Free-Floating Thrombi in the Right Heart

Diagnosis, Management, and Prognostic Indexes in 38 Consecutive Patients

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Background—Floating right heart thrombi (FRHTS) are a rare phenomenon, encountered almost exclusively in patients with suspected or proven pulmonary embolism and diagnosed by transthoracic echocardiography. Their management remains controversial.

Methods and Results—We report on a series of 38 consecutive patients encountered over the past 12 years. Thirty-two patients were in NYHA class IV, 20 in cardiogenic shock. Echocardiography usually demonstrated signs of cor pulmonale: right ventricular overload (91.7% of the population), paradoxical interventricular septal motion (75%), and pulmonary hypertension (86.1%). The thrombus was typically wormlike (36 of 38 patients). It extended from the left atrium through a patent foramen ovale in 4 patients. Pulmonary embolism was confirmed in all but 1. Mortality was high (17 of 38 patients) irrespective of the therapeutic option chosen: surgery (8 of 17), thrombolytics (2 of 9), heparin (5 of 8), or interventional percutaneous techniques (2 of 4). The in-hospital mortality rate was significantly linked with the occurrence of cardiac arrest. Conversely, the outcome after discharge was usually good, because 18 of 21 patients were still alive 47.2 months later (range, 1 to 70 months).

Conclusions—Severe pulmonary embolism was the rule in our series of FRHTS (mortality rate, 44.7%). The choice of therapy had no effect on mortality. Emergency surgery is usually advocated. However, thrombolysis is a faster, readily available treatment and seems promising either as the only treatment or as a bridge to surgery. In patients with contraindications to surgery or lytic therapy, interventional techniques may be proposed. **(*Circulation*. 1999;99:2779-2783.)**

Key Words: embolism ■ echocardiography ■ thrombus ■ thrombolysis ■ surgery

Floating right heart thrombi (FRHTS) are uncommon but probably underdiagnosed in patients with pulmonary embolism.[1] Previous studies suggest that they occur in 7% to 18% of patients.[2,3] FRHTS are in transit[4] from the legs to the pulmonary arteries and thus are a form of venous thromboembolic disease.[2] They can embolize at any moment[5] and thus require emergency treatment, especially in view of their documented high (>40%) mortality rate.[6] However, reported series are small, and because the only meta-analysis involved a heterogeneous group of patients, the most appropriate therapy remains unclear.[6,7] We report on our own experience of 38 consecutive cases of mobile right heart thrombi in patients with suspected or proven pulmonary embolism. Immobile thrombi that develop in situ,[4,6] favored by blood stagnation in patients with cardiomyopathies or with foreign bodies, have a generally good prognosis when treated with heparin[6] and were not included in this study.

Methods

Patient Population

This is a retrospective, monocentric, consecutive study, over the period from January 1986 to January 1998, of patients admitted to our intensive care unit for suspected or confirmed pulmonary embolism. We identified 38 patients with FRHTS, diagnosed by transthoracic (TTE) or transesophageal (TEE) echography in most patients (n=35) and during surgical pulmonary embolectomy, angiography, or autopsy in the remaining 3. FRHTS were defined as mobile masses, moving freely inside the right heart, and not attached to an intracardiac structure. Masses such as vegetations, primary or metastatic tumors, embryological remnants, or thrombi attached to the wall of the right atrium or right ventricle or to an intracardiac device were excluded. Two-dimensional and Doppler echocardiography was performed in the parasternal, apical 4-chamber, and subcostal views with a Vingmed with a 3.5-MHz transthoracic transducer or a 5-MHz transesophageal transducer in the intensive care unit.

