

CASE REPORTS

Anesthesiology

1998; 89:1257-62

© 1998 American Society of Anesthesiologists, Inc.

Lippincott Williams & Wilkins

Left Ventricular Thrombi: Intraoperative Detection by Transesophageal Echocardiography and Recognition of a Source of Post CABG Embolic Stroke: A Case Series

Andrew Maslow, M.D.,* Edward Lowenstein, M.D.,† John Steriti, M.D.,* Robert Leckie, M.D.,* William Cohn,‡
Michael Haering, M.D.*

INTRACARDIAC sources of systemic thromboembolism are well known. We present a series of four patients undergoing coronary artery bypass surgery (CABG), in which unknown postinfarct left ventricular thrombi (LVT) were detected intraoperatively by transesophageal echocardiography (TEE). Our experience with detection and management of LVT in cardiac surgical patients is discussed.

Case Reports

Case 1

A 65-yr-old man was admitted with an acute anterior wall myocardial infarction (MI). Cardiac catheterization showed an LV ejection fraction of 0.45 with anterior and apical akinesis. No abnormalities were seen on the ventriculogram to suggest a mass effect. The patient was brought to the operating room for CABG. Transesophageal echocardiography (multiplane TEE probe; Hewlett Packard, Andover, MA) was performed after induction of general anesthesia. The examination revealed LV systolic dysfunction with apical akinesis and suggested a nonmobile protruding apical mass approximately 1 cm in diameter (figs. 1A and 1B). After institution of hypothermic cardiopulmonary bypass (CPB), a three-vessel bypass was performed. Care was taken to avoid vigorous manipulation of the ventricle and no LV vent was placed. During removal of the aortic cross clamp, a spontaneous sinus rhythm developed with a mean arterial pressure recorded from the right radial artery of

40–60 mmHg. After several ventricular ejections, the arterial pressure decreased suddenly to 20 mmHg. A 20-gauge catheter was inserted directly into the ascending aorta, revealing a mean arterial pressure in excess of 100 mmHg. A presumptive diagnosis of right radial artery embolus was made. During this time, the patient's cardiac rhythm deteriorated to ventricular fibrillation. This was treated successfully with lidocaine and electrical defibrillation. After obtaining an atrially paced rhythm, there were several unsuccessful attempts at separation from cardiopulmonary bypass. Evaluation by TEE revealed severe LV dysfunction. The apical mass was no longer visualized. Successful separation from CPB was achieved after placement of an LV assist device. On the first postoperative day, a dense, left hemiplegia was noted. On the second postoperative day, the patient became unresponsive, his pupils were fixed and dilated, and he subsequently died. Autopsy revealed coronary artery emboli and cerebral infarct consistent with embolic injury. Emboli were reported to be organized. No thrombus was found in the cardiac chambers or on the LV assist device cannulae.

Case 2

A 73-yr-old man with coronary artery disease and S/P MI was admitted with postinfarct angina. Left ventriculography, performed during cardiac catheterization, did not suggest any mass effect. During catheterization, the patient experienced recurrent myocardial ischemia, and intraaortic balloon counterpulsation was instituted. The patient was taken to the operating room for CABG. Prebypass TEE evaluation revealed moderate to severe LV systolic dysfunction with anterior and apical wall akinesis and moderate to severe right ventricular systolic dysfunction. A 1.5-cm diameter protruding mass with an echolucent center, consistent with thrombus, was seen in the apex (fig. 2A). After institution of CPB, a three-vessel bypass was performed. Care was taken to avoid vigorous manipulation of the ventricle and no LV vent was placed. After rewarming and before removal of the aortic cross-clamp, the heart was allowed to fill with blood and TEE evaluation was performed to determine the location of the mass. The mass was observed freely floating in the left atrium (fig. 2B), but no mass was apparent in the LV (fig. 2C). The heart was emptied, and while undergoing CPB with the aortic cross-clamp in place, the thrombus was removed *via* a left atriotomy and CPB was subsequently discontinued. The patient was discharged neurologically intact.

Case 3

A 72-yr-old man with a history of a transmural MI 9 months prior to admission, was scheduled for elective CABG. Pre-CPB TEE evaluation

* Instructor in Anesthesia.

