An Analysis of Fast Idioventricular Rhythm in the Dog

MARIO VASSALLE, RANDOLPH E. KNOB, MICHAEL CUMMINS, GUSTAV A. LARA, CARLOS CASTRO, AND JACKSON H. STUCKEY

SUMMARY  Fast idioventricular rhythm was studied in dogs with and without recently-induced complete atrioventricular block. The following results were obtained. The fast idioventricular rhythm (1) has an average rate of 92 ± 7.7 beats/min, (2) is either intermittent or continuous, (3) originates from either ventricle, (4) is initiated suddenly by a beat that usually has a different electrocardiogram configuration, (5) may undergo a moderate deceleration before ceasing abruptly, (6) inhibits normal pacemaker activity, (7) when no longer present, can be brought back by a short period of fast driving, (8) can be suppressed by fast driving but the suppression is often preceded by a transient acceleration, (9) requires a longer period of driving than a normal idioventricular rhythm to be suppressed, (10) is accelerated by a short period of driving, (11) is "reset" by driving, (12) can be induced at progressively faster rates by repeated periods of driving during recovery from a prolonged overdrive, (13) is accelerated by sympathetic stimulation or norepinephrine administration, and (14) is accelerated by short periods of driving during submaximal sympathetic stimulation. We conclude that the fast idioventricular rhythm is a form of repetitive activity requiring initiating beats and thus is less sensitive to overdrive suppression. The behavior of this rhythm differs from that of normal idioventricular pacemakers and suggests that this rhythm does not result from an enhancement of a normal pacemaker process but rather from a different mechanism. This mechanism is affected by sympathetic stimulation and by norepinephrine administration and this may result in ventricular tachycardias.

TWENTY-SIX dogs were studied shortly after induction of a complete atrioventricular (AV) block. The dogs could be divided into two categories on the basis of the idioventricular rate. In one category the idioventricular rate was below and in the other above 65 beats/min. The reason for selecting this rate to separate the dogs in two groups is that normal idioventricular pacemakers rarely show a rate of discharge higher than 65 beats/min even during maximal sympathetic stimulation. Therefore, it seemed unlikely that in the dogs with the higher rate the idioventricular rhythm could have resulted from an increment in normal idioventricular pacemaker activity. In view of the results obtained in the present study, this abnormal rhythm will be referred to as fast idioventricular rhythm. Several aspects of this arrhythmia are not understood. For this reason, dogs with complete block and a fast idioventricular rhythm were studied. The presence of complete AV block offers several advantages because the behavior of the fast idioventricular rhythm can be contrasted to that of normal pacemakers. For example, one can determine whether this abnormal rhythm is suppressed by overdrive. Also, it is possible to determine both the mode of initiation and of cessation of the fast idioventricular rhythm. In addition, the degree of control of the sympathetic system on these rhythms can be studied by stimulating the cardiac sympathetic nerves and administering norepinephrine. In another series of dogs without complete AV block, idioventricular pacemaker activity was explored by suppressing the sinus node by vagal stimulation.

Methods

The dogs investigated in the present experiments were anesthetized with thiopental sodium (Abbott), 15-20 mg/kg, iv, and complete AV block was induced by placing a suture ligature around the His bundle during venous flow occlusion. At 1-3 days after the operation the dogs were anesthetized with morphine sulfate (Lilly), 5 mg/kg, im, and a-chloralose (Fisher), 75 mg/kg, iv. They were ventilated with an Engstrom respirator (model 200, MIVAB Co.) and the thorax was opened through a midternal incision. A lead II electrocardiogram (ECG) was recorded and aortic blood pressure was measured using a polyethylene catheter connected to a Statham pressure transducer (model P23Db). Silver electrodes for recording bipolar electrograms were sutured to the epicardium of the right atrium (one electrode) and of the left ventricle (four electrodes). The ventricles were driven by means of electrical stimuli delivered through silver electrodes sutured to the epicardium of the right or left ventricle. Traces were recorded with an Electronics for Medicine DR 8 recorder on photographic paper moving at speeds of 10-25 mm/sec. Esophageal temperature was monitored by a Digitel Thermister Thermometer (model 581B) and was maintained at about 37°C with a Thermorite heating system. The ventricles were driven intermittently by means of an American Electronics Laboratories stimulator (model 104-A) connected to a stimulus isolation unit. The stimulus characteristics were 1 msec, 8 V, and a frequency of 12-240 pulses/min. The left stellate ganglion was isolated from all connections except the cardiac branches and was stimulated at selected frequencies by means of a bipolar gold electrode connected to a second American Laborato-
ties stimulator via a stimulus isolation unit. The stimulus characteristics were 1 msec, 8 V, and 5-20 pulses/sec. Norepinephrine (Levophed, Winthrop) was infused through a polyethylene catheter into the femoral vein by means of a motor-driven syringe (Harvard infusion pump, model 600).

