Mechanical Complications of Acute Myocardial Infarction

37

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Key Points

- Mechanical complications after myocardial infarction are very difficult to diagnose especially in patients who present with severe respiratory distress and shock.
- Quick diagnosis is vital, and it is obtained by echocardiography.
- Patients should undergo emergency surgical treatment.
- The infarcted myocardium is friable, and the endocardium may be covered in part by mural thrombi. Therefore, before aortic cross-clamping, carefully manipulate the heart in order to prevent dislodging necrotic tissues and thrombi.
- The use of the intra-aortic balloon pump and vasodilator drugs decreases both systolic arterial pressure (afterload) and left ventricular end-diastolic pressure (preload), increasing at the same time cardiac output. In this way, chances of survival are significantly improved until surgical treatment.

37.1 Introduction

The main acute mechanical complications (MC) of acute myocardial infarction (AMI) are:

- Ventricular septal rupture (VSR)
- Free wall rupture (FWR)
- Ischemic mitral regurgitation (IMR)

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The occurrence of the MC after AMI is associated with an increased early and late mortality [1]. Anyway, the clinical introduction of primary percutaneous coronary intervention (PCI) has markedly declined the incidence of MC to less than 1%, including VSR (0.17%), FWR (0.52%), and IMR (0.26%). MC are very difficult to diagnose, especially in an emergency situation in patients who present with severe respiratory distress and shock. As matter of fact, echocardiography allows a rapid and precise diagnosis followed by emergency surgery. Intra-aortic balloon pump (IABP) insertion and vasodilator infusion reduce afterload increasing, at the same time, cardiac output.

37.2 Ventricular Septal Rupture (VSR)

Although percutaneous coronary intervention has been making VSR after AMI increasingly rare, mortality remains high. Postmyocardial infarction VSR is most commonly localized in the anterior or apical section of the ventricular septum (VS) as a consequence of anterior AMI. In about 20–40% of patients who had inferior AMI, the VSR is localized in the posterior portion of VS. VSR often occurs with complete occlusion of a coronary artery, in the majority of cases, the left anterior descending coronary artery (LAD), and usually collateral coronary circulation is poor [2]. In the past, before thrombolysis and PCI, VSR occurred in 1–2% of patients who had an AMI, generally within a week the onset of infarction [3, 4]. VSR determines shunting of blood from the left to the right ventricle, and the overall shunt volume depends on the pressure gradient across it and on the size of the defect. VSR eventually produces right ventricular failure and cardiogenic shock; left ventricular failure may be caused by AMI extent.

Physical examination reveals a holosystolic murmur, best heard at the left sternal edge, and a parasternal thrill may be present; it is important a differential diagnosis with mitral insufficiency due to papillary rupture where the murmur is

P. Aseni et al. (eds.), *Operative Techniques and Recent Advances in Acute Care and Emergency Surgery*,
https://doi.org/10.1007/978-3-319-95114-0_37

