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Chronic sinoatrial disorder (sick sinus syndrome): a possible result of cardiac ischaemia

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SUMMARY Postmortem angiography was used to examine the blood vessels supplying the sinoatrial node in 25 subjects with chronic sinoatrial disorder (group 1). The results were compared with similar studies in 54 subjects who died of heart block and in whom sinus node function was normal (group 2). Although no significant lesion obstructing the blood flow to the sinus node was seen in the majority of those in group 1, there were abnormalities in seven cases, with reduced filling of the sinus node artery in five. In group 2 the sinus node artery filled normally in all cases despite major disease of the parent vessel in three. The combination of contralateral coronary artery disease with extensive atrial anastomoses was actively sought because this arrangement might predispose to a steal phenomenon. Such conditions were fully met in three cases in group 1 and two cases in group 2, and were found to a lesser extent in a further two cases in group 1 and three in group 2.

Although coronary artery disease was unlikely to be the principal cause of sinus node dysfunction in most of the cases studied it was relatively common and may have been a factor in about one third. Improved survival after myocardial infarction may increase the number of patients with chronic sinoatrial disorder of ischaemic origin.

Chronic sinoatrial disorder (sick sinus syndrome) is now one of the commonest conditions treated by pacemaker implantation, yet its aetiology remains in doubt. Acute dysfunction of the sinus node is commonly seen in the early stages of myocardial infarction but it usually disappears within the first 24–48 hours[1] and initially coronary artery disease was assumed to be responsible for most cases of chronic dysfunction.[2–4] More recently this has been disputed and the long term follow up of patients showing sinus node dysfunction in the acute phase of myocardial infarction has produced conflicting results.[5 6] Currently necropsy studies have shown few major lesions of the blood vessels supplying the sinus node,[7–9] but the numbers of cases studied are small and even fewer have been investigated by postmortem angiography. Reviewing the subject Becker concluded that the aetiology remained in doubt and that a full angiographic study of the blood supply to the sinoatrial node was required to assess the role of coronary artery disease.[10] As an additional factor he suggested that there might be a steal phenomenon through atrial collateral vessels, a possibility which had not been previously assessed. Another factor is the improved survival of patients with extensive cardiac infarction in which there may be major obstruction to the sinoatrial artery or its parent vessel.

We have investigated the blood supply to the sinus node by postmortem angiography in patients with chronic sinoatrial disorder. Patients dying of chronic heart block were used as controls because both they and the patients with sick sinus syndrome had been followed up in an identical manner in our department.

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Table 1 *Basic data in the two groups (further details for group 1 are given in the appendix)*

Group	No	Age (yr)		Follow up in months		Sex (M/F)	Paced
		Mean	Range	Mean	Range		
1	25	75	56–92	55	1–186	20/5	19
2	54	78	49–96	37	1–140	39/15	36

Patients and methods

PATIENTS

Most of the material was obtained from patients who died while in the Devon Heart Block and Bradycardia Survey.[11] All subjects were known to have suffered from either sinoatrial disorder or heart block for 28 days or more. For the purposes of analysis they were divided into the following groups:

Group 1 consisted of 25 subjects with chronic sinoatrial disorder. All were known to have had features of sinus node dysfunction for more than a month before death and 23 had been followed up in the Devon Survey. The angiographic findings in four cases were briefly described in 1977.[8]

Group 2 consisted of 54 subjects with normal sinus node function but chronic second or third degree atrioventricular block and who had been followed up in the Devon Survey for one month or more.

Table 1 gives the age at the time of death, the follow up time, and the number of subjects paced in the two groups. The appendix gives further details of the patients in group 1 and table 2 gives electrocardiographic data. Sinus bradycardia was seen in 21 patients and bradycardia with junctional rhythm was present in another three patients. One case (No 207) did not have clinically significant bradycardia but did have episodes of sinoatrial block with sinus pauses lasting up to eight seconds. A further 18 patients had sinus pauses of $\geqslant 1{\cdot}8$ s on the standard

ble 2 *Electrocardiographic findings in patients with sinoatrial disorder (group 1)*

