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DIAGNOSTIC TECHNIQUES

Right Atrial Thromboemboli: Clinical, Echocardiographic and Pathophysiologic Manifestations

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In six patients with clinically unsuspected right atrial thromboemboli the diagnosis was made with two-dimensional echocardiography. Five patients had pulmonary emboli, and one had systemic embolization. Three patients had congestive cardiomyopathy, two with tricuspid regurgitation; of the remaining three, one had cor pulmonale complicated by tricuspid regurgitation, one had thrombophlebitis and one had no discernible cardiac illness. Four patients had dizziness or syncope, four had dyspnea, three had chest pain, three had hypotension and two had cyanosis. Five patients were treated with thrombolytic or anticoagulant therapy, or a combination of the two. In three patients, surgical removal of the thrombus was undertaken because of recurrent pulmonary emboli or tricuspid regurgitation, or both, and progressive right heart failure. The thromboemboli were removed in all three, but one patient died.

On two-dimensional echocardiography, four of the six patients' thromboemboli were snake-like, unattached to the right atrium and prolapsed freely across the tricuspid valve into the right ventricle in diastole and back into the right atrium in systole. The other two patients' thromboemboli were attached to the right atrium and did not prolapse across the tricuspid valve.

Our cases, together with a review of other reports, suggest that right atrial thromboemboli: 1) can be accurately diagnosed by two-dimensional echocardiography; and 2) result from two different pathophysiologic mechanisms developing a) in situ, either on a foreign body or secondary to reduced cardiac output, or b) as a result of an embolus from systemic vein thromboses.

The awareness of the clinical significance of right atrial thromboembolism is a direct consequence of the use of two-dimensional echocardiography. There have been several reported cases (1–9) diagnosed by two-dimensional echocardiography. Before two-dimensional echocardiography, however, the diagnosis was generally made only at autopsy (10–13). Over the past 10 months we have seen six patients with clinically unsuspected right atrial thromboemboli diagnosed by two-dimensional echocardiography. A brief description of our cases and a review of previous reports on this unusual subject will show that there appear to be two varieties of right atrial thromboemboli with different clinical manifestations and different echocardiographic appearances.

Methods

Study patients. Analysis of the clinical, echocardiographic and surgical or pathologic data from six patients with right atrial thromboemboli form the basis of this report. Four of the patients were from Emory University Affiliated Hospitals, one from St. Joseph's Hospital and one from Georgia Baptist Hospital.

Echocardiography. All patients were studied by using two-dimensional echocardiography with recording of parasternal long- and short-axis views, apical four and two chamber views and subcostal four chamber and short-axis views. All M-mode studies were derived from the two-dimensional image. Alterations in gain and reject settings were employed to optimally define the margins of the right atrial mass and the right atrial wall.

Results

Clinical data (Table 1). Six patients, four men and two women with an age range of 16 to 60 years, had a right atrial thromboembolus detected by two-dimensional echocardiography. Their clinical presentation usually suggested a pulmonary embolic event or a low cardiac output state, or both. Five patients had pulmonary emboli, and one had systemic embolization. Three patients had congestive cardiomyopathy, complicated in two by tricuspid regurgitation,

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Table 1. Clinical Findings in Six Patients With a Right Atrial Thromboembolus

Case	Age (yr) & Sex	Cardiovascular Conditions	Associated Lesions	Presenting Symptoms	Pertinent Physical Findings	Emboli
1	16M	None found	None found	Chest pain, cyanosis, hemoptysis	BP < 90 mm Hg; S_3 gallop; loud P_2	Pulmonary (lung scan and pulmonary angiogram; multiple large defects)
2	44M	CCM; chronic RHF with acute decompensation and new TR; hypertension	New embolic CVA; chronic alcoholism	Weakness, confusion	BP 170/100 mm Hg; atrial fibrillation; expressive aphasia; hemiparesis; peripheral edema	Cerebral
3	57M	Cor pulmonale and TR (chronic)	Bronchogenic CA; nephrotic syndrome	Dyspnea, cyanosis, dizziness, edema	BP 90/60 mm Hg; rales; pleuropericardial friction rub; anasarca	Pulmonary (lung scan: large segmental defect)
4	50F	CCM; chronic RHF with acute decompensation and new TR; hypertension; old MI	Recent CVA	Chest pain, dyspnea, near syncope	BP 100/80 mm Hg; prominent jugular venous "CV" waves; mitral regurgitation; pulsatile liver; marked peripheral edema	Cerebral and pulmonary (lung scan: multiple large defects)
5	60F	CCM; hypertension	Lymphoma; bilateral BK amputee	Dyspnea, syncope	BP 90/70 mm Hg; rales; sustained apical impulse; S_3 gallop	Pulmonary (chest X-ray: multiple infiltrates; Po_2 38)
6	38M	Probable thrombophlebitis	Pancreatitis; recent complicated cholecystectomy	Chest pain, syncope, dyspnea, calf pain	BP 100/70 mm Hg; prominent jugular venous *A* wave	Pulmonary (lung scan: multiple large defects)

BK = below knee; BP = blood pressure; CA = carcinoma; CCM = congestive cardiomyopathy; CVA = cerebrovascular accident; MI = myocardial infarction; Po_2 = partial pressure of oxygen; RHF = right heart failure; TR = tricuspid regurgitation.

one had cor pulmonale complicated by tricuspid regurgitation, one had thrombophlebitis and the remaining patient had no discernible cardiovascular disease. The presenting complaints included syncope or dizziness in four patients, dyspnea in four and chest pain in three. Hypotension was present in three patients and cyanosis in two.

