en résultaient. On a enfin remarqué que l'exérèse de la tumeur et de ses métastases supprimait la diarrhée et rétablissait l'équilibre électrolytique.

Les auteurs présentent leur observation d'une malade accusant de la diarrhée chronique et souffrant d'hypokaliémie. Elle présentait une ulcération œsophagienne qui évolua vers une sténose. Cette malade mourut finalement de déshydratation et de déficit potassique. A l'autopsie on découvrit une tumeur dans la queue du pancréas. Ce n'est qu'alors que les auteurs prirent connaissance de l'article de Verner et Morrison.

Le tableau clinique qu'offrent les diarrhées causées par une tumeur pancréatique ressemble quelque peu à celui des états pathologiques de l'intestin (recto-colite, entérite régionale, dysenterie bacillaire, sprue, maladie de Whipple etc.) et aussi à celui du syndrome du carcinoïde. Dans le cas rapporté ici, on avait songé à l'aldostéronisme de Conn. Dans la discussion de l'étiopathogénèse de ce symptôme, les auteurs attirent l'attention sur une certaine similarité entre les tumeurs carcinoïdes et celles du pancréas. Ils prétendent qu'il n'est pas impossible que les secondes, à l'instar des premières, peuvent secréter à l'excès la 5-hydroxy-tryptamine, ce qui expliquerait la diarrhée et l'hypotension chez ces malades. D'autre part une secrétion exagérée de glucagon pourrait expliquer les pertes d'eau et d'électrolytes dans les selles. La laparotomie exploratrice est donc indiquée dans les diarrhées chroniques incurables avec déshydratation et déficit potassique dans l'espoir de trouver un adénome langerhansien dont l'exérèse entraînerait la guérison.

MYXOMA OF THE HEART*

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SINCE CARDIAC myxomas may be surgically removed, their clinical diagnosis is now imperative. Not one of the myxomas before 1951 reported by Prichard[1] had been suspected during life. Since then, at least 38 cases have been diagnosed and 17 had successful excision.

The purpose of this paper is to discuss the subject with special emphasis on the clinical diagnosis. Because of the increasing interest in these tumours since they became amenable to surgery, we felt that the analysis of cases reported in recent years might improve our diagnostic approach. We have reviewed 63 case reports published during the past five years. Two cases, reported herein, have been added to this group, making a series of 65. The clinical and hæmodynamic data have been analyzed and will be presented.

REVIEW OF THE LITERATURE

Incidence

Half of the primary heart tumours, which according to Leach[2] are found in 0.05% of unselected autopsies, are myxomas. Prichard,[1] in 1951, estimated the reported cases at 127, and Gleason,[3] in 1955, added 23 more. In spite of its low incidence, at least 215 myxomas have been so far reported in the literature. They are found almost exclusively in adults between 30 and 60 years of age. Only six have been described in children.[4-6]

Pathological Features

Myxomas were previously thought to be degenerated, œdematous thrombi, but their true neoplastic nature is now generally recognized.[1, 7] They are usually polypoid, gelatinous to firm in consistency, and from a few millimetres to 10 cm. in diameter. Almost exclusively found in the atria, 75% are found in the left atrium.[1] Their pedicle is most frequently attached near the rim of the fossa ovalis and arises rarely from the atrial wall or valve leaflets. In a few cases, the pedicle originated from the ventricular endocardium.[1] Rare cases have been described with multiple myxomas. Separate tumours were found in each atrium in one patient,[8] and in another, two growths were observed in the left atrium.[9] Paquet[10] described a case with multiple small myxomas in the wall of the right atrium and pulmonary arteries.

Physiopathology and Clinical Manifestations

Hæmodynamic disturbances depend on the size of the tumour, its shape, and its position with respect to portals of atrial inflow and outflow. It may obstruct the venous return, encroach upon the atrial cavity or occlude the A.V. (atrioventricular) orifice, resulting in an elevation of the venous pressure and decreased cardiac output. Depending on the mobility of the tumour and the length of it pedicle, its position may vary with respect to the venous inflow or A.V. orifice, and thus lead to varying degrees of occlusion, particularly with changing posture.

Various clinical syndromes may accompany this specific lesion and the resultant hæmodynamic disturbance. Excellent clinical descriptions have been presented by numerous authors, among others Yater,[11] Mahaim[12] and Goldberg.[13] Rarely, they present the almost pathognomonic ball-valve occlusion syndrome.[12] Unexplained attacks of dyspnœa, cyanosis, palpitations or syncope may result from transient but almost complete occlusion of the mitral or tricuspid orifice. Similar episodic disturbances are due to intermittent obstruction of the pulmonary or systemic venous return. Transient shock and coma may result from longer periods of occlusion. These symptoms may be spontaneous or brought about by changing posture. Similarly, variable results on auscultation with time and with

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changing posture may be observed. A diastolic rumble may be heard best in the sitting position, whereas an apical systolic murmur may be more intense in recumbency, again owing to the varying position of the tumour with respect to the mitral or tricuspid orifice in these different postures.