e No	Sinus bradycardia	Sinus* pause	Junctional rhythm	Tachycardia	ECG lowest rate	AV conduction	ST/T wave abnormalities on entry
	+	+	+	Atrial fibrillation	34	RBBB, 1° block, intermittent CHB	
	+	+			45	CHB	
	+	+	+		31	Normal	ST depression I aVL, V5–6, T inversion I, aVL
	+				50	RBBB, intermittent CHB	
	+	+	+		33	Intermittent 1° block	T inversion I, III, aVL, V3–6
	+			Atrial tachycardia and fibrillation	24	LAHB, 1° block	
	+	+		Atrial tachycardia	30	1° block	
	+		+		37	Normal	ST depression V4–6
	+	+			38	Normal	
	+	+		Atrial flutter	25	Normal	
	+	+		Atrial flutter	50	Normal	
	+	+	+	Atrial fibrillation	28	Normal	
	+			Atrial tachycardia	39	Normal	ST depression I, V4–6 T inversion I, II, aVL, V3–6
	+			Atrial flutter	49	Normal	
	+	+		Atrial fibrillation	44	Normal	ST depression V4–6, T inversion II, III, aVF,
-6							
	+	+	+	Atrial flutter	36	Normal	
	+	+			39	RBBB, 1° block	
	+	+			46	Normal	
	+	+	+	Atrial tachycardia; ventricular tachycardia	42	Normal	
	+	+	+		34	LAHB	
		+	+		63	Normal	ST depression V4–6
			+		41	Normal	
		+	+	Atrial tachycardia	34	Normal	
		+	+	Atrial fibrillation	37	Intermittent 1° block	
	+	+			46	RBBB, intermittent 1° block	

1·8 s on standard electrocardiogram (ECG) or $\geqslant 2$ s or more on ambulant ECG.
BB, right bundle branch block; CHB, complete heart block; LAHB, left anterior hemiblock.

electrocardiogram, or ⩾2 s or more on ambulant electrocardiography. In three patients the relation of the duration of the pauses to the preceding PP intervals was compatible with sinoatrial block. Periods of junctional rhythm occurred in 12 patients and paroxysmal tachycardia was seen in 14. There were major conduction disturbances in three patients and lesser abnormalities such as bundle branch block or first degree block in seven. No conduction abnormalities were seen in 15. By definition second or third degree block was present in all the subjects in group 2 and none had evidence of sinus node dysfunction.

During the period of the study postmortem angiography was carried out on four additional patients who had been seen during the acute stages of myocardial infarction with evidence of sinus node dysfunction. All had periods of sinus arrest and gross bradycardia which persisted up to the time of their death at 4, 15, 16, and 17 days after the acute event.

METHODS

Most of the patients died at home and permission for necropsy was only obtained by the speedy cooperation of the patient's family doctor. Usually the family agreed to only a limited procedure; this was carried out as soon as possible after death. When the heart was removed particular care was taken to sever the superior vena cava 2 or 3 cm above its junction with the right atrium so that the sinoatrial node and its blood supply were not damaged. After initial inspection of the heart the coronary ostia were examined and then cannulated. Both coronary arteries were flushed with saline at low pressure to remove postmortem sludge and then the right coronary was perfused with a radio-opaque medium for 5 min at a pressure of 130 mm Hg. Radiographs of the heart were taken in three planes on a fine grain film (Mamoray RP3) with an Elema-Schonander skull unit with 40 kV at 400 mA, a focal spot of 0·6 mm, and a focal film distance of 32 inches. Films were developed and if they were satisfactory, then the left coronary artery was perfused and a further set of *x* rays was taken. The standard views were anterior-posterior, left lateral, and left anterior oblique; but in a proportion of patients additional views were taken. The injectate usually consisted of a barium suspension mixed with Colourpaque to which a little dissolved gelatine had been added. This mixture penetrated into vessels with diameters of between 25 and 30 μm.