† Henry Isaiah Dorr Professor of Anaesthesia and Professor of Medical Ethics, Harvard Medical School; Provost, Department of Anaesthesia and Critical Care, Massachusetts General Hospital, Boston, Massachusetts.

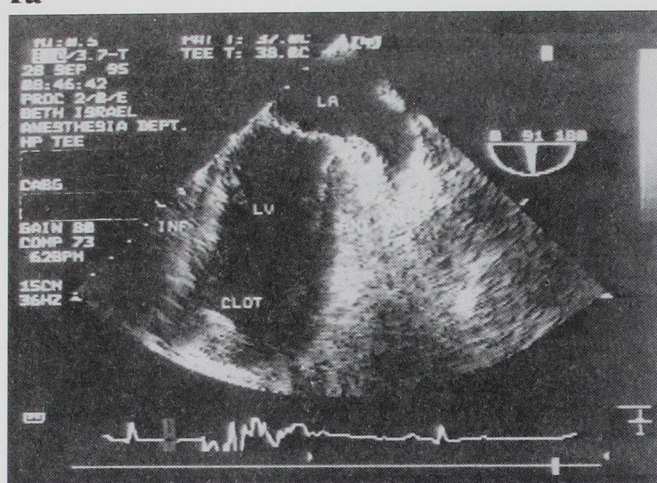
‡ Instructor in Surgery.

Received from the Departments of Anesthesia and Critical Care* and Surgery, Beth Israel-Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts. Submitted for publication February 12, 1998. Accepted for publication June 11, 1998. Supported in part by the Beth Israel Anesthesia Foundation.

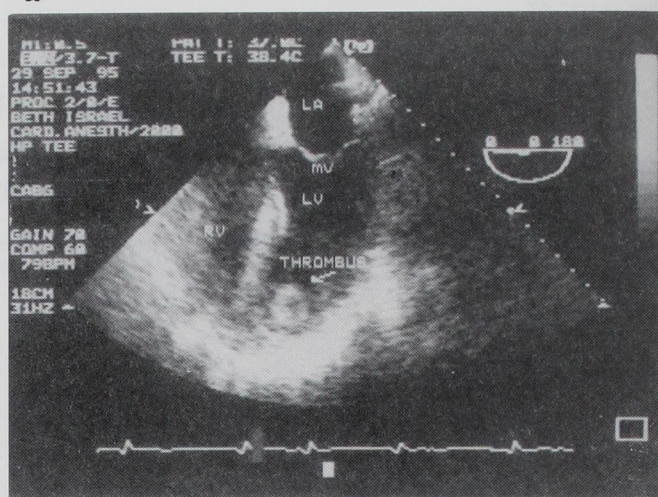
Address reprint requests to Dr. Maslow: Department of Anesthesia ST 308, Beth Israel-Deaconess Medical Center, 330 Brookline Avenue, Boston, Massachusetts 02215. Address electronic mail to: amaslow@bidmc.harvard.edu

Key words: Cardiopulmonary bypass; left ventricle; thrombi; transesophageal echocardiography.