Most frequently, however, the symptoms are constant and the clinical course is steadily progressive. The clinical manifestations appear when the tumour almost fills the atrial cavity, and progress relentlessly as the tumour steadily grows. In fact, most tumours are rather large when discovered and usually after a short illness. Partial but more persistent occlusion of the mitral orifice may produce symptoms and signs of mitral valve disease, particularly of mitral stenosis. The relentless and rapid course, as the tumour grows and encroaches further upon the atrial cavity, distinguishes it from mitral stenosis. The left atrium is rarely grossly enlarged, probably because compensatory dilatation and hypertrophy do not take place owing to the relatively sudden, rapidly progressive obstruction of the mitral orifice. This slight left atrial dilatation and the absence of myocardial involvement as found in rheumatic carditis may also explain the low incidence of atrial fibrillation in contrast to mitral stenosis.

Predominant pulmonary vein obstruction may lead to severe pulmonary hypertension in the absence of or with slight left atrial enlargement. A loud P_2 (second pulmonic sound) may be the only abnormal auscultatory finding. This pulmonary hypertension, in the absence of obvious left heart disease or other specific etiology, should arouse suspicion. In fact, many left atrial myxomas have been erroneously diagnosed as cor pulmonale, Hamman-Rich syndrome, or pulmonary hypertension due to multiple pulmonary emboli.

Emboli of myxomatous tissues or of thrombi formed on the surface of the myxoma may produce bizarre and puzzling neurological[14] and abdominal syndromes,[15, 16] particularly if there is no demonstrable heart disease and atrial fibrillation is absent. True myocardial ischæmia and infarction due to coronary emboli have been reported.[17] Anginal pain may also be produced by a decreased cardiac output or, rarely, by external pressure on one of the coronary arteries. Kroopf and Peterson[18] reported a case with an electrocardiogram showing an acute anterior myocardial infarction, but without evidence of coronary thrombosis at autopsy. It was felt that direct compression of the left coronary artery by the tumour was responsible for these changes. Pain related to posture should be carefully evaluated. Repeated small emboli may give rise to fever, petechiæ and splinter hæmorrhages, which suggests subacute bacterial endocarditis,[17] particularly in the presence of heart murmurs.

In a similar fashion, right atrial myxomas may resemble tricuspid valve lesions. The selective right heart failure which invariably develops may suggest constrictive pericarditis. Repeated pulmonary emboli may give rise to pulmonary hypertension, and further confuse the clinical picture. In rare cases of atrial myxoma, unexplained heart failure and generalized cardiomegaly without other specific findings will be present; myocarditis or heart disease of unknown etiology will remain as the unsatisfactory, but only possible, diagnosis.

CASE REPORTS

CASE 1.—This 63-year-old housewife was relatively well until six months before her admission on May 29, 1958, when she complained of progressive exertional dyspnœa, a swelling abdomen and pedal œdema. Her appetite was poor and marked weakness had developed.

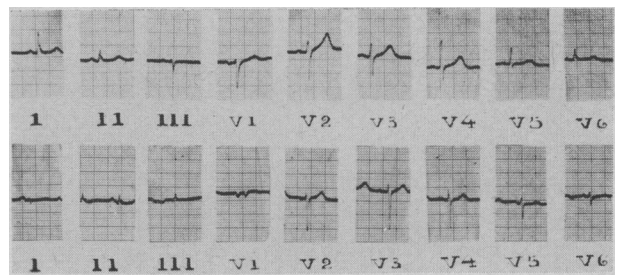

Fig. 1.—Top tracing, taken two years before present illness; bottom tracing, taken six months after onset of illness (Case 1).

On examination this obese patient appeared chronically ill. Her general appearance suggested hypothyroidism. Her skin was dry; her eyebrows were thin. She could lie flat in bed without difficulty but was dyspnœic on sitting up and with the slightest activity. Blood pressure was 110/70 mm. Hg. Neck veins were grossly distended. The thyroid gland was not enlarged. Subcrepitant rales were present at both lung bases.

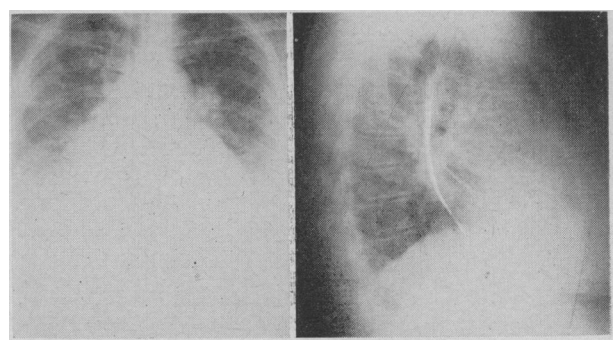

Fig. 2.—Postero-anterior and lateral chest films (Case 1).