After postmortem angiography of the heart in toto, we removed the block of tissue containing the junction between the superior vena cava and right atrium, pinned it out on a board, and *x* rayed it to demonstrate the sinoatrial node artery and its branches. The heart was fixed for two or three weeks and blocks taken from the heart and main coronary arteries for histological examination.[8] We used a modification of the method described by Davies.[12] The angiograms were studied with an overhead projector which gave a ninefold magnification. We concentrated on the right coronary up to the origin of the posterior descending branch (where present) and the left coronary, including the initial parts of its two main branches proximal to the first diagonal and second obtuse marginal. The widths of the arteries were measured and in narrowed areas the reduction in lumen was expressed as a percentage of the width of the preceding sections; obstructions of less than 20% were classified as normal. We looked for vessels running between branches of the sinoatrial node artery or its parent vessel and other atrial arteries, particularly those arising from the contralateral coronary artery. To be accepted as anastomotic vessels they had to be seen on all three *x* ray projections (fig 1). One or two connecting vessels were designated as "some" and three or more as "many". If filling of atrial arteries stemming from the right coronary artery was poor after this vessel had been perfused, the appearances were reviewed after perfusion of the left coronary artery for evidence of an increased flow of contrast material via anastomoses. The potential for a steal phenomenon was identified only if there was an anastomosis between the sinus node artery proximal to the node and a branch of an obstructed (50% or more) contralateral artery.

Filling of the sinoatrial node artery was assessed by examination of the initial angiograms of the whole heart and subsequent films of the tissue block containing the sinoatrial node. Filling was classified as "normal" where the vessel and its tributaries could be seen easily with sharp outlines on a standard *x* ray viewing box and "reduced" if the vessel was blurred with indistinct or barely visible tributaries. Filling was termed "poor" if the sinus node artery could only be discerned with difficulty using a bright light and "nil" if the artery was invisible on *x* rays of the whole heart or tissue block containing the sinus node.

The right coronary artery was identified as being dominant if it gave rise to the posterior descending artery.[13]

Results

The left coronary artery was dominant in 10 of the 25 cases in group 1 and 15 of the 54 cases in group 2. The origin of the sinus node artery was easily identified in all patients except case 187 in group 1, in whom the left coronary artery was normal but did