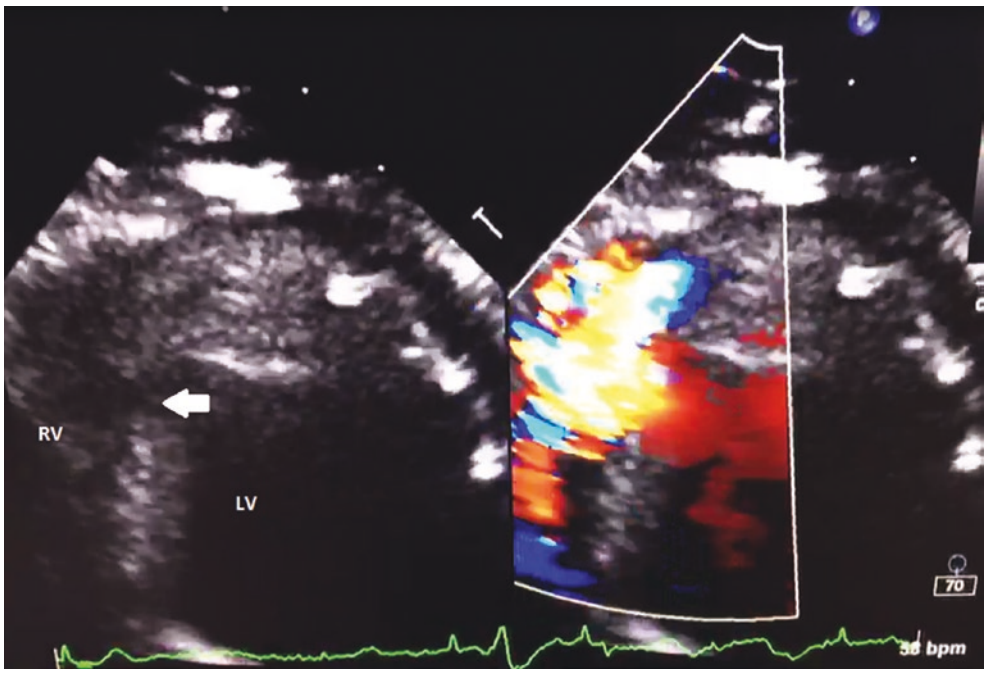


Fig. 37.1 In the left panel, the white arrow shows VSR; in the left panel, color Doppler flow demonstrates a large turbulent left-to-right flow. *VSR* ventricular septal rupture, *RV* right ventricle, *LV* left ventricle

loudest at the apex. Electrocardiogram shows anterior or inferior infarction with ST elevation (STEMI), and chest X-ray may highlight an enlarged heart and pulmonary congestion. Echocardiography with color Doppler flow (Fig. 37.1) is mandatory for diagnosis, showing the VSR site/size and estimating the left-to-right shunt volume.

37.2.1 VSR Surgical Repair

Surgery is done on an emergency basis after diagnosis. IABP insertion and vasodilator drug infusion can stabilize the patient determining an afterload reduction and cardiac output increase.

After median sternotomy, cardiopulmonary bypass is established via venous bicaval cannulation and arterial cannulation of the distal portion of the ascending aorta. The aorta is cross-clamped; cardioplegic solution is infused in ascending aorta but also through the coronary sinus route due to the acute coronary occlusion. Concomitant myocardial revascularization should be done whenever significant coronary artery disease is present and the vessel periphery is suitable. Then, the left ventriculotomy is carried out through the infarcted area in the anterior or apical aspect of the ventricle. The infarcted area is easily recognized by placing a suction probe after the institution of cardiopulmonary bypass: the recent infarcted area and/or a thinned scar of an old myocardial infarct pucker defining the limits within the incision is made. When VSR is located anteriorly, ventriculotomy is done about 1 cm lateral to the left anterior descending (LAD) coronary artery (Fig. 37.2). The septal defect is located below the incision (Fig. 37.3). At this point two surgical repairs are most commonly used.

(a) *Daggett technique*. This technique was introduced in 1977 by Willard M. Daggett [5]. The defect is repaired by suturing a patch of autologous or bovine pericardium. The ventricular septum is not resected, and the patch is tailored for the shape of the defect, covering the adjacent intact septum. The patch is sutured on the LV side of the septum using pledgeted mattress sutures, generally 3.0 polypropylene suture, with pledgets positioned on the right ventricular aspect of the defect. The patch can also be sutured with a continuous 3.0 or 4.0 polypropylene suture. Infarctectomy or aneurysmectomy is performed, and the ventriculotomy is closed using buttressed sutures; if the excised area is too large, a polyester or pericardial patch is sutured.

