BENIGN POLYPOID TUMOURS OF THE LEFT AURICLE

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IN May 1952 a female patient aged 47 died in hospital and at autopsy she was found to have a polypoid tumour in the left auricle arising from the inter-auricular septum. The correct diagnosis was not made before death.

CASE REPORT

Mrs A. B., aet. 47 years, housewife. First symptoms occurred in May 1951; there was no previous history of rheumatic fever and the patient's exercise tolerance had always been good. She had three healthy children, all the pregnancies being normal.

She first complained of the sudden onset of pain, redness and swelling of the right great toe; this settled with rest, but some weeks later, immediately following exertion, she developed transient diplopia and pain in both shoulders which radiated down the arms to the finger tips and was followed by pain and swelling of both hands. The pain and swelling lasted only for a few days, but subsequently similar episodes of pain and swelling of the hands and also of the legs and feet occurred, particularly after exertion. Following these attacks she noticed a painful blotchy erythema of the skin of the affected limbs.

Quite independently of the attacks described above, small red, tender cutaneous nodules appeared in the finger tips and under the finger nails, lasting for five or six days.

In July 1951 she noticed for the first time that she had become more easily tired and breathless on exertion, and while climbing a hill she was suddenly struck with an almost complete paraplegia, associated with severe pain in the knees, calves and feet and the appearance of a red mottled rash on both legs. After four days she was once again able to walk, but walking caused a constricting pain in both legs, and ankle swelling. With six weeks' rest, she completely recovered apart from a little residual dyspnœa on exertion. This, however, was not sufficient to prevent her resuming strenuous activities.

The transient pains in the limbs and skin lesions persisted and in October 1951 she was admitted to hospital for investigations. While in hospital she remained afebrile, had no symptoms whatsoever, and clinical examination, X-ray of the chest, plasma protein, capillary fragility, blood uric acid and full blood count were all normal. W.R. was negative and the agglutination reactions for typhoid, paratyphoid group and brucellosis were negative. The only persistent abnormality was a raised B.S.R., varying from 33 to 40 mm./hr. (Westergren). Vigorous exercise failed to produce limb pain or skin lesions, and she was discharged home.

During the next three months, she was observed as an out-patient and her general condition gradually deteriorated, she became more breathless on exertion and she lost weight. The small red, tender skin lesions in both hands occurred frequently and she noticed a constricting pain in the right arm and

some swelling of the fingers. These complaints were now not always related to exertion.

The B.S.R. was persistently raised and clinical examination of the heart and lungs remained normal.

The patient was readmitted to hospital as an emergency on 19.2.52. During the previous week she had been well, apart from one attack of palpitations and one attack of severe paroxysmal dyspnœa. This occurred when the patient was preparing for bed, was partly relieved by morphine and wore off completely during the night. There was no chest pain.

A similar attack occurred on the night of admission but the dyspnœa became progressively worse until after about one hour she was fighting desperately for breath and ultimately lost consciousness altogether.

On examination she was found to be comatose, cyanosed and orthopnœic. T. 99·4, P. 140 per min. and regular, R. 30 per min. Signs of acute pulmonary œdema were present. Occasionally a little thick, tenacious, dark brown sputum was coughed up with great difficulty. At no time was the sputum either pink or frothy. There was a trace of ankle œdema and moderate engorgement of the neck veins. The heart was not clinically enlarged. The mitral first sound was very markedly accentuated and there were no murmurs. B.P. 120/80.

Apart from the state of coma, no abnormalities were detected in the central nervous system. There was no palpable enlargement of lymph glands, liver or spleen. Optic fundi were normal.

Further investigations revealed :—Hb. 96 per cent., W.B.C. 8,600, B.S.R. 56 mm./hr. The urine contained no abnormal constituents. Sputum culture produced only a scanty mixed growth, mainly streptococcus viridans. Portable X-ray of chest showed extensive patchy opacities of both lung fields and electrocardiogram a normal sinus tachycardia.