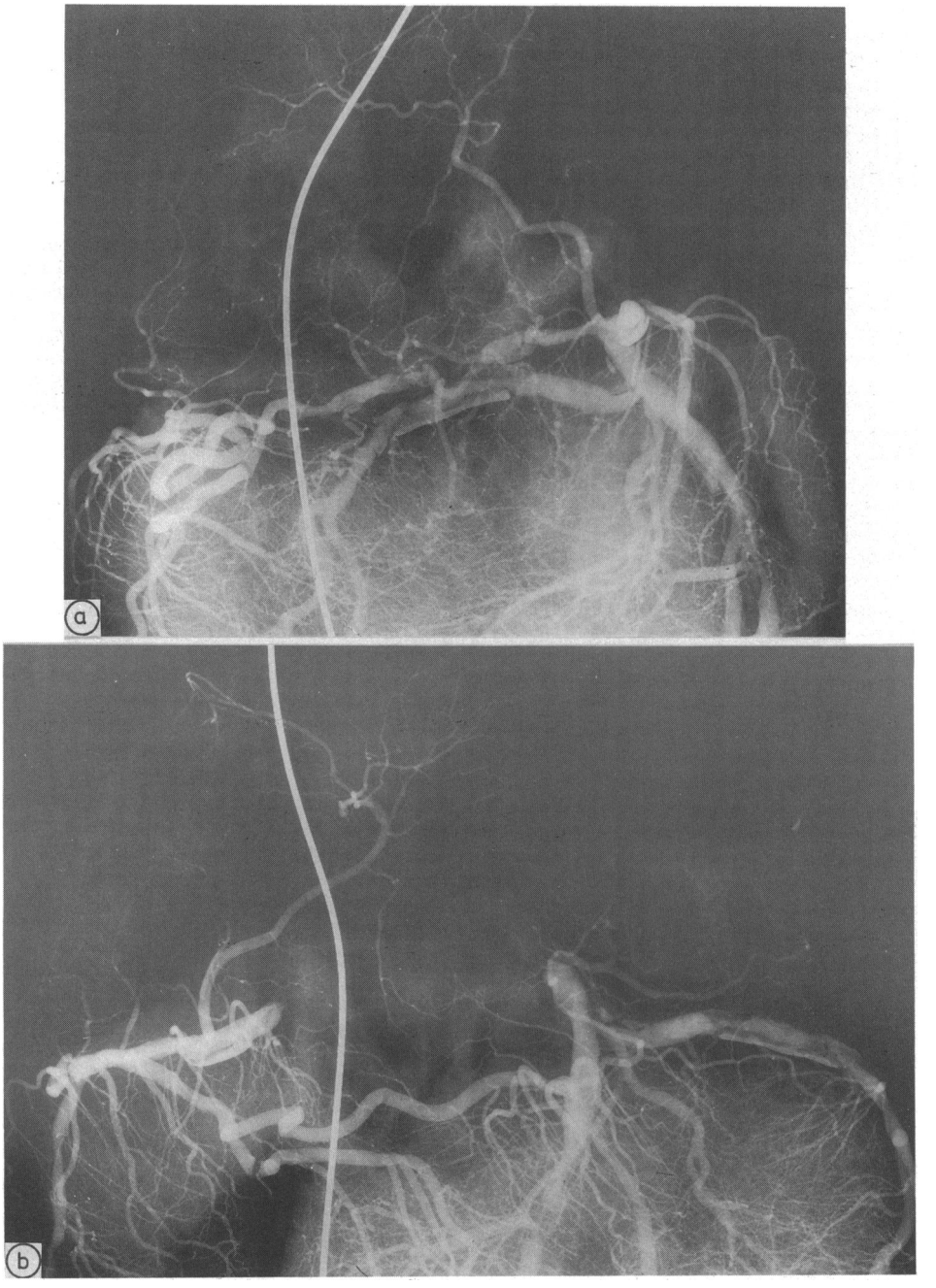

Fig 1 *Angiograms of case 118 with both coronary arteries perfused and showing a large atrial artery arising from the right coronary artery which supplies the sinus node area. The oblique view (a) appears to show a series of anastomoses but the arterior-posterior view (b) shows separation of the vessels particularly in the upper atrium.*

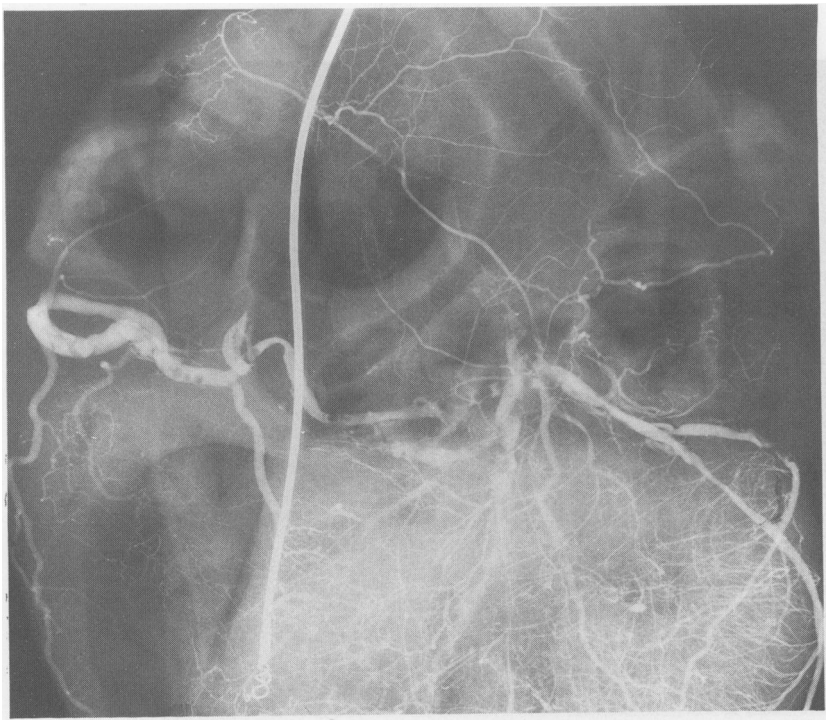

Fig 2 *Anterior-posterior angiogram of case 243 after perfusion of both coronary arteries, showing gross general arterial disease but normal filling of the sinoatrial node artery which arises close to the origin of the left circumflex coronary artery.*