Five of the six patients were treated for pulmonary emboli with thrombolytic or anticoagulant therapy, or both. In three of these patients, the right atrial thrombus was surgically removed. The operation was performed in two of the three because of the sudden onset of severe hypotension in the presence of recurrent massive pulmonary emboli despite treatment with heparin or urokinase or both; in the third patient, it was performed because of progressive right heart failure with severe acute tricuspid regurgitation. One of the three surgically treated patients died of progressive right heart failure. The three medically treated patients survived for at least 6 months of follow-up.

Echocardiographic findings (Table 2). Each right atrial thromboembolus was imaged in at least two standard two-dimensional echocardiographic views and was also identified on derived M-mode studies of the tricuspid valve. All were acoustically homogeneous. In four of the six patients (Cases 1, 2, 4 and 6) the thromboembolus was very long, snake-like, extremely mobile and prolapsed across the tricuspid valve (Fig. 1 and 2). In the other two patients (Cases 3 and 5) the thromboembolus was shorter in length, did not prolapse across the tricuspid valve and was attached to the lower lateral right atrial wall by a broad-based stalk in one (Fig. 3) and a narrower stalk in the other (Fig. 4). The four elongated, fusiform clots occupied much of the right atrium, assumed a variety of forms, occasionally curling or corkscrewing either in the right atrium or as they prolapsed along or across the tricuspid valve into the right ventricle. In each of these four patients (Cases 1, 2, 4 and 6), the clots had no visible stalk or point of attachment to the right atrial

Table 2. Echocardiographic Findings, Therapy and Pathologic Features of Study Group

Case	Echocardiographic Findings	Therapy	Surgical/Pathologic Correlations	Outcome
1	Dilated RA and RV. Mobile right atrial mass prolapses across TV and is partly embedded in TV apparatus. Multiple masses entwined in RV trabeculations.	Thromboembolectomy (RA, RV and PA) because of sudden hypotension and cyanosis while taking heparin and urokinase. Also IVC ligation and closure of F. ovale.	1st operation: multiple clots removed from RA and PA; largest clot 13 cm long, "appeared to have originated from a femoral vein" (Fig. 6). 2nd operation: removed clots that extended into IVC and SVC; no attachment point found; clot projected into F. ovale. Histologic examination: layering of fibrin, platelets and red blood cells with leukocytes, compatable with thromboembolic material; no evidence of myxoma.	Discharged on Coumadin
2	Four chamber dilation. Multiple grape-like masses in RA prolapse across TV.	Heparin and Coumadin.	None.	Discharged on Coumadin.
3	Markedly dilated RA and RV with paradoxical septal motion. Large pleural and pericardial effusion. Long finger-like right atrial mass attached to posterior RA wall by a narrow stalk.	None.	None.	Recurrent hospital admissions; alive at 6 month follow-up.
4	Markedly dilated RA and RV and moderately dilated LA and LV. Mobile right atrial mass prolapses across TV and is partially embedded in TV apparatus.	Thromboembolectomy (RA) because of acute RHF and TR while taking heparin.	Removed a 32 cm long right atrial clot that was entangled in chordae tendineae of TV; "clot had early divisions similar to systemic veins." Histologic examination: focal, early scattered marrow particles that represent degenerated entrapped blood elements.	Died at surgery due to progressive RHF.
5	Four chamber dilation with massive RA. Small pericardial effusion. Long finger-like right atrial mass attached to posterior right atrial wall by broad base.	Heparin and Coumadin.	None.	Discharged on Coumadin.
6	Dilated RV and markedly dilated RA. Mobile mass in RA prolapses across TV.	Thromboembolectomy (RA and PA) because of marked respiratory distress while taking heparin. Also IVC interruption.	Removed multiple PA clots (longest 18 cm) and a 25 cm long RA clot that straddled TV; the right atrial clot had "divisions similar to iliac veins."	Discharged on Coumadin.

F. ovale = foramen ovale; IVC = inferior vena cava; LA = left atrium; LV = left ventricle; PA = pulmonary artery; RA = right atrium; RV = right ventricle; SVC = superior vena cava; TV = tricuspid valve; other abbreviations as in Table 1.

wall, interatrial septum, tricuspid valve or inferior vena cava. In two of these four patients (Cases 1 and 4), the clots appeared to be entangled in the tricuspid valve apparatus and in one of them thromboembolic material was also present in the right ventricle. Dense, shaggy echoes in the right atrium adjacent to the posterior leaflet of the tricuspid valves were seen on each of the M-mode studies (Fig. 5).

At surgery, all three clots were extremely large with adherence to, or entangled within, the chordae tendineae of the tricuspid apparatus. In two patients (Cases 1 and 6), the clots were described as "appearing to have originated from a femoral vein, with definite evidence of tributaries" (Fig. 6). Gross pathologic inspection of the thromboembolic fragments suggested that they were organized and probably came from the femoral veins. Microscopic examination revealed layering of fibrin, platelets and red blood cells with leukocytes extending among them, compatible with thromboembolic phenomena. There was no histologic evidence of myxomatous tissue.