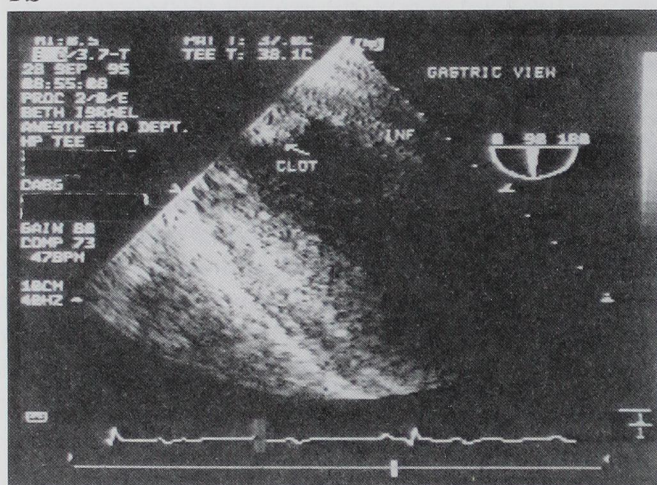
1a



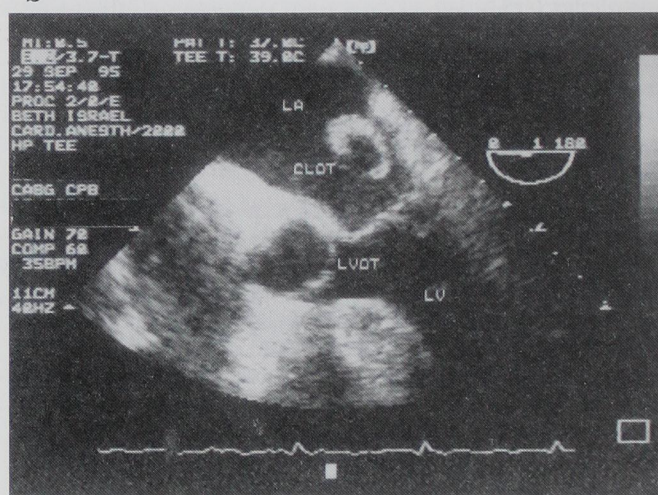
a



1b



b



c

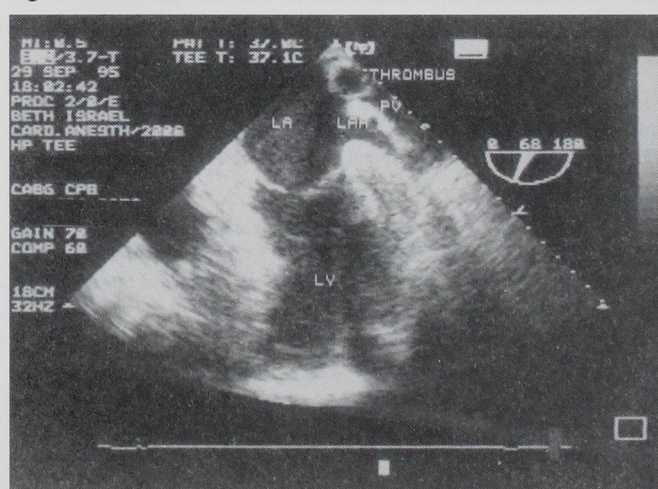


Fig. 1. Transesophageal images of the left ventricular (LV) thrombus seen in case 1. (A) Thrombus in the LV apex (along the inferior wall, as imaged from the esophagus). (B) Thrombus in the apex, as imaged from the stomach. LA = left atrium; LV = left ventricle; inf = inferior wall.

showed an aneurysmal akinetic apex and a 1.0-cm diameter, well-demarcated, protruding, nonmobile echodense mass in the LV apex (fig. 3). Cardiopulmonary bypass and grafting proceeded uneventfully. Vigorous manipulation of the LV was avoided and no LV vent was placed. The heart chambers were examined by TEE before removal of

Fig. 2. Transesophageal echocardiography images of case 2. (A) Represents the precardiopulmonary bypass image of the apical thrombus in the left ventricle (LV). (B) Shows the thrombus in the left atrium (LA) before removal of the aortic cross-clamp. (C) The thrombus is seen at the junction of the pulmonary vein and the LA, and no thrombus seen in the LV. RV = right ventricle; RA = right atrium; mv = mitral valve; LVOT = left ventricular outflow tract; PV = pulmonary vein; IAA = left atrial appendage.

