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The serendipitous discovery of fulguration (high energy DC ablation)

The patient involved in the first fulguration was a 47 year-old man who had experienced a myocardial infarction ten years previously. This had been long forgotten until he experienced an abrupt syncope, followed quickly by a second episode. In light of these episodes, an electrophysiological exploration was performed in 1979 with the intention of highlighting a probable disorder of atrio-ventricular conduction. This disorder was presumed to be located below or inside the His bundle because the PR interval was not lenghtened. Apart from some extra-systoles, there were no disorders of intraventricular conduction resulting from the old myocardial infarction.

The insertion of an endocavitary exploration catheter identified a good His bundle potential associated with a normal HV interval of 40 ms. However, despite meticulous exploration by means of moving the electrodes on the atrio-ventricular junction, it was impossible to obtain the 2nd Hisian potential which could have documented a disorder of conduction located in the trunk of the His bundle. Such an interruption of conduction could have then led to a period of heart standstill and would have explained the symptoms.

During the second part of the study, while following a well-planned protocol, a dynamic study of atrio-ventricular conduction was done by programmed stimulation delivered on the atrium, which showed normal effective and functional refractory periods of atrio-ventricular conduction in the anterograde direction. At this point of the examination, the conduction system was cleared of any suspicion. However, in accordance with the regular electrophysiological study protocol used at that time, the atrial catheter was moved into the ventricle to practice a programmed ventricular stimulation in order to study retrograde conduction as well. It was not completely impossible that apparently normal anterograde conduction could have been revealed by an abnormal retrograde conduction (the reverse of what one generally observes).

By principle, this programmed ventricular stimulation created an extra-stimulus increasingly closer to the basic stimulations in order to trigger an artificial ventricular extra-systole applied progressively closer to the top of the 'T'-wave of the preceding QRS complexes, which were already of 'extra-systolic' type, having been induced by ventricular stimulation. However, during this protocol, an episode of ventricular tachycardia was obviously induced by the stimulation. We then used a short period of fast stimulation to stop the ventricular tachycardia at once, as is routinely done. But instead of stopping, the tachycardia accelerated. This is a known event which can itself be stopped by another burst of even faster stimulation, but in this case, the tachycardia had taken an extremely fast rate associated with hemodynamic failure promptly leading to loss of consciousness.

Although impressive, this was not completely exceptional. It had been known since the methods of programmed stimulation were introduced by a team of famous doctors in Amsterdam in 1972 that it was indeed possible to trigger an episode of ventricular tachycardia in the chronic phase after myocardial infarction. It was therefore possible to explain the syncopes of the patient as due to spontaneously recurring episodes of fast ventricular tachycardia with cerebral hypoxia, and not to a disorder of atrio-ventricular conduction!

It wasn't possible to discuss this reasoning in more detail because the tachycardia had quickly transformed into ventricular fibrillation. This is sometimes experienced in the electrophysiology lab, and is always something for which suitable measures must be taken as soon as possible, such as applying a defibrillation shock. At present, when one practices an electrophysiological exploration at the Institute of Cardiology in the Salpêtrière Hospital on a patient of this type, one always takes the precaution of placing two self-adhesive electrodes of defibrillation on the patient, one anterior and one posterior, both connected to a defibrillator with which such an incident can be quickly controlled. Indeed, as soon as ventricular fibrillation is authenticated on the electrocardiography and as soon as the patient has lost consciousness, the doctor or the nurse nearest the defibrillator can easily and immediately deliver a shock which restores normal sinus rhythm at once. But in those days, such electrodes did not yet exist. Then, in the event of syncope, one had to quickly grab the 'emergency reanimation cart', seize the manual electrodes, dab a good quantity of conducting paste onto the active metal plaque and apply them manually to the thorax of the patient.