Discussion

Previous reports of right atrial thromboemboli. Although they have been recognized for a long time, the occurrence of right atrial thromboemboli is rare. In their

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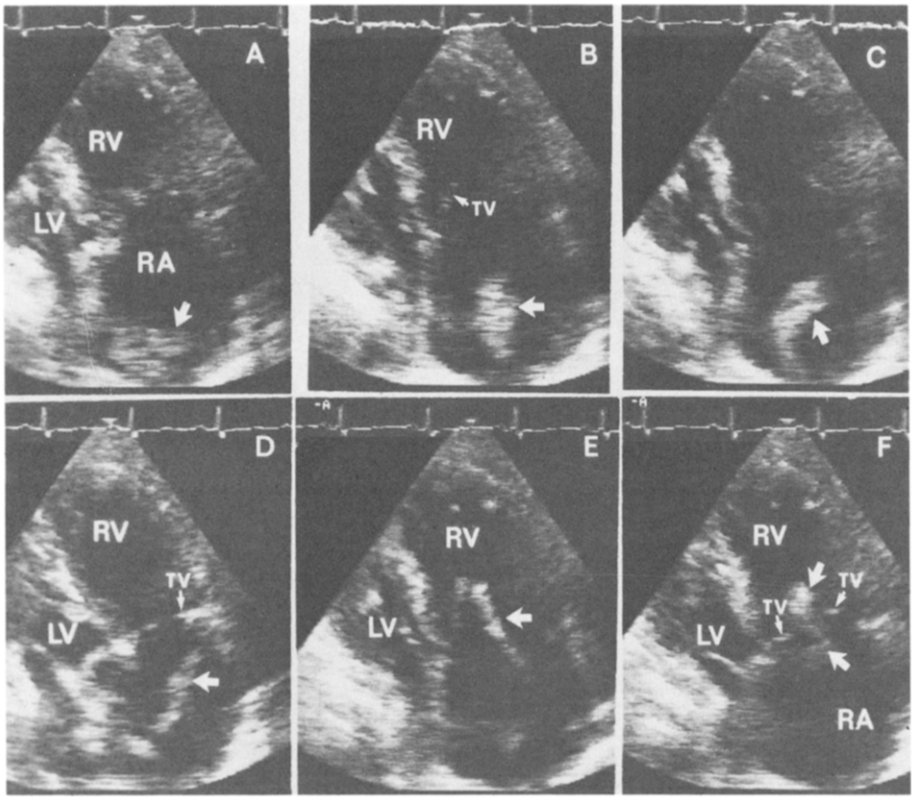
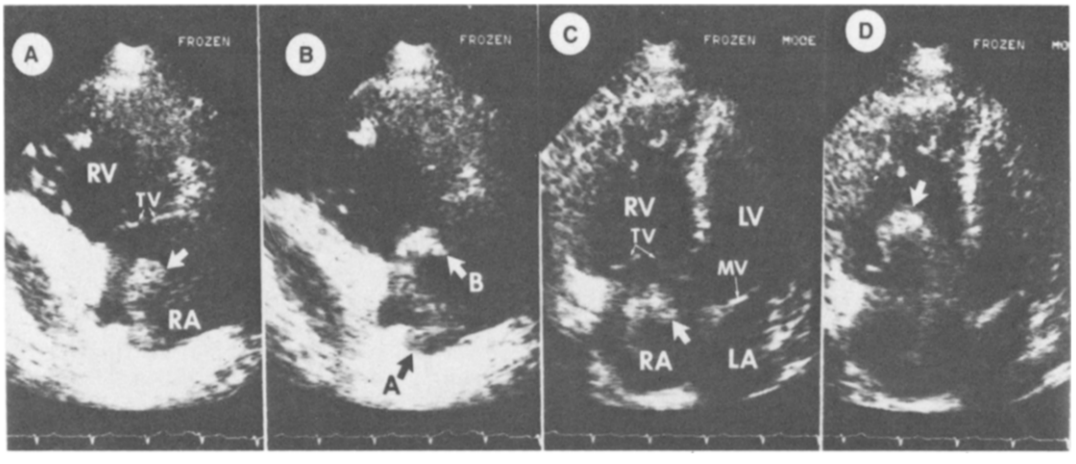


Figure 1. Case 4. Two-dimensional sequential frames of the parasternal long-axis right ventricular inflow tract view. A long, serpentine-like right atrial (RA) thromboembolus (**arrow**) moves from its coiled position in the posterior portion of the right atrium (**A**), toward the tricuspid valve (TV) (**B,C,D**) and into the right ventricle (RV) (**E**). In **F**, the thromboembolus twists through the tricuspid valve with its distal end in the right ventricle (**top arrow**) and its proximal end in the right atrium (**bottom arrow**).

review of more than 2,000 autopsies, Wartman and Hellerstein (14) found only 14 cases of right atrial thromboembolus and 4 cases of right ventricular thromboembolus. Right atrial thromboemboli have been even less frequently recognized antemortem. In our review of previous reports (1–9,15–17), we found only a few patients with right atrial thromboemboli that were suspected during life and that were not associated with a foreign body in the right atrium. Table 3 shows 15 reported cases of right atrial thromboemboli described as mobile, because they prolapsed across or were embedded in the tricuspid valve apparatus. Since 1976, all but 1 of these 15 thromboemboli were diagnosed by echocardiography and 11 were confirmed pathologically. Similar cases (18–30) of right ventricular thromboemboli documented by echocardiography are listed in Table 4.