(b) *David technique*. This is the infarct exclusion technique introduced in 1987 by Tirone E. David [6]: a patch of autologous or bovine pericardium or Dacron is employed to separate the left ventricle from the VSR. Essentially, a new left ventricle is made realizing a new septum beside VSR that is inglobated in the right ventricle. The patch is sutured starting from the inferior border of VSR, and the suture is continued cephalad; stitches are passed through normal myocardium, and also the mitral annulus may be used to secure the patch to healthy tissue (Fig. 37.4) [7]. The ventriculotomy is eventually closed using buttressed sutures (Fig. 37.5).

In case of posterior VSR, it is important to know right ventricular function because the right ventricle is often involved; furthermore, mitral valve function should be assessed and, consequently, evaluated prior to a valve repair/substitution procedure. Posterior VSR is more difficult to expose and repair: the heart is lifted out of pericardial cavity,

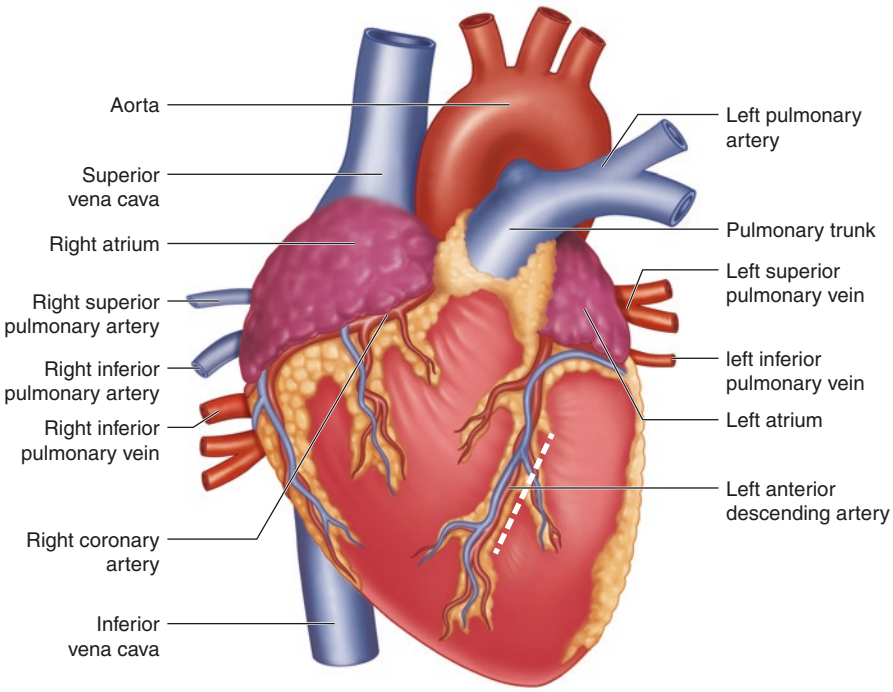


Fig. 37.2 Dashed line marks the incision area, about 1 cm beside the left anterior coronary artery