P_2 was loud; no murmur was heard. Heart rhythm was regular at 120 per minute. Her liver was enlarged; ascites and œdema of the legs were noted. Deep tendon reflexes had a delayed relaxing phase. Routine laboratory examinations were normal. The electrocardiogram (Fig. 1) showed sinus tachycardia, right axis deviation, low voltage, and marked clockwise rotation. A routine electrocardiogram taken two years previously was normal. The chest radiograph (Fig. 2) revealed congested pulmonary vessels and

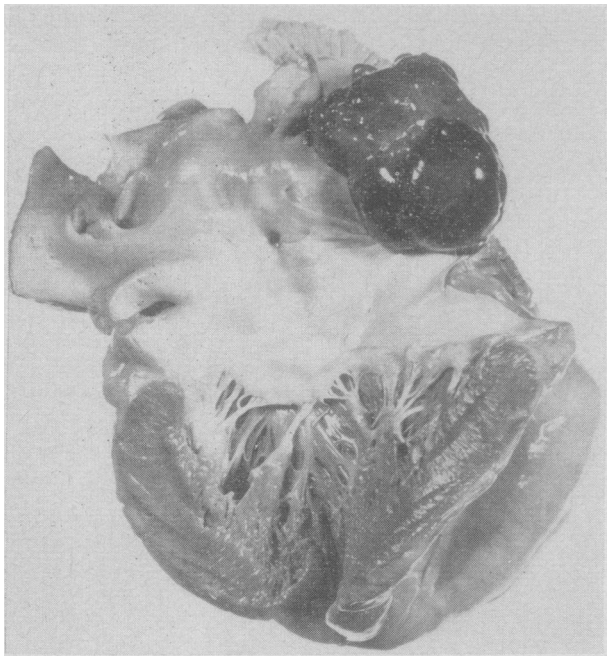

Fig. 3.—Left atrial polyp (Case 1).

bilateral pleural effusions. The heart was grossly enlarged with a cardio-thoracic ratio of 15/26 cm. All cardiac cavities appeared to be involved, but particularly the right side. On fluoroscopy, the cardiac pulsations were almost absent, suggesting pericardial effusion, but supine and upright films did not show significant change in the width of the mediastinum. The I^{131} thyroid uptake was 7.2%. It was suspected that she had arteriosclerotic and myxœdematous heart disease. She was given digitalis, chlorothiazide, and thyroid extract and improved slowly, lost 25 lb. in a period of four weeks, and became œdema-free. Her heart rate slowed to 60-80 per minute. She was discharged but did poorly at home, complaining particularly of marked weakness, anorexia, and dyspnœa whenever she got out of bed. She was readmitted two months later, and on examination was found again to be in right heart failure. The only new finding was a grade III, high-pitched, systolic murmur at the apex. During the first two weeks, she improved again, lost 13 lb. and became œdema-free. She suddenly became very agitated and markedly dyspnœic. The blood pressure fell to 90/80 mm. Hg; urinary output decreased sharply. She became progressively comatose and died three days later.

*Post-Mortem Findings**

The heart weighed 450 g. The right ventricle was hypertrophied with a wall thickness of 1 cm. Both atria were slightly dilated, but the left ventricle appeared normal. A polypoid tumour (Fig. 3) filled the left atrium. It was dark red to yellow-white and gelatinous in consistency. It measured 6 x 5 x 5 cm. and was attached by a short thin pedicle to the interauricular septum, above the fossa ovalis. Its position and mobility could permit occlusion of the mitral

*We are indebted to Paul Maheux and Nicolas Aerichide for the pathology study.

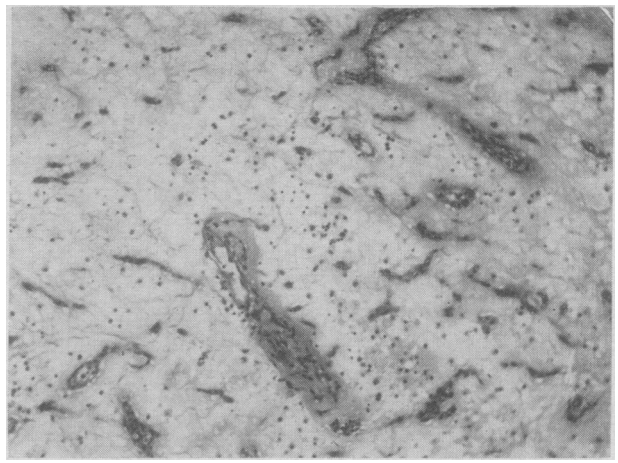

Fig. 4.—Conjunctival framework filled with mucoid substance (Case 1). (H.F.S. × 200.)

orifice in a ball-valve fashion. In fact, its lower pole was cone-shaped as if moulded by the mitral orifice. On cut section, the centre was necrotic and hæmorrhagic. Except for passive congestion of most organs, and an atrophic fibrous thyroid gland, the remainder of the post-mortem examination was not remarkable. Histological studies (Fig. 4) revealed a conjunctival framework filled with mucoid substance and blood. It was considered to be a myxoma.

CASE 2.*—This 47-year-old male draftsman was referred to us in April 1957, with the diagnosis of possible constrictive pericarditis. He had noticed during the past year a progressively enlarging abdomen, and, several months before admission, had slight exertional dyspnœa and pedal œdema.