Statistical Analysis

The following variables were analyzed: age, sex, previous venous thromboembolism, predisposing factors for thromboembolism, symptoms (chest pain, syncope or faintness, dyspnea), arterial hypotension <100 mm Hg if not caused by hypovolemia or sepsis, paradoxical embolism, shock (hypotension as defined above with cold and clammy extremities and urine output <30 mL/h), cardiac arrest with need for cardiopulmonary resuscitation, in-hospital mortality, first-day mortality, ECG signs, arterial gas analysis, TTE or TEE findings (end-diastolic right ventricular diameter, paradoxical

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ventricular septal motion, pulmonary artery systolic pressure determined from the velocity of tricuspid regurgitation, location and shape of the thrombi, patent foramen ovale), diagnosis of phlebitis (by venography or B-mode and Doppler ultrasonography), diagnosis of pulmonary embolism, and treatment. Then we tried to identify factors that predicted in-hospital mortality among age, sex, NYHA class IV dyspnea, shock, cardiac arrest, severe hypoxemia (arterial oxygen pressure <65 mm Hg or oxygen saturation <90%), and treatment. Analysis was performed in univariate form with χ^2 testing because of the size of the population. Statistical significance was defined as $P<0.05$.

Treatment

Treatment was not standardized, and the choice of therapy was based on the judgment of the attending physicians. When a patient received a second treatment after the first one was unsuccessful, we considered the first one in our analysis, according to the intention-to-treat principle. Four options were discussed: (1) surgical embolectomy during cardiopulmonary bypass; (2) intravenous thrombolysis with recombinant tissue plasminogen activator (rtPA; the choice of the dosing regimen was based on the judgment of the physicians involved, ranging from 60 to 100 mg over a period of 2 hours); (3) intravenous heparin alone, with the dose adjusted to keep the activated partial thromboplastin time at 2 to 3 times normal; and (4) interventional techniques. The interventional procedure has already been described by our institute[8,9] and was performed when surgery and thrombolytics were initially contraindicated because of the age of the patient or the presence of coexisting disease. Briefly, the thrombus was trapped in a basket device (Cook, Inc), advanced to the right atrium via the femoral approach, and then withdrawn into the inferior vena cava; subsequently, a caval filter (LGM) was placed above the entrapped thrombus via the jugular vein. The basket was then removed, leaving the thrombus below the filter. After a period of intravenous heparin therapy, all patients were discharged on oral anticoagulation, adjusted to maintain an international normalized ratio between 2 and 3 times control.

Follow-Up

The survival rate of the population was established on January 1998 by phone contact with the patients or their physicians. Causes of death were based on autopsy reports when available or on the judgment of the physician involved in each case.

Results

Clinical Findings

Among patients admitted to our department for suspected pulmonary embolism from January 1986 to January 1998, 38 patients (19 men and 19 women) were found to have an FRHT. Ten FRHTS were diagnosed in the period from 1986 to 1991 and 28 from 1992 to 1997. The prevalence of FRHTS among proven pulmonary embolisms in our institute from 1992 to 1997 was 28 of 341 (8.2%); data are lacking before 1992. The mean age (±SD) was 64.6±12.2 years (range, 34 to 86 years). Clinical data are summarized in Table 1; the mode of presentation was in most cases suggestive of severe pulmonary embolism, because a cardiac arrest occurred in half of the population and was fatal in all cases but 1, despite resuscitation, thrombolysis, or surgery.

Imaging Data

Echocardiography usually demonstrated acute cor pulmonale (Table 2). The thrombus was serpentine in 36 patients and spherical in 2; it was located in the right atrium in 30 patients, in the right ventricle in 6, and in both chambers in 2. Deep venous thrombosis was found in 28 of 31 patients (90.3%) who underwent venography or ultrasonography. Pulmonary embolism was confirmed in all cases but 1 (34 of 35) when a diagnostic procedure could be performed (Table 3).

TABLE 1. Clinical Characteristics

Clinical Characteristics	n (%)
No. of patients	38
Previous thromboembolism	9 (23.7)
Predisposing factors for venous thromboembolism	28 (73.7)
Symptoms	
Chest pain	8 (21.1)
Syncope or faintness	14 (36.8)
NYHA class IV dyspnea	32 (84.2)
No symptoms	1 (2.6)
Clinical presentation	
Blood pressure <100 mm Hg	21 (55.3)
Paradoxical embolism	4 (10.5)
Shock	20 (52.6)
Cardiac arrest	18 (47.4)
Before treatment	6 (15.8)
During treatment	9 (23.7)
After treatment	3 (7.9)
In-hospital mortality	17 (44.7)
First-day mortality	8 (21.1)

