Recent Advances in

CARDIOLOGY

EDITED BY

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Preface

This volume of *Recent Advances in Cardiology* follows the eighth issue after only 3 years. Despite this shortened refractory period the major editorial concern continues to centre on the question of which topics to omit rather than which to include.

The importance of the discipline of anatomy is reflected in the joint chapter from the departments of Professors Anderson and Becker with the co-operation of Siew Yen Ho. The major role of cross-sectional echocardiography in the cardiological assessment of neonates and young children is comprehensively described and generously illustrated by Professor Macartney and Dr Smallhorn. The increasing impact of salvaged congenital heart disease on the spectrum of heart disease in the adult is emphasised by Professors Alpert and Dalen. The ever-expanding role of drug treatment in the management of heart disease is covered by Dr Gray Ellrodt and Dr Singh (calcium antagonists), Drs Steele, Fuster, Chesebro, Badimon and Gorlin (platlet-inhibiting drugs) and by John Hamer (management of heart failure). The fundamental tool in evaluating drug effectiveness (the clinical trial) is itself carefully scrutinised by Professor John Hampton. The desire to reduce the progression (or hopefully even to produce regression) of atheroma in man is both understandable and urgent. Dr Malinow looks at the evidence for possible regression and Drs Palac and Hwang with Professors Loeb and Gunnar review the very important question of the influence of medical and surgical therapy on progression of atheroma in the native coronary vessels. Psychological factors in the development of coronary disease are discussed by Drs Weiss, Krantz and Matthew and psychological problems in relation to cardiac intensive care by Dr Cay. The increasingly important technique of percutaneous transluminal coronary angioplasty is handled by Drs Bourassa, David and Guiteras Val and the modern management of arrhythmias using pacemakers by Drs Rowland and Krikler. Finally the area of cardiac investigation is covered by Drs Testa, Shields and Rowlands (radionuclide diagnosis of venous thrombosis and pulmonary embolism) and by Drs Brunt, Love and Rowlands (objective analysis of left ventricular cine-angiograms). Whilst it cannot be pretended that this list comprehensively covers recent advances in cardiology it is hoped that it will provide some illumination of important and developing areas.

This volume of *Recent Advances in Cardiology* is the first for more than 10 years which has not been produced under the editorship of John Hamer. Dr Hamer has skilfully and wisely guided the publication through three issues. Important and far reaching advances in cardiology have occurred during this period, including the acquisition of extensive experience with the beta-blocking drugs, the mature assessment of the role of coronary care units and coronary ambulances, the development of isotopic techniques and of cardiac ultrasound in diagnosis and experience with coronary artery bypass grafting and cardiac transplantation. Dr Hamer has

contributed to this present issue, the influence upon this publication of his years in office persists and it is hoped that he will contribute to future issues. He is most sincerely thanked for the work he has done and is wished well for the future.

Finally I wish to record my gratitude to my secretary, Judy Carling, for her help in the production of this book.

Manchester, 1984 D.J.R.

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Contents

1. The clinical anatomy of the cardiac conduction system

Robert H. Anderson Siew Yen Ho Anton E. Becker

INTRODUCTION

Like cardiology itself, study of the cardiac conduction system is predominantly a twentieth-century event. Although our knowledge of cardiac anatomy is much older, the specialised muscular connection linking the atrial and ventricular myocardial masses was not discovered until 1893 (His, 1893) while the cardiac pacemaker was not found until the century turned (Keith & Flack, 1907). Throughout its brief history, the system has been surrounded by controversy. At the same time that His described the bundle which now bears his name, Kent (1893) described a series of muscular connections which he claimed linked the atrial and ventricular musculature. This led to a confused understanding of the atrioventricular junction such that Keith, subsequently to discover the sinus node, described in detail in his autobiography how he was unable to confirm the description of His (Keith, 1950). It was the publication of Tawara's monumental monograph which clarified the situation for Keith (Tawara 1906), although misunderstanding of Kent's precise findings have persisted until the present day (Sealy, 1979; Anderson & Becker, 1979). Working in the laboratory of Aschoff in Marburg, Tawara showed how the bundle described by His originated in the specialised atrioventricular node ('knoten') in the base of the atrial septum and terminated in the network of false tendons described half a century before by Purkinje (1845). Tawara's illustrations in tinted drawings are superbly accurate (Fig. 1.1). Despite such explicit demonstrations, together with the equally accurate description of the sinus node by Keith & Flack (1907) and its verification by the anatomico–electrophysiological correlations of Lewis et al (1910), the conduction system continued to be attacked. In a series of papers, the Glomsetts questioned the very existence of the entire system (Glomsett & Glomsett, 1940a, 1940b). It was the advent of intracardiac surgery which resolved this controversy, since surgeons worldwide learnt only too painfully that one injudiciously placed suture could interrupt the atrioventricular bundle which truly is, in most instances, the only muscular connection between the atria and the ventricles. Indeed, it has been the vast improvements in cardiac surgery over the past few decades, coupled with the more recent explosion of knowledge of cardiac electrophysiology, which has emphasised the value of thorough knowledge of the position and structure of the specialised cardiac musculature which constitutes the conduction system of the heart. In this review we will attempt to summarise this information, concentrating particularly on its vascular supply. It is not possible in the space of a single chapter to begin to consider all the highly significant aspects of disease of the conduction system. Instead, we will concentrate on pure morphology. Should any reader find his or her interest kindled by this limited coverage and be desirous of more information, we recommend him or her to the recently published monograph on pathological aspects (Davies et al, 1983).