In another series of experiments, dogs without AV block were anesthetized with morphine and chloralose. A thoracotomy was performed and electrodes were placed on the atrium and the ventricles as indicated above. The right vagus was isolated in the neck and severed. The peripheral end was stimulated as described above for the stellate ganglion. Vagal stimulation was carried out intermittently to suppress the sinus node or to induce atrioventricular block in order to study idioventricular pacemaker activity. During vagal stimulation, the ventricles were driven electrically, as described above. The dogs of this series will be referred to as "dogs with vagally induced AV block."

Experimental interventions usually were repeated more than once and the results averaged. The beats elicited by electrical stimuli delivered directly to the ventricles will be referred to as "driven beats." The extra beats induced either during or after a sequence of driven beats will be referred to as "induced beats." Stimulation of the stellate ganglion and infusion of norepinephrine will be referred to as "adrenergic enhancement." The idioventricular rhythm usually found in complete idioventricular block is characterized by (1) an uninterrupted series of beats, (2) a relatively slow rate (about 40 beats/min), (3) a prolonged period of quiescence following a period of fast driving, and (4) a gradual resumption of activity after a period of overdrive suppression: this rhythm is referred to as "normal idioventricular pacemaker activity." In contrast, the fast idioventricular rhythm will be referred to as an "abnormal rhythm."

Results
RATE AND SITE OF ORIGIN OF THE FAST IDIOVENTRICULAR RHYTHM

The average rate of the fast idioventricular rhythm, measured at the beginning of each experiment was 92 ± 7.7 (mean ± SE) beats/min (range, 66–132). It was regular in eight dogs either for part of the experiment (two dogs) or for the whole experiment (six dogs). In the two remaining dogs, it was irregular at all times as a result of the presence of two different pacemaker sites. The existence of two sites was suggested by different configurations of the QRS complexes and of the electrograms. Whether regular or not, the rhythm was continuous in five dogs and intermittent in the other five.

The QRS complex in lead II was positive in seven, diphasic in one, and negative in two dogs. If the polarity of the QRS complex can be taken as an indication of the origin of the impulses, the site of origin was located in either ventricle.

CESSATION AND RESUMPTION OF THE FAST IDIOVENTRICULAR RHYTHM

The spontaneous intermittence of the fast idioventricular rhythm observed in some dogs allowed an analysis of the relationship between this rhythm and the normal idioventricular pacemaker activity. This relationship is illustrated in Figure 1, in which three pairs of traces are shown. Each of the three pairs consists of the electrogram LVd (recorded from the distal area of the anterior wall of the left ventricle) and lead II of the ECG. In the top pair of traces, the fast idioventricular rhythm ceased abruptly after the 15 beats shown and was followed by a prolonged (19.4 seconds) period of ventricular standstill. The ventricular standstill was terminated by the onset of another rhythm characterized by 3 beats (the first ECG complex is diphasic, the second positive, and the third again diphasic) at a rather slow rate (average, 9.5 beats/min). The 3rd of these slow beats was followed by the sudden resumption of the fast idioventricular rhythm. The configuration and the rate of the QRS complexes of the resumed fast rhythm are similar to that seen prior to its abrupt cessation. Therefore, not only the cessation but the resumption of the fast idioventricular rhythm is an abrupt process. It should be noted that resumption of the fast idioventricular rhythm was initiated by a normal idioventricular beat. This is seen in more detail in the middle pair of traces and in the adjacent inset. Again, the abrupt cessation of the fast rhythm is followed by a prolonged period of ventricular standstill and by the delayed onset of normal idioventricular pacemaker activity. The normal idioventricular pacemaker activity is represented by 4 beats, the last 3 of which are labeled with the letters a, b, c. The fourth spontaneous
beat after the ventricular standstill (beat labeled c) was followed by resumption of the fast idioventricular rhythm. The inset shows three traces with electrograms recorded from the posterior wall of the left ventricle (LVp), the proximal (LVp), and middle (LVm) areas of the anterior wall of the left ventricle. The electrograms labeled a, b, c in the inset were recorded simultaneously with the QRS complexes labeled with the same letters. The electrograms in the inset recorded after those labeled with letters were simultaneous with the first 3 beats of the resumed fast idioventricular rhythm. Although both the normal and abnormal rhythm show negative (but not identical) QRS complexes, the electrograms recorded in LVm are obviously different and suggest a different site of origin for the two types of rhythms. In the bottom pair of traces of Figure 1 a similar sequence of events is shown again.