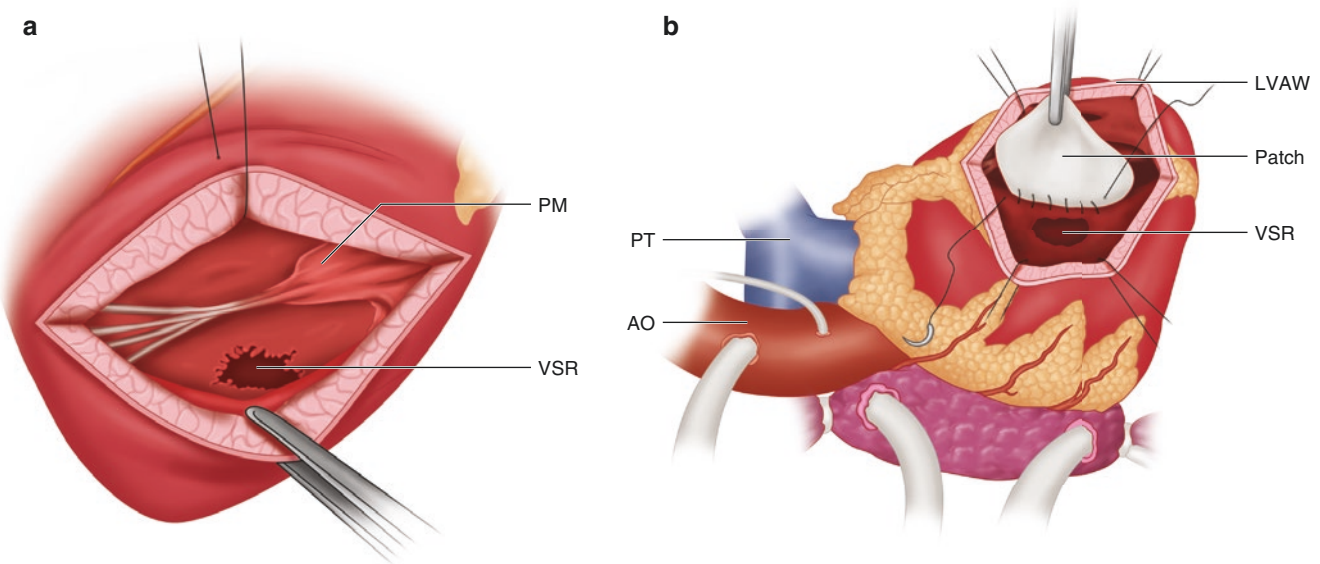


Fig. 37.3 Surgeon view. (**a**) The left ventricle is opened 1 cm lateral to left anterior descending coronary artery ventricular septal rupture (*VSR*) is located underneath the incision. (**b**) the patch is sutured starting for the posterior border of VSR using a 3-0 continuous polypropylene (papillary muscle (*PM*), left ventricle anterolateral wall (*LVAW*))

