Reentry in the Atrium

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The concept of reentry as a mechanism for inducing ectopic activity in the heart is already old. The necessary conditions for reentry are:

1. a unidirectional block of the impulse
2. the conduction of the impulse over a circuitous or alternative route has to take so much time that the excitability of the tissue proximal of the site of block is restored
3. re-excitation of the tissue proximal of the site of block

In the second condition we use the term "excitability" rather than refractory period, since the time needed by fibers proximal of the site of block to restore their excitability in such an amount that the efficacy of the reentrant impulse is enough for excitation, will outlast the refractory period of these fibers.

One might expect that in the atrial myocardium reentry will occur only when slowly conducting fibers are involved in the circuitous route and therefore we first have to look at the sino-auricular node and the atrio-ventricular node.

Reentry in the SA node

HAN, MALLOZI and MOE [9] demonstrated in the isolated right atrium of the rabbit that an early atrial premature beat can invade the SA node and reenter the atrium. In their experiments they stimulated the atrium and after every tenth beat a premature atrial stimulus was given. Therefore the SA node did not discharge spontaneously ,but was overdriven via the stimulating electrode on the atrium.

BONKE et al. [6, 7] have investigated the effect of an atrial premature beat in the isolated spontaneously beating right atrium of the rabbit. They made multiple and simultaneous impalements with micro-electrodes in fibers of the SA node and were not able to demonstrate a reentrant activation of the atrium after an early premature beat.

The impulse of a premature beat, elicited by stimulation of the atrium shortly after the end of the refractory period, penetrates only a small part of the SA node, since the refractory period of nodal fibers lasts longer than of

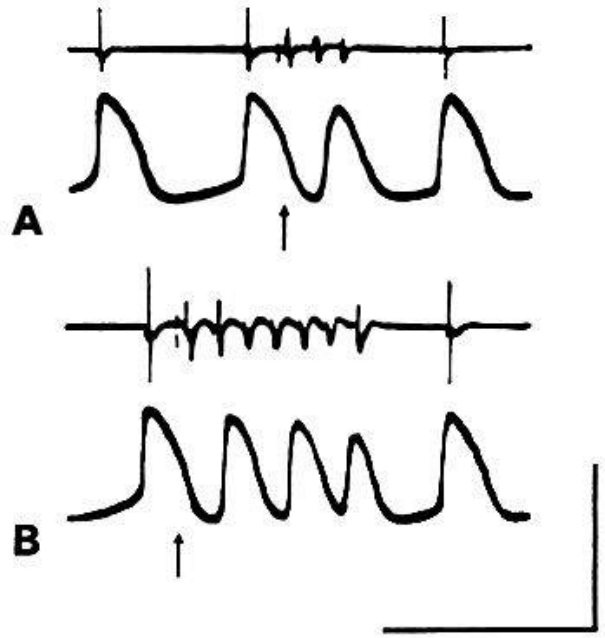

Fig. 1. The effect of an early atrial premature beat induced in the spontaneously beating right atrium preparation of the rabbit. In both recordings (A and B) the atrial electrogram, recorded with a surface electrode, is given above and the change of the transmembrane potential, recorded with a microelectrode impaled into a fiber of the SA node, below. The arrow indicates the moment the stimulus, inducing the early atrial premature beat, is given via a stimulating electrode on the atrium.
In A the atrial premature beat is followed by two atrial discharges of which the first preceeds obviously the nodal discharge. There are three atrial discharges and only one nodal discharge.
In B the same activation pattern is shown, but now the atrial premature beat is followed by six atrial discharges.
In this case there are seven atrial discharges including the premature beat and only three discharges of the impaled nodal fiber.
Although a number of simultaneous impalements of the SA node is necessary to draw conclusions about the activation pattern (see reference 7), it seems reasonable to conclude from these recordings that the atrial premature beat is followed by a number of "spontaneous" atrial discharges, whereas a SA node entrance block occurred allowing a 2:1 penetration of the SA node.
Calibrations: vertical bar: 100 mV; horizontal bar: 500 msec.

atrial fibers. Therefore there is an entrance block at the border of the SA node [8, 14], as shown in figure 1.