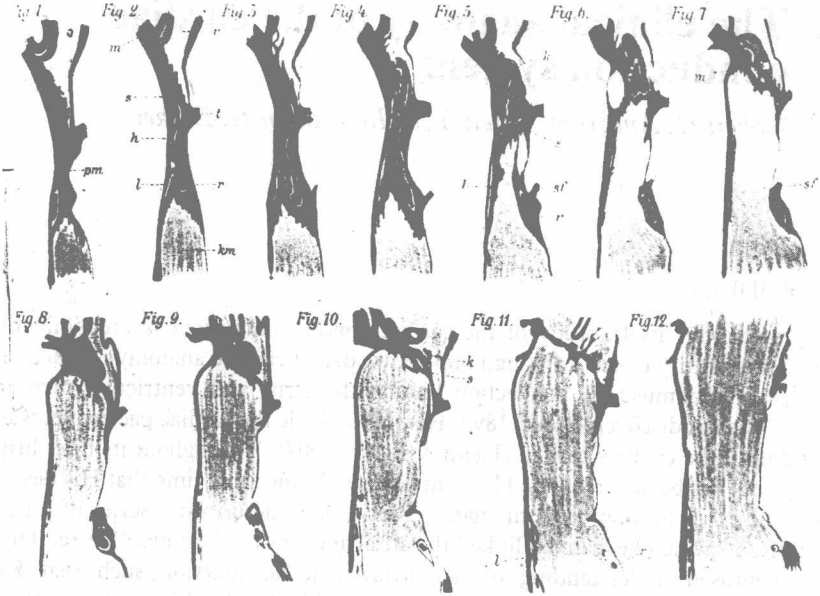

Fig. 1.1 Reproduction of a plate from Tawara's monograph (1906) depicting serial sections through the human atrioventricular junctional area. Although published in tinted colours, even in this black and white copy the accuracy of the drawings can be appreciated.

THE SINUS NODE

The most significant feature of the morphology of the sinus node is its position. Surgical damage to the atrioventricular bundle is certainly an event to be avoided, but damage to the sinus node or its blood supply can have equally disastrous consequences, the results of which are only now becoming fully appreciated (Gillette et al, 1980). The node is a small spindle-shaped structure, usually set around the prominent nodal artery. It lies immediately sub-epicardially within the terminal sulcus lateral to the crest of the right atrial appendage. Since this feature is of most significance to surgeons, Figure 1.2a is orientated so as to display the position of the node as it would be seen at operation, while Figure 1.2b is a microscopic section taken at right angles to the long axis of the node and illustrate its immediately sub-epicardial position. Although the node is most usually in the lateral position shown in Figure 1.2, on occasions it may extend in horseshoe fashion across the crest of the appendage so that its head becomes a medial limb which continues into the interatrial groove (Anderson et al, 1979). Frequently the tail of the node is also extensive, running down the terminal sulcus towards the mouth of the inferior caval vein before burrowing into and merging with the musculature of the terminal crest. As seen in Figure 1.2b, in most cases the node is arranged around a prominent nodal artery, considerably larger than needed for mere nutrition of the node. This feature has prompted James (1967, 1973) to suggest a servo-mechanism function. Certainly in animals such as the rat ultrastructural studies have shown a particular specialisation of the adventitial coat of the artery, which is composed of nodal cells (Taylor, 1980). However, if it exists the servo-mechanism function is not universal because there is considerable variation in