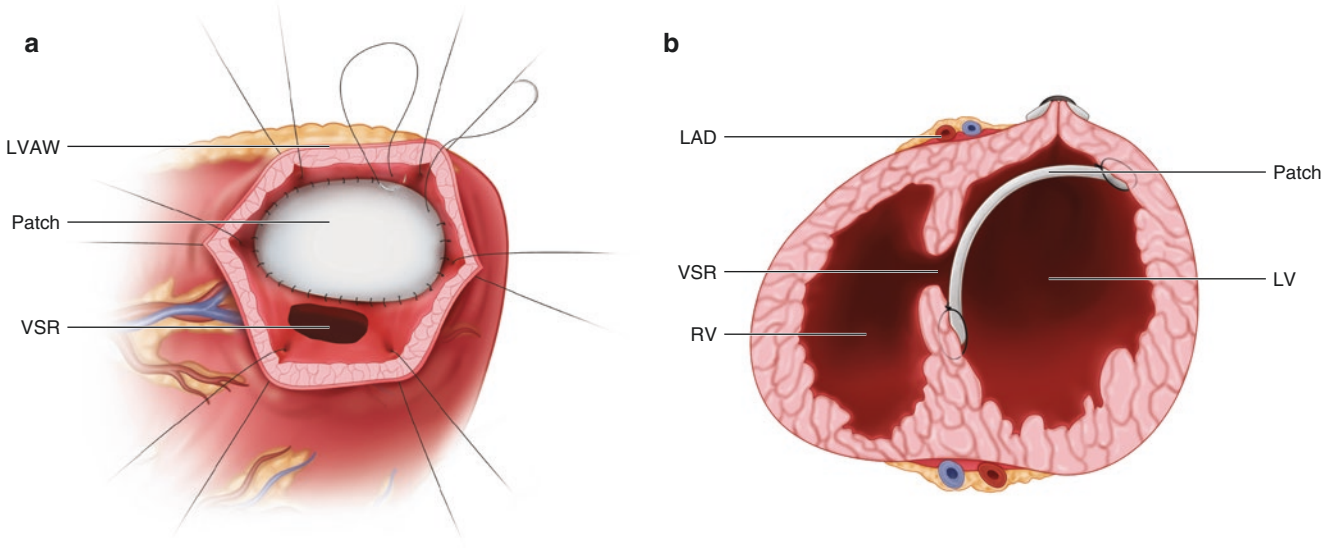
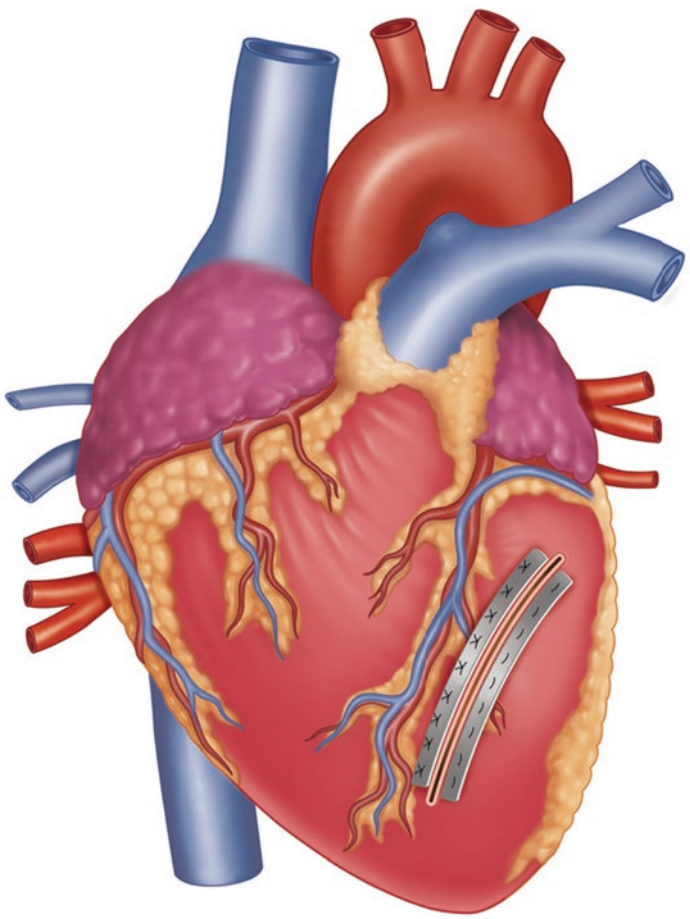


Fig. 37.4 (**a**) Suture line is continued into the LVAW. (**b**) Transverse section of the heart: David's technique allows to exclude LV from the infarcted myocardium (right ventricle (*RV*), left ventricle (*LV*))