Table 5 lists 10 of the more recently described cases of

Figure 2. Case 6. Two-dimensional images of the parasternal right atrial (RA)-right ventricular (RV) view (**A,B**) and the apical four chamber view (**C,D**). **A**, The thromboembolus (**arrow**) is coiled in the right atrium. **B**, The mobile thromboembolus has uncoiled and its proximal end is located at the level of the lateral right atrial wall (**arrow A**), while its distal end is at the level of the tricuspid valve (TV) (**arrow B**). **C**, The thromboembolus (**arrow**) is completely contained within the right atrium. **D**, The thromboembolus (**arrow**) has prolapsed across the valve with the distal end curled in the right ventricle. LV = left ventricle; MV = mitral valve.



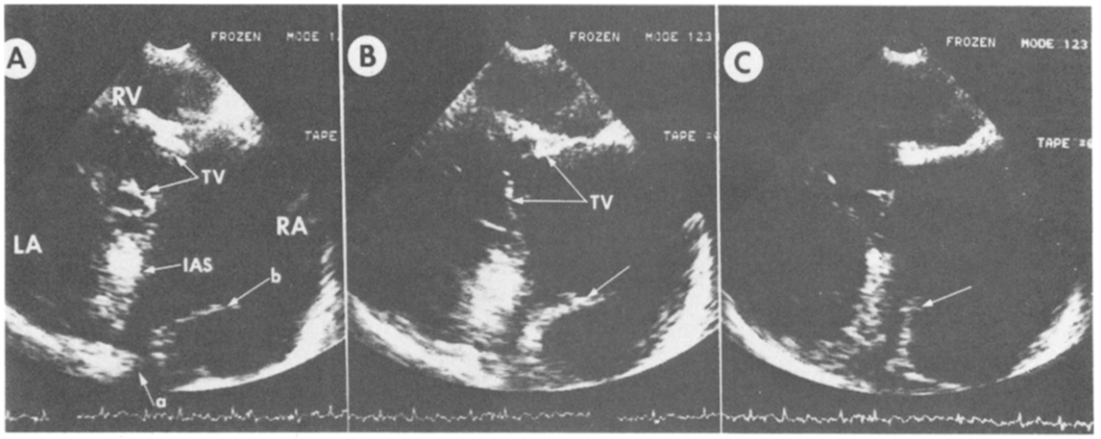


Figure 3. Case 5. Two-dimensional sequential frames of the parasternal right atrial (RA)-right ventricular (RV) view. A curvilinear thromboembolus is attached to the posteromedial right atrial wall by a broad-based stalk (**arrow a**). It is 3 cm long and does not prolapse across the tricuspid valve (TV), but its tip (**arrow b**) whips back and forth in the right atrium (**A,B,C**). IAS = interatrial septum; LA = left atrium.

nonprolapsing right atrial thromboemboli that developed in situ, forming on a foreign body (for example, central venous catheter, pacing wire, and so forth) or adhering to the atrial wall secondary to stasis of blood flow. Seven were diagnosed by two-dimensional echocardiography and all but two were confirmed pathologically.

Clinical sequelae of right atrial thromboemboli. Patients with right atrial thromboemboli frequently have a history of congestive heart failure, pelvic or femoral vein thrombophlebitis, or both, and frequently develop acute pulmonary embolism. They may present with abrupt onset of syncope, hypotension, dyspnea, chest pain and evidence of massive or recurrent pulmonary emboli or tricuspid regurgitation. In our series, five of the six patients had multiple pulmonary emboli, some with massive obstruction of the pulmonary artery resulting in acute pulmonary hypertension, cor pulmonale and right heart failure. Baum and Fisher (31) found 19 patients with right heart thromboemboli in their 1,135 necropsies, 9 of whom manifested with pulmonary emboli and cor pulmonale.

Three of our patients had tricuspid regurgitation; in two it appeared to have been acute and in at least one it presumably developed secondary to a clot that was found at surgery to be lodged between the chordae tendineae of the tricuspid valve, preventing it from closing. Large, coiled thromboemboli have been reported (10,11,16) to lodge in the tricuspid valve apparatus and to result in tricuspid regurgitation or sudden cardiac death. They have also been reported to have been trapped in anomalies of the venous