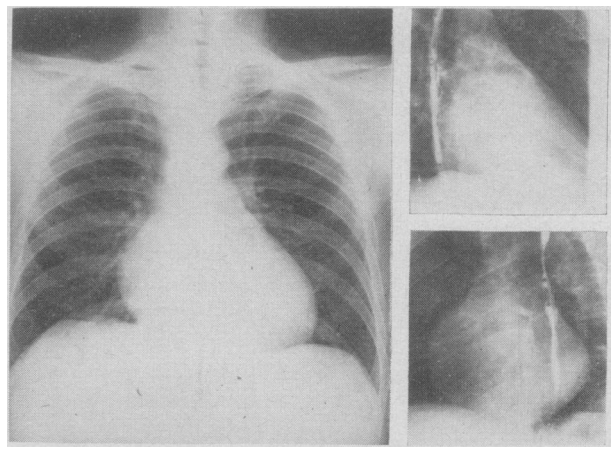

Fig. 5.—Postero-anterior, left and right oblique chest films (Case 2).

On examination, he appeared chronically ill. The neck veins were grossly distended. Lungs were clear; P_2 was reduplicated, but not accentuated. Harsh systolic and high-pitched diastolic murmurs were heard at the fourth intercostal space to the left of the sternum. Four heart sounds were heard at the apex. The rhythm was regular. Blood pressure was

*Previously reported in part.[19]

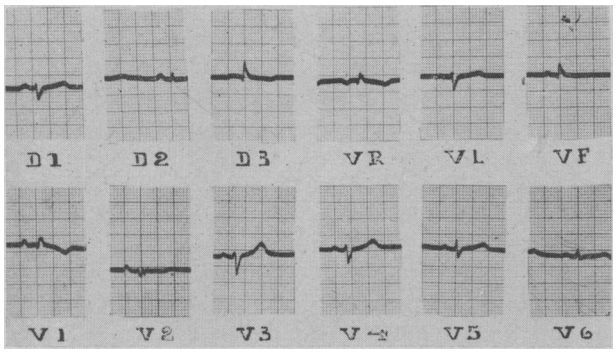

Fig. 6.—Electrocardiogram, April 1957 (Case 2).

120/76 mm. Hg. The liver was enlarged and ascites was present but no pedal œdema noted. Routine laboratory tests were within normal limits. The sedimentation rate was 44 mm. in one hour; BSP excretion 28%. The chest radiograph (Fig. 5) revealed moderate right heart enlargement; the pulmonary vasculature appeared normal. At fluoroscopy, the right atrium showed striking systolic expansions. The electrocardiogram (Fig. 6) had low voltage, right axis deviation and incomplete right bundle-branch block. At right heart catheterization, a simultaneous pressure recording of the right atrium and ventricle gave a diastolic pressure gradient of 8 mm. of mercury, compatible with tricuspid stenosis (Fig. 7). The right ventricular curve had an early diastolic dip with a diastolic plateau, the "square root sign". The patient was discharged, and readmitted in September 1957 for an exploration of the tricuspid valve and possible commissurotomy.

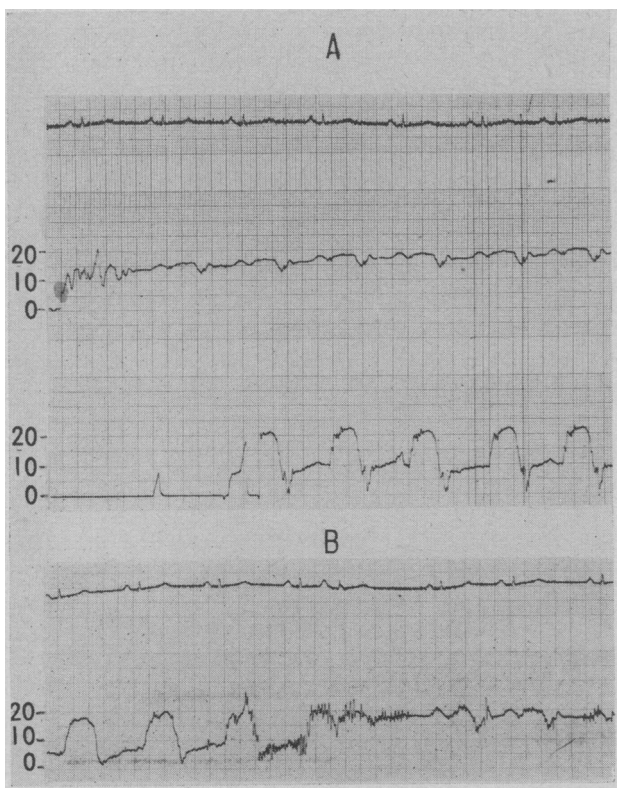

Fig. 7.—A: Simultaneous right atrial and right ventricular pressure. B. Pull back from right ventricle to right atrium.