Fig. 37.5 The left ventriculotomy is closed using buttressed sutures



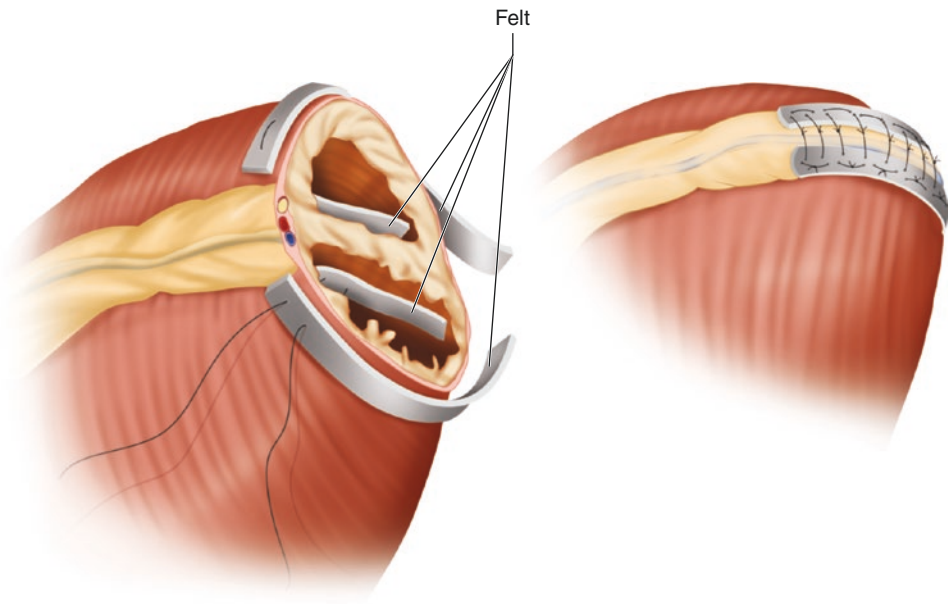


Fig. 37.6 VSR in the apical portion of the septum: the procedure is directly done by amputating the apex of the heart, together with the involved portion of the ventricular septum. The closure is performed by applying strips of polytetrafluoroethylene felt on each side of the septum and on edges of both ventricles

and the left ventricle is incised; VSD is approached through an incision in infarcted left ventricle parallel to the posterior descending coronary artery. The patch is positioned in the same fashion on the left ventricular side.

When the VSR is situated in the apical portion of the septum with an apical AMI, the procedure is directly done amputating the apex of the ventricle, together with the involved portion of the ventricular septum (Fig. 37.6).

It is remarkable to read what Dr. David said in 2012 at the 48th Annual Meeting of the Society of Thoracic Surgeons, Fort Lauderdale, FL, Jan 28–Feb 1, 2012: "… I was able to change the classical technique of infarctectomy and patches with a stiff Dacron graft (DuPont, Wilmington, DE) developed by Bill Daggett in Boston to a more conservative approach whereby we simply opened the infarct and excluded the infarcted muscle and ruptured septum by using a soft patch such as pericardium secured to the healthy endocardium around the infarct. We named the technique "infarct exclusion," and we were able to dramatically reduce, at least my personal mortality, particularly in patients with posterior septal rupture, who had the highest mortality 20–30 years ago …" [8].

37.3 Free Wall Rupture (FWR)

Left ventricular FWR is one of the most lethal complications following myocardial infarction. It occurs 4–10 times more often than rupture of the interventricular septum or of a papillary muscle, two conditions for which surgical treatment is widely applied. However, surgical treatment of cardiac rupture remains a surgical challenge for the high mortality even before the diagnosis is made.

FWR generally occurs between 1 and 7 days after AMI, a *blow out type rupture* determines the sudden patient's death for cardiac tamponade; FWR is more often a gradual process, starting with small areas of endocardial necrosis, allowing formation of hematomas that progressively dissect through the necrotic myocardium into the pericardium, resulting in tamponade and cardiogenic shock. FWR occurs in most cases on the lateral or anteroapical left ventricle. In the FRW the myocardial necrotic tissue appears weakened and friable, not ensuring stable anchoring sites for sutures; therefore, it is often necessary to sacrifice viable areas of myocardium with the result of significant reduction of LV dimensions. Various surgical techniques have been utilized, including pericardial patch placement with epicardial sutures or applying biological glue, infarctectomy with patch placement and ventricular wall reconstruction, pledgeted sutures without infarctectomy, and pericardial, Dacron, Gore-Tex, or Teflon patches adhered with biologic glue or sutures [9]. New techniques allow to preserve the left ventricular cavity size and viable myocardial tissue around the FWR. Sutureless techniques include gelatin-resorcinol-formaldehyde (GRF glue: Cardial, Saint-Etienne, France) applied to a bovine pericardial patch (Impra, Tempe, AZ, USA) [10] and collagen fleece with fibrinogen-based impregnation [11] (Tachocomb; Nycomed Pharma, Linz, Austria).