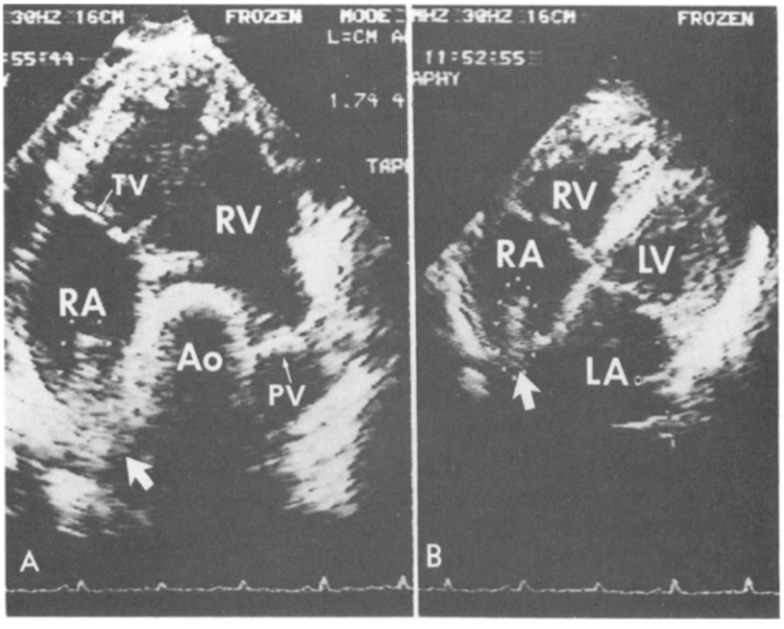


Figure 4. Case 3. Two-dimensional images of the subcostal short-axis (**A**) and four chamber (**B**) views. **A**, A finger-like thromboembolus (**circled**) measuring 1.74 cm in length is attached to the junction of the posterior right atrial wall and interatrial septum (**arrow**) and projects into the right atrium (RA). **B**, The thromboembolus has a narrow base as it emerges from its attachment point in the posterior right atrium (**arrow**). It does not prolapse across the tricuspid valve (TV). Ao = aorta; LA = left atrium; LV = left ventricle; PV = pulmonary valve; RV = right ventricle.

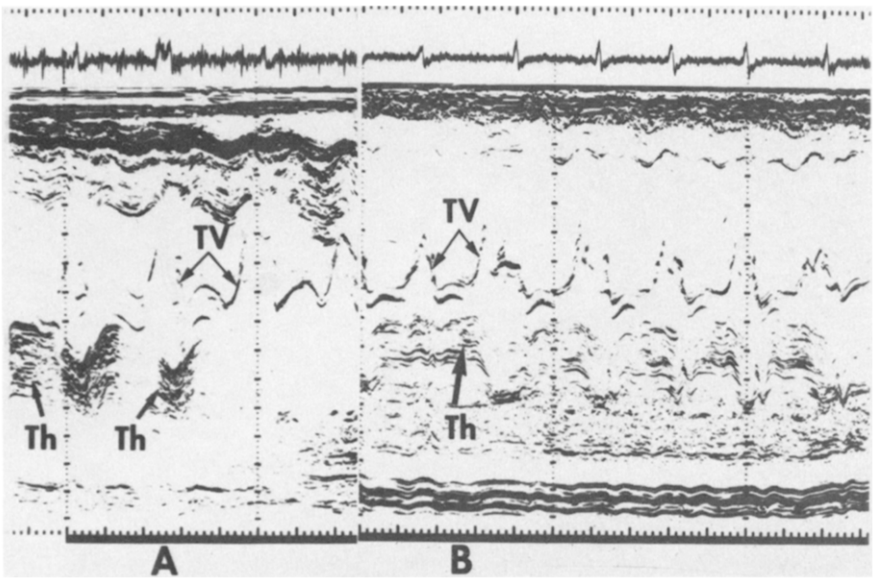


Figure 5. Case 3. M-mode echocardiogram, derived from the two-dimensional image. The cursor crosses the right ventricle, tricuspid valve (TV) and the mass of echoes in the right atrium representing a thromboembolus (Th). **A**, Dense mass of echoes on the atrial side of the tricuspid valve. **B**, The echoes from the thromboembolus appear to obscure the posterior tricuspid leaflet.

valves of the right atrium (including Chiari's network)(32) or to result in a superior vena caval syndrome (33). In addition, they have been reported (10,34) to occlude the pulmonary valve and to cause changing basilar systolic murmurs. Usually, however, most emboli pass rapidly through the right heart chambers and enter the pulmonary artery system. They may, however, embolize to the periphery if there is a patent foramen ovale (4,16). The absence of any true attachments of the thromboembolus to the tricuspid valve apparatus is against primary intracardiac formation. This type of obstruction of the tricuspid orifice differs from that described (35) in cases of right atrial "mass" thrombi or myxomas that, owing to their large size and peculiar location, impede blood flow by plunging through the tricuspid orifice. Cyanosis (Cases 1 and 3) may have also been due to occlusion of the tricuspid valve orifice by a clot causing right atrial hypertension with right to left shunting through a patent foramen ovale. This phenomenon has been previously described (10,34,35) in patients with right atrial thromboembolus, and is further evidence for the changing shape and extreme mobility of the thromboembolus.

Cause of right atrial thromboembolism. It was originally believed that most right atrial thromboemboli occurred in patients with dilation of the right atrium (36), low output syndrome (9) or foreign bodies in the right atrium (for example, central line catheters for total parenteral nutrition [23,24,28,30], ventriculoatrial shunts for hydrocephalus [28], transvenous pacing electrodes [29,37], Swan-Ganz catheters [38], or suture material [26]). The potential is present in any condition with endothelial damage, relative stasis of blood (that is, heart failure, atrial arrhythmias, and so forth), alteration in the coagulating mechanism of blood and an enlarged right atrium.