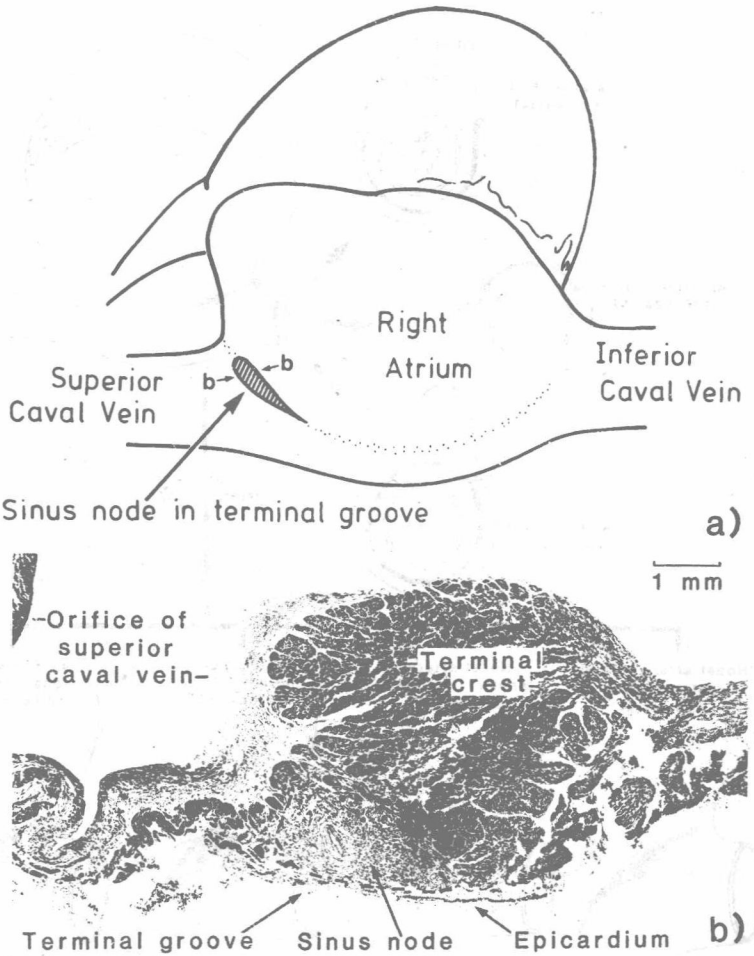

Fig. 1.2 The usual site of the sinus node as might be viewed by the surgeon. Figure 1.2b is a section in the indicated plane and shows the immediately sub-epicardial position of the node.

the human in the pattern the artery takes as it courses through the node. Figure 1.3 illustrates the findings in twenty-five infant hearts we studied by serial section techniques (Anderson et al, 1979). As can be seen, in some hearts there is no prominent artery at all. There is additional highly significant variability in the course taken by the nodal artery. The studies of James (1961a) suggested that it was an early branch of either the right (55%) or left circumflex (45%) arteries. We have recently studied a series of post-mortem angiograms from people dying suddenly and undergoing autopsy at St George's Hospital, Tooting. These show much more variability in the origin of the nodal artery. When arising from the right coronary it often took off several centimetres away from the aortic sinus, while in some cases of circumflex origin, the nodal artery did not originate until the parent artery approached the crux of the heart (Fig. 1.4). James (1968) has pointed to the significance of left main coronary arterial disease in obstructing the nodal artery and