37.3.1 Clinical Case

A 78-year-old man was transferred from a smaller hospital with cardiac tamponade and aortic dissection diagnosis. Central venous pressure was 20 mmHg, he had anuria, and

his face and his upper chest were extensively cyanotic. The chest was entered through a median sternotomy (Fig. 37.7). The neck vessels were isolated before pericardial incision (Figs. 37.8 and 37.9). Peripheral extracorporeal circulation was started after femoral vessels' cannulation. The pericardium was opened, no aortic dissection was found, and blood and clots were removed. A fissuration was found on the lateral aspect of the left ventricle (Fig. 37.10). Repair was accomplished on the beating heart in order to minimize ischemia and to allow assessment of wall function: a wide Teflon patch was applied using gelatin-resorcinol-formaldehyde biological glue (Figs. 37.11 and 37.12).

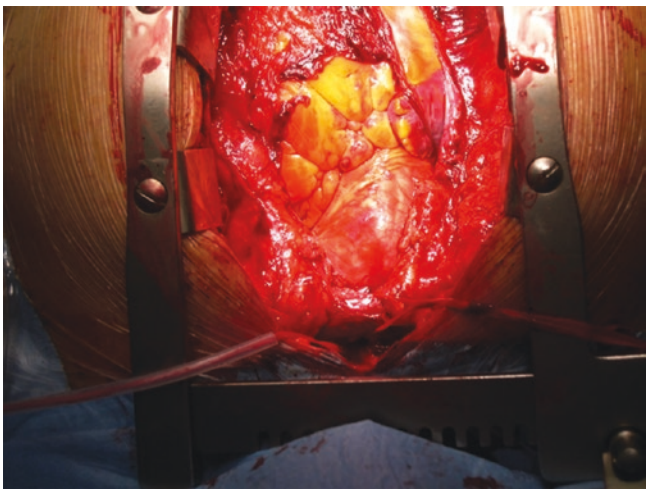


Fig. 37.9 The heart, anterior aspect, after blood and clots removal

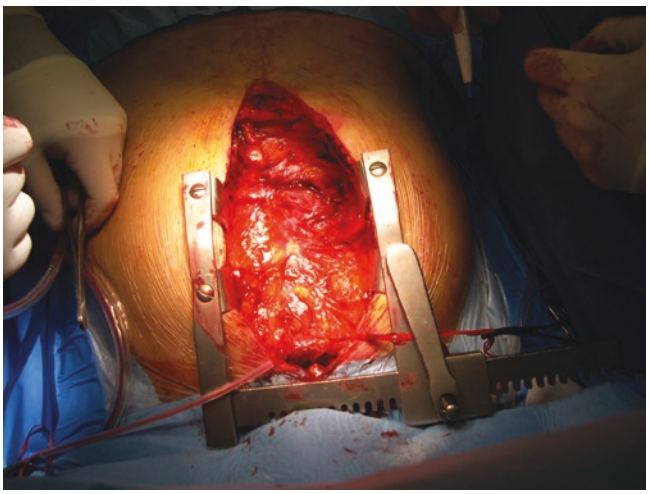


Fig. 37.7 After median sternotomy left innominate vein and brachiocephalic artery are encircled