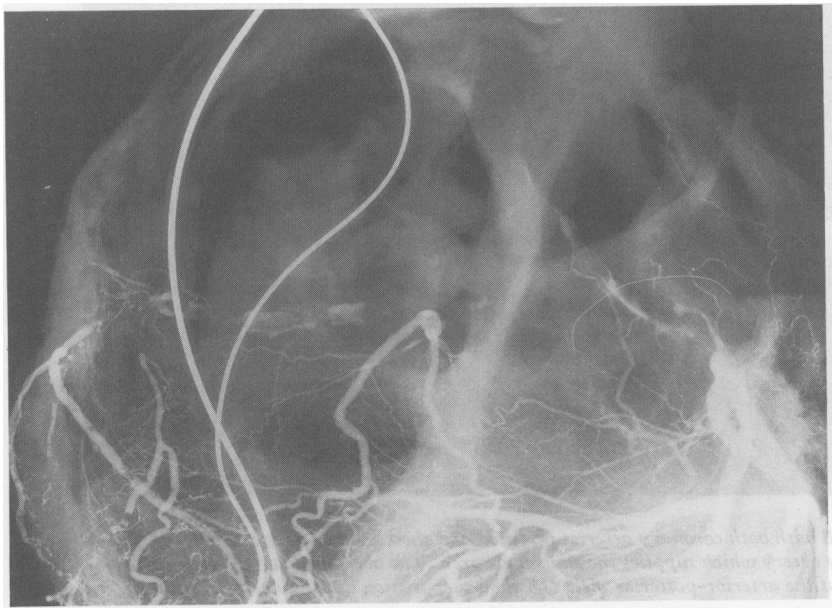

Fig 3 *Anterior-posterior angiogram of case 187 after perfusion of both coronary arteries. The right main coronary immediately distal to the conus artery was completely blocked. There was no filling of the atrial arterial branches on the right side.*

ıle 3 *Arteriographic findings in all the patients in group 1 and in the 18 cases from group 2 showing arterial obstructions of ⩾20% in or more of the major vessels*

No	Dominant coronary artery	SANA filling	Coronary supplying sinoatrial node			Contralateral coronary artery	Atrial anastomoses
			Side	Proximal*	Distal*		
				Group 1			
	L	Normal	L	50%	50%	90%	Some
	L	Normal	L	Normal	Normal	Normal	Some
	R	Normal	R	Normal	Normal	LAD blocked	Many
	L	Nil	R	Blocked	Blocked	Normal	None
	R	Normal	R	Normal	Normal	LAD blocked, circ 30%	Some
	R	Normal	R	Normal	Normal	Normal	None
	R	Normal	L	Normal	Blocked	20% proximal	Many
	L	Normal‡	R	>90%	20%	40% main	Many†
	R	Normal	R	Normal	Normal	Normal	None
	R	Normal	R	Normal	Normal	Normal	Some
	R	Normal	L	Normal	Normal	Normal	Some
	R	Normal	R	Normal	Normal	Normal	None
	L	Reduced	L	75%	Circ 60%, LAD blocked	40%	None
	R	Normal	R	Normal	35%	Normal	Some
	L	Normal	R	Normal	50%	80% main, 40% circ, 80% LAD	Some
	R	Poor	R	>90%	60%	Circ blocked	None
	L	Normal	L	Normal	Normal	Normal	Some
	R	Normal	R	Normal	Normal	Normal	Some
	R	Normal	R	Normal	35%	50% main, circ blocked	Many
	L	Nil	L	35%	Normal	Normal	None
	R	Poor	L	90%	80%	Normal	Some†
	L	Normal	R	Normal	35%	50% circ	None
	L	Normal	R	Normal	Normal	Normal	Some
	R	Normal	R	Normal	Normal	Normal	Some
	R	Normal	R	Normal	Normal	Normal	Many
				Group 2			
	L	Normal‡	R	90%	Blocked	50% LAD	Many†
	R	Normal	L	80%	70%	50%	Some
	R	Normal	R	Normal	40%	60% LAD, 40% circ	Some
	R	Normal	R	Normal	Normal	LAD blocked, 80% circ	Some
	R	Normal	R	Normal	Normal	40% circ	Some
	L	Normal	R	Normal	50%	50% LAD	Some
	R	Normal	R	Normal	65%	50% LAD	None
	L	Normal‡	R	Blocked	Normal	30% LAD	Many†
	L	Normal	R	Normal	20%	40% LAD	Some
	R	Normal	R	Normal	Normal	20% LAD	Some
	L	Normal	R	50%	Normal	30% LAD	Some
	R	Normal	L	Normal	Normal	50%	None
	R	Normal	R	Normal	50%	Normal	None
	L	Normal	R	Normal	Normal	50% LAD	Some
	R	Normal	R	25%	Normal	50% main, 25% circ	Some
	R	Normal	L	Normal	20%	Normal	None
	L	Normal	L	40%	Normal	Normal	None
	R	Normal	L	30%	90% LAD	20%	Some

o sinoatrial node artery; †apparent filling towards SANA; ‡normal filling after perfusion of left coronary artery.
.D, left anterior descending artery; circ, circumflex artery; SANA, sinoatrial node artery.