When the impulse of the premature beat invades part of the margin of the node, it is possible that the group of fibers, that is the functional pacemaker, is influenced electrotonically [4, 5, 7]. The first atrial activation after the premature beat occurs more or less at the expected moment as if there was no premature beat. Such an atrial premature beat is an "interpolated beat" [8]. When the premature beat is elicited somewhat later in the atrial cycle, the impulse reaches the pacemaker fibers, although with decrement, and discharges them. This causes a shift of the pacemaker within the SA node and only after a number of beats the original pacemaker will take over the function of pacemaker again.

In this case the premature beat causes an explicit change in rhythm.

We therefore will make the statement that in the spontaneously beating heart one atrial premature beat will not cause an echo beat in the atrium, at least in the rabbit. On the other hand, when the SA node is paced from the atrium, the situation is different[1] and a premature impulse can bring the SA node in a favourable position for reentry and therefore might elicit a tachycardia.

Reentry in the AV node

A circus movement within the AV node has already often been postulated as the mechanism responsible for a reciprocating tachycardia.

In the isolated right atrium of the rabbit including the AV node and a small part of the interventricular septum, JANSE et al. [10] using a tenfold microelectrode brush to impale into the AV nodal area, demonstrate that the impulse of an early atrial premature beat can be blocked somewhere in the AV node, whereas the impulse finds another route in the node from which both a reentry of the atrium and an activation of the His bundle is possible. Such a reentrant impulse can start a tachycardia based on a circus movement of the impulse within the AV node. The very slow conduction of the impulse within the AV node enables the occurrence of a circus movement in a very small part of the heart.

Reentry in atrial myocardium

BONKE et al. [7] found that an early atrial premature beat elicited in the spontaneously beating right atrium of the rabbit can be followed by a number of atrial activations. These are, as shown in figure 1, real atrial discharges and the SA node is activated only once per two atrial discharges. One may call this a 2:1 SA entrance block.

What mechanism underlies the occurrence of such atrial discharges?

There are two alternative possibilities:

1. The premature beat induces spontaneous activity in a group of normal atrial myocardial fibers.

Such a focus then acts as a temporary pacemaker.

2. The impulse of the premature beat is not conducted in the atrial myocardium in all directions as well and is blocked into one or more directions. Then the primary conditions for the occurrence of reentry and therefore for a circus movement exist.

We [1, 7] decided to investigate this phenomenon using the isolated left atrium of the rabbit. In this preparation there are, after cutting the entrances of the pulmonary veins, no spontaneously discharging fibers nor slowly conduct-

1 The course of the transmembrane potential, as well as the time relation between the moments of activation of the different fibers, are strikingly changed if the spontaneously discharging SA node is overdriven by an ectopic pacemaker.