CASE REPORTS

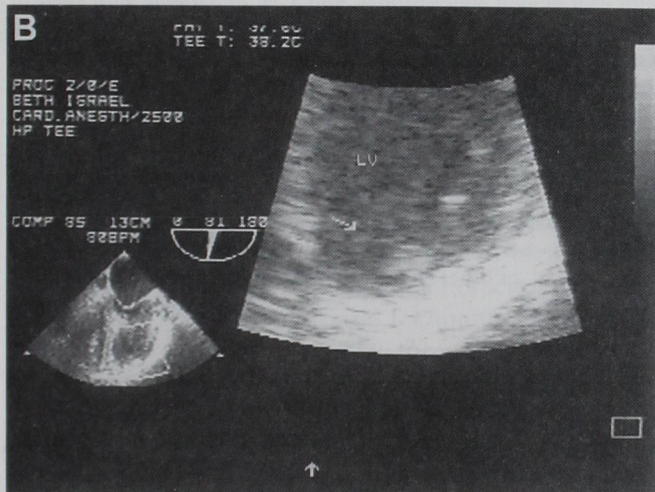
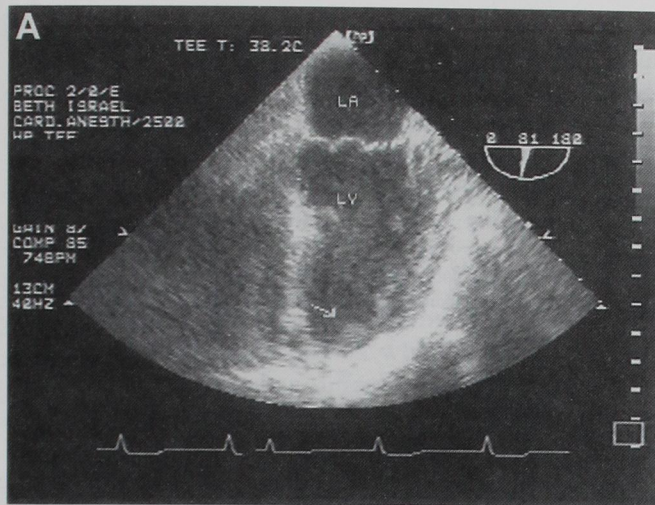
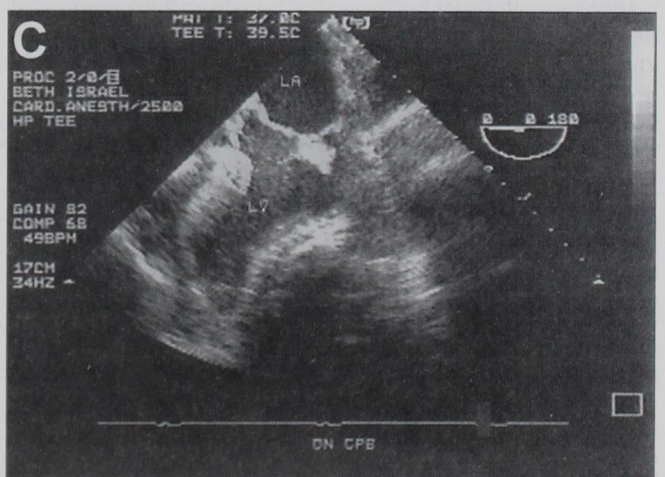
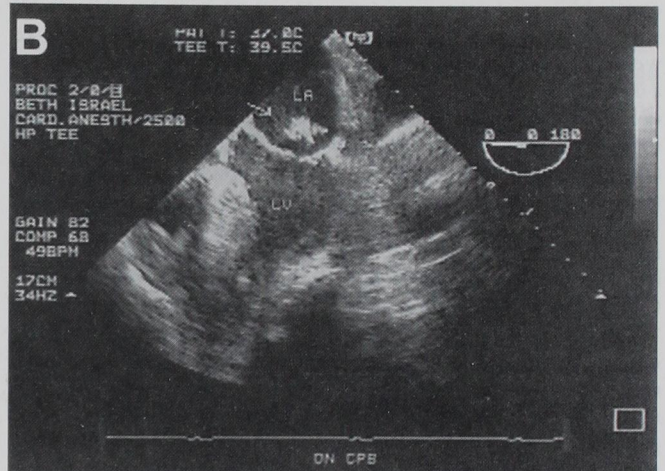
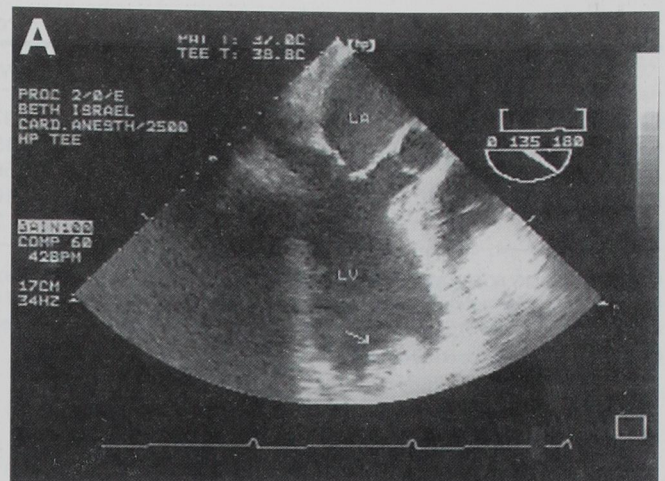