not give rise to any large atrial branches while the right coronary was completely blocked close to its origin. In this case the right coronary was believed to be the parent vessel supplying the sinoatrial node artery. So the blood supply to the sinus node came from the right coronary artery in 17 (distal in one) and the left coronary artery in eight cases (distal in three) in group 1 and from the right coronary artery in 35 (distal in three) and the left coronary artery in 19 cases (distal in one) in group 2.

SINUS NODE ARTERY AND ITS PARENT VESSEL

In most subjects from both groups the sinus node artery filled normally, often despite evidence of extensive coronary artery disease (fig 2). In group 1, however, narrowing of ⩾50% was present in seven cases and sinus node artery filling was abnormal in six (table 3). Figure 3 shows examples in which there was no filling of the sinus node area and fig 4 shows the sinus node artery filled through anastomotic connections from the contralateral coronary vessel.

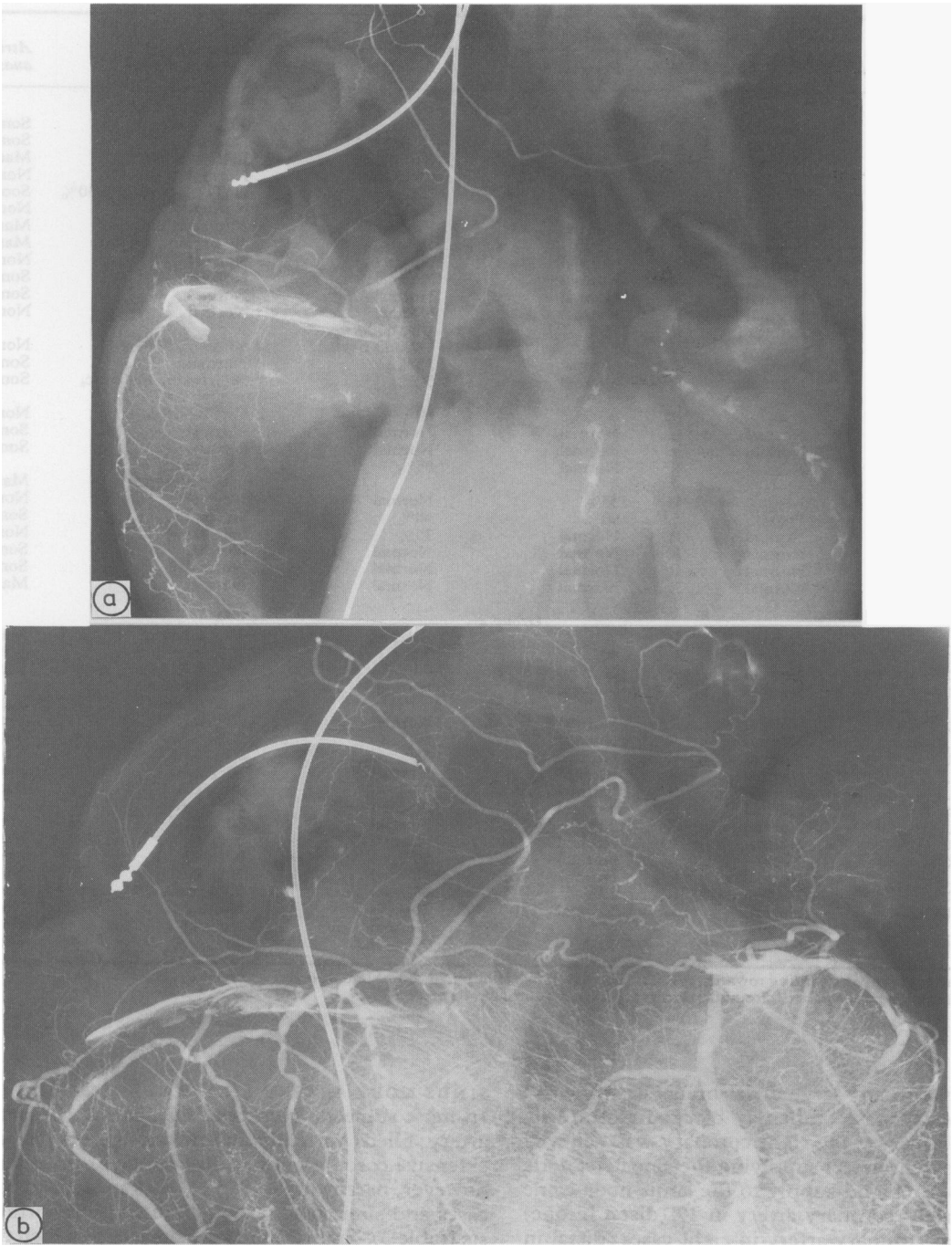

Fig 4 *Anterior-posterior angiograms of case 169: (a) perfusion of the right coronary shows that this was almost completely obstructed close to its origin. The sinus node artery filled but to a reduced extent and smaller tributaries cannot be seen; (b) taken after both coronaries had been perfused, showing increased filling of the sinus node artery and adjacent vessels.*

Table 4 *Origin of the sinoatrial node artery in groups 1 and 2 compared with that described by James[18] and Hutchinson[19]*

	Group 1		*Group 2*		*James*		*Hutchinson*	
Artery	*No*	%	*No*	%	*No*	%	*No*	%
Right proximal	16	64	32	59	55	52	19	48
Right distal*	1	4	3	6	2	2	7	18
Left proximal	5	20	18	33	43	41	11	28
Left distal*	3	12	1	2	2	2	3	8
Total	25		54		106†		40	

*The second (intermediate) and third (posterior) parts of the artery.
†Including two with dual supply and two with unknown origin.

Case 185 was unusual because the atrial artery supplying the branch to the sinus node was blocked at a point approximately two thirds along its course.

The sinus node artery filled normally in all of the 54 subjects in group 2. There was a major obstruction of the parent vessel in three, however, and in cases 162 and 146 the right main coronary artery was completely blocked and the sinus node artery filled via extensive anastomotic networks coming from the left coronary. In case 86 the sinus node artery was a branch of the left coronary, which was 80% narrowed; initial injection of the right coronary artery produced some filling by anastomotic channels and filling through the left coronary was normal.

