as multiples of the shorter ones. All time intervals are in milliseconds. (A) Typical VP. There is no evidence of exit block. (B) Concealed VP from the same focus, after 3 weeks of amiodarone. Parasystolic discharges become manifest only under the form of ventricular escape of varying coupling or as a regular idioventricular rhythm, during carotid sinus massage. The four discontinuous strips, from the same study, were rearranged according to decreasing values of the first escape interval. The exit refractory period of the ectopic center is longer than 1030 msec (longest concealed coupling, fourth strip) and shorter than 1350 msec (shortest manifest coupling, fourth strip). (C) Restoration of manifest VP during isoproterenol infusion (2 µg/min), but there is still some degree of exit block. The exit refractory period is now shorter than 730 msec (shortest manifest coupling, first strip), and longer than 630 msec (longest concealed coupling, second strip).**
**Figure 2.** Case 2. Concealed ventricular parasystole (VP) occurring spontaneously or under the effect of lidocaine. Conventions are as in figure 1 (A) Concealed VP, 7 days after a typical VP from the same focus (not shown) had been recorded. Parasystolic discharges become manifest only during carotid sinus massage, under the form of ventricular escapes of varying coupling or as a regular idioventricular rhythm. The three discontinuous strips were rearranged according to increasing values of the first escape interval. The exit refractory period is longer than 1690 msec (longest concealed coupling, third strip) and shorter than 1880 msec (shortest manifest coupling, first strip). (B) Manifest VP with exit block recorded 1 month later, during spontaneous sinus depression. The exit refractory period is now longer than 800 msec (first concealed discharge, second strip), and shorter than 1020 msec (second manifest discharge, first strip). (C) Typical VP without exit block, recorded 3 months after B. (D) An i.v. bolus of lidocaine (100 mg) administered right after C restores transiently conditions for the occurrence of concealed VP.
ExRP longer than the sinus cycle length. The third concealed discharge in the third strip indicated that the ExRP was longer than 1690 msec. The two strips in figure 2B were recorded 1 month later, during a spontaneous sinus depression, and showed automatic beats with the same QRS configuration. The coupling intervals were again variable but much shorter. The presence of a VP was obvious, but a high degree of exit block was still apparent. Three months later, the same automatic focus gave rise to a typical VP without exit block (fig. 2C). Under the latter conditions, the administration of a bolus of lidocaine evoked the features of the concealed VP (fig. 2D). The automatic beats were initially suppressed but reappeared after a few minutes (during vagal stimulation) under the form of VE of varying coupling (1560-2520 msec) or as a regular idioventricular rhythm (IEI 2260 msec). The effect of lidocaine persisted for 25 minutes. Several studies during the next 12 months showed absence of parasystolic activity even during vagally induced sinus depression, followed by a short period during which typical VP reappeared, and by a final period in which automatic activity could not be evoked even under the effects of isoproterenol.

Case 3
Several ECGs recorded during a 6-month period showed a typical VP (fig. 3A). In several subsequent studies the VP was no longer present, but automatic beats with the same QRS contour reappeared during vagal stimulation (fig. 3B), under the form of VE with variable coupling (2070-3960 msec). Although the manifest VP first disappeared after propranolol (to treat hypertension), after discontinuation of the drug the arrhythmia did not reappear, and several studies 5 months later were similar (fig. 3B). In studies during the next 21 months, automatic beats did not occur even after extremely long pauses or under isoproterenol action.

Case 4
Several ECGs recorded during a 2-month period showed a VP with a long IEI (2830-2940 msec) and no exit block (fig. 4A). Two months later the VP was not manifest during spontaneous sinus rhythm (fig. 4B), but became apparent during vagal stimulation under the form of VE of variable coupling (1480-3770 msec), and as a remarkably slow idioventricular rhythm (rate of 18 or 19 beats/min). No further follow-up was available.