Fig. 3. Transesophageal echocardiography image of case 3. The small protruding thrombus (arrow) appears to be organized and sits in the apex of the left ventricle. LV = left ventricle; LA = left atrium.

the aortic cross-clamp. This examination revealed that the mass was no longer in its original position. It was not possible to identify the mass elsewhere in the heart. Cardiopulmonary bypass was discontinued without difficulty and the operation was completed uneventfully. On postoperative day 1, the patient had not awakened. Computerized tomography of the head revealed a large cerebral infarction consistent with embolization. There was also evidence of uncal herniation. The patient was subsequently declared brain dead.

Fig. 4. Transesophageal echocardiography images of case 4. The location of the thrombus is indicated by the arrow. (A) The thrombus in the antero-apical position of the left ventricle before cardiopulmonary bypass (CPB). During CPB a four-vessel bypass, aneurysmorrhaphy, and removal of the thrombus were performed. (B,C) Clot on both sides of the mitral valve after removal of the aortic cross-clamp and before discontinuation of CPB. These fragments were removed before discontinuation of CPB. The patient recovered without neurologic sequelae. LV = left ventricle; LA = left atrium.



CASE REPORTS

Table 1. Types of Surgical Procedures in Which TEE Was Performed

Procedure	N	No WMA	Hypokinesis	Akinesis/Dysk	LVT
CABG with CPB	473	197	137	139	5
CABG without CPB (mid CABG)	25	15	7	3	0
AVR/CABG	76	56	11	9	0
MVR/CABG	33	16	9	8	1
Other	17	7	5	5	2

CABG = coronary artery bypass graft surgery; CPB = cardiopulmonary bypass; AVR = aortic valve replacement; MVR = mitral valve replacement/reconstruction; No WMA = no wall motion abnormalities; Hypokinesis = patients with areas of hypokinesis and no areas of akinesis/dyskinesis; Akinesis/Dysk = all patients with akinesis/dyskinesis with or without hypokinesis; LVT = left ventricular thrombus.

Occurrence of wall motion abnormalities are reported.

Case 4

A 73-yr-old man had congestive heart failure. Preadmission echocardiographic examination revealed a dilated, globally hypokinetic LV with an estimated ejection fraction of 0.15. No intracardiac masses were seen. Cardiac catheterization showed global LV hypokinesis and a dyskinetic apex. Ventriculography of the left side was not suggestive of an intracardiac mass. Examination by TEE after induction of anesthesia revealed a dilated, severely hypokinetic LV with an aneurysmal apex. Within the aneurysm, a 2-cm diameter protruding mass, consistent with thrombus, was seen (fig. 4). The patient underwent four-vessel CABG and ventriculoendoneurysmorrhaphy with removal of the apical thrombus. After removal of the aortic cross-clamp and before discontinuation of CPB, echocardiographic reevaluation showed a mobile mass consistent with a fragment of thrombus in the left atrium. The heart was arrested and the aorta was cross-clamped. A left atriotomy was performed and the thrombus was removed. Subsequently, CPB was discontinued and the patient required pharmacologic and mechanical support. No intracardiac masses were seen during postbypass echocardiographic evaluation. The patient was discharged from the hospital neurologically intact on postoperative day 8.