Treatment with oxygen, penicillin, morphine, aminophylline, neptal and atropine led to gradual improvement. The signs of cardiac failure and pulmonary œdema disappeared and the tachycardia was gradually controlled. Six days after admission the pulse and respiratory rate were normal and the signs in the lungs consisted of basal crepitations and a few rhonchi only.

It was now discovered that ausculatory signs of mitral stenosis had developed. At the apex, the first sound was accentuated and slapping preceded by a short presystolic murmur and thrill. A mid-diastolic murmur was also heard at the apex, but no systolic murmur. Over the third and fourth left intercostal spaces in the parasternal line a faint high pitched systolic murmur and a third heart sound were heard. The pulmonary second sound was split, but the characteristic " opening snap " of mitral stenosis was not present. The sounds at the aortic area were normal.

Screening of heart and lungs revealed slight generalised cardiomegaly, a normal aorta and a normal pulmonary artery. The left auricle was slightly enlarged in the right oblique view, and showed systolic expansion.

Six blood cultures were negative. Cardiac catheterisation revealed the following mean pressures, expressed in mm. of mercury above the sternal angle :—

Right auricle	= − 4
Right ventricle	= +12
Main left pulmonary artery	= +32
Left pulmonary artery (capillary pressure) .	= +28

The patient recovered completely and was discharged from hospital on 25.3.52, symptom free with a diagnosis of rheumatic mitral stenosis.

Following discharge, the patient remained well and complained only of a slight degree of exertional dyspnœa. She had one moderately severe attack of paroxysmal dyspnœa, for which there was no obvious precipitating factor.

