Ventricular-triggered Pacemaker Arrhythmias*

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There have been few published reports dealing with the electrocardiographic changes produced by ventricular-triggered pacemakers (Center, Samet, and Linhart, 1968; Dreifus *et al.*, 1968; Furman, Escher, and Solomon, 1967; Furman and Escher, 1968; Murphy, 1968; Faivre *et al.*, 1968; Scheppokat *et al.*, 1968; Sowton, 1968a). This contrasts with the more extensive information with atrial-triggered units. Since the former are apparently being used with increasing frequency, it seemed of interest to present the fundamental electrophysiological characteristics of those pacemakers which are synchronized to the ventricular deflections. The complex disorders of rhythm that they can produce have created a new chapter in the field of iatrogenic electrical arrhythmias. Their significance should be well known, and the distinction drawn between the ones indicating pacemaker failure and those seen during the normal function of these units.

Sowton (1968b) has emphasized that non-competitive pacemakers may be of two types: ventricular-triggered (Neville *et al.*, 1966) or ventricular-inhibited demand (Lemberg, Castellanos, and Berkovits, 1965; Parsonnet *et al.*, 1966; Chardack *et al.*, 1968). Moreover, he also stressed that there are two different modalities of ventricular-triggered pacemakers. Sowton's classification will be followed in the present communication.

VENTRICULAR-TRIGGERED PACING WITH RELATIVELY LONG R-TO-SPIKE INTERVAL

The first attempts in *intentional* ventriculo-synchronization were performed in patients in whom an atrial-triggered unit was attached to a right ventricular endocardial catheter. Both sensing and stimulation were performed through the ventricular electrode. This arrangement resulted in uni-

polar ventricular-triggered pacing during which the R-to-spike distance was in the same range as the P-to-spike distance in instances of conventional atrial-triggered stimulation.

Fig. 1 was obtained from a patient with complete AV block. Small diphasic P waves appear at different moments of the cycle. The ventricular complexes are followed by large unipolar spikes. Note that the spike which follows a ventricular extrasystole (arrow) falls close to the peak of the T wave. This spike is ineffective, but it would probably have been able to stimulate the ventricles if it had fallen a few msec. later. These pacemakers had a refractory delay of 0.5 sec.; a maximum rate of 125 a minute, and an escape rate of 60 a minute.

VENTRICULAR-TRIGGERED PACEMAKERS WITH SHORT DELAY BETWEEN ONSET OF DEPOLARIZATION AND EMISSION OF SPIKE

Our experience has been obtained through the use of the "Ectocor" pacemaker manufactured by the Cordis Corporation, Miami, Florida, U.S.A. (*Ectocor Synchronous Standby Pacemaker*, 1968).

General Characteristics of Ectocor Pacemaker. The main feature of this pacemaker is that the interval between the onset of ventricular depolarization and the spike is very short. When a QRS complex is detected, the electrical stimulation to the heart is applied when the *ventricles* are totally refractory. Artificial escapes occur if natural beats fail to appear for an interval of around 0.86 sec. (corresponding to a rate of 70/min.). This first model has a built-in refractory period of 0.50 sec. Spontaneous ventricular deflections falling during this interval will not be detected. An increase in the spontaneous ventricular rate to 125 immediately produced a 2:1 ventriculo-pacemaker block. Data presented in this communication apply only to a

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FIG. 1.—Ventricular-triggered pacemaker with a relatively long R-to-spike interval (Atriacor sensing through a ventricular lead). The spike synchronized to a premature ventricular contraction falls on the peak of the antecedent T wave.

FIG. 2.—Testing of a ventricular-triggered (Ectocor) pacemaker.

pacemaker with these characteristics, because the latest model has a refractory delay of only 0.40 sec., so that 2:1 responses will not occur until a rate of 150 a minute is achieved.

The presence of spikes during the absolute refractory period of natural beats does not assure that they can stimulate the ventricles effectively if they fall in the responsive portion of the cycle. Therefore, the manufacturers include a magnetic switch in the pacemaker circuitry. A magnet placed over the pacemaker will short out the detecting function, changing the ventriculo-synchronized pacer into an asynchronous one firing at a fixed rate (Fig. 2). It is recommended that this manoeuvre be tried first in evaluating the performance of the Ectocor pacemaker in patients with sinus rhythm. Slowing of the rate by means of carotid sinus massage or edrophonium chloride (Sowton, 1967), performed with the purpose of producing pacemaker escapes, should be attempted only when the observer is reasonably sure that the spikes will be able to stimulate the ventricles when falling outside the refractory period. The size of the spike is independent of its stimulating abilities (Siddons and Sowton, 1967).

