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Atrial fibrillation: one more sporting inconvenience?

See page 477, doi:10.1053/euhj.2001.2802 for the article to which this Editorial refers

In a paper I submitted some years ago, I stated that every arrhythmia had something to do with the autonomic nervous system. Then I was asked by the reviewer to withdraw such a statement unless I could produce evidence of its reality. I asked, respectfully, for the referee to provide the evidence of a single arrhythmia that was free of any autonomic influence. Apparently the referee could not satisfy my request and, as a result, the initial statement was left in the final version. The philosophy of the story is that one cannot always provide evidence of a statement but we can stick to any theory as long as it does not contradict experiments or observations.

An arrhythmogenic substrate must be responsible for an arrhythmia even though its very existence cannot be demonstrated. There is no spontaneous arrhythmia genesis, and only transient substrates are conceivably undetectable. This leads to an analysis of the relationships between the two essential ingredients of any arrhythmia, the substrate and its modulating factors. As to the third ingredient, namely the initiating factor, it also depends on the autonomic nervous system, but not necessarily according to the same rules.

Every time an equilibrium exists between two factors, one should consider the situation created by the extremes of an imbalance, a scenario in which the role of one of the two factors largely predominates over the other. This leads to a discussion of the notion of a substrate's sensitivity to autonomic influences, as opposed to its strength. A curious paradox can then occur. It consists of the fact that the more visible one of the two factors the less important it is in arrhythmia determinism. A corollary is that the less easily detectable one factor, the more important it may be for the patient in terms of therapeutic consequences. As an example, what we called the adrenergic paradox^[1] supposes that any clinical arrhythmia triggered by strenuous exercise should correspond to an arrhythmogenic substrate relatively insensitive to adrenergic stimulation: the adrenergic stimulation must be strong to produce an effect. On the other hand, any clinical arrhythmia preceded by a limited heart rate increase should be suspected of being very sensitive to sympathetic stimulation.

These theoretical notions have practical consequences; for instance beta-blockade is indicated much more in the latter case compared to the former. The adrenergic paradox explains why rhythmologists have overlooked the therapeutic aspects of beta-blockade: a large body of evidence now indicates that adrenergic-dependent tachyarrhythmias must be more common in heart failure than in the absence of

myocardial impairment. The former actually benefit more than the latter from the beta-blocking treatment although the adrenergic mechanisms of their arrhythmias are not easy to see^[2]. Logically, the same sort of paradox should apply to the vagal side of the autonomic nervous system. The trouble is that vagolytic drugs cannot be used as easily as beta-blockers. Not only are we missing vagolytic drugs that could be active by the oral route, but their pharmacological action is often ambivalent: at low dosages, atropine or scopolamine are clearly vagotonic rather than vagolytic. Finally it should be recalled how complex is the interaction between the two facets of the autonomic nervous system and how cautious should be our hypotheses, which appear simple compared to reality^[3]. The notion of accentuated vagal antagonism proposed by Levy^[4] is probably as important in our understanding of vagally-mediated arrhythmias as the adrenergic paradox is in the management of adrenergically-mediated tachyarrhythmias.

It is necessary to keep in mind the above considerations when reading and interpreting the interesting report by Mont *et al.*^[5] in this issue. They studied the incidence and behaviour of paroxysms of AF in two cohorts of males less than 65 years, actively practising in sport or not. Sportsmen started their episodes of AF at a younger age and their attacks were predominantly vagal, in contrast to the sedentary patients. This notion would be easily accepted by clinicians with a large experience of paroxysmal idiopathic AF. Many times we reported on vagally-mediated AF, a clinical entity that is now largely accepted, although it is difficult to provide evidence of its reality^[6].

The clinician should be aware that at each and every step of vagally-mediated atrial fibrillations (AF), paradoxes or at least contrasts are observed. For instance, the clinical history is very suggestive when it shows the predominance of nocturnal or post-meal attacks, but it should be noted that the *absence* of any attack between breakfast and lunch-time is regularly observed, most probably because the predominance of the sympathetic activity exerts a protective effect. The predominance of arrhythmia onset at rest is also a good indication, but the onset of attacks during the period of relaxation that follows exercise or emotional stress is also common. The phenomenon of accentuated vagal antagonism is operative in these conditions and the protective effect of activity is frequently mentioned by patients who can escape from an impending arrhythmia just by walking. To conclude on the influence of sport, it is known that in well-trained people suffering from vagal AF, the first step of therapy should be deconditioning by discontinuing high-level training. It may

be sufficient to bring about an improvement in the patient and it is often a necessary adjuvant to facilitate pharmacological therapy. In this regard, among type I antiarrhythmics, flecainide and its vagolytic effect^[7] is much more effective than propafenone and its beta-blocking properties. Not only are beta-blockers ineffective, but they usually make patients worse and inhibit the efficacy of antiarrhythmics.