The cessation and resumption of the fast idioventricular rhythm was consistently abrupt whenever intermittence was present. The resumption of the fast rhythm was characterized by a first beat of different configuration, except for one experiment. This exception could be due to a close proximity of the normal and abnormal pacemakers.

In eight dogs the fast idioventricular rhythm ceased either spontaneously or as a consequence of a period of fast driving (see below), and the frequency of the normal idioventricular pacemaker activity then was 37 ± 6.7 beats/min. The difference between the rate of the fast idioventricular rhythm and that of the normal idioventricular pacemaker activity was statistically significant (P < 0.001). These results suggest that (1) the mechanism responsible for the fast idioventricular rhythm is different from that responsible for normal idioventricular pacemaker activity, (2) the fast idioventricular rhythm suppresses normal idioventricular pacemaker activity, and (3) the resumption of the fast idioventricular rhythm requires an initiating beat.

If a normal idioventricular beat is required to initiate a fast idioventricular rhythm, a short period of driving also might initiate a fast idioventricular rhythm. This was tested by allowing the fast idioventricular rhythm to subside spontaneously and normal idioventricular pacemaker activity to initiate. A brief (5 seconds) period of drive at 180/min resulted in the reappearance of the original abnormal rhythm. The resumed fast rhythm was fastest immediately after driving and slowed somewhat before ceasing abruptly.

**ELECTRICAL DRIVE OF THE VENTRICLES AT SLOW RATES**

Ventricular driving at rates varying from 12 to 60/min was carried out in eight dogs. Because the driving rate was slower than that of the fast idioventricular rhythm, only a fraction of the electrical stimuli excited the ventricles. When the stimulus induced an extrasystole, invariably the extrasystole reset the rhythm of the spontaneous beats. Thus, in each instance, the site of origin of the idioventricular beat was discharged by the driven beat. The interval following the driven beat, however, was not necessarily the same as the interval between spontaneous beats (Fig. 2). The rate of the fast idioventricular rhythm was 78 beats/min and that of the driven beats labeled with a dot was 60/min. The first driven beat slowed the fast rhythm enough for a second driven beat to occur. A slowing of the fast idioventricular rhythm by driven beats was observed in five experiments. In three experiments the postextrasystolic interval was little different from the interval between spontaneous beats. In every case the driven beats reset the fast idioventricular rhythm and indicated that the site of origin of this rhythm was not “protected” from the driven beat.

**THE EFFECT OF PROLONGED DRIVE ON FAST IDIOVENTRICULAR RHYTHM**

The question of whether a prolonged period of rapid driving would suppress a spontaneously occurring fast idioventricular rhythm was investigated next. In Figure 3, two traces are shown: the upper trace is lead II and the lower trace is a reference line. Two breaks divide the traces into three sections. The first section shows the normal idioventricular pacemaker activity (48 beats/min). The second section begins with a fast idioventricular rhythm which developed spontaneously. At the upward deflection of the reference line, a 15-second period of ventricular driving at 240/min was initiated. The stimulus artifacts are visible on the shifted reference line: the first and second stimuli failed to excite the ventricles but the subsequent stimuli consistently did so. The third section shows the last 6 driven beats after which the drive stimulus

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**Figure 2.** Effect of slow drive on the fast idioventricular rhythm. The interval between the 2 driven beats was 20.8% longer than the interval between 2 spontaneous beats and 14.8% longer than the cycle immediately following the 2nd driven beat. The two electrical stimuli were delivered through a stimulating electrode placed in the His bundle region at the time atrioventricular block was produced.
FIGURE 3  Suppression of a fast idioventricular rhythm by overdrive.