Treatment

Seventeen patients underwent emergency surgery as initial therapy (Figure 1), and 9 of them were discharged well. Nine patients received lytic drugs as initial therapy, and 7 survived (1 had successful surgical intervention after failed thrombolysis). Of the 8 patients initially treated by heparin alone, 3 died soon after treatment, 2 subsequently received

TABLE 2. Complementary Investigations

Complementary Investigations	n (%)
ECG	38 (100)
Tachycardia >100 bpm	29 (76.3)
S_1Q_3	17 (44.7)
Right bundle-branch block	15 (39.5)
Normal	3 (7.9)
Arterial gas*	27 (71.1)
Severe hypoxemia (P $_{O_2}$ <65 mm Hg or $_{O_2}$ saturation <90%)	21 (77.8)
TTE*	36 (94.7)
End-diastolic right ventricular diameter >30 mm	33 (91.7)
Paradoxical septal motion	27 (75.0)
Systolic pulmonary arterial pressure	31 (86.1)
Pulmonary artery thrombus	4 (11.1)
TEE*	11 (28.9)
Patent foramen ovale	6 (54.5)
Pulmonary artery thrombus	5 (45.5)
Thrombus entrapped on the foramen ovale	4 (36.4)

*Number of patients who underwent these investigations is <38 because of unknown data.

TABLE 3. Diagnosis of Pulmonary Embolism

Investigations	n=35
Surgery*	9
High-probability lung scan	7
Angiography	6
CT scan	6
TTE*	3
TEE*	2
Autopsy*	1
Pulmonary embolism	34

*Diagnosis was based on presence of thrombus in the pulmonary arteries. Emergency did not allow additional investigations.

lytic drugs because of hemodynamic deterioration and died, and 3 were discharged well. A percutaneous interventional procedure was performed in 4 cases because of relative contraindications to lytics or surgery. This was successful in 1 patient; in a second, it failed, and rescue thrombolysis was associated with a favorable outcome; in the last 2 patients, the thrombus was entrapped in the basket, but intractable right heart failure occurred afterward.

Thus, the therapeutic management changed in a number of cases. As explained in the Methods section, we considered in our outcome analysis the first treatment administered. Table 4 displays mortality rates according to the therapeutic option.

Prognostic Factors

Prognostic factors are summarized in Table 5. Only severe hypoxemia and the occurrence of cardiac arrest were significantly related to in-hospital mortality.

Follow-Up

Follow-up was obtained for all patients at a mean delay of 47.2±37.7 months (range, 1 to 70 months). Of the 21 patients who were discharged alive, 3 died during follow-up: 1 because of a metastatic tumor at 46 years, 1 secondary to a severe stroke at 73 years, and the last 1 because of dementia at 59 years; these conditions existed before the occurrence of FRHTS.

TABLE 4. Mortality According to Therapeutic Management

Treatment	No. of Treated Patients	No. of Deaths (%)
Surgery	17	8 (47.1)
Thrombolysis	9	2 (22.2)
Heparin	8	5 (62.5)
Interventional techniques	4	2 (50.0)
All treatments	38	17 (44.7)

Discussion

Free-floating right heart thrombi are a rare phenomenon, generally diagnosed when echocardiography is performed in patients with suspected or proven pulmonary embolism, and have a dismal prognosis, as documented by previous reports of small series of patients. This study underlines the importance of echocardiography as a key examination in this setting. It is a simple, noninvasive, painless investigation that is widely available and that can be performed at the bedside in an intensive care unit. FRHTS are mostly wormlike,[4] as confirmed by our series, arising from the lower-limb veins. Sometimes their shape is spherical or grapelike.[5] Severe pulmonary embolism usually coexists with FRHTS.[3] Serial echocardiographic examinations are useful when the clinical status deteriorates because they may demonstrate thrombus that was not detected on the initial examination. This was the case in 2 patients in the present series in whom an FRHT that was not present at the first examination was detected on a second examination performed by the same operator.