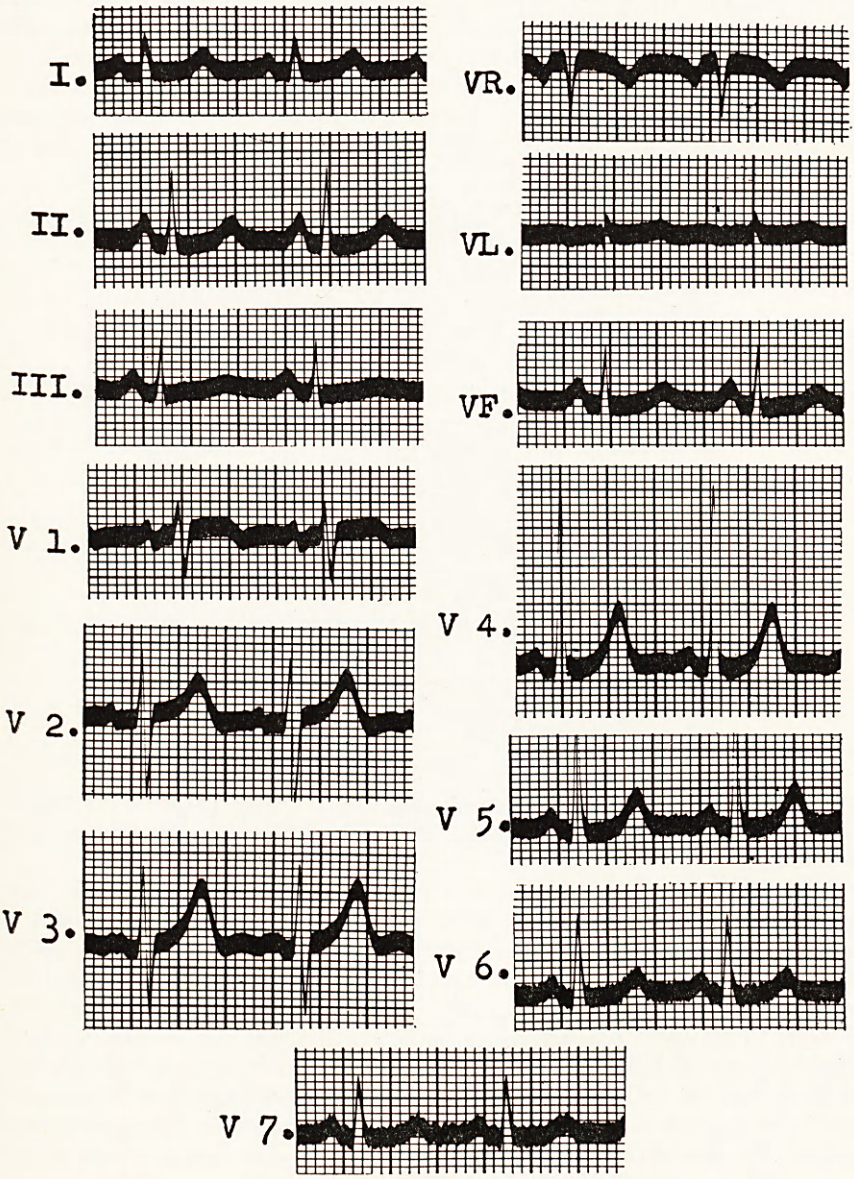

FIG. 1.—Electrocardiogram. May 1952.

She also had several recurrences of the erythematous patchy skin lesions on the fingers and hands and some vague joint pain. Accordingly she was readmitted to hospital on 1.5.52 with the presumptive diagnosis of active rheumatism or subacute bacterial endocarditis.

On examination the physical signs were unchanged. There was no evidence of congestive cardiac failure although occasional basal crepitations and scattered rhonchi were heard in the lungs. As before X-ray of the chest showed slight prominence of the left auricle with moderate hilar congestion. E.C.G. (13 leads) was normal. (Fig. 1). B.S.R. was 55 mm./hr. Hb. 85 per cent. R.B.C. 4·13 m. W.B.C. 9,200, normal differential. Blood cultures were again negative and urinary examination revealed no abnormality. Prothrombin concentration and serum electrolytes were normal.

A therapeutic trial of sodium salicylate was commenced. On the seventh evening after admission she was distressed and became restless and emotional.

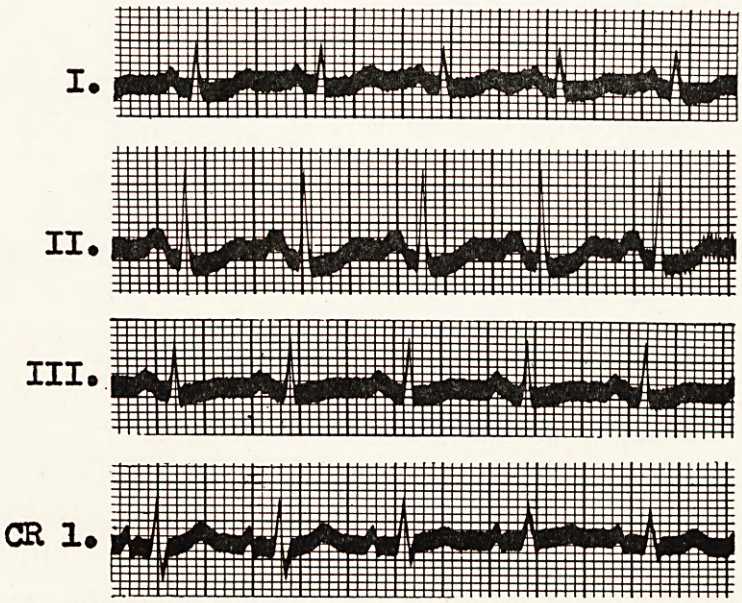

FIG. 2.—Electrocardiogram taken shortly before death. Showing sinus tachycardia.

Dyspnœa and tachycardia developed followed by the onset of acute pulmonary œdema. Within an hour of the onset of this acute episode she was desperately ill and in much the same state as when previously admitted.

She became comatose, cyanosed and dyspnœic. Sinus tachycardia (rate 120) was confirmed by electrocardiogram (Fig. 2). On auscultation no cardiac murmur could be heard owing to the tachycardia but the mitral first sound was accentuated. There was no peripheral venous distension or œdema and no hepato-jugular reflux. B.P. 125/85.

