Relato de Caso

Anatomical and clinical aspects of the blood supply of the sinoatrial node

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SUMMARY — BACKGROUND. The recent study of the variations of the origin of the sinoatrial node and on the "arterial network of the perinodal sinusal area" in normal hearts points out the importance of this network.

PURPOSE. Report on a case of patient with syncope of ischemic etiology.

CONCLUSION. In this patient the arterial network did not protect the node from the ischemia caused by the obstruction of the artery of the sinoatrial node.

KEY WORDS: Sinoatrial node. Artery of the sinoatrial node. Sick sinus node syndrome. Syncope. Perinodal arterial network.

INTRODUCTION

Holden, in 1984[1], emphasized the correlation between the variability of the pacemaker blood supply and its clinical and surgical aspects. DiDio *et al*., in 1995[2], studied the origin of the artery of the sinoatrial node and the "perinodal arterial network", to support the activity of the sinoatrial (SA) node when the nodal artery is obstructed and causes ischemia. A patient with severe occlusion of the SA node artery and ischemia is reported as an exceptional case in which the collateral circulation from the arterial perinodal network was not sufficient to prevent the nodal dysfunction.

CASE HISTORY

A 44 year old, male, Caucasian patient, from São Paulo, presented syncopes after median and great efforts. The physical examination was normal and the electrocardiogram showed sinusal rhythm with 83 bpm cardiac frequency, disturbance in the conduction at the level of the right branch of the atrioventricular bundle (His bundle), left atrial enlargement and primary and diffuse alterations of the ventricular repolarization.

During the ergometric test the cardiac frequency, while remaining in sinus rhythm dropped from 130 bpm to 35 bpm without alterations of the T wave and ST segment.

The Holter test exhibited an average cardiac frequency of 60 bpm, ranging from 44 to 100 bpm, the minimum occurring during median stress. The cardiac electrophysiologic study detected the presence of a double pathway of the atrioventricular nodal conduction, without induction of tachycardia. The coronary angiography showed 40% obstruction of the lumen of the circumflex artery and the artery of the sinoatrial node originating at the level of the obstruction. The patient was submitted to the implantion of a programmable double chamber pacemaker. He became asymptomatic for 2 months, and then he began to present angina after walking 200m. A new electrocardiogram during effort displayed important ischemic alterations. The pacemaker maintained the cardiac frequency at 130 bpm without symptoms of cerebral ischemia but with precordial pain of typical angina. A subsequent coronary angiography showed 90% obstruction of the lumen of the circumflex artery (Fig.1). The patient was submitted to a coronary artery bypass grafting utilizing the left internal thoracic (mammary) artery.

DISCUSSION

The correlation between the variability of the cardiac pacemaker blood supply and clinical as well as surgical aspects, the latter emphasized by Holden[1] and Busquet *et al*.[3], was described by several authors. In fact, anatomical findings were correlated (a) to the sinoatrial heart block by Levine[4]. (b) to the tachycardia-bradycardia or sick sinus node syndrome by Kaplan *et al*.[5] and Pfeferman *et al*.[6], the etiology of which is variable[7] and not always determinable.