It was impossible to know exactly what had occurred, but the fact was that when the first shock was applied and the spot (which had been driven off the screen) returned to view, it was obvious that the patient was still in ventricular fibrillation. Yet at first, this was something relatively banal, as it is known that the threshold of defibrillation is variable and that it is necessary in certain cases to apply a second shock to correct this rhythm disorder. The defibrillator was thus reloaded and a second shock applied, but when the spot quickly returned to the screen once more, it showed the patient was still in ventricular fibrillation. This was a situation encountered rarely in practice, but known to be possible. Under these conditions, the energy was increased. In fact, the maximal energy was used, which caused the defibrillator to take a noticeably longer time to reload. Once reloaded, a third shock was applied, the effect of which was exactly the same as that of the preceding two. The heart still remained in ventricular fibrillation. One could imagine that concern seized the operator because it was known that defibrillation became increasingly more difficult to correct as time passed.

The defibrillator was reloaded again with the same negative result. The fourth shock, and even the fifth, remained ineffective, even though the defibrillator functioned perfectly well, causing the patient to 'jump' with each discharge. At that point, it had become necessary to make instant decisions because the situation had turned life-threatening.

It was necessary to improvise hastily by tearing off the sterile fields, pushing back the radiology apparatus, applying a larger amount of paste to the electrodes and tightening the thorax between them. Then a sixth shock was given. This time, when the spot returned to the screen, ventricular fibrillation had disappeared. But after a moment of surprise, it was evident that the small round waves which appeared on the screen were not QRS complexes flattened by the effect of the shock on the electrocardiography amplifiers, but indeed authentic P-waves not followed by the expected QRS complexes. It became evident that there did not exist either a junctional escape or spontaneous ventricular rhythm, and that the patient remained inert. He was now in cardiac standstill with a complete 'solemn' atrio-ventricular block.

Very quickly, the ventricular catheter was moved to the ventricle and connected to the stimulator. After a few seconds, the patient made some convulsions, opened his eyes and gradually returned to consciousness. After a few more minutes of stimulation, he was able to answer the questions of the nurses more coherently, and everyone was reassured.

Yet that was not the end of it, because each time the stimulation was stopped, the electrocardiogram showed only P-waves, not followed by the normal ventricular complexes. They seemed sometimes to be followed by a QRS complex with a weakened, widened and sometimes fragmented His potential. After approximately one and a half hours of waiting, the patient was put back into His bed under electronic monitoring, with a catheter connected to an external stimulator fixed upon his thigh. He would remain there for nearly a week, at the end of which the complete atrio-ventricular block was as 'solemn' as during its first appearance. A pacemaker was implanted, which would be changed several times over the subsequent years. At present, the patient is with his seventh device and continues to carry out a normal life!

This entire incident, which could have easily turned into a catastrophe, was at first difficult to understand. Why had a complete atrio-ventricular block occurred during a series of external electric shocks, followed by a delayed but acceptable recovery? To this question, there was no clear answer in the literature of the time, especially considering the beginning of electrophysiological exploration which proved that the patient had a normal atrio-ventricular conduction. In addition, this complete block with absolutely no 'ventricular escape beats' was also in itself surprising.

At first, it was impossible to understand the electrical phenomenon that led to this surprising event. But, I remembered from having read several detective novels that a very important aspect of a police investigation is to reconstitute the environment of a crime at the place where it occurred. So, I gathered all the personnel who had attended the event and asked each of them to describe exactly what happened from their point of view. The only interesting item that emerged from this reconstitution was that one nurse said that she had heard a spark!

A spark? That point immediately attracted my attention and I asked what she meant by the word 'spark'. She said that 'spark' was the common term referring to an 'electric spark' that can sometimes be observed during the use of electrical apparatus. This was intriguing! How could a spark have occurred in such an environment?

The answer came several years later, probably when I was manipulating catheters and connector leads, the same equipment which was used during the initial event.

The 'spark' could have burst near one of the electrodes of defibrillation during the last shock. This spark occurred when the electrodes were correctly applied against the thorax with a large quantity of conducting jelly. In this case, it was known

that the current runs out between the broad surfaces of the two electrodes of defibrillation to cover the ventricular mass in near totality, thus allowing for an effective defibrillation. But how, under these conditions, could a spark have been possible?