Once again, with oxygen, morphine, aminophylline, neptal and this time with full doses of digitalis, she slowly recovered. Three days later when much improved she suddenly vomited and lost consciousness. On examination, she was found to have a complete right sided hemiplegia. The left optic fundus showed the picture characteristic of occlusion of the central artery of the retina. The right pupil was dilated.

Over the twelve hours following the onset of hemiplegia, the patient at first improved, although the paralysis remained and she was unable to speak. Later, however, her condition deteriorated and she developed signs of intracranial compression. Coma deepened and she died eighteen hours after the onset of the hemiplegia.

Post-mortem Findings.—The heart was slightly enlarged and showed several patches of fibrosis of the epicardium. The myocardium of all chambers was of normal thickness and the only abnormality seen there was a few flakes of fibrosis in the left ventricle. The only chamber which appeared to be enlarged was the left auricle. Both chambers on the right side were healthy and the endocardium on that side was also healthy. The left auricle contained a large soft gelatinous mass firmly attached to the septum closing the foramen ovale, by a pedicle about 2 cm. in diameter. The adjacent auricular endocardium was thickened in places. The mass measured about $8 \times 6 \times 4$ cm. and although it was gelatinous, it contained many areas of hæmorrhage and was covered by a smooth lining. It had the naked-eye appearance of a myxoma (Fig. 3).

FIG. 3.—Photograph of autopsy specimen. Polypoid tumour seen lying in the left auricle.

The mitral valve was not dilated. The cusps were slightly thickened by fibrous tissue, but not shortened. A few of the chordæ were also thickened by fibrous tissue. The aortic valve was healthy. Coronary vessels showed only a moderate degree of atheroma.

Microscopic examination showed the intra-auricular mass to be an œdematous organising thrombus. Much round cell infiltration was present at its base. Fibrosis was confirmed in the mitral valve and the chordæ, but no other evidence of active or previous rheumatic heart disease was seen. The only lesions found in other organs were an extensive organising pneumonia with patchy œdema, a recent small infarction in the spleen and hæmorrhagic infarction in the left basal ganglia.

DISCUSSION

The earliest recorded discussion of the occurrence and ætiology of intra-auricular polypi is to be found in Allan Burns' *Observations*

on some of the most Frequent and Important Diseases of the Heart. This was published in 1809 and is quoted by Wood in a letter to the *Edinburgh Medical and Surgical Journal* of 1814, when describing the case and autopsy findings in a girl aged 15 with mitral stenosis and a ball thrombus. Wood states that intra-auricular polypi were well recognised at that time and could be distinguished from ante-mortem and post-mortem thrombi, either free or adherent to the endocardium.

Strouse (1938) quoted a case described by Pavlowski in 1894 and in more recent years numerous cases have been reported by both clinicians and pathologists.

Prichard (1951) states that approximately 125 cases of cardiac myxoma have been recorded, roughly 75 of these occurring in the left auricle. Mahaim (1945) gives the number of cases described as nearer 280. Kirkeby and Leren (1952) and Goldberg *et al.* (1952) both record cases where a correct diagnosis had been made during life, but successful surgical removal has not yet been reported. In Goldberg's case, operative removal of a left auricular polyp was attempted, but their patient, a child of three, did not survive.

In addition to the case described in this report 29 other patients were found to have been described in adequate detail and the main clinical features of these 30 cases are presented in table form. It is hoped that the information provided may be of value in diagnosing future cases at a stage when surgical treatment might be possible.

In some of the 30 cases described the tumour was believed to be a degenerated mural thrombus and in others a myxomatous new growth, macroscopically they were indistinguishable and the site of attachment was always near the fossa ovalis or its rim, but whatever the nature of its origin once the tumour had formed its effect on cardiac function and symptomatology was the same.

The pathological aspect of the problem is dealt with in full by Yater (1931) and Prichard (1951) and is still much debated. Since no attempt has been made to discuss pathological details or enter into the controversy over the origin of these tumours the term " benign polypoid tumour " is used in preference to " myxomatous polyp " suggested by Mahaim in 1945.