Case 5
This was a case (illustration not shown) in which lidocaine changed a manifest VP into a concealed VP. The latter became apparent only during vagal stimulation, under the form of VE of variable coupling. Findings were similar to those presented in figure 2D, although in the present case concealment of the VP lasted for 3 hours.

Case 6
VEs of variable coupling (1420-1620 msec) were observed in a patient with second-degree atrioventricular block (fig. 5A). At the end of the same tracing, during 2:1 atrioventricular block, automatic beats from the same focus showed a parasystolic behavior, with some degree of exit block (fig. 5B). The ExRP was longer than 650 msec (longest coupling of a concealed discharge) and shorter than 860 msec (shortest coupling of a manifest discharge).
SUMMARY

of six of them coupling, high degree the latter VP typical cycle length.

minutes (fig. 6B) a VE of variable coupling occurred, but there was a substantial shortening of the idioventricular cycle length. After 5 minutes (fig. 6C), a typical VP became manifest (IEI 2040–2090 msec).

Case 7

During a routine study of a patient with left bundle branch block, VEs of variable coupling (2640–3040 msec) and a very slow idioventricular rhythm (19 beats/min) occurred during carotid sinus massage (fig. 6A). A concealed VP was suspected and proved by the administration of isoproterenol. During the first 4 minutes (fig. 6B) a VE of variable coupling occurred, but there was a substantial shortening of the idioventricular cycle length. After 5 minutes (fig. 6C), a typical VP became manifest (IEI 2040–2090 msec).

Summary of the Cases (table 2)

The seven patients showed VEs of highly variable coupling, six of them during carotid sinus massage and one as a result of second-degree atrioventricular block. An idioventricular rhythm was present in five patients. The VE interval could be either shorter or longer than the IEI of the idioventricular rhythm, and the latter never showed warming-up. The automatic focus was protected by entrance block and showed a high degree of exit block. The ExRP was always (in the six cases in which it could be estimated) longer than the sinus cycle length, giving rise to a concealed VP. A typical VP from the same focus that provoked the concealed VP was documented in the seven patients.

Discussion

Mechanism of Concealed Parasystole

An ectopic pacemaker can produce a manifest VP, provided it is protected from the activation of a dominant pacemaker by entrance block. However, if the zone of block is great enough, both entrance and exit block will be present in such a way that a parasystolic focus could fire rhythmically but never become manifest.2 In our patients, a VP had lost its ability to manifest itself during sinus rhythm, while retaining the ability to become apparent under the form of VE when sinus rhythm was depressed. This suggests that the sinus impulses could adequately penetrate the parasystolic area, so as to leave in their wake a state of refractoriness responsible for the occurrence of an apparently complete exit block. Exit block from a parasystolic focus is recognized when expected automatic discharges fail to become manifest well after the T wave of the preceding sinus beat.5–8 In the present study, however, this is the first time that the ExRP is
shown to last longer than the sinus cycle length, giving rise to persistently concealed rhythmic discharges. While the regular firing of the concealed parasystolic focus is independent of the sinus rhythm, the first manifest discharge occurring as a VE must necessarily keep a random relationship to the preautomatic beat. This explains the highly variable coupling and the fact that the VE interval can be either shorter or longer than the regular IEI. The absence of warming up when the concealed automatic activity becomes apparent as an idioventricular rhythm also reflects independence of the automatic focus.

Spontaneous and Provoked Modifications of Exit Block

In patients 2, 3 and 4, a prolonged ExRP causing concealment of a previously manifest VP developed spontaneously during follow-up studies. In patients 6 and 7, the prolonged ExRP was present when they were first examined, and the parasystolic mechanism was only suspected from the occurrence of VE with varying coupling. In case 2, the VP changed back and forth from concealed to manifest several times. This dynamic character of the abnormality that caused the exit block is emphasized by the fact that the ExRP can be manipulated pharmacologically. Thus, we could show that in manifest VP, exit block and concealed VP can be induced with conduction-depressant drugs. This was seen in patients 2 and 5 after administration of i.v. lidocaine, and in patient 1 during treatment with oral amiodarone. Both drugs prolong refractoriness in abnormal tissues.  On the other hand, a prolonged ExRP causing concealed VP was shortened by the administration of isoproterenol in case 1 and probably in case 7, thereby restoring a manifest VP. While isoproterenol may be useful for uncovering or proving the existence of VP in cases in which this arrhythmia is concealed, conduction-depressing drugs can be used to provoke or increase exit block, and both may become important tools for understanding this physiologic abnormality.