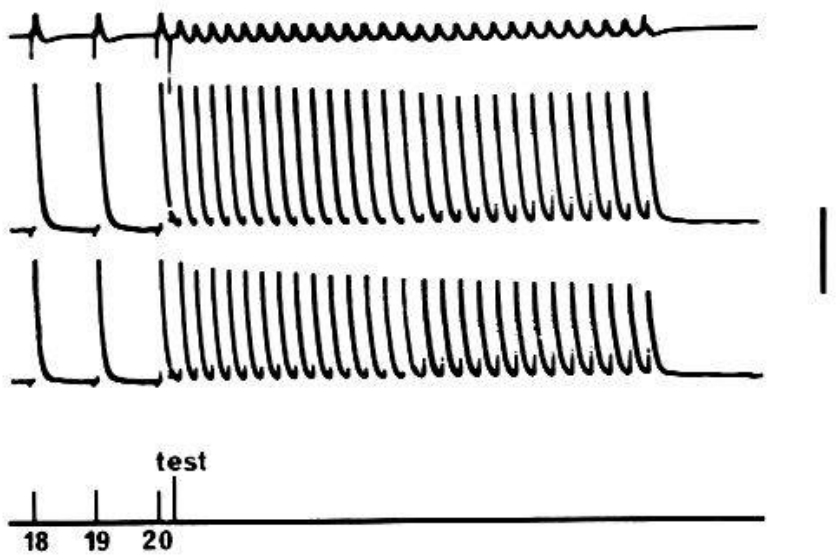

Fig. 2. The effect of an early premature beat in the left atrium of the rabbit. The electrogram, recorded with a surface electrode, is given uppermost. In the middle two recordings of microelectrode impalements of fibers of the left atrium are shown (The vertical bar at the right gives the calibration of 50 mV).
At the bottom the stimulation program is indicated. The preparation is stimulated 20 times with a rhythm of 3 Hz and an intensity of twice the threshold. The test stimulus is given after the 20th basic beat just after the end of the refractory period with an intensity of three times the threshold. Thereafter the preparation is not stimulated anymore.
In this figure the last three basic beats are shown. The premature beat is followed by a tachycardia of 26 beats, that stopped spontaneously.

ing fibers. The preparation was driven regularly and after every 20th beat an extra stimulus was given, followed by a period of at least 2 seconds without stimulation. It turned out that an extra stimulus, given just after the end of the refractory period, can be followed by a number of atrial discharges, as shown in figure 2.

The period in the atrial cycle in which a premature beat resulted in a series of rapid atrial discharges was very critical, often not more than 1 or 2 msec. Furthermore, the phenomenon could not be elicited everywhere in the left atrium preparation, but sometimes only at one or two places and sometimes nowhere.

On the other hand, when we had found a proper position for the stimulating electrode and the exact timing for the extra stimulus, these tachycardias could be induced reproducibly. We used a tenfold recording electrode and moved this device after each period of tachycardia to another site. So we were able to record the extracellular electrical activity of at least 300 sites on the preparation during the onset of tachycardia.

We checked by means of a control electrogram whether the sequence of the activations was the same during the subsequent tachycardias.

In figure 3 a complete map of the spread of activation is given. All sites activated at about the same moment (within 5 msec) are indicated by different

