in the event of myocardial ischemia, SACE increases markedly at maximum effort as well as during recovery. Our data on SACE activity in nonischemic subjects support similar results obtained by Milledge and Catley,11 who found SACE to remain unchanged during extended stress testing (up to 60 minutes) in normal healthy subjects. In light of our findings, we propose that SACE elevation could reflect acute endothelial vascular damage (both in general and in particular in the cardiac vasculature) in subjects with clinical and ECG signs of myocardial ischemia during stress testing (regardless of the duration of exercise).

REFERENCES

Protruding left ventricular thrombus formation following blunt chest trauma

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Following myocardial contusion, attention has been traditionally directed toward the hemodynamic derangement that it produces as well as life-threatening ventricular arrhythmias. Little attention has been devoted to the possible formation of thrombus and the potential thromboembolic hazards thus evoked. A case is presented of a young patient with normal coronary arteries in whom an unnoted blunt trauma to the chest gave rise to septoapical dyskinesia and protruding left ventricular (LV) thrombus formation.

A 35-year-old man with no history of heart disease or cardiac risk factors was referred to a surgical department of our hospital following maxillofacial trauma. He was pushed from a one-story structure, falling on a slanted surface scattered with fairly large poles. An open fracture of the left zygomatic bone was accompanied with swelling and the appearance of hematoma around the lateral aspect of his left eye. Apart from pain at the site of trauma, the patient complained of blurred vision and troublesome irritation in his eye. At the time of admission, no history or evidence of chest trauma was obtained. Three days later, in the operating room and just before surgical repair and repositioning of the zygomatic fracture, he complained of sharp precordial pain on deep inspiration, radiating to his left arm. An electrocardiogram (ECG) performed in the operating room showed a right bundle branch block–left anterior hemiblock pattern and QS complex in the precordial leads (Fig. 1). At this stage, surgery was postponed, and the patient was transferred to the coronary care unit. When seen for the first time in the unit, his blood pressure was 120/80 mm Hg and the pulse rate was 64 beats/min. On examination, there was tenderness over the left precordium and a fourth heart sound was audible. As the possibility of chest trauma was considered, careful ammnesia elicited that the patient had probably suffered bruising of the left anterior chest wall. A chest x-ray film was normal. Continuous monitoring revealed frequent ventricular premature beats and short runs of ventricular tachycardia. Cardiac enzyme levels including creatine kinase MB fraction were indicative of infarction. Two-dimensional echocardiography performed the next day showed septoapical dyskinesia and an LV mass protruding into the chamber cavity with marked mobility (Fig. 2). The mass length from apex to the mobile end reached 1.2 cm. Cardiac catheterization demonstrated patent coronary arteries. Ventriculography was not performed for fear of dislodging the thrombus. The patient was treated by continuous intravenous heparin infusion (1000 units per hour), followed by warfarin treatment. Follow-up echocardiographic examination at 1 week, 2 weeks, and 1 month later showed gradual regression of the thrombus dimensions, with diminished protrusion and mobility. At a 3-month follow-up, echocardiographic findings were compatible with mural nonprotruding apical thrombus. No sequelae of embolization or further ECG changes were noted during the 4-month follow-up period.

Ventricular thrombus, logically expected in the setting of myocardial contusion, is surprisingly a relatively rare echocardiographic finding. In three prospective echocardiographic studies involving 279 individuals, thrombus was documented in none of the patients suffering myocardial contusions.
Fig. 2. Two-dimensional two-chamber apical view showing a protruding thrombus in the dyskinetic septoapical area of the left ventricle.

However, there is more than anecdotal evidence to suggest that in those patients demonstrating wall motion abnormalities related to cardiac contusion, a high index of suspicion should be maintained to ensure that no clinical sequelae of ventricular thrombus may occur. In this context, it has been recently concluded by Wisner et al. that most of the patients who are prone to life-threatening cardiac complications from blunt cardiac injury can be identified soon after the traumatic event in an emergency room setting. However, one should bear in mind that thrombus could easily be missed if echocardiography is performed immediately after blunt contusion. As in myocardial infarction, it is not inconceivable that ventricular thrombus, either mural or protruding, will be seen 2 or 3 days following myocardial contusion, giving rise to a delayed embolic complication. As thrombus formation is promoted by severe wall motion abnormality, echocardiographic follow-up is strongly recommended in this subset of patients demonstrating early wall motion abnormalities.