Analysis of Reported Cases of Benign Polypoid Tumours of the Left Auricle

Table I indicates the sources from which the case history was taken. It shows the age and sex incidence, the lack of a previous history of rheumatic fever in most instances, the duration of illness and the post-mortem findings. The cases are numbered to facilitate reference to them in the text.

It is worth while noting that most patients were under observation for several weeks or months, and in Case 23 symptoms had lasted for as long as seven years. Diagnosis, therefore, although difficult, should be possible.

TABLE I

Case No.	Case described by	Sex.	Age.	Previous History of Rheumatic Fever.	Duration of Symptoms in Months.	Autopsy Findings other than Auricular Polyp and its direct Effects.
1	Norton (1919) . .	M.	29	No	3	None
2	Houck and Bennett (1930)	F.	44	,,	2	,,
3	Yater (1931) . .	F.	23	,,	24	,,
4	Schwartz and Biloon (A) (1932)	F.	44	Yes	108	Rheumatic mitral and aortic disease
5	Schwartz and Biloon (B) (1932)	M.	63	No	2¼	Hypertensive heart disease
6	Kaplan and Hollingsworth (1935)	M.	38	,,	60	None
7	Gilchrist and Millar (1936)	M.	57	,,	3	,,
8	Bennett, Konigsberg and Dublin (1938)	F.	37	,,	? " days "	,,
9	Benjamin (1939) .	M.	17	,,	¾	,,
10	Fawcett and Ward (1939)	F.	60	,,	10	,,
11	Dexter and Work (1941)	F.	63	,,	12	Slight thickening of mitral valve. Some micro. evidence of old rheumatic carditis
12	Hamilton-Paterson and Castleden (1942)	M.	46	?	? " years "	None
13	Maun (1943) . .	F.	?	?	7	,,
14	Thomson (1944) .	F.	33	No	12	,,
15	Burnett and Davidson (1945)	F.	29	,,	15	,,
16	Field, Donovan and Simon (1945)	F.	48	,,	? " months "	,,
17	Brown (1946) . .	F.	35	,,	24	,,
18	Brewin (A) (1948) .	F.	47	,,	8	,,
19	Brewin (B) (1948) .	F.	31	,,	2	,,
20	Brewin (C) (1948) .	F.	37	?	18	Doubtful micro. Evidence of old rheumatic carditis only
21	Brewin (D) (1948) .	F.	49	No	26	None
22	Alison and Susman (1949)	F.	20	,,	2½	Ante-mortem clot in both ventricles
23	Von Reiss (1949) .	F.	37	,,	84	None
24	Weinstein and Arata (1949)	F.	64	Yes	24	,,
25	Coulter (1950) .	F.	68	No	3	,,
26	Mills and Philpott (1951)	M.	44	?	7	,,
27	Block, Parker and Edwards (1952)	M.	57	No	18	Slight thickening of mitral valve. No micro. evidence of rheumatic carditis
28	Goldberg *et al.* (1952)	F.	2	,,	19	None
29	Kirkeby and Leren (1952)	M.	50	,,	14	,,
30	Morton (1953) .	F.	47	,,	12	Slight mitral valve thickening. Minimal micro. evidence of old rheumatic lesions

Only in Cases 4 and 5 was there any autopsy evidence of a significant degree of organic heart disease. In Cases 11, 20 and 30 doubtful or minimal evidence of previous rheumatic carditis was found, but in these 3 cases the rheumatic lesions were insufficient to cause clinical signs of mitral disease or cardiac embarrassment.

Table II gives an analysis of the commonest clinical signs and symptoms in this series of cases, and the conclusions drawn from their detailed study are given.

TABLE II

The Commonest Clinical Signs and Symptoms in 30 Cases of Left Auricular Polyp.