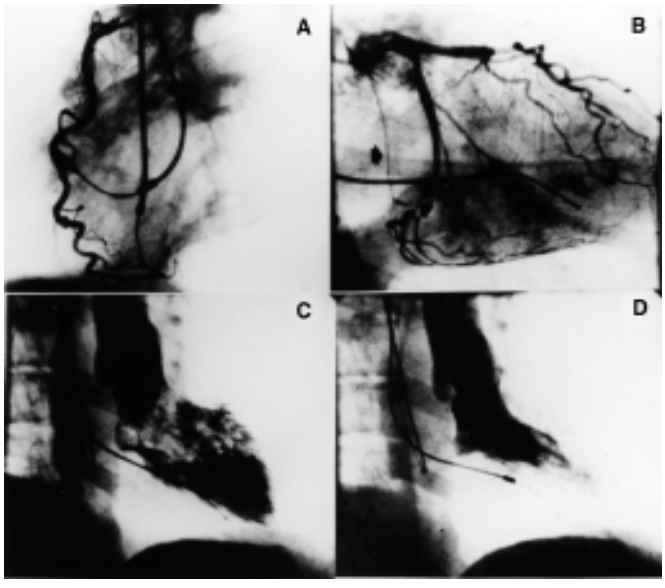

Fig. 1 – A) *Left coronary angiography in let anterior oblique view (LAO).* B*) Left coronary angiography in right anterior oblique view (RAO) shows 90% obstruction of the lumen of the circumflex artery* (▲). *The artery of the sinoatrial node given off at the level of the lesion displays less contrast (slow coronary flow)* (◗). C) *Left ventricular diastole (RAO) with normal function.* D) *Left ventricular systole (RAO) with little dysfunction of the diaphragmatic wall.*

Having studied the artery of the sinoatrial node[2], in a series of 100 normal human hearts, we showed that it originated more frequently from the right coronary artery (52%) than from the left (42%), confirming the results obtained by most authors. Such a collection of hearts constituted a unique series because the specimens were obtained from suicidal individuals or victims of automobile accidents, in whom the cause of death, as ascertained by pathologists evaluation, had no influence on the morphology of the heart, and they were considered normal. In this paper, both the origin of the sinoatrial nodal artery and the perinodal arterial network are considered, as the later may play an important role when there is a narrowing or an occlusion of the former.

The fact that the thick-walled artery of the sinoatrial node crosses the node may not mean that it nourishes the pacemaker, limiting its function to a periodic mechanical stimulus. The artery may have rami that branch in the vicinity of the node and contribute to the formation of what we call the "arterial perisinusal network". Such a network contains arterioles and capillaries which are the main source of blood supply of the cardiac pacemaker[2]. This assumption might explain the clinical cases in which the obliteration of the sinoatrial nodal artery does not cause any cardiac dysfunction because the perisinusal arterial network supplies the sinoatrial node, thus compensating for the occluded main artery. We are led to go even further and state that it is the network and not the artery of the pacemaker the major responsible for the nutrition of the sinoatrial node. Once confirmed the assumption, the term "artery of the sinoatrial node" becomes a misnomer, since though closely related to the node it does not provide directly blood supply to the pacemaker.

Our observation suggests that in this patient the sinusal bradycardia was caused by ischemia affecting the sinoatrial node since the angiogram indicated the origin of this artery from the circumflex artery at the level where it presented an occlusive lesion reducing the lumen by 90%.

Although the perinodal arterial network protects the SA node from ischemia[2], caused by the obstruction of its artery, our patient showed ischemia of the SA node after physical efforts and subsequent bradycardia; in this case the perinodal arterial network is formed likely only by the artery of the SA node or, if there is anastomosis between this artery and another one, the former prevails. Another explanation might be the absence of the network or its underdevelopment to the point that it is unable to protect the node against ischemia. Pfeferman *et al.*[6] emphasized the ischemic etiology of the sick sinus node syndrome. Further investigations are needed to explain the clinical consequences from the variability of the origin of the perinodal arterial network.

RESUMO

Aspectos clínicos e anatômicos da irrigação sanguínea do nó sinoatrial

Estudo recente sobre as variações da origem da artéria do nó sinoatrial demonstra sua importância na rede arterial perinodal da área sinusal em corações normais.

OBJETIVO. Relatar o caso de um paciente com síncope de etiologia isquêmica.

CONCLUSÃO. No paciente apresentado, a rede arterial perinodal não protegeu o nó da isquêmia causada pela obstrução da artéria do nó sinoatrial.

[Rev Ass Med Brasil 1998; 44(1): 47-9.]

UNITERMOS: Nó sinoatrial. Artéria do nó sinoatrial. Rede arterial perinodal. Doença do nó sinoatrial. Síncope.

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