![Table 2. Duration of Different Intervals as Measured During Concealed Ventricular Parasystole](image)

<table>
<thead>
<tr>
<th>Case</th>
<th>Spont. SCL (msec)</th>
<th>VE coupling (msec)</th>
<th>IVCL (msec)</th>
<th>Ex RP during CVP (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>800–1000</td>
<td>1350–2640</td>
<td>1830</td>
<td>&gt; 1030; &lt; 1350</td>
</tr>
<tr>
<td>2</td>
<td>800–1000</td>
<td>1880–3840</td>
<td>2130–2150</td>
<td>&gt; 1690; &lt; 1880</td>
</tr>
<tr>
<td></td>
<td>800–940*</td>
<td>1560–2520</td>
<td>2260</td>
<td>&gt; 620; &lt; 1560</td>
</tr>
<tr>
<td>3</td>
<td>680–850</td>
<td>2070–3960</td>
<td>2110</td>
<td>&gt; 1840; &lt; 2070</td>
</tr>
<tr>
<td>4</td>
<td>800–1000</td>
<td>1480–3770</td>
<td>3130–3270</td>
<td>&gt; 1010; &lt; 1480</td>
</tr>
<tr>
<td>5</td>
<td>650–880</td>
<td>1320–2280</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>6</td>
<td>750–850</td>
<td>1420–1620</td>
<td>?</td>
<td>&gt; 650; &lt; 860</td>
</tr>
<tr>
<td>7</td>
<td>820–1000</td>
<td>2640–3040</td>
<td>3160–3200</td>
<td>&gt; 830; &lt; 2640</td>
</tr>
<tr>
<td>8</td>
<td>650–800†</td>
<td>1180–2640</td>
<td>2420–2520</td>
<td>&gt; 710; &lt; 1180</td>
</tr>
</tbody>
</table>

*During i.v. lidocaine.
†During i.v. isoproterenol.

Abbreviations: Spont. SCL = spontaneous sinus cycle length (before vagal maneuvers); VE = ventricular escape; IVCL = idioventricular cycle length; Ex RP = exit refractory period; CVP = concealed ventricular parasystole.
Ventricular Escapes of Variable Coupling and the "No Warming-up" Phenomenon

A VE is usually an expression of the inherent normal automaticity of a nonprotected ventricular pacemaking cell, and its occurrence presupposes that a quiescent, inhibited subsidiary pacemaker may break out of the control of the dominant pacemaker.1 How the dominant rhythm exerts its inhibitory action has been extensively studied.15,16 The pertinent point is that a nonprotected normal ectopic center will give rise to VE of fixed or scarcely variable coupling,11 provided activity of the dominant rhythm is relatively constant. While each impulse of the sinus rhythm constantly discharges and resets the subsidiary pacemaker, the escape interval must necessarily depend on the last time the pacemaker was discharged by a sinus impulse. On the contrary, the escape interval cannot be constant if regeneration of the ectopic impulses occurs earlier or later than the preceding sinus beat, as is the case in concealed VP, in which independent ectopic discharges occur regularly all the time but only become manifest when sinus activity is depressed. In the same way as variable coupling is a sign of protection in typical VP, it is also a sign of protection when exhibited by VEs. VEs of variable coupling are thus the hallmark of concealed VP.