In our series, however, the original source of the right

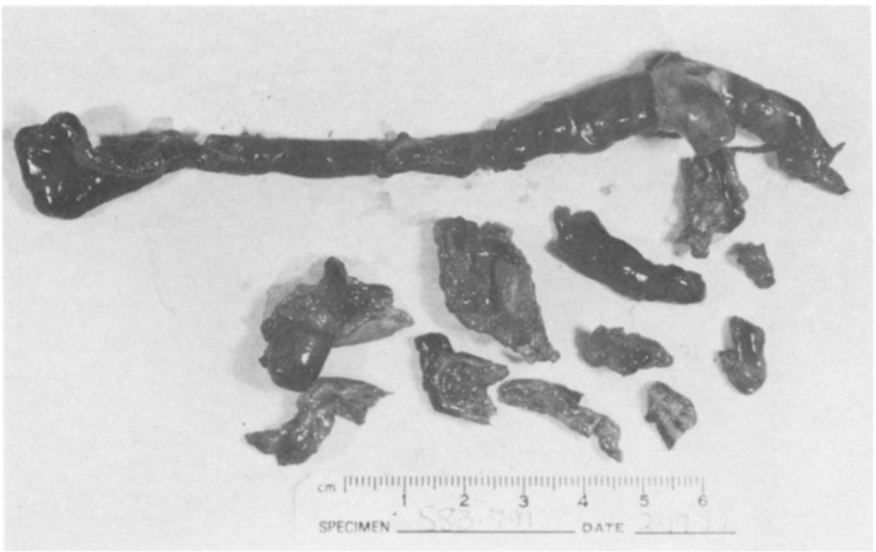


Figure 6. Case 1. Several fragments of organized thromboembolus removed at operation. The fragments were found in the right atrium, right ventricle and pulmonary artery. The longest fragment (13 cm in length) was removed from the pulmonary artery and shows indentations conforming to the contour of valves in the leg veins.

Table 3. Summary of 15 Previously Reported Cases of Mobile Right Atrial Thromboembolus

Reference	Year	Predisposing Condition	Tricuspid Regurgitation	Echo	Clot Description	Surgery	Sudden Death
Buckingham et al. (1)	1984	Femoral thrombophlebitis	Present	2-D	Prolapsing across TV	Successful	No
Reddish et al. (2)	1983	CCM	–	2-D	Prolapsing across TV	None	Yes
Ouyang et al. (3)	1983	CVA; prolonged bed rest	–	2-D, M-mode	Prolapsing across TV	None	No
Starkey et al. (4)	1982	CCM	–	2-D	Prolapsing across TV; wedged into F. ovale	None	Yes
Arvan et al. (5)	1982	Femoral thrombophlebitis	–	2-D, M-mode	Prolapsing across TV	None	No
Rosenzweig et al. (6)	1982	AMI and CHF	–	2-D	Prolapsing across TV	None	Yes
Oldershaw et al. (7)	1982	None	–	2-D, M-mode	Prolapsing across TV	Successful	No
Come (8)	1982	AMI and atrial fibrillation	–	2-D	Prolapsing across TV; wedged into F. ovale	None	No
Whitford et al. (15)	1982	Femoral thrombophletitis	–	M-mode	Prolapsing across TV	None	No
Covarrubias et al. (16)	1977	Probable thrombophlebitis	Present	M-mode	Prolapsing across TV; wedged into F. ovale	None	Yes
Johnson et al. (10)	1977	Femoral/pelvic thrombophlebitis	Present	–	Trapped in TV apparatus	None	Yes
Broadbent et al. (17)	1976	Pelvic thrombophlebitis	Present	M-mode	Extended from IVC and prolapsed across TV	Successful	No
Spencer et al. (11)	1971	Pelvic thrombophlebitis	Present	–	Trapped in TV apparatus	None	Yes
Goodman et al. (12)	1966	Femoral thrombophlebitis; cor pulmonale	–	–	Multilocular masses; some embedded in TV apparatus	None	Yes
Hudnut et al. (13)	1962	AMI and femoral thrombophlebitis	–	–	Trapped in TV apparatus	None	Yes

AMI = acute myocardial infarction; CCM = congestive cardiomyopathy; CHF = congestive heart failure; CVA = cerebrovascular accident; Echo = echocardiography; F. ovale = foramen ovale; IVC = inferior vena cava; 2-D = two-dimensional; TV = tricuspid valve.

atrial thromboembolus was probably a propagating thrombus in a leg or pelvic vein in three patients (Cases 1, 4 and 6); in two patients (Cases 2 and 3), the thromboembolus may have primarily developed in situ as a complication of relative stasis of blood flow (right heart failure and atrial fibrillation) and in Case 5 it probably developed as a combination of the two mechanisms. The femoral and pelvic veins are the primary source of right heart thromboemboli and the majority of pulmonary emboli, especially if they are large and cause acute cor pulmonale, even if no known precipitating cause, associated illness or cardiac disease is identified. Less than half of the patients with pulmonary emboli have clinical evidence of thrombophlebitis (39).

The original source of the thromboembolus in Case 1 was an embolus from a thrombus in a leg or pelvic vein. An embolus can also originate from renal veins, especially in conjunction with the nephrotic syndrome (40). The potential for a hypercoagulable state also existed in our patient with the nephrotic syndrome, since this lesion can cause urinary loss of protein (for example, antithrombin III) that is necessary to prevent and reverse clotting (41). It has also been reported (42) that patients with pancreatitis may exhibit decreased fibrinolysis, increased fibrogen concentration and increased tolerance to heparin and that these coagulation changes may predispose to deep-vein thrombosis. Since pathologic/anatomic correlations were not obtained in three of our six patients, including two with documented malignancy (lymphoma and bronchogenic carcinoma), the possiblity of a tumor being responsible for the right atrial mass in these patients cannot be excluded.