CONTRALATERAL CORONARY ARTERY

In seven cases in group 1 the contralateral artery or one of its major branches was obstructed by $\geqslant 50\%$. Two of these (216 and 144) had no apparent anastomotic vessels, but in the other five the findings were compatible with a steal phenomenon because there were some or many anastomotic vessels associated with major obstruction of the contralateral coronary artery.

In group 2 there was $\geqslant 50\%$ obstruction of the contralateral artery or its main branches in nine, including two of those with major disease of the other coronary artery. None of the remaining seven cases had many anastomotic vessels: five had some and two had none.

In the four patients dying soon after acute myocardial infarction postmortem angiography showed that the parent vessel to the sinus node artery was completely blocked in two and that there was severe bilateral coronary disease in the other two cases, one of which also fulfilled the criteria for a potential steal phenomenon.

Discussion

The anatomy of the blood supply to the atria and sinus node is very variable[14-16] and if sinus node dysfunction were usually the result of ischaemia it might be expected that certain anatomical patterns would be unduly common in this condition because they predisposed the area to ischaemia.[17] Conversely other vascular configurations might reduce the risk of hypoxia because they give considerable scope for anastomosis between the different arterial branches to the atrium.[18 19] Nerantzis and Avgoustakis suggest that the so-called S shaped sinus node artery arising from the distal part of the left circumflex may be particularly beneficial in cases of atherosclerosis, since it can function as a bridge between the two major coronary trunks.[20] This makes the sinus node area in a heart "much less vulnerable to atherosclerotic disease than would be the case otherwise". In practice, the basic coronary anatomy of the subjects of the present study was generally similar in groups 1 and 2 and findings in both groups resembled those in other series of subjects without sinus node disease (table 4).