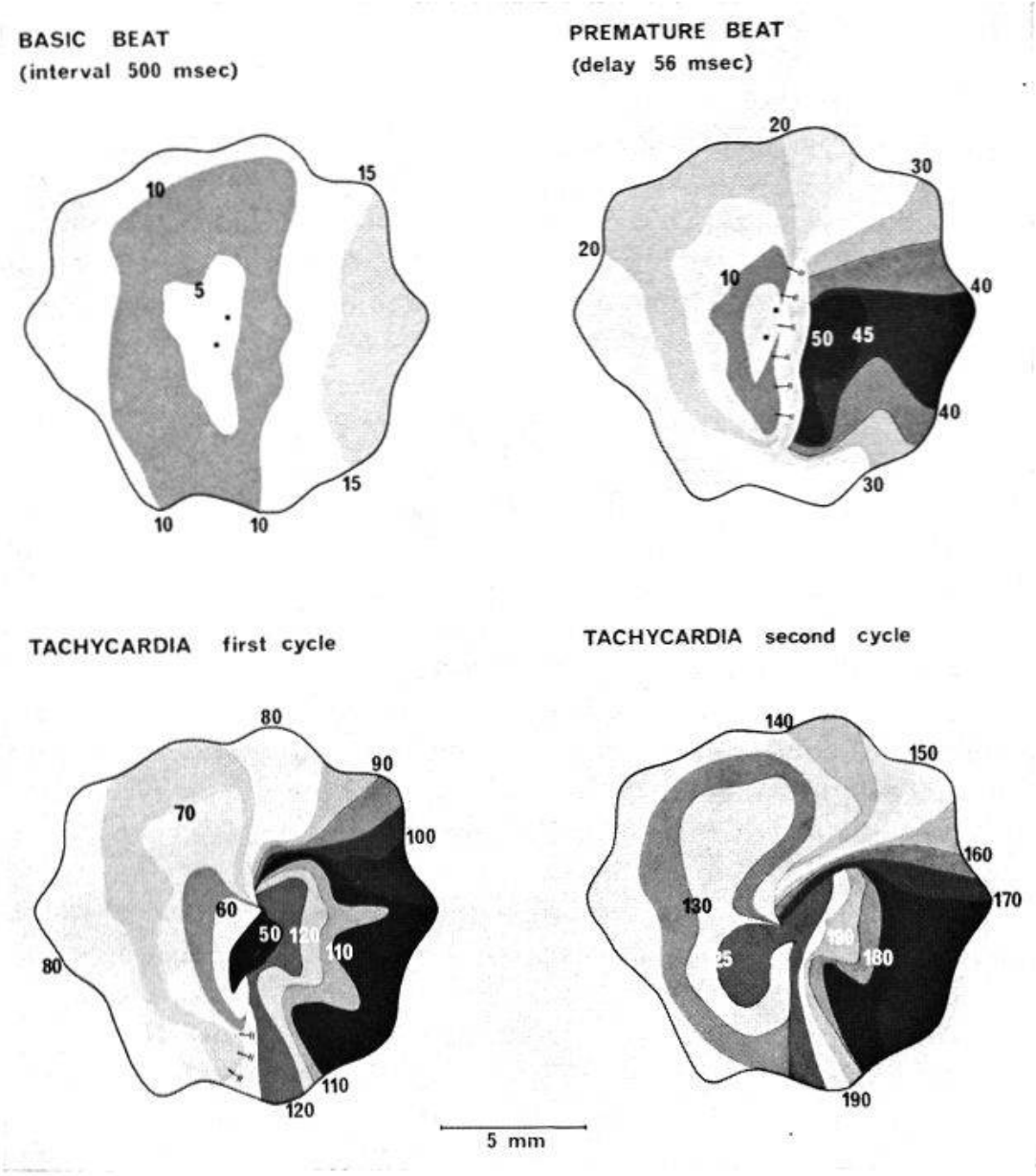

Fig. 3. Maps of the spread of activation composed of the activation times of about 300 sites during basic rhythm, the premature beat, and the subsequent two beats of the tachycardia. The site of stimulation is indicated by the two black dots in the map of the basic beat and the premature beat.
The parts of the preparation that were activated at about the same moment (with a range of 5 msec) are indicated with different colors (this figure is a black and white print of a color picture). The activation times are given at the site where the isochronal lines cross the circumference of the map and sometimes within the map for greater clarity. In the "Basic Beat" map these times concern the interval between the basic stimulus and the intrinsic deflection of the electrogram complex that was recorded, whereas in the other maps the activation times are related to the moment the test stimulus was applied.
Double bars indicate conduction block.

colors. During basic rhythm the impulse spreads from the point of stimulation – indicated by the two black dots – more or less radially. This concentric propagation was lost with the conduction of the premature beat elicited by a stimulus

56 msec after the last basic stimulus. In case of this premature beat the impulse was blocked to the right side from the stimulating electrode and was conducted about half as fast as during basic rhythm, at both sides around this blocked area. After about 60 msec the impulse reentered the site of stimulation and thus entrapped in a circuitous route. During the first cycle of the tachycardia the impulse again splitted up into two separate activation waves, but the lower one died out, apparently because the impulse traveled too fast in a circuit that was too small for every part of the route to restore its excitability. The other activation wave, however, could make another complete circle and reentered again, starting the second cycle of the tachycardia. This pattern of activation remained as long as the tachycardia persisted. In figure 3 the activation pattern during the first cycles of the tachycardia is shown.

From this figure it is obvious that these atrial tachycardias are based on the reentry of the impulse of the early premature beat. Thus a circus movement is the underlying mechanism of these atrial tachycardias.