Echocardiography in patients with right atrial thromboemboli. Echocardiographic manifestations in our patients also suggest that there are two types of right atrial thromboemboli. Thromboemboli resulting from a peripheral venous thrombus that embolizes to the right atrium are serpiginous and freely mobile. They gradually enlarge, prolapse across the tricuspid valve and do not have an obvious attachment point to the atrial wall. They may become trapped in the chordae tendineae or papillary muscles of the tricuspid valve resulting in tricuspid regurgitation (10–13). Right atrial thrombi that develop in situ are considerably shorter and nonmobile. They usually form around a foreign body or

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Table 4. Summary of Six Previously Reported Cases of Right Ventricular Thromboemboli Diagnosed by Echocardiography

Reference	Year	Predisposing Condition	Tricuspid Regurgitation	Clot Description	Therapy	Survival
Patel et al. (18)	1983	Thrombophlebitis; right heart failure	Present	Multiple, mobile "rocks," some entangled in tricuspid apparatus	Surgery	No
Ouyang et al. (3)	1983	Thrombophlebitis	Present	Attached to moderator band	Heparin	Yes
Shiu et al. (19)	1983	Probable thrombophlebitis	–	Mobile	Heparin; surgery	Yes
Woolridge et al. (20)	1983	None found	–	Mobile (entangled in tricuspid apparatus)	Heparin	No
Stowers et al. (21)	1983	Right ventricular infarction	–	In situ development	Urokinase	Yes
Kessler et al. (22)	1981	Chest wall trauma	Present	Mobile (entwined about moderator band)	Surgery	Yes

adhere to the right atrial wall and tend not to prolapse across the tricuspid valve.

Before the development of echocardiography, only right heart catheterization or angiography was available for the antemortem diagnosis of right atrial thromboemboli. This technique, however, may result in dislodgment of the clot with subsequent embolization (28). The M-mode echocardiogram has not been very helpful, since the right atrium is very difficult to visualize and a right atrial mass cannot be reliably detected unless it is large and prolapses into the right ventricle through the tricuspid valve orifice. M-mode echocardiographic descriptions of right atrial thromboemboli are therefore rare (15–17). In separate case reports, however, Covarrubias et al. (16) and Broadbent et al. (17) correctly attributed the multiple, shaggy echoes adjacent to the tricuspid valve to right atrial thrombi that were entrapped near the tricuspid valve in their patients with sudden hypotension, multiple systolic clicks and a systolic murmur at the lower left sternal edge. Our M-mode studies are very similar to their descriptions. Before two-dimensional echocardiography, however, these echoes adjacent to the tricuspid valve were apparently dismissed as artifacts and thus the M-mode clue to the presence of a right atrial thromboembolus was overlooked.

Two-dimensional echocardiography is clearly superior to M-mode echocardiography and probably superior to angiography in identifying intracardiac masses because it is safer and provides imaging of most of the right atrium, right ventricle, interatrial septum, superior and inferior venae cavae and the tricuspid valve. Its ability to visualize these areas, which are inaccessible to study with other noninvasive techniques, with multiple tomographic sections of the heart from a variety of planes make it a highly useful, if not the preferred technique, in identifying right atrial masses. In addition, it can provide information on the size, shape and mobility of intracardiac thromboemboli, differentiate an atrial from a ventricular mass and differentiate an intracardiac from an extracardiac tumor.

The patients with a right atrial thromboembolus in our series represent only a very small percent of patients studied echocardiographically who have systemic vein thrombosis, low cardiac output syndrome, dilated right atrium or intracardiac foreign bodies. The reason that right atrial thromboemboli are not more frequently seen by echocardiography is that the majority of the thromboemboli originating in systemic veins pass rapidly through the right atrium and lodge in the pulmonary arteries. Other reasons why the two-dimensional examination may "miss" a right atrial thromboembolus are technical: 1) Thromboemboli may be located in portions of the right atrium not well seen; 2) small thrombi may exceed the current resolution capabilities of this technique; and 3) the technique may be unable to differentiate nonmobile, flat thrombi from the right atrial wall. Previously, it was thought that biologic features of thrombi (for example, the acoustic impedance of recently clotted blood being similar to that of surrounding blood or endocardium [43]) may also limit their detection by echocardiography. Mikell et al. (44), however, showed experimentally that tissue acoustic properties of recently formed thrombi are not a primary limitation to their echocardiographic detection.

Echocardiographic differentiation of intracardiac masses. Echocardiographically, all cardiac thrombi are not identical. A *left atrial thrombus*, for example, is a nonhomogeneous, immobile mass with a broad base of attachment to the atrial wall and a distinct intracavitary margin (45). A *right atrial thromboembolus*, however, may be: 1) a mobile, serpentine-like structure that prolapses through the tricuspid valve and lacks any apparent attachment to the endocardium; 2) a finger-like projection attached to the atrial wall by only a narrow stalk; or 3) superimposed on a foreign body. The difference between the physical characteristics of these left and right atrial masses is that right atrial thromboemboli frequently do not originate from the atrium itself, but rather have embolized from the deep venous system and resemble casts of these veins. This was apparent in our three surgical specimens. Because of this characteristic a major echocardiographic problem is the differentiation between a mobile right atrial thromboembolus and other right atrial