After encountering these four cases, we retrospectively reviewed all recorded TEE studies of patients undergoing CABG ($n = 624$), since the start of our formal perioperative TEE program (May 1995–

August 1997). Wall motion abnormalities were seen in 333 (53%) of the studies. One hundred sixty-four patients (26%) had areas of akinesis/dyskinesis. We detected LVT in eight patients. This represents 1.3% of patients undergoing CABG and TEE, 2.4% of patients with wall motion abnormalities, and 4.7% of patients with areas of akinesis/dyskinesis. Four of eight patients had nonmobile laminar masses. Three of the four patients with nonmobile laminar masses were known to have LVT before the intraoperative TEE study, one of which had a previous neurologic injury believed to be caused by emboli. Two of these four patients underwent LV aneurysmectomy, in addition to CABG. The other two patients with nonmobile laminar masses had a history of large MIs. All LVT were located in akinetic/dyskinetic segments. All patients with LVT had nonmobile aortic atheroma. There were no embolic sequelae (neurologic, cardiac, renal, or other) recognized in any of the four patients with laminar masses.

Discussion

Left ventricular thrombi have been reported to occur in up to 30–45% of patients after acute anterior wall MI and in less than 5% of patients after inferior wall MI.^{1–4}

Table 2. Breakdown of the 16 Cases Under the Heading of "Other" in Table 1

Procedure	N	No WMA	Hypokinesis	Akinesis/Dysk	LVT
AVR/MVR/CABG	5	3	1	1	0
AVR/CABG/aortic root repl	2	1	1	0	0
MVR/aneurysmectomy/CABG	1	0	0	1	1
Aneurysmectomy/CABG	1	0	0	1	1
VSD/CABG	1	0	0	1	0
AVR/PDA/CABG	1	1	0	0	0
CEA/CABG	2	0	1	1	0
TVR/CABG	1	0	1	0	0
MVR/TVR/CABG	1	0	1	0	0
ASD/CABG	1	1	0	0	0
MVR/ASD/CABG	1	1	0	0	0

CABG = coronary artery bypass graft surgery; CPB = cardiopulmonary bypass; AVR = aortic valve replacement; MVR = mitral valve replacement/reconstruction; VSD = ventricular septal defect; PDA = patent ductus arteriosus; CEA = carotid end-arterectomy; ASD = atrial septal defect; TVR = tricuspid valve replacement/repair; No WMA = no wall motion abnormalities; Hypokinesis = patients with areas of hypokinesis and no areas of akinesis/dyskinesis; Akinesis/Dysk = all patients with akinesis/dyskinesis with or without hypokinesis; LVT = left ventricular thrombus.

Data includes the type and number of procedures, number of cases in which left ventricular thrombi (LVT) was seen, and occurrence of wall motion abnormalities.

CASE REPORTS

Systemic embolism has been reported to occur in up to 36% of patients and is more likely with protruding or mobile thrombi than with laminar thrombi.^{5,6} The majority of thrombi occur in the apex and are associated with an abnormality of ventricular wall motion, particularly akinesis or dyskinesis.¹⁻⁴ Thrombi typically appear within 10 days^{1,4} of an MI and 96% are present within 2 weeks.⁴

Echocardiography has proven to be a useful tool in the diagnosis and evaluation of cardiac masses.⁷⁻¹¹ A mass is considered to be a thrombus when it has a separate endocardial surface, has different acoustic properties, and is seen in at least two different echocardiographic windows. Thrombi typically occur in areas of low flow, such as akinetic or dyskinetic myocardial segments.³ The differential diagnosis includes normal tissue (e.g., trabeculae), tumor, and artifact. Transthoracic echocardiography has been considered the preferred modality to visualize ventricular thrombi.⁵⁻⁹ Difficulties in detection of LVT have come from poor acoustic windows (transthoracic echocardiography, 25%),^{9,11} problems in obtaining true apical views, and differentiation from normal tissue structures such as apical trabeculae and false tendons. Laminar thrombi may be more difficult to identify than protruding thrombi. Although transthoracic echocardiography has been the preferred technique, two studies showed the superiority of TEE for detection of LVT, including those in the LV apex.^{1,2}