Suppression of a fast idioventricular rhythm by overdrive. The imposed overdrive resulted in the suppression of the abnormal rhythm. The normal idioventricular pacemaker activity was also inhibited as shown by the long pause following cessation of driving. However, it must be pointed out that in other instances a fast prolonged driving failed to induce suppression of a fast idioventricular rhythm. One of these instances is illustrated in Figure 4. The figure shows three traces. In the top trace, a fast idioventricular rhythm (90 beats/min) was overdriven at 240/min for 1 minute as indicated by the horizontal bar above the trace. The driven beats are labeled with a dot. Cessation of the driving was followed by suppression of the fast idioventricular rhythm; on the contrary, this rhythm initially was accelerated (95 beats/min). Yet, this fast idioventricular rhythm occasionally ceased spontaneously as shown in the middle and bottom traces. In both the middle and bottom traces, the resumption of the rhythm was characterized by a beat of a different configuration (labeled with an asterisk), possibly a normal pacemaker beat. Prolonged fast driving was carried out in eight experiments. In six of them it was possible to obtain suppression by suitably increasing the duration of the fast driving (180 or 240 stimuli/min) often in excess of 1 minute. When suppression was induced, it usually was preceded by a few fast spontaneous beats.

INDUCTION OF INHIBITION BY INTERMITTENT DRIVE

Overdrive suppression is obtained not only with prolonged fast driving but also with intermittent short periods of driving. This is due to the fact that inhibition summates with successive periods of driving provided the interval between them is not too long. It was of interest to study the effect of repeated short periods of driving on the fast idioventricular rhythm since a short period of driving may induce such a rhythm, as reported above. In Figure 5, four pairs of traces are shown. Each pair consists of the electrograms recorded from the distal (apical) area of the anterior wall of the left ventricle (LV a) and lead II. The top pair of traces began with a fast ventricular rhythm at a rate of 113 beats/min. After the fourth beat, a 5-second period of driving at 240/min was initiated. The driving stimulus was terminated, in the postdrive period the fast idioventricular rhythm (instead of slowing) accelerated to an average rate of 124 beats/min. A second period of driving was initiated as shown by the last 3 beats of the top pair of traces. When this was discontinued 5 seconds later, at the beginning of the second pair of traces, the fast idioventricular rhythm was still faster than control (average rate, 119 beats/min). However, after the third period of driving (third pair of traces), the fast idioventricular rhythm not only was slower (average rate, 101 beats/min) but seemed to cease altogether after 6 beats. This is confirmed in the bottom pair of traces. The last period of driving was followed by 5 beats at an average rate of 66/min and then by the abrupt cessation of the fast idioventricular rhythm. Since driving was not renewed, it can be seen that the initial "excitation" induced by drive was followed by complete suppression of the fast rhythm. Complete suppression might not have occurred if the short period of driving had been reinstated after a 5-second interruption. The experiments suggest that the fast rhythm is (1) initially accelerated by the short periods of driving, (2) eventually suppressed as inhibition summates over repeated periods of driving, and (3) "renewed" by each period of driving.
but at a lower rate due to the increasing inhibition. As usual, the suppression of the fast rhythm was followed by a long pause and the onset of normal pacemaker activity (last beat in the bottom pair of traces). Intermittent driving caused a progressive slowing of the fast idioventricular rhythm in four out of five experiments. In the fifth experiment there was no suppression.