Sometimes, the differential diagnosis of an FRHT may be difficult. Other diagnoses, for instance, congenital structures[4] such as a Chiari network,[10] persistent eustachian or thebesian valves, or atrial septal aneurysms, or acquired conditions such as intracardiac tumors[4] or devices[11] and vegetations need to be considered.[4,12] Any doubt should lead to the performance of TEE, which is a rapid, safe,[13] semi-invasive, bedside

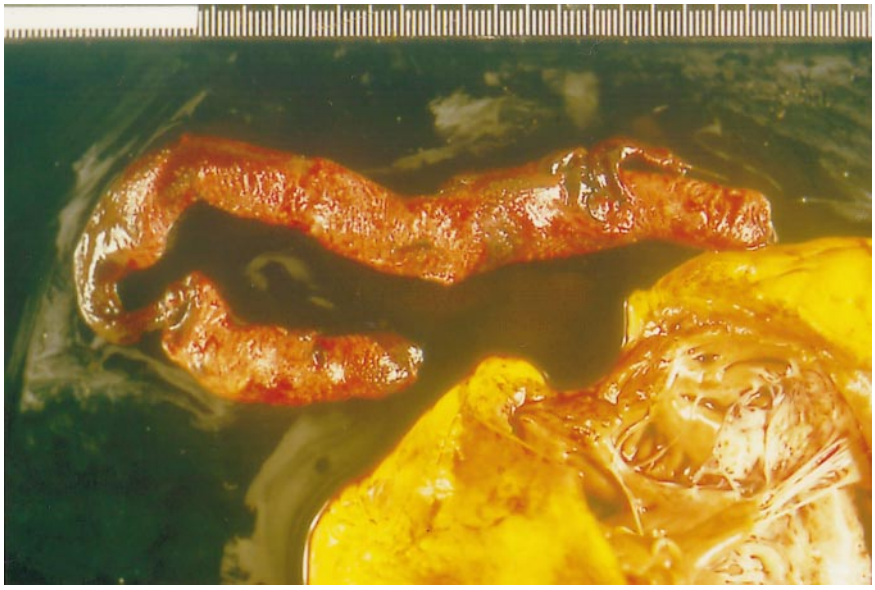

Figure 1. Huge mobile thrombus removed from right heart chambers at surgery under full cardiopulmonary bypass.

TABLE 5. Prognosis Factors According to Mortality

Characteristics	No. of Patients (%)	No. of Deaths (%)	*P*
Male	19 (50.0)	9 (47.7)	0.99
Age >65 y	22 (57.9)	11 (50.0)	0.89
NYHA class IV dyspnea	32 (84.2)	15 (46.9)	0.94
Shock	20 (52.6)	12 (60.0)	0.25
Cardiac arrest	18 (47.4)	17 (94.4)	<0.001
Severe hypoxemia	21 (55.3)	15 (71.4)	<0.001
Surgery	17 (44.7)	8 (47.1)	0.76
Thrombolysis	9 (23.7)	2 (22.2)	0.48
Heparin	8 (21.1)	5 (62.5)	0.71
Interventional techniques	4 (10.5)	2 (50.0)	0.99

investigation. Moreover, TEE may allow direct diagnosis of pulmonary embolism when it displays a thrombus in the pulmonary arteries,[14] and it may visualize a patent foramen ovale[15] and sometimes a thrombus entrapped in it. We observed these findings in 5, 6, and 4 patients, respectively (Figure 2).

Almost all the patients of our series had a venous thrombosis or a pulmonary embolism, which is consistent with published reports.[3,5,6] However, their diagnosis is useful in an emergency situation only if it modifies the therapeutic strategy. According to most authors,[1,2,16] diagnosis of FRHT by echography allows immediate treatment, and additional investigations (lung scintigraphy, CT scan, pulmonary angiography) may be performed after treatment.