	Number of Cases in which Symptom was Present.
Exertional dyspnœa	26
Congestive cardiac failure	25
Clinical cardiomegaly	22
Cough noted as a marked feature	19
Retrosternal pain—	
At rest	14 } Total 14*
On exertion	8 }
Arterial emboli	14
Paroxysmal dyspnœa	13
Pyrexia noted as a marked feature	12
Palpitations	12
Syncopal attacks	10
Postural symptoms—	
Variation of auscultatory signs	1 }
Postural dyspnœa	3 } Total 7
Postural retrosternal pain	1 }
Postural syncope	2 }
Arrhythmias—	
Auricular fibrillation	4 } Total 6
Paroxysmal arrhythmia	2 }
Hæmoptysis	5
Intermittent claudication	4

* All 8 patients who experienced pain on exertion also had retrosternal pain on occasions while at rest.

Electrocardiogram and radiological examination have been of little assistance in diagnosis, with the noteworthy exception of Case 28. Here Goldberg *et al.* had the advantage of an angiocardiogram which demonstrated a large irregular polypoid filling defect in the left auricle. This was, of course, diagnostic and is the only reported case where the diagnosis has been confirmed in this way.

Conclusions

From this study it would appear that polypoid tumours of the left auricle do occur in otherwise normal hearts. Whether or not the tumour is a true myxomatous new growth or an organised thrombus seems to make no difference to the signs and symptoms and indeed the pathological distinction between the two groups is debatable. The clinical picture they present is varied, but often follows a fairly definite pattern.

A proportion of cases die suddenly, presumably from complete obstruction of the mitral valve, without prior symptoms. Most cases seem to present with signs or symptons which suggest a diagnosis of mitral valve disease, but even in the cases where this diagnosis seems most likely, atypical features are always present. These patients complain of exertional dyspnœa, attacks of paroxysmal dyspnœa, palpitations, retrosternal pain, cough and sometimes hæmoptysis. They are usually found to be in normal cardiac rhythm. Auscultatory signs of mitral stenosis may or may not be present and these signs, if present, may be heard one day and not the next. The patients are pyrexial, blood cultures are negative and the blood sedimentation rate is high. Usually the total history is a matter of months and once symptoms appear, deterioration is progressive and fairly rapid.

The particular features which may be present and may suggest a diagnosis of left auricular polyp rather than mitral stenosis are firstly a lack of history of rheumatic fever, sore throats or chorea. Secondly, a steady and rapid progression of the congestive cardiac failure despite digitalis and mercurial diuretics. Thirdly, radiological and electrocardiographic changes of minor degree and which are not quite typical of rheumatic or other heart disease of such apparent severity. Paroxysmal arrhythmias are rare, but may occur. Fourthly, postural changes may occur in the auscultatory signs or there may be a noticeable relationship between posture and attacks of paroxysmal dyspnœa, cyanosis and fainting attacks. Fifthly, syncopal attacks are relatively common and may be followed by severe shock and peripheral circulatory failure. They may mimic Stokes-Adams attacks, but heart-block is absent. Sixthly, attacks of pulmonary œdema are unusually severe and either these or the syncopal attacks may prove fatal.

There is a small but definite group of cases where the diagnosis should be suggested by embolic episodes occurring in a patient either with an apparently normal heart, or with a heart in which some of the signs of mitral stenosis are present, but where the cardiac rhythm is normal and no obvious source of emboli can be found.

These emboli may produce signs and symptoms suggestive of peripheral vascular disease, may stimulate intermittent claudication and may lead to gangrene. They may, however, produce bizarre and often transient signs in the central nervous system, and skin lesions suggestive of subacute bacterial endocarditis.

Angiocardiography has only been used in the diagnosis of one case so far but it should be employed in the investigation of all future cases where an auricular polyp is suspected and an adequate angiocardiograph will probably be of the greatest value.

As in many of these cases the differential diagnosis lies between mitral stenosis and left auricular polyp, exploratory thoracotomy (which in practised hands is now regarded as a relatively minor procedure) might be justifiable both to confirm the diagnosis and to relieve the condition whether it be by valvulotomy or by excision of a polyp.