Table 5. Summary of 10 Recently Reported Cases of Nonmobile Right Atrial Thromboemboli

Reference	Year	Predisposing Condition	Echo	Clot Description	Confirmation
Manno et al. (9)	1983	Amyloid CM and CHF	2-D	Broad-based mass	Autopsy
Manno et al. (9)	1983	Post-ASD repair	2-D	Irregular mass	Angiocardiography
Delaplane et al. (23)	1982	RA catheter for TPN (infant)	2-D	Attached to catheter	None
Riggs et al. (24)	1981	RA catheter for TPN; CHF (infant)	2-D	Attached to catheter	Catheter specimen
Mahoney et al. (25)	1981	RA catheter for TPN (infant)	2-D/Mode	Large masses filling RA and RV	Autopsy
Formolo et al. (26)	1981	Atrial flutter; CABG with atriotomy sutures	–	Attached to suture material	Autopsy
Schmaltz et al. (27)	1980	V-A shunt catheter	M-mode	Echoes beneath TV	Autopsy
Pliam et al. (28)	1979	RA catheter for TPN (infant)	M-mode	Ball-valve thrombus attached to catheter	Surgical specimen
Kinney et al. (29)	1979	Transvenous pacemaker wire	–	Attached to pacemaker wire	Surgical specimen
Wesley et al. (30)	1978	RA catheter for TPN (infant)	–	Attached to catheter	Catheter specimen

ASD = atrial septal defect; CABG = coronary artery bypass graft; CHF = congestive heart failure; CM = cardiomyopathy; Echo = echocardiography; RA = right atrial; RV = right ventricle; TPN = total parenteral nutrition; 2-D = two-dimensional; TV = tricuspid valve; V-A = ventriculoatrial.

masses (46,47). Metastatic tumor, sarcoma and Wilms' tumor, for example, commonly extend through venous channels and present as a mass in the right atrium with occlusion of the inferior vena cava; they can be identified by imaging the inferior vena cava with two-dimensional subcostal views. Atrial myxomas are large ovoid masses that occur in the setting of a normal-sized right atrium, and usually arise from the interatrial septum and, therefore, often present a much different two-dimensional image from that of thrombus. Tricuspid valve vegetations may simulate a right atrial mass, but their movement in concert with valve opening and closure helps differentiate them from thrombi (45). Right heart catheters and pacing wires may also simulate right atrial masses, but their echocardiographic appearance is usually quite distinctive (48).

To exclude the possibility of an artifact one should identify the right atrial thromboembolus on several views with proper gain settings. If the attenuation is too high, the mass may not be detected, whereas excessively low attenuation may lead to false positive diagnosis of an intracavitary mass. To minimize false positive diagnoses, one should set the gain attenuation as high as possible while still allowing visualization of normal structures and endocardial cavitary interfaces. Interpretive errors can also be caused by structures outside the main axis of the ultrasound beam that lead to spurious echoes. Reverberation artifacts may also be present in the atria or ventricles. These artifacts frequently extend across normal tissue interfaces and are positional in nature; they are not consistently imaged in multiple transducer planes. In contrast, an intracavitary thrombus, such as those encountered in our patients, can be imaged in multiple planes and is not subject to slight alterations in transducer angulation or position.

Normal structures within the right atrium might also lead to diagnostic errors. Experience in identifying the location and mobility of several other right atrial structures, such as an aneurysm of the atrial septum associated with an atrial septal defect, dilated coronary sinus, Chiari's network or eustachian valves, should help distinguish between these structures and the thicker right atrial clots (32,45). In our series, the apical and subcostal four chamber views and occasionally the parasternal short-axis plane at the level of the aortic valve (which allows visualization of both the right and the left atrium as well as the interatrial septum) and the parasternal long-axis view of the right ventricular inflow tract were the most helpful. The finding of accompanying abnormalities such as congestive cardiomyopathy and right atrial enlargement somewhat support the possible diagnosis of right atrial thromboembolus. The length and serpentine-like motion of the prolapsing right atrial clots in our series make them unlike other masses we have seen reported.

Therapeutic considerations. Given our limited experience with right atrial thromboemboli, it is difficult to determine whether the presence of a right atrial thromboembolus should be regarded as an indication for surgical rather than medical treatment. Surgical removal was performed in three of our six patients after thrombolytic or anticoagulant therapy, or both, was started because of recurrent massive pulmonary emboli and the presence of sudden hypotension or progressive right heart failure with acute tricuspid regurgitation; one patient died. Anticoagulant therapy alone was prescribed in two of the three medically treated patients, all of whom survived for at least 6 months.

Clinical implications. Right atrial thromboemboli appear to result from two different pathophysiologic mechanisms: 1) development in situ (on a foreign body in the right atrium or secondary to reduced cardiac output); or 2) as a result of an embolus to the right atrium from systemic vein thrombosis. Two-dimensional echocardiography has been shown to be a highly useful, if not the preferred technique for the diagnosis of right atrial thromboemboli. In addition, it can differentiate the serpiginous, freely mobile throm-

boemboli that result from peripheral vein thrombosis from those nonmobile thromboemboli that probably develop in situ. Since prolapsing right atrial thromboemboli predispose to massive or recurrent pulmonary emboli and may cause tricuspid regurgitation or unexplained right heart failure, patients with these findings should have two-dimensional echocardiograms before and after therapy is begun.

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