FRHTS are an extreme therapeutic emergency,[2,6] and any delay to treatment could be lethal[4,5] ; 21.1% of the patients died within the first day after admission in our series, as in that of the European Working Group.[6] In-hospital mortality in our series is consistent with published reports, and there was no significant difference between the different therapeutic approaches. Surgical embolectomy with exploration of the right chambers and the pulmonary arteries under full cardiopulmonary bypass is the classic treatment[2] and was effective in 9 of 17 patients operated on. However, the place of surgery for severe pulmonary embolism with or without concomitant FRHT is discussed in the literature.[17] Thus, thrombolysis may be advocated first.[1,18–21] It may favorably affect the clinical outcome of patients with acute massive pulmonary embolism, even without hemodynamic compromise.[22] Its theoretical advantages are numerous[23,24]: it accelerates thrombus lysis and pulmonary reperfusion, reduces pulmonary hypertension, and because of right ventricle–left ventricle interdependence,[25] improved right ventricular function helps to increase both right and left ventricle output and to reverse cardiogenic shock. Moreover, thrombolysis may dissolve the clot in 3 locations at the same time[20]: the intracardiac thrombus, the pulmonary embolus, and the venous thrombosis. Finally, it is a simple, rapid, widely applicable treatment, and it can be administered at the bedside. Lytic therapy was efficient in 7 of 9 patients in our series. We think it may be possible to improve the survival rate by adequate management. First, monitoring by TTE[3,26] is useful during thrombolytic administration to assess the outcome of the thrombus and the right ventricle function, because the clot could embolize to the lungs at any moment; transesophageal examination[27,28] is rarely mandatory. Second, these patients are usually unstable, and worsening of their hemodynamic status should be anticipated. We found that the occurrence of a cardiac arrest was significantly linked with mortality. Thus, inotropic support with catecholamine infusion should be prepared immediately after the diagnosis of FRHT and administered as soon as blood pressure falls below 100 mm Hg or cardiogenic shock is suspected. It may be preferable to admit the patients to an intensive care unit, because sudden death is a risk and because mechanical ventilation may be required. Three lytic drugs have been approved by the Food and Drug Administration in cases of severe pulmonary embolism[24]: urokinase, streptokinase, and rtPA. It seems reasonable to choose rtPA, which allows faster hemodynamic improvement[24] and because of its fibrinospecificity[20] and its short half-life, which do not contraindicate surgical embolectomy when hemodynamic status continues to worsen. We used rtPA with an intravenous regimen of 100 mg over 2 hours, as proposed by Goldhaber.[24]

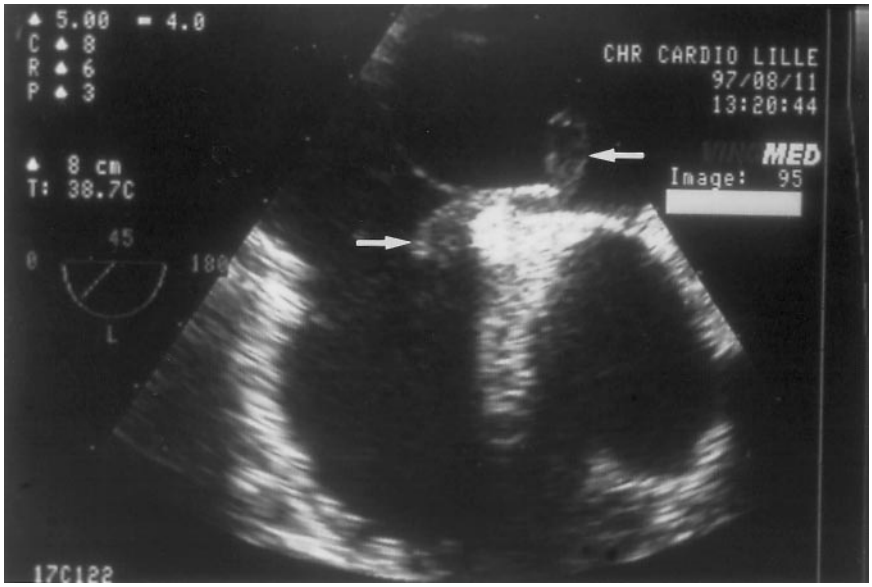

Figure 2. Transesophageal view displays thrombus (arrows) entrapped in foramen ovale.

Mortality in the heparin group is up to 62.5% in our series, more than in other reports.[5–7,29] Heparin is more an antithrombotic than a lytic drug and is inappropriate as the sole treatment of an impending pulmonary embolism[6]; it may be proposed in stable patients. Our experience with interventional techniques is recent but promising when thrombolysis and surgery are contraindicated; 2 of 4 treated patients survived. Finally, the therapeutic strategy changed in 25% of our patients when hemodynamic status deteriorated or thrombus embolization occurred. Thus, these different approaches were not exclusive but rather complementary.