A large polypoid tumour was discovered in the right atrium (Fig. 8); it filled the entire right atrium and protruded into the right ventricle through a dilated tricuspid ring. It was attached by a 2-cm. pedicle to the atrial septum near the fossa ovalis. The surgeon felt that it could not be removed with a closed-heart technique. The patient recovered without complications and was subsequently referred to the Mayo Clinic where Dr. F. Henry Ellis[19] resected the tumour, using extracorporeal circulation. The tumour was removed completely, but in multiple fragments. It measured 10 cm. in diameter and weighed 175 g. After the resection, an obvious free tricuspid insufficiency, probably related to the tremendous dilatation of the tricuspid ring, was noted. The patient had an uneventful course, has since resumed a normal life and is still doing well 1½ years after operation. Histologically the tumour was described as a cellular myxoma.

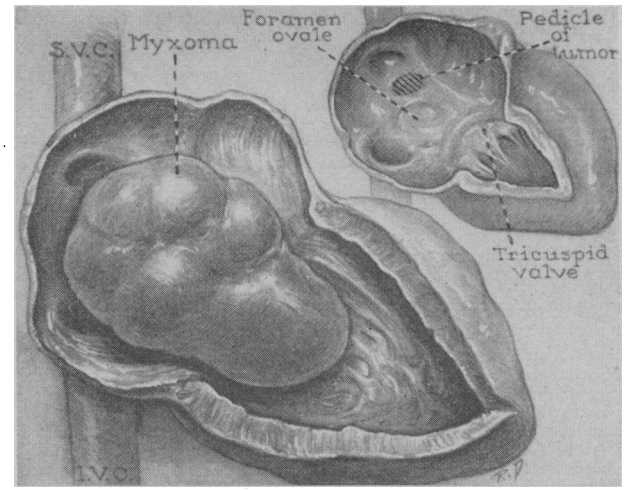

Fig. 8.—Drawing of the right atrial polyp (from Ellis *et al.*: *M. Clin. N. America*, 42: 1087, 1958).

ANALYSIS OF SIXTY-FIVE CASES

We have studied 65 cases of cardiac myxoma; two have been presented above and 63 were reported in the French and English literature between January 1954 and January 1959. Twelve articles were not available.

Of the 65, 57 were found in the left atrium, and only eight in the right. The sex distribution was equal and the median age, 45 years. The youngest patient was one month old and the oldest 73 years. The median duration of the clinical manifestations was one year, with a range varying from one month to 30 years.

Left Atrial Myxoma (Symptoms, Signs and Clinical Diagnosis)

From 45 adequately described cases of left atrial myxoma, which were analyzed in detail, it was observed that paroxysmal dyspnœa occurred in 37%, syncope in 22%, and postural symptoms in 18% (Table I). In comparison, varying symptoms

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TABLE I.—SYMPTOMS OF LEFT ATRIAL MYXOMA
(45 CASES)

Exertional dyspnœa	38	Postural symptoms	8
Orthopnœa	18	Acute pulmonary œdema	11
Paroxysmal dyspnœa	17	Hæmoptysis	9
Fever	14	Cerebral emboli	15
Syncope	10	Peripheral emboli	4
Chest pain	6		

with different body positions were observed in 37% of the cases with right atrial tumour. The relatively low incidence of orthopnœa contrasted with the much more frequent exertional dyspnœa. Symptoms and signs of cerebral or peripheral emboli were noted frequently and, in several cases, were the first clinical manifestations.[10, 20, 21] A biopsy of accessible peripheral emboli, as reported by Bland,[6] confirmed the diagnosis by revealing myxomatous tissue. Gerbode *et al.*[22] reported an interesting case in which the symptoms and signs of mitral stenosis disappeared for a period of 18 months after an embolic accident, presumably due to the decreased size of the tumour after its fragmentation. The other symptoms found were not specific and may be commonly found with left atrial or ventricular failure of various etiology.

TABLE II.—AUSCULTATION IN LEFT ATRIAL MYXOMA
(45 CASES)

Accentuated P2	28	Apical murmurs:	
Snapping M1	18	diastolic	5
Mitral opening snap	7	systolic	6
Third heart sound	6	diastolic and systolic	26
No murmur	8	Presystolic crescendo	11
		Variable: time and posture	7

The auscultatory signs in left atrial myxoma most frequently suggested isolated mitral stenosis or stenosis with regurgitation (Table II). In several cases, the auscultatory findings were typical of mitral stenosis, including an accentuated first heart sound and a mitral opening snap. Only 15% demonstrated varying auscultatory findings with time and with changing postures. Eight patients had no murmur. In seven of these, P2 was markedly accentuated, suggesting pulmonary hypertension without obvious evidence of left heart disease. These findings suggested falsely the diagnosis of Hamman-Rich syndrome, cor pulmonale, and pulmonary hypertension due to multiple pulmonary emboli.

The electrocardiogram in most cases had one or several of the features commonly observed in mitral stenosis: right axis deviation, broad or peaked P waves, right bundle-branch block and right ventricular hypertrophy. Abnormal P waves and right ventricular hypertrophy patterns were noted in approximately 50% of the cases. Arrhythmias were infrequent, being noted in only eight cases. Atrial fibrillation and flutter were observed in six, the paroxysmal type in two. One case[19] had a bigeminy with varying posture, and paroxysmal ventricular tachycardia was observed in another.[23] Two cases had an anterior myocardial infarction pattern.[17, 18] Only two tracings were considered entirely normal.