In contrast to the variable coupling of the parasympathetic VE, the parasympathetic idioventricular rhythms showed a constant or nearly constant IIEI, without the initial warming up usually seen in nonparasympathetic automatic rhythms.1,11,15 This also reflects protection and may suggest the presence of a concealed VP. It may be argued that some ventricular rhythms that are nonparasympathetic show little or no warming up.16,17 In such cases, however, the ventricular automatic rhythm is usually much more rapid than seen in typical VP and that seems to reflect an abnormally enhanced automaticity,15,18 and not the inherently slow activity of subsidiary ventricular pacemaker cells.

VEs do not often occur during carotid sinus massage in the human or when a strong and sustained vagal stimulation is provoked in the experimental animal. When they do occur, the escape interval usually lasts several seconds in the human19,20 and over 15 seconds in the animal.11,12,22 This is the normal response of unprotected, inhibited subsidiary pacemaker cells, followed systematically by gradual acceleration (warming-up) of the escaping center.1,11,16 The classic "rhythm of development"23,24 and "pre-automatic pause"23 that occur in acute clinical or experimental artioventricular block allude to an essentially similar phenomenon. Accordingly, VE occurring after relatively short pauses may suggest an alteration of ventricular automaticity. If coupling is
variable, concealed VP is a likely mechanism; if coupling is fixed, an enhanced automatic focus should be suspected.

VEs of variable coupling occur in patients with phase 4 bundle branch block and were thought to arise from the injured fascicle. It may well be that some of those VEs were due to a concealed VP, but at that time such possibility was not investigated. Others have also postulated that patients with bundle branch block may develop VP arising from the diseased fascicle, and concealed discharges from injured fascicles have been shown to occur in phase 4 bundle branch block induced experimentally.

The Role of Vagal Stimulation

Concealed VP results from an ExRP induced by sinus impulses, and its cycle length is longer than the sinus cycle length. Vagal stimulation depressed sinus activity in order for the parasystolic VE to become apparent. In patients 2 and 6, parasystolic VE occurred spontaneously. Classic ventricular asystole provoked by vagal stimulation occurs because suppression of sinus node activity reveals the rate-dependent inhibition of nonprotected ventricular pacemakers. In concealed VP, suppression of sinus activity reveals the absence of rate-dependent inhibition of a protected ventricular automatic center. However, vagal stimulation may also have a depressing action on some idioventricular or parasystolic ventricular rhythms. In seven of our 49 patients with VP, carotid sinus massage caused lengthening of the IEI, followed by a gradual return to control values. This did not occur in the present study, in which the IEI maintained its regularity during vagal stimulation.

Relationship to Intermittent Parasystole:
Natural History of Ventricular Parasystole

Concealed VP becomes apparent when the sinus rate is slowed. In this sense, it is rate-related and an intermittent arrhythmia. However, it is quite different from so-called intermittent parasystole. Intermittent parasystole results because protection of the automatic focus is only partial. Essentially, there is an entrance refractory period (determined by activity of the parasystolic center) shorter than the parasystolic cycle length, allowing intermittent discharge of the automatic focus by appropriately timed sinus impulses. On the other hand, concealed VP is due to an ExRP (determined by sinus activity) longer than the sinus cycle length, precluding manifestation of the parasystolic discharges. Another difference relates to what might be called the natural history of VP. If entrance block disappears, parasystole can no longer exist, and this may well be a form in which VP may fade away or even be cured. In this regard, the partial unidirectional entrance block of intermittent parasystole indicates a lesser deterioration of conduction, and obviously, if entrance block becomes total, VP can no longer be intermittent. VP may also disappear completely if the partial exit block of concealed VP becomes total, even if the protected automatic focus persists as a silent, rhythmically discharging center. Concealed parasystole, which implies bidirectional block, may thus be closer to terminal or more advanced stages of the natural course of VP. In fact, total extinction of the VP seemed to occur in patients 2 and 3 during follow-up; in several cases, the rate of the concealed parasystolic focus was extremely low (table 2). This may also suggest that the entire process is closer to extinction, or that the same deterioration that causes bidirectional block may even affect the automatic activity of the parasystolic nucleus.

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