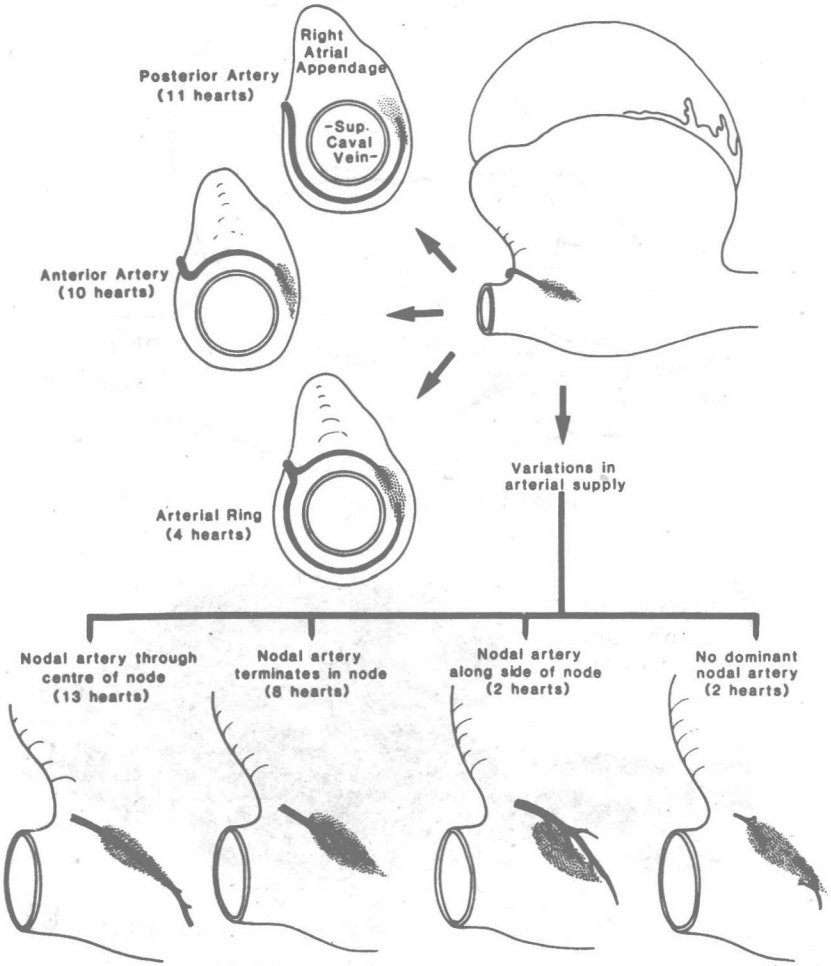

Fig. 1.3 The findings concerning the extra- and intranodal course of the sinus node artery in twenty-five infant hearts studied by serial section techniques.

resulting in atrial arrhythmias following acute myocardial infarction. However, others have denied any positive correlation between main coronary atherosclerosis and impaired sinus node function (Engel et al, 1975) while Davies & Pomerance (1972) commented that the sinus node artery itself is only rarely involved in the atherosclerotic process. There are now several factors emerging which suggest that involvement of the sinus node artery may be a more frequent event. Its origin from a more distal part of the circumflex artery as shown in Figure 1.4 places it at greater risk. In this respect it may be significant that the nodal artery was not visualised at all in a proportion of the cases studied in the St George's Sudden Death Study (Vassal-Adams, Davies & Anderson, unpublished observations). Of greater import is the finding by Jordan and his colleagues (1977), using sinoatrial conduction time as an indicator of sinus node function, that coronary artery disease does deleteriously affect

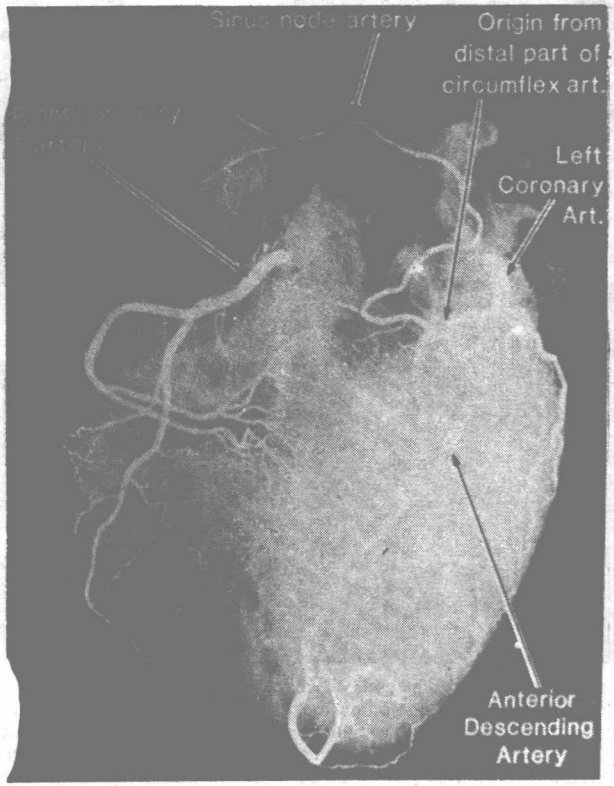

Fig. 1.4 Postmortem coronary arteriogram illustrating the origin of the sinus node artery from the left circumflex artery in the posterior atrioventricular sulcus.

sinus node performance. This correlates well with two pathological observations (Becker, 1978). Firstly, the pathologist who regularly studies postmortem coronary angiograms frequently finds good visualisation of the sinus node artery with a non-sclerotic proximal segment of the parent artery but with a marked but limited narrowing at the origin of the sinus node artery (Fig. 1.5). The good filling seen at postmortem angiography does not mean that obstruction was absent during life. Moreover, such lesions could easily be missed in clinical coronary angiography or else their significance to sinus node performance not be appreciated. The second factor which is often neglected is that the sinus node artery, irrespective of its origin, has many anastomoses with atrial arteries originating from the other coronary artery. These can be significant collateral pathways for an ischemic ventricular myocardium. The possibility therefore exists that these pathways could result in a 'coronary steal' with ischaemic effects on pacemaker functions (Fig. 1.6). But it must then be noted that postmortem angiocardiographic studies of patients known to have 'sick sinus syndrome' has revealed no obvious atherosclerotic lesions (Evans & Shaw, 1977), although whether the possibility of a steal was considered is not discussed. The relationship of subtle coronary artery lesions and atrial arrhythmias is clearly a fertile area for further study.