Already LEWIS et al. [11] postulated a circus movement of the impulse in the atrium as a possible mechanism of atrial flutter.

They were not able to map the spread of activation carefully and assumed that the impulse was conducted around the orifices of the vena cava superior and/or inferior. Therefore in the hypothesis of Lewis the basic principle was the same as already mentioned by MINES [12, 13], namely a circus movement around an anatomical obstacle.

In the isolated left atrium of the rabbit there is no anatomical obstacle. We [3] therefore postulate the model of the "leading circle". The leading circle is the smallest circle in which the impulse can be conducted. In this pathway the front of the activation wave always encounters tissue that has just restored its excitability after the foregoing activation. From this leading circle the peripheral fibers are activated, whereas the impulse invading the centre will be blocked, since the excitability is not restored in time. In contrast with a circus movement around an anatomical obstacle, neither the position nor the dimension of such a circus movement are fixed (see figure 4).

One of the consequences of this leading circle concept can be described as follows. Let us assume theoretically that during an atrial tachycardia the refractory period of the atrial fibers is shortened for some reason, without a change in the conduction velocity of the impulse. In this case, the leading circle will become smaller and the revolution time, i.e. the time necessary to complete one circus movement, will be shortened and thus the cycle length during the tachycardia will decrease. If the impulse should be conducted around an anatomical obstacle, there would be no change in revolution time since only the amount of tissue of the circuitous path that is completely excitable – the white part in the schematic drawing on the right side of figure 4 – is increased.

Since acetylcholine shortens the refractory period and causes only a very small increase in the conduction velocity, our observation [3] that adding

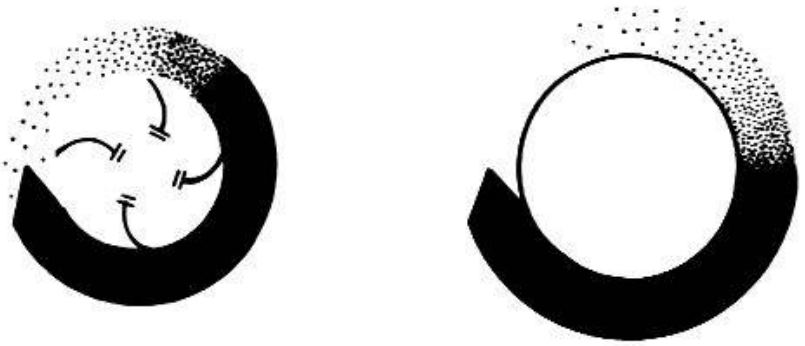

Fig. 4. Comparison of two models of circus movement.
At the right the model for a circus movement around an anatomical obstacle. The black area indicates tissue that is activated and not excitable. The dots are indicating the area that is more or less refractory, while in the white part the excitability is completely restored.
In the model at the left side there is no white area; the front of the activation wave encounters always tissue of which the excitability is restored just enough to be excited. In the centre the impulses are blocked. Such a "leading circle" is the smallest circle in which the impulse can be conducted, given a certain refractory period and conduction velocity.

acetylcholine during a long lasting atrial tachycardia causes an obvious increase in the frequency of the tachycardia is in agreement with the concept of the leading circle.

Further investigation is necessary to get more evidence for this leading circle concept [3], because in our opinion this will have consequences for the therapy of tachycardias based on a circus movement of the impulse.

Conclusion

Supra-ventricular tachycardias induced by a single atrial premature beat may be based on a circus movement of the impulse in different parts of the atrium (atrial myocardium, AV node and the paced SA node). The revolution time and the dimension of such movement depend on the electrophysiological properties of the part of the atrium where the impulse is circulating. However under normal conditions the occurrence of such a circus movement is rather rare – at least in the rabbit atrium – but under pathologic conditions a premature beat more often perhaps is associated with a reentry of the impulse followed by a circus movement.