The early Ectocor models were sensitive to external interference created by electrocauteries, diathermy machines, and electric shavers. In these instances, the maximal rate attained was limited, by the duration of the pacer's own refractory period, to 125 beats a minute.

ARRHYTHMIAS DURING NORMAL OPERATION OF VENTRICULAR-TRIGGERED (ECTOCOR) PACEMAKER

It is important to make the distinction between the disorders of rhythm seen during normal operation of this pacemaker from those indicating malfunction, which require pacer replacement. One important characteristic of this pacemaker is that, in the presence of sinus rhythm, it might be difficult to determine whether the spike was triggered by the atria or by the ventricles. This is well seen at the beginning of Fig. 2, when the Ectocor pacer was connected to the ventricles. At first glance the spike seems to be synchronized to the P wave. An important clue in the identification of the modality of synchronous pacing during sinus rhythm can be obtained from the study of the morphology of pacemaker escapes. Escapes from atrial-triggered

FIG. 3. Ectocor pacer showing: (a) difficulties in identifying the spikes during sinus rhythm; (b) pseudo-currents of injury; (c) non-sensed ventricular beat; (d) false pacemaker escape; (e) stimulation on antecedent T wave.

units yield QRS complexes almost identical in shape to those present during sinus rhythm, since, in both instances, the ventricles are totally depolarized by the pacemaker. On the other hand, escapes produced by ventricular-triggered pacemakers are different from the ventricular deflections seen during sinus rhythm; whereas the former are exclusively pacemaker induced, the latter result primarily from atrial activation propagated through the anatomical AV pathways.

FALSE CURRENTS INJURY

Another feature of unipolar, ventricular-triggered pacing is shown in Fig. 3. The large spikes produce a significant distortion of the ST segments. These pseudo-currents of injury should not be interpreted as due to pericarditis or acute coronary artery disease. In fact, this tracing was obtained one year after implantation when the patient was asymptomatic. Evaluation of ST segment changes is very different in patients with Ectocor pacemakers.

FIG. 4.—Sensed and non-sensed ventricular extrasystoles leading to true and false pacemaker escapes.

EFFECTS OF PACEMAKER REFRACTORINESS IN SENSING EXTRASYSTOLES

The fourth QRS complex in Fig. 3 is an ectopic ventricular beat with a coupling of 0.44 sec. Therefore, it is not sensed by the pacemaker since it falls in the refractory period of the latter. In consequence, the extrasystole is followed by a "false" pacemaker escape appearing at an interval of 0.84 sec. after the last synchronized beat. The escape falls in the terminal portions of the extrasystolic T wave. It is precisely for this reason that the refractory period has been shortened from 0.50 sec. to 0.40 in the latest Ectocor models. The term "false" escape has been used to indicate that the beat under consideration appears after a pause which is shorter than the previous RR cycle.

Additional features are presented in Fig. 4. In the top strip, an extrasystole with a coupling of 0.52 sec. is properly sensed. A synchronized spike is seen in its middle portion. The post-extrasystolic pause is ended by a true pacemaker escape appearing 0.84 sec. after the extrasystole. In the bottom strip the same premature contraction has a shorter coupling and falls during the period of pacemaker refractoriness. This ectopic beat is not sensed so that it is not accompanied by a synchronized spike. As in Fig. 3, the post-extrasystolic pause is terminated by a false pacemaker escape occurring 0.84 sec. after the previous, normally synchronized, beat. Non-sensed extrasystoles are not followed by compensatory pauses, as in the top strip.

EFFECTS OF PACEMAKER REFRACTORINESS DURING RAPID VENTRICULAR RESPONSES

This is seen in Fig. 5 which starts with a pure pacemaker complex. It is followed by a supra-ventricular impulse, the spike being properly synchronized to the R wave. The third (spontaneous) QRS complex occurs during the period of pacemaker refractoriness; hence, it is not sensed.

FIG. 5.—Function of a ventricular-triggered (Ectocor) pacer during atrial fibrillation. X indicates a false pacemaker escape following a non-sensed supraventricular beat. (Courtesy of Dr. W. Feder.)