Study Limitations

The first limitations of this study are its size and its retrospective and nonrandomized nature. Second, therapeutic management was based on the judgment of the physicians involved in each case and varied according to hemodynamic status and possible contraindications. Thus, comparison between the different treatments remains difficult. However, FRHT is a rare condition, and our study is the largest single-center experience that we know of; indeed, the largest multicenter experience published reported on 48 FRHTS.[3,6]

Conclusions

Our study confirms that thrombi in transit in the right heart are a severe form of venous thromboembolic disease. In-hospital mortality is as high as 44.7%. Conversely, prognosis is usually good after discharge, as shown by our follow-up. It justifies diagnosis and treatment in an emergency. TTE, or TEE in some cases, is the key investigation in suspected or proven pulmonary embolism. Additional investigations that do not modify the treatment strategy can be performed later. Our study shows no significant difference in mortality between the different therapeutic approaches. However, while we await randomized trials, it might be advisable to propose thrombolysis first, which is a simple, easily available, bedside treatment. Of course, surgery remains the classic treatment, particularly in cases with contraindications to thrombolysis or if thrombolysis is ineffective. Catheter-device removal may be an attractive alternative in patients with contraindications to thrombolysis or surgery.

References

1. Goldhaber SZ. Optimal strategy for diagnosis and treatment of pulmonary embolism due to right atrial thrombus. *Mayo Clin Proc*. 1988;63:1261–1264.
2. Chapoutot L, Nazeyrollas P, Metz D, Maes D, Maillier B, Jennesseaux C, Elaerts J. Floating right heart thrombi and pulmonary embolism: diagnosis, outcome and therapeutic management. *Cardiology*. 1996;87:169–174.
3. Casazza F, Bongarzoni A, Centonze F, Morpurgo M. Prevalence and prognostic significance of right-sided cardiac mobile thrombi in acute massive pulmonary embolism. *Am J Cardiol*. 1997;79:1433–1435.
4. Panidis IP, Kotler MN, Mintz GS, Ross J. Clinical and echocardiographic features of right atrial masses. *Am Heart J*. 1984;107:745–758.
5. Farfel Z, Shechter M, Vered Z, Rath S, Goor D, Gafni J. Review of echocardiographically diagnosed right heart entrapment of pulmonary emboli-in-transit with emphasis on management. *Am Heart J*. 1987;113:171–178.
6. European Working Group on Echocardiography. The European Cooperative Study on the clinical significance of right heart thrombi. *Eur Heart J*. 1989;10:1046–1059.
7. Kinney EL, Wright RJ. Efficacy of treatment of patients with echocardiographically detected right sided heart thrombi: a meta-analysis. *Am Heart J*. 1989;118:569–573.
8. Loubeyre C, Chartier L, Beregi JP, Asseman P, Coullet JM, Bauchart JJ, Hannebicque G, Koussa M, Prat A, Théry C. How should pulmonary embolism associated with mobile right atrial thrombus be treated? Is there a place for interventional cardiology? *Eur Heart J*. 1995;16(suppl):268. Abstract.
9. Beregi JP, Aumégeat V, Loubeyre C, Coullet JM, Asseman P, Debaecker-Steckelorom C, Bauchart JJ, Liu PC, Théry C. Right atrial thrombi: percutaneous mechanical thrombectomy. *Cardiovasc Intervent Radiol*. 1997;20:142–145.
10. Felner JM, Churchwell AL, Murphy DA. Right atrial thromboemboli: clinical, echocardiographic and pathophysiologic manifestations. *J Am Coll Cardiol*. 1984;5:1041–1051.
11. Yarnal JR, Smiley WH. Right atrial mass simulated echocardiographically by a Swan-Ganz catheter. *Chest*. 1978;74:478–479.