**INDUCTION OF A PROGRESSIVELY FASTER IDIOVENTRICULAR RHYTHM AS INHIBITION IS DISSIPATED**

If the interpretation offered above is correct, it should be possible to reverse the sequence of excitation and inhibition by suitable procedures. This was done in one experiment as illustrated by the four pairs of traces in Figure 6. The top pair of traces begins with the control fast idioventricular rhythm (81 beats/min). As indicated above the traces, the ventricles were driven at 240/min for 2 minutes: the cessation of the drive stimulus was followed by a single beat (solid triangle) and then by prolonged ventricular standstill. The ventricular standstill was terminated by a single spontaneous beat followed by the first 3 beats of a 5-second period of ventricular driving. The second pair of traces begins with the last 3 driven beats of that short period of driving. The cessation of the drive stimulus was followed by 2 induced beats (solid triangles), then a prolonged pause and eventually by the resumption of the 5-second period of driving. The third pair of traces begins with the last 3 driven beats. The cessation of the drive stimulus was followed by an increased number of induced beats (solid triangles) and a shorter ventricular standstill. The bottom pair of traces begins with the last 3 beats of the short period of driving. Cessation of the drive stimulus was followed by a larger number of induced beats and the rate of this induced rhythm is actually faster (average rate, 92 beats/min) than the control. Also, the ensuing ventricular standstill is the shortest. The explanation of these findings appears to be as follows. The prolonged fast driving results in a marked suppression, as shown by the very long pause in the top pair of traces. As the inhibition wears off as a function of time, the ability of shorter periods of driving to induce excitation increases and this becomes apparent in the form of induced rhythms which are progressively faster and last longer.

**INFLUENCE OF THE ADRENERGIC SYSTEM ON FAST IDIOVENTRICULAR RHYTHM**

The effect of adrenergic enhancement on fast idioventricular rhythms is shown in Figure 7, in which three pairs of traces are displayed. Each pair consists of the electrogram recorded from the ventricle (LVd) and lead II. The top pair of traces begins with the control fast idioventricular rhythm. After the sixth beat, sympathetic stimulation was initiated and was continued for the remainder of the top and middle pair of traces. It is apparent that the rate of the fast idioventricular rhythm increased markedly to a peak value of 247 beats/min. After sympathetic stimulation had been discontinued, the fast idioventricular rhythm returned to its control value. The bottom pair of traces begins with the control fast idioventricular rhythm. After the seventh beat, norepinephrine infusion was started and samples of recording at the 3rd, 4th, and 5th minutes of infusion are shown. Under the influence of norepinephrine, the rate of the fast idioventricular rhythm increased to a peak of 221 beats/min. In the 5-minute recording, a period of driving at 240/min, instead of suppression, induced an 11% acceleration. In six experiments the control rate prior to sympathetic stimulation was 92.3 ± 10.4 beats/min; during sympathetic stimulation it increased to 145.5 ± 28.7 beats/min (P < 0.05). In six experiments the control rate prior to norepinephrine was 109 ± 17.3 and during norepinephrine administration it increased to 123 ± 19.9 beats/min (P < 0.05). It has been demonstrated that driving during sympa-
thetic stimulation may precipitate a tachycardia. A similar phenomenon was found for the fast idioventricular rhythm. In the procedure followed, sympathetic stimulation was initiated first and the rate of the fast idioventricular rhythm allowed to increase to a new steady value. While sympathetic stimulation was continued, the ventricles were driven at 180/min for 5–30 seconds. On cessation of driving, no suppression was present but, on the contrary, there was an acceleration of the fast idioventricular rhythm. Overdrive during sympathetic stimulation or norepinephrine administration was carried out in seven experiments and it was followed consistently by an acceleration of the fast idioventricular rhythm.