A 2:1 ventriculo-pacemaker block persists until a supraventricular beat fails to appear at the expected moment, so that the corresponding pause is ended by a false pacemaker escape (marked by an X). The bizarre disorder of rhythm disappeared when the patient received enough digitalis to block AV conduction.

ARRHYTHMIAS INDICATING PACEMAKER FAILURE

In contrast to the arrhythmias presented in Fig. 5, which are seen during the normal operation of the unit under consideration, there are certain disorders of rhythms which suggest malfunction. As with all type pacemakers, conspicuous changes in pacemaker rate, as well as variations in the size and spatial orientation of the spikes, are significant. Failure of a spike to capture the ventricles might not be detected during normal ventricular synchronization. As mentioned previously, the presence of a spike falling in the ventricular refractory period (hence being unable to stimulate these chambers) does not assure that it will capture effectively when falling later in diastole. As reported by Siddons and Sowton (1967), the size of the spike does not correlate with its stimulating potential. The top strip in Fig. 6 shows normal ventricular synchronization and no evidence of pacer failure. However, the same spikes when falling during the full recovery phase (bottom strip) are unable to yield an effective ventricular beat. Proper evaluation of these units requires a study of the behaviour of the spikes falling outside the refractory period. This tracing also shows another sign of pacemaker failure, i.e. a spontaneous change to a continuous mode of operation, with loss of synchronization.

Sensing of T wave is a usual manifestation of pacemaker failure. The top strip in Fig. 7 shows Ectocor escapes during sinus arrest. The RR intervals measure 0.84 sec. A conspicuous sinus tachycardia is seen in the bottom strip. The first QRS complex shows that the ventricular-triggered spike seems to be synchronized to the P wave. The second QRS complex is not sensed since it falls

FIG. 6.—Malfunction of a ventricular-triggered (Ectocor) pacemaker: (a) failure to capture the ventricles in late diastole and (b) spontaneous change to fixed rate.

within the period of pacemaker refractoriness. Therefore it does not trigger a spike. 2:1 ventriculo-pacemaker block occurs until the eighth QRS complex, which is produced by a spike falling a few msec. after the T wave of the preceding, non-sensed, beat. This spike appeared 0.56 sec. after the last synchronized spike, indicating that it is not a true pacemaker escape, since the latter should occur only after an interval of 0.84 sec.

EFFECTS OF ELECTRICAL STIMULATION DURING "ABSOLUTE" REFRACTORY PERIOD

Stimuli falling in the so-called refractory period are apparently ineffective. Yet, since the early days of electrocardiography, various investigators have been aware that they need not always be so. For instance, Drury and Love (1926), working in the laboratory of Sir Thomas Lewis, showed that

FIG. 7.—Failure of a ventricular-triggered (Ectocor) pacemaker: sensing of T waves.

FIG. 8.—Exposure of concealed digitalis extrasystoles by diastolic (X) and early systolic (X') pacemaker impulses (dog experiment).

properly timed stimuli delivered during the irresponsive period were able to inhibit those occurring somewhat later in the cycle, in spite of the fact that the former did not produce a detectable response in the peripheral leads. According to Brooks *et al.* (1955), stimuli delivered slightly after the onset of depolarization can create some sort of a long-lasting state. Orias *et al.* (1950) stressed that stimuli could produce excitatory effects during the irresponsive period even though not evoking a response. An ineffective local state thus created could be summated with the enhancing effects of a second, subthreshold, stimulus to produce an eventual response. Other investigators have stated that the excitatory effects of electrical stimuli delivered during the refractory period can outlive the latter (Forbes, Griffith, and Ray, 1923; Lucas, 1911; Moe, Harris, and Wiggers, 1941).

Lown, Kleiger, and Williams (1965), Childers, Rothbaum, and Arnsdorf (1967), and Ten Eick *et al.* (1967) showed that electrical stimulation in the countershock range can expose concealed digitalis arrhythmias even when delivered a few msec. after the R wave. In our laboratory we have seen the same phenomenon (Castellanos *et al.*, 1969), with pacemaker stimuli falling either after the onset of depolarization (therefore, producing a fusion beat) or shortly after the R wave (Castellanos *et al.*, 1969). A typical example is shown in Fig. 8. In the control strip diastolic impulses of twice threshold values produce a single response, as expected. A ventricular tachycardia was then induced with acetyl-

strophanthidin. Immediately after disappearance of the arrhythmia, diastolic impulses of a similar intensity (marked with an X) were able to reintroduce ectopic QRS complexes similar to those seen during the glycoside tachycardia. Finally, toward the end of the lower strip (X) an impulse falling 0.08 sec. after the R wave induced a similar phenomenon.