The radiological appearance of the heart in most instances simulated mitral stenosis. Left atrial enlargement was present in 80%. This was the only enlarged cardiac cavity in 20%. The absence of or slight enlargement of the left atrium observed in some cases was an important atypical feature that helped rule out mitral stenosis. At fluoroscopy, powerful systolic expansion of the left atrium, believed to be significant by Van Buckem *et al.*,[8] was noted in four cases;[10, 24, 25] half had right ventricular enlargement. Generalized cardiomegaly was observed in one-fourth of the cases, and normal heart size was reported in five instances only.

Most patients resembled those with other more common heart diseases. They simulated mitral stenosis in 70% of the patients, and, in fact, the resemblance was so perfect that half of them were erroneously operated on for this lesion. Several atypical features were pointed out by the reporters or in retrospect were believed by us to be significant in ruling out mitral stenosis. The late onset, the short duration of the illness and the rapidly progressive and relentless course—all unusual in mitral stenosis—were observed in most cases. Cerebral or peripheral emboli in the absence of atrial fibrillation, and postural symptoms and signs were found in 42 and 35% of these cases respectively. Only one of these was confirmed by angiocardiography.

Eight cases or 18% presented with pulmonary hypertension without obvious cause. This diagnosis was not always considered as such by the reporters but it was frequently our impression after reviewing the cases. There was no evidence of left heart disease in the absence of apical murmurs and slight or no left atrial enlargement. The atypical features were most commonly the absence of a satisfactory explanation for the hypertension, slight left atrial enlargement, paroxysmal or postural symptoms and signs. Four of these cases were suspected on clinical grounds and two confirmed by angiocardiography.

Subacute bacterial endocarditis was falsely suspected in eight cases. Anorexia, weight loss, fever, petechiæ, splinter hæmorrhages, elevated sedimentation rate and white blood cell count have been noted, and only the absence of splenomegaly and negative blood cultures were suspicious. A puzzling case, reported by Dick and Mullin,[26] had positive blood cultures due to an associated *Staphylococcus aureus* septicæmia. The two patients suspected of having myocarditis were both infants. Stephen[27] reported three asymptomatic cases, but it impressed us that only two could be so qualified. One of these patients suffered for four years preceding his death from "bronchial asthma" which could have been cardiac asthma related to the myxoma. Of the 45 left atrial tumours, four were

suspected clinically, three were confirmed by angiocardiography, 16 were discovered during surgery for presumed mitral stenosis, and 22 were post-mortem discoveries.

Right Atrial Myxoma (Symptoms, Signs and Clinical Diagnosis)

Postural symptoms and signs were observed in three of eight cases.[10, 28, 29] Signs of right heart failure were the chief findings in one-half of the cases. The electrocardiogram frequently showed right axis deviation, tall peaked P waves, right bundle-branch block and low QRS voltage. (These features, as recently suggested by Belle,[30] are not unlike the electrocardiographic ones obtained in Ebstein's anomaly.) Radiography showed most commonly right-sided heart enlargement. Systolic expansion of the right atrium was observed in three cases (Case 2).[10, 31] Calcifications of radiological significance were present in two cases,[32, 33] and indeed established the diagnosis. In Krcilkora's[31] case, a well-circumscribed ball-shaped mass could be seen moving within the shadow of the right atrium. Two cases simulated tricuspid stenosis, and two, insufficiency. One patient was suspected of having constrictive pericarditis and another Ebstein's anomaly. Two simulated subacute bacterial endocarditis. All these tumours were diagnosed during life. One was discovered because of tell-tale calcifications, another during exploratory thoracotomy, and six by angiocardiography.

Cardiac Catheterization Findings

At least 28 patients, three-quarters with left atrial tumour, underwent right heart catheterization. An elevated P.C.V. and P.A. pressure was generally observed in cases of left atrial tumour, reflecting an impaired pulmonary venous return. Broad swings in the pressure levels, as described by Ellis,[19] were felt to be characteristic. These changes are probably due to varying positions of the tumour with respect to the A.V. orifice or pulmonary veins. Van Buckem[8] felt that an X dip followed by a tall V wave in the P.C.V. tracing was of diagnostic value. This tall V wave, similar to the regurgitant wave of mitral insufficiency, is particularly significant in the absence of other signs of mitral regurgitation. Ellis described a case with a P.C.V. pulse showing such a tall V wave but with a dye dilution curve that did not suggest mitral regurgitation. This P.C.V. pattern is thought to be due to the rapid filling and decreased capacity of the atrium. It may also be produced by true regurgitation in instances where the tumour interferes with valvular function. In two cases,[20, 25] direct left atrial pressures were recorded of an elevated pressure with tall C and V waves.