Summary

Studies with isolated atrial preparations of the rabbit showed that the occurrence of a single early premature beat may cause reentry not only in nodal tissue (SA node and AV node) but also in working myocardial tissue.

In the SA node an early premature beat will cause a reentrant activation of the atrium only when the SA node is driven by an ectopic pacemaker.

If the SA node is discharging spontaneously, no reentry could be demonstrated. In this situation the early impulse can not reach the center of the SA node because of a sinoatrial entrance block.

Since the AV node fibers normally do not discharge spontaneously, an atrial premature beat may find an alternative route through the node and reenter the atrium. Such a reentrant beat or echo beat can start a tachycardia based on a circus movement of the impulse through the AV node.

A supraventricular tachycardia can be started too by an early premature beat in the isolated left atrium, containing only working myocardial fibers and no slow conducting fibers as the nodal fibers are. By careful mapping the spread of activation during the premature beat and the subsequent beats of the tachycardia, a unidirectional block of the impulse of the premature beat was demonstrated.

The impulse then turned around and invaded the blocked area retrogradely and reentered the area where it originated. This circus movement of the premature impulse was maintained during the subsequent tachycardial beats, showing that even in a small area of atrial muscle, containing no anatomical obstacle, a circus tachycardia can take place. To describe this kind of circus movement a new model (the "leading circle" concept) is introduced and briefly discussed.

Zusammenfassung

Untersuchungen mit isolierten Vorhofpräparaten vom Kaninchen zeigten, dass das Auftreten eines einzelnen früheinfallenden vorzeitigen Schlages Wiedereintreten der Erregung nicht nur im Knotengewebe (SA- und AV-Knoten), sondern auch im arbeitenden Myokardgewebe zur Folge haben kann.

Im SA-Knoten verursacht ein früher vorzeitiger Schlag nur dann eine wiedereintretende Vorhofaktivierung, wenn der SA-Knoten von einem ektopischen Schrittmacher gesteuert wird.

Wenn der SA-Knoten sich spontan entlädt, zeigt sich kein Wiedereintritt. In dieser Situation kann der frühe Impuls das Zentrum des SA-Knotens wegen einer sinoatrialen Eintrittsblockierung nicht erreichen.

Da sich in der Regel die Fasern des AV-Knotens nicht spontan entladen, kann ein vorzeitiger Vorhofschlag einen andern Weg durch den Knoten finden und wieder in den Vorhof eintreten. Ein solcher wiedereintretender Schlag oder Echo-Schlag kann eine Tachykardie auslösen, bedingt durch eine kreisende Erregung des Impulses durch den AV-Knoten.

Eine supraventrikuläre Tachykardie kann aber auch ausgelöst werden durch einen frühen vorzeitigen Schlag im isolierten linken Vorhof, welcher nur Arbeitsmyokard enthält und keine langsam leitenden Fasern, wie es die Knotenfasern sind.

Mittels sorgfältiger Aufzeichnung der Erregungsausbreitung während des vorzeitigen Schlages und der folgenden Schläge der Tachykardie konnte ein unidirektionaler Block des Impulses des vorzeitigen Schlages gezeigt werden.

In der Folge kreiste der Impuls, drang retrograd in die blockierte Zone ein und betrat erneut sein Ursprungsgebiet.

Diese kreisende Erregung des vorzeitigen Impulses wurde auch während der folgenden tachykarden Schläge aufrechterhalten und zeigte, dass sogar in einer schmalen Zone atrialer Muskulatur ohne anatomische Hindernisse ein tachykardes Erregungskreisen Platz greifen kann.

Zur Beschreibung dieser Art von kreisender Erregung wird ein neues Modell (das Leit-Kreis-Konzept) eingeführt und kurz diskutiert.

Résumé

Les recherches faites sur des préparations isolées d'oreillettes de lapin ont montré que l'apparition d'un battement précoce isolé provoque la stimulation de l'excitation non seulement dans le tissu nodal (sino-auriculaire ou atrio-ventriculaire) mais aussi dans le tissu myocardique fonctionnel.