Embolic complications in patients with thrombus-related myocardial contusion have been sparse. It remains debatable whether an echocardiographic finding of a mural thrombus warrants prophylactic anticoagulation. The therapeutic approach might be entirely different when protruding thrombus is apparent, mainly because of its higher propensity (up to 60% of cases) toward embolization. Although anticoagulation has questionable benefits and is often contraindicated in patients with multiple trauma, one should consider the potential embolic risk versus the hazards associated with prophylactic anticoagulation. In the present case, there was a relatively minor contraindication for systemic anticoagulation, while the risk of embolization seemed high in view of the thrombus configuration. Anticoagulation proved to be efficient, decreasing the size of the thrombus and presumably reducing the potential risk for embolization. Thrombus formation following myocardial contusion may be related either to direct myocardial insult or to compression of a coronary artery, resembling myocardial infarction. The first seems the more plausible explanation in our patient, who was thought to have myocardial contusion based on elevation of serum cardiac enzymes, electrical instability, mechanical dysfunction, and a normal coronary angiogram. We have been able to find in the literature only two reported cases of ventricular protruding thrombus induced by blunt chest trauma.

Cardiac injury is a common and frequently unsuspected visceral injury. Patients may be free of any cardiac symptoms or may have transient complaints. In the present case, chest trauma was probably masked by the major maxillofacial injury. As we noted, even "innocent" trauma may result in protruding thrombus formation, and had the patient not complained of chest pain, the thrombus could have gone undetected, with the patient exposed to both operative and a major delayed embolic risk.

REFERENCES
Sinus of Valsalva aneurysms (SVA) involving a single sinus are felt to be caused by localized congenital absence of the media of the aorta. Patients with this condition are generally asymptomatic unless aneurysm rupture occurs. Rarely, patients with unruptured aneurysms may present with right ventricular outflow obstruction, myocardial infarction as a result of coronary artery compression, conduction disturbances, or endocarditis. We report a patient with an unruptured SVA who presented with central nervous system embolization. We also describe the utility of transesophageal echocardiography, both for diagnostic and intraoperative evaluation of this condition.

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A previously healthy 45-year-old woman presented with the sudden onset of temporal visual field loss in the right eye. Diagnosis of acute occlusion of a branch of the right retinal artery was made by an ophthalmologist. The patient related a similar episode 3 months before in which she experienced transient right-sided hemiparesis. There was no history of fever, chills, night sweats, or anorexia. Her past medical history was significant for patent ductus arteriosus requiring surgical ligation at age 5 years. On physical examination, her temperature was 98.6°F, the blood pressure was 120/74 mm Hg, and the heart rate was 76 beats/min. There were no petechiae, splinter hemorrhages, carotid bruits, jugular venous distention, or organomegaly. Funduscopic examination revealed occlusion of a branch of the right retinal artery. Cardiovascular examination was normal. The remainder of the examination was unremarkable. Laboratory studies revealed a white blood cell count of 9400/mm³, packed cell volume of 39.6%, platelet count of 168,000/mm³, and an erythrocyte sedimentation rate of 13 mm/hr. Blood cultures and Venereal Disease Research Laboratory (VDRL) were negative. The chest roentgenogram was normal. The electrocardiogram showed normal sinus rhythm and left atrial abnormality. Carotid duplex ultrasonography and cranial computed tomography were normal.

M-mode and two-dimensional transthoracic echocardiography showed normal chamber dimensions and left ventricular systolic function. The aortic valve was trileaflet without thickening or vegetations. A large aneurysm measuring 3 x 3 cm originated from the noncoronary sinus of Valsalva and protruded into the right atrium above the septal leaflet of the tricuspid valve. Thrombus was evident within the aneurysm (Fig. 1). Mild aortic insufficiency was seen by color flow imaging and by continuous wave Doppler. No perforation was seen by Doppler or by contrast echocardiography. Transesophageal echocardiography (TEE) better visualized the ascending aortic root, the sinuses of Valsalva, the aortic valve, the proximal coronary arteries, and right-sided chambers. TEE confirmed the presence of thrombus within the noncoronary SVA (Fig. 2). Examination of the right atrium and right ventricle with a four-chamber frontal plane view demonstrated protrusion of the SVA into the right atrium just above the tricuspid valve (Fig. 3). Color flow turbulence and a pulse wave Doppler gradient were seen across the tricuspid valve, indicating aneurysmal impingement on right ventricular filling. Pulse wave Doppler and color flow imaging excluded aneurysmal rupture. This was further confirmed by the absence of a negative contrast effect during saline contrast injection. The patient underwent corrective surgery. At operation, visual inspection confirmed an aneurysm of the noncoronary sinus of Valsalva filled with thrombus. The aortic valve was structurally normal and the right coronary artery was not involved. A Dacron patch was sutured over the orifice of the SVA producing a blind pouch. Intraoperative TEE was employed to assess the degree of residual aortic insufficiency and left ventricular function after patch placement. The postoperative course was uneventful, and at 2-year follow-up the patient remained asymptomatic.