In right atrial myxomas, an elevated right atrial pressure with a diastolic gradient of tricuspid stenosis or with a tall tricuspid insufficiency wave has been described. These findings may vary considerably during the procedure, suggesting intermittent obstruction or a varying degree of regurgitation. Bahnson[34] observed in one case that the lowest point in atrial pressure in diastole occurred just before ventricular contraction, at which time the pressure should rise rather than decrease in true tricuspid stenosis. The atrial pressure curve may also assume a ventricular pattern, simulating Ebstein's disease, as noted by Coates and Drake.[28] This finding, we believe, may be due to severe tricuspid regurgitation or to a complete loss of valvular function, resulting in a common functional chamber, not unlike Ebstein's anomaly. A pattern not previously described, and observed in our Case 2, is the diastolic dip and plateau of the right ventricular tracing, the so-called "square root sign". We believe that this may be due to restriction of the ventricular diastolic volume by the tumour protruding through the tricuspid orifice and encroaching upon the ventricular cavity.

Coates and Drake[28] reported a case where cyanosis and peripheral desaturation varied with different body positions. The variable occlusion of the tricuspid valve by a tumour associated with an atrial septal defect explained the fluctuating right-to-left shunt. Similar cases were reported by Paquet[10] and Hanlon.[35] These hæmodynamic findings are not specific but may suggest that a condition other than valvular disease is present, and prompt further investigation with angiocardiography.

Value of Angiocardiography

Angiocardiography, well described by Steinberg,[9] is the only method of confirming the diagnosis. And yet, the filling defect so typical of a space-occupying tumour may also be due to massive thrombus, so that a definite diagnosis may be made only during cardiac surgery. Of the 57 left atrial myxomas, 30 were autopsy discoveries, four were said to have been suspected clinically and 17 were discovered during operation mistakenly performed for mitral stenosis. Only six were definitely diagnosed by angiocardiography. In all right-atrial tumours, except two, the diagnosis was confirmed by angiocardiography. Two right and one left atrial myxomas had been proved by this technique before January 1954, giving a total of 15 clearly demonstrated by this method.

Incidence of Operation

In this five-year period, 17 atrial myxomas were successfully resected. There have been at least 25 reported attempts. Crafoord[36] was the first to resect a left atrial tumour using extracorporeal circulation, while Bigelow[37] was the first to remove such a tumour successfully under hypothermia. Nichols's[38] patient, operated on mistakenly for mitral stenosis, survived partial excision without

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having the benefit of planned surgery (not included in the operated group). Two others[32, 39] were operated on by the closed-heart method and one survived. Of the 18 cases accidentally operated on for mitral stenosis or tricuspid stenosis, eight had the benefit of subsequent planned surgery under hypothermia or extracorporeal circulation, and five survived. Of the 17 patients operated on for left atrial myxomas, 11 survived, whereas six of the eight patients with right atrial tumours had successful resection. Hypothermia was used in 13 patients with eight survivals (60%), and 10 were performed with the aid of cardiopulmonary bypass, with eight successful resections (80%).

DISCUSSION

The diagnosis of left atrial myxoma is indeed difficult. Although approximately half of the 57 cases studied were diagnosed during life, only ten (17%) were so diagnosed clinically. Many such patients will undoubtedly continue to be operated on erroneously for mitral stenosis. It appears to us that emboli without atrial fibrillation is the most frequent suspicious finding (42% in our series); this has not been sufficiently stressed in the literature. Postural symptoms and variable murmurs occurred in one-fifth of the cases but it appears that these findings were not considered too seriously. In fact, their significance was frequently recognized only after the tumour had been found at operation or at autopsy. Morton,[24] in a study of 30 cases of left atrial myxomas described adequately in the literature from 1919 to 1953, reported syncope and postural symptoms in 33 and 20% of the cases respectively. Gleason[3] found symptoms and signs varying with posture in only two out of 23 cases. Another syndrome highly suggestive of left atrial tumour that has not been adequately recognized, is pulmonary hypertension of unknown etiology. In this series, half of these cases were suspected clinically. However, in the absence of the ball-valve occlusion syndrome, the diagnosis rests on keeping a high index of suspicion and an awareness of its possibility in mind in all atypical cases of mitral stenosis and in pulmonary hypertension without obvious left heart disease. The following atypical features should arouse suspicion: (1) late onset of illness; (2) a rapidly progressing course; (3) cerebral and peripheral emboli without atrial fibrillation, particularly in the absence of demonstrable heart disease; (4) unexplained paroxysmal symptoms, such as dyspnœa, syncope and tachycardia, in a patient with normal exercise tolerance; (5) postural symptoms; (6) absence of orthopnœa in a patient with moderate to severe exertional dyspnœa, or dyspnœa relieved by lying down; (7) the appearance of heart murmurs in a previously silent heart; (8) murmurs varying in time and with posture; (9) regular sinus rhythm in a severely incapacitated patient when atrial fibrillation would be expected; (10) absent or slight left atrial enlargement in the presence of severe pulmonary congestion; (11) systolic expansions of the left atrium on fluoroscopy, and (12) broad swings in the pulmonary wedge pressure on cardiac catheterization.