The clinical counterpart of this experiment is presented in Fig. 9. The records were obtained during co-action of a regular sino-atrial rhythm and a slower, fixed-rate, pacer. The large, unipolar, spikes are followed by very small QRS complexes. It is interesting to observe that ectopic beats appeared *exclusively* after spikes falling 0.12 to 0.18 sec. after the R wave of supraventricular origin since they were not seen in any other circumstance, though the electrocardiogram was recorded for long periods of time. The intervals between the spikes and the ectopic R wave ranged from 0.36 to 0.46 sec. It is possible that the spikes, including the one falling immediately after the end of the S wave (fourth strip), presumably in the so-called "absolute" refractory period, could have stimulated the ventricle after a very long latency. Whether the latter was a "true" latency (Brooks *et al.*, 1955) or included some degree of conduction or delay is irrelevant for the proposed interpretation of the tracing. If this were the case, stimulation would have occurred during the "relative" refractory period, not in the absolute refractory period, for a response, though delayed, did occur. The R-to-spike intervals were in the same range as that of some ventriculo-triggered pacemakers used clinically (Atriacor sensing through a ventricular lead, Fig. 1). Hence, stimuli falling close to the R wave can produce more significant after-effects than commonly accepted. Though these could be devoid of clinical importance, it has to be remembered that the vulnerable phase is located early in the relative refractory period.

SIGNIFICANCE OF VENTRICULAR-TRIGGERED PACEMAKER ARRHYTHMIAS

In the present report, we have not attempted to evaluate the clinical usefulness of ventricular-triggered pacemakers. A longer period of observation is necessary for this purpose, yet several authors are already satisfied with their performance (Center *et al.*, 1968; Furman *et al.*, 1967; Furman and Escher, 1968; Murphy, 1968; Faivre *et al.*, 1968; Scheppokat *et al.*, 1968; Sowton, 1968a).

However, subtle electrophysiological differences existing between the various types of pacers should be emphasized. For instance, arrhythmias that indicate malfunction of ventricular-inhibited demand

FIG. 9.—Ectopic beats (X) produced by pacemaker stimuli falling in early systole. The spikes fall in that part of the cycle usually considered to be “absolutely” refractory.

units can be seen during the normal operation of ventricular-triggered pacemakers. The reverse can also be true. Hence, a complete understanding of the specific parameters of a given instrument is necessary to determine whether the unit has to be replaced in a given circumstance. The iatrogenic disorders of rhythm shown in Fig. 1-7 appear to be complicated. Moreover, some of them have no spontaneous counterpart in the human heart. The most important consideration regarding iatrogenic pacemaker arrhythmia is that they have to be analysed in context, i.e. in relation to the type of pacing being used. Presentation of these arrhythmias is justified so that the physician using ventricular-triggered pacemakers can recognize the nature of events occurring in some patients. A new chapter has been opened in the field of arrhythmias—a brave new world that must be fully understood to enhance the safety with which these pacemakers can be used in the treatment of symptomatic AV conduction disturbances.

SUMMARY

Arrhythmias related to the use of ventricular-triggered pacemakers were presented. Differences

between older and newer models were stressed. The possibility that pacemaker stimuli occurring at the early part of systole could have some after-effects was considered.

A distinction between disorders of rhythm seen during the normal operation of these pacemakers and those indicating malfunction is necessary. Their recognition enhances the understanding, safety, and clinical usefulness of ventricular-triggered pacemakers. Confusion with atrial synchronization, non-sensing of early ventricular extrasystoles, false pacemaker escapes, significant false currents of injury, and stimulation during the antecedent T wave do not require pacemaker replacement. Malfunction is suggested by a spontaneous conversion to fixed rate, with persistent loss of synchronization, sensing of T waves, and failure to capture the ventricles. The latter possibility cannot be fully assessed during sinus rhythm, even when the spikes appear to be normally synchronized.

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