Management options for LV thrombi include anticoagulation without surgery^{12,13} or aneurysmectomy with thrombectomy.¹⁴ Studies are not consistent with regard to the benefits of anticoagulation. Spontaneous resolution of the thrombus without anticoagulation occurs in 30-76% of patients over 2 years.³ The perioperative mortality rate associated with aneurysmectomy may be as high as 22%¹⁴ and has been reserved for cases of failed maximal medical therapy for heart failure and embolism.¹⁵

Failure to remove protruding or mobile LVT may increase the risk of systemic embolism.⁵⁻⁷ Even minimal manipulation of the ventricle may dislodge, fracture, or dislodge and fracture the thrombus. Reevaluation of the thrombus before removal of the aortic cross-clamp necessitates filling the heart to allow echocardiographic visualization of the chambers. Retrieval of the thrombus may prolong bypass time and necessitates atriotomy, aortotomy, ventriculotomy, or each, all of which have attendant morbidity. However, prevention of systemic embolization may justify the increased risk.

We reported our experience with LVT. In these four

cases with protruding thrombi, the thrombi were no longer *in situ* at the discontinuation of CPB. When the thrombi were removed there were no neurologic sequelae. In the patients in cases 1 and 3 the thrombi could not be found after discontinuation of CPB. Both patients had fatal neurologic events. Case 4 is a good example of how a thrombus can be fragmented when manipulated.

Our practice includes a standard pre-CPB TEE evaluation in patients with a history of large MI, severe ventricular dysfunction, or both, to, among other things, evaluate for the presence of thrombus. If present before CPB, we will reexamine the cardiac chambers before discontinuing CPB, with the cross-clamp still present. The heart is filled to allow TEE imaging. If fragments are found to be freely mobile, the heart is emptied and the mass is removed. If imaging is inconclusive, epicardial ultrasound imaging is performed.

In conclusion, post MI LVT detected intraoperatively represents a risk factor for serious/fatal systemic emboli. These thrombi invariably occurred in areas of akinesis/dyskinesis and may go unnoticed despite preoperative ventriculography, transthoracic echocardiography, or both. Because protruding LVT may be dislodged or fragmented during CPB, TEE (or epicardial echo or both) evaluation before removal of the cross-clamp may prevent neurologic injury.

References

1. Reeder GS, Khandheria BK, Seward JB, Tajik AJ: Transesophageal echocardiography and cardiac masses. *Mayo Clin Proc* 1991; 66:1101-9
2. Chen C, Koschik D, Hamm C, Sievers B, Kupper W, Bleifeld W: Usefulness of transesophageal echocardiography in identifying small left ventricular apical thrombus. *J Am Coll Cardiol* 1993; 21:208-15
3. Asinger RX, Mikell FL, Elsparger J, Hodges M: Incidence of left ventricular thrombosis after acute transmural myocardial infarction. *N Engl J Med* 1981; 305:297-302
4. Funkekupper AJ, Vereugt FWA, Peels CH, Galema TW, Roos JP: Left ventricular thrombus incidence and behavior studied by two-dimensional echocardiography in acute anterior myocardial infarction: Left ventricular wall motion systemic embolism and oral anticoagulation. *J Am Coll Cardiol* 1989; 13:11514-20
5. Jugdutt BI, Sivaram CA, Wortman C, Trudell C, Penner P: Prospective two-dimensional echocardiographic evaluation of left ventricular thrombus and embolism after acute myocardial infarction. *J Am Coll Cardiol* 1989; 13:554-64
6. Visser CA, Kan G, Meltzer RS, Dunning AJ, Roelandt J, Corler MV, DeKoning H: Embolic potential of left ventricular thrombus after myocardial infarction: A two-dimensional echocardiographic study of 119 patients. *J Am Coll Cardiol* 1985; 5:1276-80
7. Stratton JR, Lighty GW Jr, Pearlman AS, Ritchie JL: Detection of left ventricular thrombus by two-dimensional echocardiography: Sen-

CASE REPORTS

sitivity, specificity, and causes of uncertainty. *Circulation* 1982; 66: 156-66

8. Mugge A, Daniel WG, Haverich A, Lichtlen PR: Diagnosis of noninfective cardiac mass lesions by two-dimensional echocardiography: Comparison of the transthoracic and transesophageal approaches. *Circulation* 1991; 83:70-8