The diagnosis of right atrial myxoma, in contrast, appears less puzzling, as suggested by the fact that all except one in this series were diagnosed clinically. In fact, signs of isolated tricuspid disease, which is frequently present, are almost pathognomonic of such tumours. Ebstein's anomaly, which may be simulated quite closely, including the electrocardiographic and cardiac catheterization findings, should also arouse suspicion.

The correct diagnosis was suspected in our Case 1, but only during her second admission when a loud systolic apical murmur was heard. The initial impression of myxœdema heart disease was never satisfactory but no other suitable diagnosis could be found. The severe pulmonary hypertension, believed by one observer to be due to repeated pulmonary emboli, and the predominant right-sided failure were easily explained by left atrial tumour. Unfortunately she died before she was considered well enough to tolerate selective angiocardiography.

In Case 2, the correct diagnosis should have been suspected, since it simulated tricuspid stenosis. On the other hand, we were impressed by the right ventricle pressure curve which gave a "square root sign". This pattern has been described in constrictive pericarditis and subendocardial fibroelastosis.[40] White[41] has reported a case of pericarditis with selective constriction of the auriculoventricular orifice resulting in functional stenosis. Fibro-elastosis may also be associated with valvular lesions.[42] For these reasons, we believed that tricuspid stenosis due to selective constrictive pericarditis or associated with subendocardial fibro-elastosis was a definite possibility. This specific pressure pattern, to our knowledge, has never been described in cases of right atrial tumour. We suggest that the restriction of the right ventricular cavity by the protruding tumour and the marked dilatation of this cavity were responsible.

In conclusion, this study merely points out again that the clinical diagnosis of atrial myxoma is possible and should always be kept in mind, since the removal of these tumours is now feasible and frequently life-saving.

SUMMARY

Two cases of atrial myxomas are reported with a study of 63 cases collected from the literature between 1954 and 1959. The symptoms, signs, hæmodynamic data and clinical course are reviewed. Over half were diagnosed during life by angiocardiography or operation for presumed mitral stenosis, and 26% of the tumours were successfully removed with the aid of hypothermia or cardio-pulmonary by-pass technique.

We are grateful to Drs. Jean de L. Migneault, Edward Gagnon and F. Henry Ellis and associates for permission to report Case 2.

ADDENDUM

Since this paper was written, an excellent review of the literature and report of a right atrial myxoma diagnosed preoperatively and successfully treated has appeared in the *Canadian Journal of Surgery* (Padhi *et al.*: *Canad. J. Surg.*, 2: 414, 1959).

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RÉSUMÉ

Deux observations cliniques de myxome sont présentées. Une tumeur auriculaire droite fut découverte au cours d'une intervention pour une prétendue sténose tricuspidienne; cette tumeur fut enlevée avec succès par la suite. La présence d'un myxome auriculaire gauche, soupçonnée cliniquement, fut confirmée à l'autopsie d'une malade de 63 ans. Nous avons retrouvé 63 observations de myxome publiées durant les cinq dernières années. Les auteurs présentent une analyse détaillée des manifestations cliniques, des examens spécialisés et de l'évolution de 45 cas adéquatement décrits de myxome auriculaire gauche et huit de myxome auriculaire droit. Cette étude souligne encore les difficultés que présente le diagnostic du myxome surtout si la tumeur siège dans l'oreillette gauche. En effet, seulement 17% des cas étudiés furent soupçonnés cliniquement. Ils ont simulé la plupart du temps une sténose mitrale et plus rarement une hypertension pulmonaire d'étiologie inconnue. Les embolies périphériques et cérébrales en l'absence d'une fibrillation auriculaire ainsi que les symptômes déclenchés par les changements de position et l'auscultation variable se sont avérés des indices fort suggestifs. Par contre, le diagnostic du myxome auriculaire droit est beaucoup plus facile; en effet, sept des huit cas rapportés ont été découverts cliniquement. Avec l'aide de l'hypothermie ou de la circulation extra-corporelle, 26% de ces tumeurs auriculaires ont été réséquées sans encombre. Cette étude démontre que les myxomes du cœur, quoique rares et difficiles à reconnaître cliniquement, doivent continuer à stimuler l'intérêt du clinicien, maintenant que l'angiocardiographie peut les démontrer avec certitude et que le chirurgien peut les enlever avec succès. L.C.

SPECIAL ARTICLE

THE EXECUTIVE HEALTH PLAN OF THE ROYAL VICTORIA HOSPITAL

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THE HEALTH of the executives in our Canadian industries is now awakening an interest on the part of the medical profession, which until recently viewed with little concern the annual trek to the American clinics of a large segment of management personnel. It is becoming more and more evident that as our country becomes larger and more industrialized the problem of the health of the executives of corporations, large or small, will be increasingly thrust on to the shoulders of the medical profession throughout the country. In this article I am going to attempt to bring into focus for the Canadian doctor viewpoints which prevail in our country with reference to this program, some of the opinions of management and the industrial medical officer, some of the problems facing the medical profession in the choice of the medical examiner, the examination and its medico-legal aspects, as well as the various opinions about the results of these examinations and, finally, an outline of our policies in the Royal Victoria Hospital and our methods for meeting this need in our community.

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