12. Covarrubias EA, Sheikh MU, Fox LM. Echocardiography and pulmonary embolism. *Ann Intern Med*. 1977;87:720–721.
13. Daniel WG, Erbel R, Kasper W, Visser CA, Engberding R, Sutherland GR, Grube E, Hanrath P, Maisch B, Denning K, Schartl M, Kremer P, Angermann C, Iliceto S, Curtius JM, Mugge A. Safety of transesophageal echocardiography. *Circulation*. 1991;83:817–821.
14. Nixdorff U, Erbel R, Drexler M, Meyer J. Detection of thromboembolus of the right pulmonary artery by transesophageal two-dimensional echocardiography. *Am J Cardiol*. 1988;61:488–489.
15. Nelessen U, Daniel WG, Matheis G, Oelert H, Depping K, Lichtlen PR. Impending paradoxical embolism from atrial thrombus: correct diagnosis by transesophageal echocardiography and prevention by surgery. *J Am Coll Cardiol*. 1985;5:1002–1004.
16. Goldhaber SZ, Morpurgo M, for the WHO/ISFC task force on pulmonary embolism. Diagnosis, treatment, and prevention of pulmonary embolism. *JAMA*. 1992;268:1727–1733.
17. Gray HH, Morgan JM, Paneth M, Miller GAH. Pulmonary embolectomy for acute massive pulmonary embolism: an analysis of 71 cases. *Br Heart J*. 1988;60:196–200.
18. Fournier P, Pacouret G, Charbonnier B, Marchal C, Rioux P. Thrombus des cavités droites: intérêt de la thrombolyse dans le traitement des thrombi mobiles. *Arch Mal Coeur*. 1992;85:877–882.
19. Cuccia C, Campana M, Franzoni P, Faggiano P, Volterrani M, Musmeci G, Visioli O. Effectiveness of intravenous rt-PA in the treatment of massive pulmonary embolism and right heart thromboembolism. *Am Heart J*. 1993;126:468–472.
20. Leclercq F, Messner-Pellenc P, Beigbeder JY, Albat B, Thévenet A, Grolleau-Raoux R. Thrombus flottant de l'oreillette droite et embolie pulmonaire grave: intérêt de la thrombolyse intraveineuse. *Arch Mal Coeur*. 1994;87:805–811.
21. Goldhaber SZ, Nagel JS, Théard M, Levine JD, St John Sutton MG. Treatment of right atrial thrombus with urokinase. *Am Heart J*. 1988;115:894–897.
22. Konstantinides S, Geibel A, Olschewski M, Heinrich F, Grosser K, Rauber K, Iversen S, Redecker M, Kienast J, Just H, Kasper W. Association between thrombolytic treatment and the prognosis of hemodynamically stable patients with major pulmonary embolism. *Circulation*. 1997;96:882–888.
23. Levine MN. Thrombolytic therapy in acute pulmonary embolism. *Can J Cardiol*. 1993;92:158–159.
24. Goldhaber SZ. Thrombolysis for pulmonary embolism. *Prog Cardiovascular Dis*. 1991;34:113–134.
25. Jardin F, Dubourg O, Guéret P, Delorme G, Bourdarias JP. Quantitative two-dimensional echocardiography in massive pulmonary embolism: emphasis on ventricular interdependence and leftward septal displacement. *J Am Coll Cardiol*. 1987;10:1201–1206.
26. Lee KS, Pashkow FJ, Homa DA, Salcedo EE. Transthoracic echocardiography documents prompt resolution of right atrial thrombus after thrombolytic therapy. *J Am Soc Echocardiogr*. 1992;5:541–543.
27. Rittoo D, Sutherland GR. Acute pulmonary artery thromboembolism treated with thrombolysis: diagnostic and monitoring uses of transoesophageal echocardiography. *Br Heart J*. 1993;69:457–459.
28. Dall'Aglio V, Nicolosi GL, Zanuttini D. Transthoracic and transoesophageal echocardiographic documentation of disappearance of massive right atrial and pulmonary artery thromboemboli after fibrinolytic therapy and normalization of left ventricular dimensions and function. *Eur Heart J*. 1990;11:863–865.
29. Crowell RH, Adams GS, Koilpillai CJ, McNutt EJ, Montague TJ. In vivo right heart thrombus: precursor of life-threatening pulmonary embolism. *Chest*. 1988;94:1236–1239.

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