A hard-and-fast pre-operative diagnosis may not therefore be regarded as essential, as, if an auricular polyp is suspected, surgical excision would appear to be the only way of obtaining a radical cure.

Summary

1. A case of benign polypoid tumour of the left auricle is described with post-mortem findings.

2. A survey of the English literature reveals a further 29 cases where the case history is given in some detail.

3. The signs and symptoms of these 30 cases are present in Table IV and discussed in the text.

4. No case has yet been successfully diagnosed and treated, but the surgical removal of such a tumour is considered to be possible and a plea is put forward for earlier diagnosis.

5. The employment of angiocardiography should be of the greatest assistance and exploratory thoracotomy is suggested as a diagnostic as well as a therapeutic measure.

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References

Alison, D. R., and Susman, W. (1949), *Lancet*, **2**, 11.
Benjamin, H. G. (1939), *Arch. Path.*, **27**, 950.
Bennett, D. W., Konigsberg, J., and Dublin, W. (1938), *Amer. Heart Journ.*, **16**, 117.
Block, W. J., Parker, R. L., and Edwards, J. E. (1952), *Proc. Mayo Clin.*, **27**, 361.
Brewin, T. B. (1948), *Guy's Hosp. Rep.*, **97**, 64.
Brown, W. O. (1946), *Amer. Heart Journ.*, **31**, 373.
Burnett, W., and Davidson, J. I. (1945), *Brit. Heart Journ.*, **7**, 180.
Burns, Allan (1809), " Observations on some of the most frequent and important Diseases of the Heart," quoted by Wood.
Coulter, W. W. (1950), *Arch. Path.*, **49**, 612.
Dexter, R., and Work, J. L. (1941), *Arch. Path.*, **32**, 995.
Fawcett, R. E. M., and Ward, E. M. (1939), *Brit. Heart Journ.*, **1**, 249.
Field, M. M., Donovan, M. A., and Simon, H. (1945), *Amer. Heart Journ.*, **30**, 230.
Gilchrist, A. R., and Millar, G. W. (1936), *Edin. Med. Journ.*, **43**, 243.
Goldberg, H. P., Glenn, F., Dotter, C. T., and Steinberg, I. (1952), *Circulation*, **6**, 762.
Hamilton-Paterson, J. L., and Castleden, L. I. M. (1942), *Brit. Heart Journ.*, **4**, 103.
Houck, G. H., and Bennett, G. A. (1930), *Amer. Heart Journ.*, **5**, 787.
Kaplan, D., and Hollingsworth, E. W. (1935), *Journ. Amer. Med. Assoc.*, **105**, 1264.
Kirkeby, K., and Leren, P. (1952), *Acta med. Scand.* CXLIII, fasc. 5, 385.
Mahaim, I., *Les Tumeurs at Les Polypes due Coeur*, Paris, 1945.
Maun, M. E. (1943), *Amer. Heart Journ.*, **26**, 549.

MILLS, P., and PHILPOTT, M. (1951), *Brit. Heart Journ.*, **13**, 115.
NORTON, W. H. (1919), *Amer. Journ. med. Sci.*, **158**, 689.
PAVLOWSKI, R. A. (1894), *Ztschr. F. Klin. Med.*, **26**, 482, quoted by Strouse.
PRICHARD, R. W. (1951), *Arch. Path.*, **51**, 98.
SCHWARTZ, S. P., and BILOON, S. (1932), *Amer. Heart Journ.*, **7**, 84.
STROUSE, S. (1938), *Arch. int. Med.*, **62**, 401.
THOMSON, R. B. (1944), *Brit. Heart Journ.*, **6**, 23.
VON REISS, G. (1949), *Acta. med. Scand.* CXXXIII, fasc. **3**, 214.
WEINSTEIN, M. S., and ARATA, J. E. (1949), *Amer. Heart Journ.*, **38**, 781.
WOOD, W. (1814), *Edin. Med. Surg. Journ.*, **10**, 50.
YATER, W. M. (1931), *Arch. Int. Med.*, **48**, 627.