Alcohol is also a precipitating factor, possibly by a direct toxic effect. We think, however, that it has probably been overemphasized in the 'holiday heart syndrome'^[8], published by coincidence in the same year we identified vagal fibrillation^[9]. Another study^[10] is less firm about the direct and exclusive role of alcohol. The habits of the French differ somewhat from the populations of other countries. Not only is it recognized that French people do not restrict alcohol consumption to the weekend, but they predominantly drink wine rather than other forms. Patients can usually distinguish the wines that provoke their attacks: red more than white (with the noticeable exception of Champagne probably because of the mechanical effects of bubbles on the stomach and oesophagus) and the stronger Burgundy wines more than the lighter Bordeaux. We do not know why the direct toxic effect of alcohol appears to vary, although it is clear that in terms of digestive tolerance there is a difference.

In order to prove the vagal mechanism of initiation, the minutes and hours preceding arrhythmia onset need to be recorded in order to demonstrate the relationship between the attack and the heart rate decrease. The latter is still the best marker of vagal predominance, even though the increase in respiration-related heart rate oscillations is even more suggestive of vagal activity. The absence of documented arrhythmias at Holter and the absence of heart rate variability data are limitations of the Mont study^[5]. We can, however, take for granted that bradycardia and an increase in high-frequency heart rate oscillations are characteristic of trained people. What is more important is to note that no heart disease was present in any of the sports patients. Such an observation may look somewhat pleonastic against the notion of athlete's heart. In fact it is not. The point is that in our experience the presence of any myocardial disease is not compatible with a vagal profile of AF because the first effect of heart disease (not only heart failure) is to depress vagal activity.

The known causes of AF should not contradict the vagal profile of the arrhythmia, with the exception just mentioned on the usual incompatibility of a heart disease (or any cause of enhanced sympathetic stimulation) and vagal AF. But the now well known mechanism of ectopic foci located in the left atrium or

the pulmonary veins^[1] is quite compatible with any vagal or adrenergic profile. Ectopic foci in fact form the arrhythmia trigger, the behaviour of which may be sensitive preferentially to vagal or to adrenergic influences. It is our experience that vagal arrhythmias can be cured by a focal ablation. These foci, however, are more often sensitive to adrenergic than to vagal stimulation.

Waxman *et al.*^[12] made some interesting observations some years ago on the mechanism of the occurrence of AF as a consequence of paroxysmal reciprocating tachycardias. They nicely demonstrated that AF was the result of strong adrenergic stimulation evidenced by the rise in blood pressure and the acceleration of the rate of the tachycardia. Both reflected the sympathetic reaction to the initial fall in blood pressure resulting from sudden tachycardia onset. This type of AF can be efficaciously prevented by beta-blockers, which can, however, have a counter-productive corollary: by preventing blood pressure peak and its vagal consequences via the baroreflex, beta-blockers also prevent the vagal mechanism which allows the reciprocating tachycardia to block the atrioventricular node. No treatment is perfect.

Sport in general, and strong exercise in particular, are dangerous from the cardiologists' point of view, although their reservations about excessive sporting activities may not be popular and are frequently ignored. Usually the danger comes from ventricular tachyarrhythmias in the context of cardiac disease, such as cardiomyopathy, right ventricular dysplasia or the long QT syndrome, in apparently healthy young people. Compared to such severe accidents, AF looks benign but should not be neglected: its recurrence may be extremely disabling, but it is now

well established that the risk of ischaemic cerebral accidents is always present.


P. COUMEL

*Hôpital Lariboisière,
Paris, France*

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Tilt-induced asystole: a useful prognostic marker or clinically irrelevant finding?

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Vasovagal syncope (also called neurocardiogenic syncope) is a common clinical problem. It may occur at

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any age and accounts for more than one third of all causes of syncope^[1]. The diagnosis of vasovagal syncope is relatively easy in the presence of characteristic triggering factors (fear, severe pain, medical instrumentation, tiredness, prolonged standing, crowded places, warm environment) and prodromal symptoms