UNMASKING A FAST IDIOVENTRICULAR RHYTHM IN DOGS WITH VAGALLY INDUCED AV BLOCK

Repeated vagal stimulations were carried out in five dogs with the AV conduction system intact. In three dogs vagal stimulation led to an immediate ventricular standstill and then to the onset of normal idioventricular pacemaker activity. In a fourth dog there was the usual sequence but intermingled with the normal pacemaker activity there were bouts of a fast rhythm. The fifth dog also showed an intermingling of normal and abnormal idioventricular activity. The findings in this last dog are illustrated in Figures 8 and 9. In figure 8, panel A shows lead II, the blood pressure tracing, and the zero reference line. At the arrow vagal stimulation was initiated and led to the suppression of sinus rhythm. However, instead of the usual ventricular standstill, an idioventricular rhythm was immediately present, discharging initially at an average rate of 62 beats/min and later on (panel B) at 72 beats/min. Toward the end of panel B, a 10-second period of right ventricular driving at 180/min was initiated. The last 3 driven beats are shown at the beginning of panel C and it is apparent that the cessation of the drive stimulus was followed by little inhibition of the idioventricular pacemaker. This lack of suppression by overdrive shows why the abnormal rhythm came into play immediately at the beginning of vagal stimulation: this rhythm was neither suppressed by the faster sinus rhythm nor by the electrical overdrive. In panel D, the cessation of vagal stimulation at the arrow was followed by the immediate resumption of sinus node dominance. As shown above in the dogs with complete AV block, a fast idioventricular rhythm may cease spontaneously. Therefore, it is not surprising that on repetition of vagal stimulation (panel E), the usual marked inhibition of normal idioventricular pacemaker activity was present, instead of a fast idioventricular rhythm. In panel E (which shows lead II, the blood pressure tracing, and the zero reference line), vagal stimulation was initiated at the arrow and the sinus rhythm was immediately suppressed. Ventricular standstill followed: excluding the first (sinus escape) beat, the standstill lasted 14.8 sec. Toward the end of panel E, normal idioventricular pacemaker activity appeared (last 3 ventricular beats) which typically accelerated toward a steady value. In panel F, the rate for the first 2 normal idioventricular beat was 49 beats/min. After these beats a fast idioventricular rhythm appeared (large negative complexes with a rate of 86 beats/min). In panel G, the fast idioventricular rhythm was overdriven at 180 beats/min for 10 seconds and the driving was followed by a slight acceleration, instead of suppression. In panel H, vagal stimulation was terminated at the arrow and the sinus rhythm promptly reappeared. The persistence of the fast idioventricular rhythm was demonstrated by renewing
vagal stimulation as illustrated in Figure 9, in which three sets of traces are shown. The first set includes lead II, the blood pressure tracing, and the zero reference line. The other two sets of traces consist of lead II and a reference line. The top set of traces shows that shortly after the beginning of maximal vagal stimulation, the fast idioventricular rhythm (seen previously at the bottom of Fig. 8) was present immediately at a rate of 67 beats/min. The cessation of vagal stimulation is indicated by the return of the reference line to the original level and this was followed shortly by the resumption of sinus rhythm. Several beats after the cessation of vagal stimulation, one sinus impulse failed to propagate to the ventricles (spontaneous AV block) and a ventricular extrasystole (marked by an asterisk) occurred. The extrasystole had a QRS complex similar to those of the fast idioventricular rhythm. In the middle pair of traces, graded vagal stimulation was begun at the arrow. The sinus rhythm progressively slowed and this eventually allowed the fast idioventricular rhythm to emerge temporarily. In the bottom pair of traces, the first three complexes are of sinus origin, the fourth is obviously a fusion beat, and the following five are caused by the fast idioventricular rhythm. After 3 sinus beats, the fast idioventricular rhythm emerged once more. At the arrow, graded vagal stimulation was terminated and the sinus rhythm resumed. These findings demonstrate that fast idioventricular rhythm is inhibited neither by fast driving by sinus rhythm nor by vagal stimulation. In other runs (not shown) during a 3-minute vagal stimulation, the ventricles were driven for 10, 20, and 30 seconds at 240/min and no ventricular standstill followed. Although in none of the instances discussed there was a ventricular standstill as a consequence of fast driving, the fast idioventricular rhythm in Figure 8 was faster in panel B than in panel A, suggesting that a degree of inhibition was present.

Discussion

The results of this study suggest that the fast idioventricular rhythm (1) is not due to acceleration of normal pacemaker activity, (2) is due to a mechanism requiring initiating beats, (3) is a form of repetitive activity which can be induced or accelerated by fast driving of optimal duration, (4) is subject to overdrive suppression but far less than normal pacemaker activity, and (5) is accelerated by adrenergic enhancement and, as a consequence, may become a ventricular tachycardia.