Dans le nœud sino-auriculaire on ne voit une nouvelle activation auriculaire, lors d'un battement précoce, que lorsque le nœud S-A est dirigé par une impulsion ectopique.

Lorsque le nœud S-A se décharge spontanément, on ne voit aucune réactivation. Dans ce cas l'impulsion précoce ne peut pas atteindre le centre du nœud S-A à cause d'un blocage sinoatrial.

Cependant, comme dans la règle les faisceaux du nœud atrio-ventriculaire ne se déchargent pas spontanément, un battement auriculaire précoce peut trouver une autre voie à travers le nœud A-V et revenir dans l'oreillette. Une pulsation pareille qui a réapparu, dite «pulsation en écho», peut déclencher une tachycardie provoquée par une excitation rotative dans le nœux A-V.

Une tachycardie supraventriculaire peut aussi être déclenchée par une impulsion précoce dans l'oreillette gauche isolée, qui ne contient que du myocarde contractile et point de fibres conductrices lentes, comme le sont les fibres des nœuds.

Grâce à un enregistrement soigneux de la transmission de l'excitation pendant un battement prématuré et les battements suivants d'une tachycardie, on a pu constater un blocage unidirectionnel de l'impulsion dans l'extrasystole.

Par la suite l'impulsion tournoyait, puis en pénétrant par voie rétrograde dans la zone de blocage elle revenait de nouveau dans sa zone de départ.

Ce courant rotatoire de l'impulsion prématurée a persisté encore durant les pulsations tachycardiques qui ont suivi, et cela a montré que même dans une mince zone de musculature atriale sans obstacles anatomiques une impulsion rotatoire tachycardique peut prendre place.

Pour décrire cette espèce d'excitation rotatoire on a créé un nouveau modèle (le concept de «leading circle») et les auteurs le discutent brièvement.

Riassunto

Gli studi su dei preparati di tessuto atriale di coniglio hanno mostrato che l'apparizione di una extrasistole precoce isolata provoca un fenomeno di rientro non solo nei tessuti dei nodi seno-atriale e atrio-ventricolare ma pure nel tessuto miocardico non specifico. Nel nodo seno-atriale una extrasistole precoce provoca un'attivazione dell'atrio attraverso un fenomeno di rientro solo se il nodo seno-atriale è controllato da un segnapasso ectopico; se esso ha un'attività spontanea, il fenomeno di rientro non avrà luogo. In tale situazione, infatti, l'impulso precoce non può raggiungere il centro nodale a causa di un «blocco di entrata» seno-atriale. Dato che le fibre del nodo atrio-ventricolare non hanno normalmente un'attività spontanea, una extra-sistole atriale può trovare una via diversa attraverso il nodo e rientrare nell'atrio. Tale sistole da «rientro» o sistole da «eco» può essere all'origine di una tachicardia basata su un moto circolare dell'impulso attraverso il nodo atrio-ventricolare. Una tachicardia sopraventricolare può anche venir provocata da una extrasistole precoce nell'atrio sinistro isolato, che contiene solo muscolatura non specifica e nessuna fibra di conduzione lenta come il tessuto nodale. Esaminando accuratamente il propagarsi dell'attivazione durante l'extrasistole e le successive sistoli della tachicardia, si è dimostrata la presenza di un blocco unidirezionale dell'impulso extrasistolico; questo si diffonde allora all'intorno e invade l'area bloccata per via retrograda, per poi rientrare nell'area da cui ha preso origine. Questo movimento circolare dell'eccitazione prematura viene mantenuto durante le sistoli successive della tachicardia, ciò che mostra come anche in un'area ristretta del muscolo atriale, priva di ostacoli anatomici, possa originarsi una tachicardia per eccitazione a moto circolare. Per descrivere questo tipo di eccitazione circolare, viene presentato e discusso brevemente un nuovo modello (il concetto di «leading circle»).

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