9. Visser CA, Kan G, David GK, Lie KI, Durrer D: Two dimensional echocardiography in the diagnosis of left ventricular thrombus. *Chest* 1983; 83:228-32

10. Stratton JR, Ritchie JL, Hammermeister KE, Kennedy JW, Hamilton GW: Detection of left ventricular thrombi with radionuclide angiography. *Am J Cardiol* 1981; 48:565-72

11. Ezekowitz MD, Wilson DA, Smith EO, Burrow RD, Harrison LH Jr.,

Parker DE, Elkins RC, Peyton M, Taylor FB: Comparison of indium-111 platelet scintigraphy and two-dimensional echocardiography in the diagnosis of left ventricular thrombi. *N Engl J Med* 1982; 306:1509-13

12. Ebert RV: Anticoagulants in acute myocardial infarction. *JAMA* 1973; 225:724-9

13. Keating EC, Gross SA, Schlamowitz RA, Galssman J, Mazur JH, Pitt WA, Miller D: Mural thrombi in myocardial infarctions. *Am J Med* 1983; 74:989-95.

14. Graber JD, Oakley CM, Peckering BN, Goodwin JF, Raphael MJ, Steiner RE: Ventricular aneurysms: An appraisal of diagnosis and surgical treatment. *Br Heart J* 1972; 34:830-8

15. Cohen M, Packer M, Gorlin R: Indications for left ventricular aneurysmectomy. *Circulation* 1983; 67:717-22

Anesthesiology

1998; 89:1262-4

© 1998 American Society of Anesthesiologists, Inc.

Lippincott Williams & Wilkins

Massive Pyramidal Tract Signs after Endotracheal Intubation: A Case Report of Spondyloepiphyseal Dysplasia Congenita

Gerhard Redl, M.D.*

SURGERY in children with dwarfism is often a challenge to the anesthesiologist because of craniocervical abnormalities, respiratory problems, and behavioral disturbances. I report a case of a young patient with spondyloepiphyseal dysplasia in whom massive pyramidal tract signs developed after surgery and tracheal intubation.

Case Report

An 18-yr-old man with spondyloepiphyseal dysplasia congenita was scheduled for surgery to remove retained hardware after surgical correction of a genu valgus. His height was 120 cm and his weight was 30 kg. Medical history included diabetes mellitus type I treated with insulin. Former surgery of the genu valgus, 3 months previously, occurred uneventfully. Preoperative physical examination revealed thoracic kyphoscoliosis, a short neck, systolic murmur, and respiratory arrhythmia. Laboratory data were within the normal range with the exception of the blood glucose level. Plain radiography of the cervical spine (no flexion/extension series) noticed only "little abnormality

caused by spondylosis." Surgery was planned using a balanced anesthetic technique. After administration of a nondepolarizing muscle relaxant (vecuronium) and controlled ventilation for 3 min by mask, the intubation was performed during direct laryngoscopy with a McIntosh 3 laryngoscope blade (Rüsch, Vienna, Austria). The procedure was performed by an experienced staff anesthesiologist without using a cervical collar. During induction and intubation, the head was maintained in moderate flexion. The anesthesiologist could visualize only the posterior commissure, but nevertheless placed the tube, with a guide wire inside, on the first pass. He described the procedure as "not very difficult." The intraoperative course occurred uneventfully. The patient was extubated, and the early postoperative period in the recovery room and at the ward was uneventful. On the first postoper-



Fig. 1. Computerized tomography of the cervicooccipital region.

* Associate Professor.

Received from the Department of Anesthesiology, Orthopedic Hospital Vienna-Speising, Vienna, Austria. Submitted for publication March 10, 1998. Accepted for publication June 11, 1998.

Address reprint requests to Dr. Redl: Orthopedic Hospital Vienna-Speising, Department of Anesthesiology, Speisinger Strasse 109, A-1134 Vienna, Austria. Address electronic mail to: gredl@aon.at

Key words: Difficult airway; dwarfism; fiberoptic intubation.