The explanation lies in the particular environment in which the incident occurred. The proximal part of the endocavitary United States Catheter Instrument Company (USCI) catheter, pushed up by right femoral way, was connected to the electrophysiology amplifiers, while passing by a connection box assembled on a metal bar, located behind the head of the patient. A significant space was present between the proximal male part of the USCI catheter and the connecting box. However, it was found by chance that an interconnection cable made in France by a company called L'Electronique Appliquée, especially designed on my request to connect endocardial catheters to external stimulators for temporary pacing, could provide the explanation! The same equipment proved suitable to connect the junction box to the USCI catheter. On this side everything functioned correctly, except that because of the differences between inches and centimeters the male part of the USCI catheter was not going deep enough inside the female part of the interconnecting wire. This was sufficient to ensure a good electrical contact, but unfortunately left nearly one and a half centimeters of the metal connector of the catheter outside the connection and therefore exposed to any external contact (Fig. 1).

I realized that when the sterile fields had been withdrawn, the catheter was of course not disconnected and the exposed part of the connector displaced during the emergency move of the surgical fields came into electrical contact with the paste overflowing from one of the defibrillation electrodes. It happened that this connector was precisely connected to the intra-cardiac electrode exactly located on the His bundle. Part of the electric field intended for defibrillation could have been diverted through the paste towards the exposed connector, which had led to the creation of a spark and had explained the leakage of the current in the direction of the electrode placed on the His bundle. It is now well known that energies from a few joules to a few tens of joules are sufficient to definitively deteriorate atrio-ventricular conduction. The absence of escape rhythm can be also explained by the fact that the his bundle and its branches are more sensitive to current than the normal myocardium.

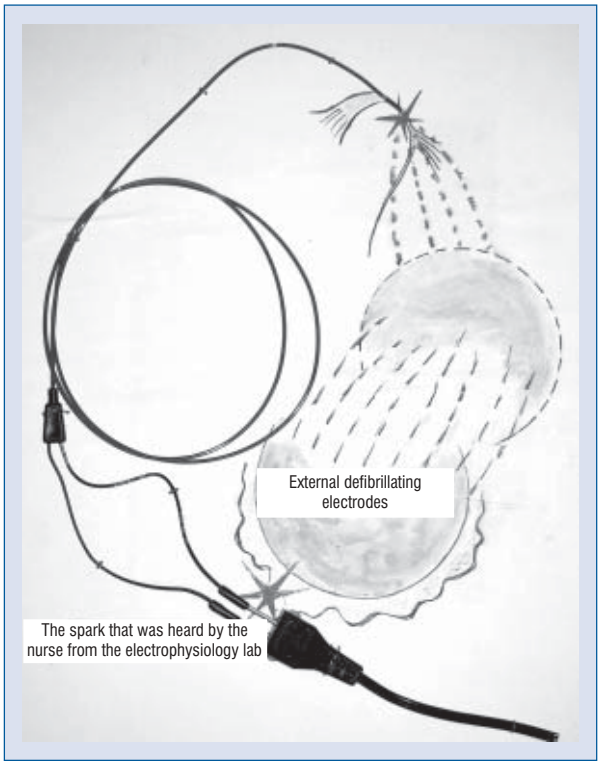

Figure 1. The mechanism of accidental His bundle ablation.

All of this is now clear, but the credit goes to one of us (Dr. Jacques Vedel) who was to write about it in an important scientific publication which would not pass unnoticed. Indeed, at that time, surgical methods were used to interrupt atrio-ventricular conduction, but when these methods were carried out on a purely anatomical basis, even in open heart surgery, they were not always assured of success. One knows now that the anatomical location of the His bundle can be located on the left side of the interventricular septum in certain cases. In such a case, it is necessary to approach the his bundle from the left part of the heart, which is possible, but more difficult, because the catheter must be introduced through the femoral artery and must cross the aortic valves with counter-current to reach the critical zone.

It was thus interesting to exploit the possibilities of the use of an electric current to obtain a voluntary interruption of atrio-ventricular conduction among patients with incessant supraventricular arrhythmias resistant to anti-arrhythmic drugs, and those who had a myocardium unable to tolerate open heart surgery.

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