It is unwise to make dogmatic deductions from necropsy angiograms about the likely state of the circulation in life, because the static filling pressure applied to dead vessels must be very different from the pulsatile flow occurring in the live circulation. The steady pressure of 130 mm Hg that was used would tend to fill peripheral vessels even if a major obstruction was present, yet under such circumstances in life tissue ischaemia might be expected, particularly when under increased load. Also the degree of coronary spasm cannot be assessed and there is evidence that spasm is often present in atheromatous heart disease. The influence of extracardiac anastomotic vessels[21] could not be assessed in this study and even intercoronary anastomoses may have been difficult to identify. Radiographs taken in a single plane may be misleading since vessels appear to criss-cross each other and the scope for anastomosis may seem very great. Further views, however, will disclose the true arrangement and with three projections it is usually possible to confirm the presence of collateral channels. In some instances we studied up to six views after each injection but the additional plates did not materially add to the information. When collaterals are found it may be difficult to assess their importance because

they vary considerably in size, length, and morphology.

In most patients with chronic sinoatrial disorder in the present study the blood supply to the sinus node was not obstructed by disease of the large arteries. Significant arterial lesions were relatively common, however; and these caused reduced filling of the sinoatrial node artery in five of 25 cases and the potential for a steal phenomenon in a further five cases. There is some uncertainty about the effects of the lesions in two of these cases (215 and 150) where the principal contralateral arterial lesions were in the anterior descending branch of the left coronary artery, a vessel which contributes less to the atrial anastomotic system than do the circumflex and right coronary arteries. All of the cases in group 2 showed satisfactory filling of the sinus node artery, although in three collateral vessels from the contralateral coronary artery made a major contribution. The angiographic appearance suggested a potential for a steal phenomenon in five cases, but in three the predominant obstruction was in the left anterior descending artery. There had been no evidence of sinus node dysfunction during life in any of these cases. The findings in the four cases of acute sinus node dysfunction after cardiac infarction suggest that, had they survived, at least three would have been left with ischaemia of the sinus node. Improved methods of treatment may increase the number of patients with impaired sinoatrial function who survive the acute episode.

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Appendix *Basic data in group 1*

Case No	*Age at death*	*Sex*	*Follow up (mth)*	*Symptoms and signs*	*Paced/unpaced*	*Cause of death*
243	92	M	186	Syncope, dyspnoea	Paced	Cerebrovascular accident
214	80	F	155	Syncope, dyspnoea	Paced	Cerebrovascular accident
215	69	F	144	Syncope, dyspnoea	Paced	Myocardial infarction
187	79	M	121	Dizziness, dyspnoea, chest pain	Paced	Carcinoma/bronchus
150	85	M	97	Syncope, dyspnoea	Paced	Cerebrovascular accident
36	64	F	89	Syncope, dyspnoea, chest pain	Paced	Left ventricular failure
186	82	M	79	Syncope, dyspnoea	Paced	Bronchopneumonia
169	63	M	78	Cardiac failure, chest pain	Paced	Myocardial infarction
118	56	M	71	Dizziness, dyspnoea	Paced	Unknown
82	60	F	57	Syncope, dyspnoea	Paced	Cerebrovascular accident
226	77	M	49	Syncope	Paced	Carcinomatosis
4	65	F	58	Syncope, dyspnoea, cardiac failure	Paced	Pacemaker failure
35	69	M	58	Dyspnoea, chest pain	Unpaced	Myocardial infarction
101	87	M	30	Syncope	Unpaced	Bronchopneumonia
210	70	M	29	Syncope, dyspnoea	Paced	Cardiac failure
216	70	M	14	Dizziness	Paced	Carcinoma/oesophagus
39	88	M	12	Dizziness	Unpaced	Bronchopneumonia
209	77	M	9	Syncope	Paced	Cerebrovascular accident
88	76	M	8	Syncope, cardiac failure	Unpaced	Cardiac failure
185	85	M	7	Syncope	Unpaced	Unknown
207	78	M	5	Syncope, cardiac failure	Paced	Renal failure
144	82	M	4	Dizziness, dyspnoea, chest pain	Unpaced	Unknown but sudden
184	65	M	2	Syncope, dyspnoea	Paced	Cardiac failure
135	76	M	1	Syncope, cardiac failure	Paced	Unknown
75	83	M	1	Syncope	Paced	Cardiac failure