Irrespective of the origin of the nodal artery, there is further significant variability

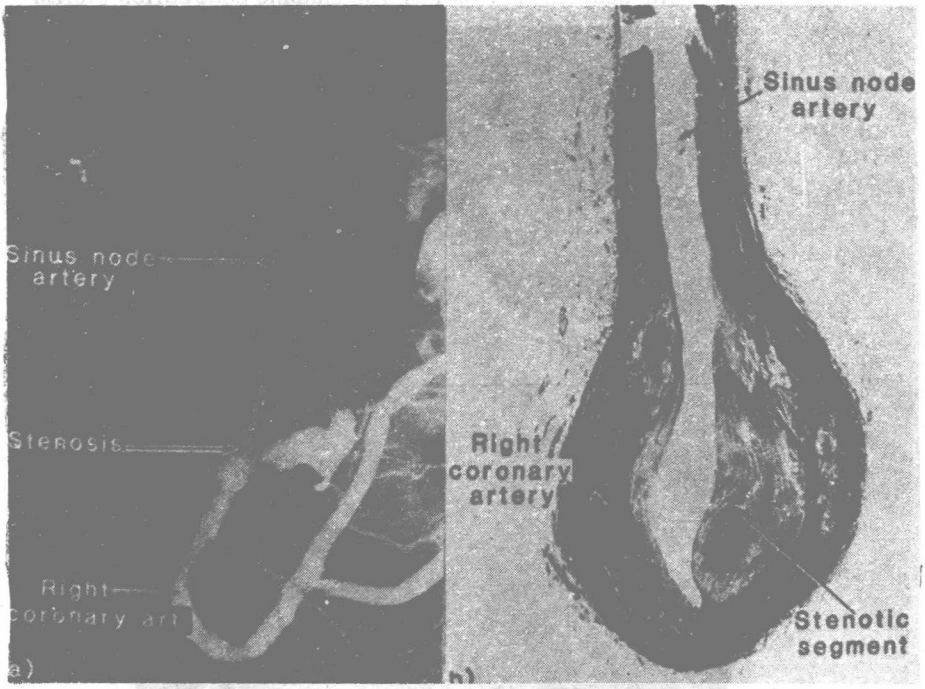

Fig. 1.5 Illustrates how coronary atherosclerosis may affect the sinus node artery. (a) shows a detail of a postmortem coronary angiogram after injection into the right coronary artery. The sinus node artery originates from the proximal segment of the right coronary artery. A severe narrowing of the lumen is present at the site of origin but nevertheless a good contrast filling of the sinus node artery has been obtained. However, this does not exclude impaired flow in vivo. Such localised stenoses can be misinterpreted on clinical angiograms, as illustrated in a histological section, (b) of this segment. The right coronary artery shows eccentric stenosis at the site where the sinus node artery originates. Because in certain views the lumen would appear not to be narrowed, this type of luminal narrowing can cause difficulties in being recognised angiographically. Elastic tissue stain; (×10.5).

in its course as it approaches the cavoatrial junction. In some hearts the artery curves over the appendage crest and enters the cephalic end of the node. In others it passes retrocavally to penetrate the tail while in still further examples there is an arterial circle around the cavoatrial junction with the node receiving an arterial supply from both ends. All of this is highly pertinent to post-operative arrhythmias in congenital heart disease. It has been known for some time that these are particularly frequent following atrial repair of complete transposition. Indeed, their frequency has been forwarded as a reason for avoiding atrial repair. Of late it has become increasingly evident that these arrhythmias result from damage either to the sinus node or its blood supply (Gillette et al, 1980). Scrupulous avoidance of the entire cavoatrial junction (coupled with the observation that some so-called 'post-operative' electrocardiographic events were present prior to operation) now permit Mustard's operation to be performed with a gratifyingly low incidence of post-operative rhythm problems (Turley & Ebert, 1978; Ullal et al, 1979; Southall et al, 1980).

Microscopic examination of the sinus node shows it to be composed of a network of small nodal cells aggregated into interweaving fasciculi, themselves set in a dense fibrous tissue matrix (Fig. 1.7a). The fibrous tissue is already present in the fetal node