The factors responsible for the presence of the idioventricular rhythm in the dogs studied is not clear. The adre-
nergetic enhancement brought about by operative and post-
operative stress\(^4\) is a possible factor as sympathetic stimu-
lation and catecholamine administration can lead to ab-
rupt ventricular tachycardias in the absence\(^5, 6\) and in the
presence\(^6, 7\) of complete AV block. It is possible, however,
that other changes associated with the operation for the
induction of the AV block also may play a role in the
development of the fast idioventricular rhythm.

Whatever the factors involved, there are many similari-
ties between the fast idioventricular rhythm studied in the
present experiments and that first described by Harris\(^8\) and
termed differently by different investigators.\(^9-11\) Thus,
both (1) are of ventricular origin, (2) have a rate much
faster than that of normal idioventricular rhythm but usu-
ally below 100 beats/min, and (3) lack protection in that
the ectopic focus is discharged by a capturing beat. There-
fore, it seems possible that the abnormal rhythm studied
here may be related to the fast idioventricular rhythm
observed clinically. In the present experiments, of course,
the AV dissociation was present at all times in the form of
complete AV block and the capture beats were obtained
by means of electrical stimulation.

The ability of the driven beats to reset the abnormal
pacemaker is demonstrated in each of the figures in which
driving is shown (Figs. 2–8). Figures 8 and 9 show that the
sinus node also resets the fast idioventricular rhythm, as
one would expect.

It is apparent that the fast idioventricular rhythm does
not result from an acceleration of normal idioventricular
pacemaker activity. This is demonstrated by several find-
ings. First, cessation of the abnormal rhythm reveals an
inhibition of the normal idioventricular pacemaker ac-
tivity, indicating that the normal idioventricular pacemaker
in actuality is suppressed (because it is overdriven) by the
fast idioventricular rhythm. Second, both the onset and
cessation of the fast idioventricular rhythm are abrupt
while normal idioventricular pacemakers tend to show
more gradual changes in rate. Thus, as a ventricular stand-
still is terminated, normal pacemakers never attain their
steady state rate in one cycle (see Figs. 1, 3, and 8, panel
E) and this should be contrasted with the behavior of the
fast idioventricular rhythm (see Figs. 1, 4, and 8, panel
A). Third, overdrive leads only to inhibition of normal
pacemaker activity while it induces or accelerates the fast
idioventricular rhythm. Overdrive also may suppress a fast
idioventricular rhythm but this usually is preceded by an
acceleration and the driving needs to last longer and be
faster than for normal pacemakers. Fourth, there is no
gradual transition between the rate of the normal and the
abnormal idioventricular pacemaker activity: the rate is
either typical of a normal idioventricular pacemaker or of
a fast idioventricular rhythm (Figs. 1, 3, and 8). Finally,
the fast idioventricular rhythm needs an initiating beat
while a normal idioventricular pacemaker initiates its own
activity. In fact, it must be assumed that the beat initiating
the fast idioventricular rhythm is due to the spontaneous
discharge of a normal idioventricular pacemaker (Figs. 1
and 4). Since the fast idioventricular rhythm is not due to
an acceleration of normal idioventricular pacemaker activ-
ity, the use of the term accelerated idioventricular rhythm
in this connection would be misleading. On the other
hand, it does not seem appropriate to define this rhythm as
a ventricular tachycardia. For these reasons, the term fast
idioventricular rhythm was used here.

The present findings show that the fast idioventricular
rhythm may be intermittent. This implies that, in the
absence of AV block, the sinus node may regain domi-
nance either because it becomes faster than the idioven-
tricular rhythm (Fig. 9) or because the latter undergoes
periods of quiescence. The intermittence was demon-
strated here also in dogs with vagally induced AV block
(Fig. 8). When the vagal stimulation was initiated during
the period of quiescence, the usual prolonged suppression
of normal idioventricular pacemaker activity was found
(Fig. 8, panel E). When, on the contrary, vagal stimula-
tion was initiated and the mechanism underlying the fast
idioventricular rhythm was operative, this abnormal
rhythm was present immediately (Fig. 8, panel A). Fur-
thermore, the fast idioventricular rhythm appeared during
vagal stimulation when normal idioventricular activity was
established already (Fig. 8, panel F).

A peculiar characteristic of the fast idioventricular
rhythm is its response to overdrive. This is a key point in
differentiating normal from abnormal idioventricular
rhythms. A normal idioventricular rhythm is inhibited by
overdrive whether the overdrive is slow or fast, short or

![Figure 9](https://example.com/figure9.png)
prolonged; the only difference is quantitative in that faster driving and longer periods of driving cause a more marked inhibition.\textsuperscript{16-17} The fast idioventricular rhythm, instead, responds to overdrive in different ways depending on the characteristics of overdrive. Thus, fast driving may induce (Figs. 5 and 6) or accelerate (Figs. 4 and 5) a fast idioventricular rhythm, and this effect is favored in the presence of adrenergic activation. Very prolonged fast driving may be needed to suppress the fast idioventricular rhythm and the suppression is often preceded by the induction of several spontaneous beats. Even then, as the suppression wears off, fast driving induces the onset of a fast rhythm (Fig. 6). In other words, overdrive causes only inhibition of normal idioventricular pacemakers and simultaneous inhibition and excitation of abnormal idioventricular pacemakers. Because driving favors the events which lead to spontaneous repetitive discharge of abnormal pacemakers, the abnormal pacemakers are less sensitive to the suppression by overdrive. In fact, if the period of driving is short (and therefore overdrive suppression small), excitation prevails and acceleration follows. This is because it takes fewer beats to induce the changes responsible for repetitive activity than those which suppress activity, normal or abnormal.\textsuperscript{2}

The mechanism responsible for the fast idioventricular rhythm is unknown. An important point in this regard is that the fast idioventricular rhythm, in contrast to normal idioventricular pacemakers, requires initiating beats generated either by normal idioventricular pacemaker discharge or by applied stimuli. This suggests that the induced repetitive activity could be due to afterpotentials (for a discussion of afterpotentials see Cranefield\textsuperscript{18}) and that these afterpotentials could progressively increase in size after each successive action potential of a fast drive. A short period of driving would then enhance the magnitude of the afterpotential and this would account for onset or acceleration of spontaneous activity. For longer periods of driving, the process of overdrive suppression is brought into play. Thus, both normal and abnormal idioventricular pacemakers are subject to overdrive suppression, but only abnormal idioventricular pacemaker activity responsible for fast idioventricular rhythm is enhanced by driving.

An afterpotential by definition requires a preceding action potential to be produced. This would explain the necessity of an initiating beat. The cessation of the fast idioventricular rhythm may involve a moderate reduction in the amplitude of the afterpotentials before the threshold is abruptly missed. Overdrive suppression may act just by shifting such an afterpotential to more negative values.

An alternative explanation is that the fast idioventricular rhythm results from a reentry mechanism. Many of the features described are compatible with the characteristics found in reentry rhythms elicited in vitro in partially depolarized fibers.\textsuperscript{19} While it is not possible at the present time to conclude that one or the other mechanism is operative, it must be pointed out that there are no reasons to believe that areas of partially depolarized tissue were present in the ventricles of the dogs with or without complete AV block. Furthermore, the QRS complexes of the fast idioventricular rhythm are not longer than those of a normal idioventricular rhythm, indicating that an obviously exten- sive reentry pathway is unlikely to be involved. This concept is reinforced by the fact that recordings from several sites on the left ventricle show that the activation of these different sites occurs within the expected time.

As to the autonomic control, the fast idioventricular rhythm is clearly not inhibited by the vagus. In contrast, it is accelerated by the sympathetic nerves and norepinephrine. It is of interest that the acceleration of the fast idioventricular rhythm by adrenergic enhancement is gradual except when it is precipitated by superimposed overdrive. The high rates often attained suggest that under the effect of adrenergic enhancement a fast idioventricular rhythm may evolve into a ventricular tachycardia.

In conclusion, the present experiments suggest that fast idioventricular pacemaker activity may result from a process which is different in several respects from normal idioventricular pacemaker activity. The outstanding characteristic of this abnormal pacemaker activity is its frequency dependence, a point which may account for its ability to escape overdrive suppression by the sinus node under certain circumstances.

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