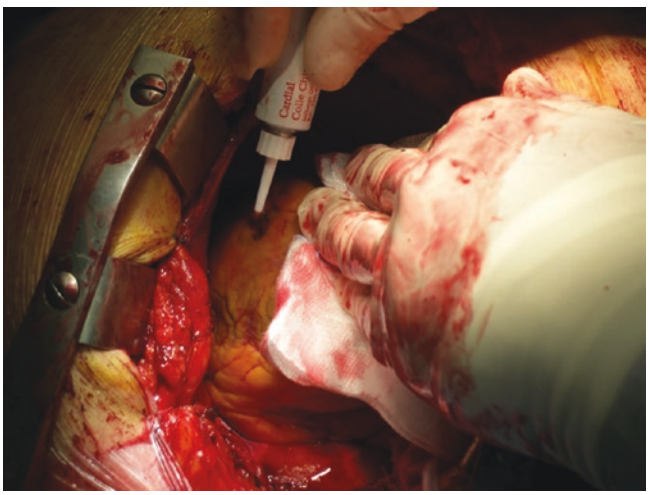


Fig. 37.10 A fissuration was found on the lateral aspect of the left ventricle

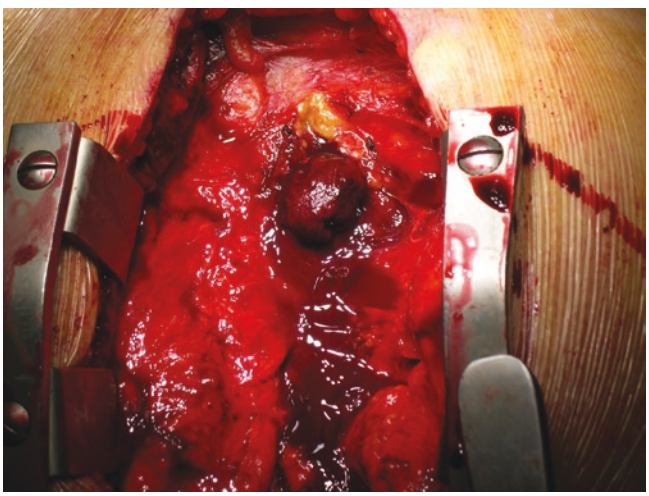


Fig. 37.8 The pericardium is incised and large amounts of blood and clots are removed

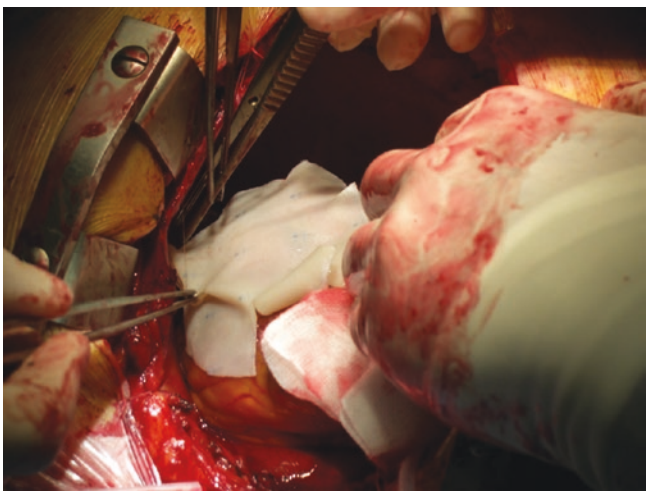


Fig. 37.11 Repair was accomplished on the beating heart in order to minimize ischemia and to allow assessment of wall function: a wide Teflon patch was applied using gelatin-resorcinol-formaldehyde biological glue

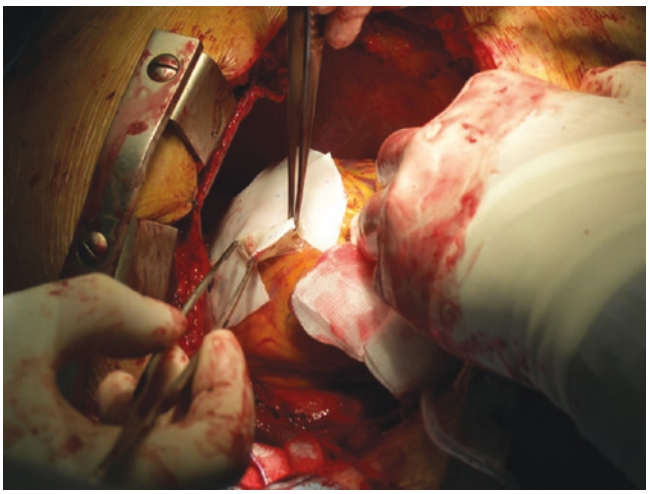


Fig. 37.12 Teflon patch applied to the left ventricle

37.4 Ischemic Mitral Regurgitation (IMR)

In the acute phase of acute myocardial infarction (AMI), IMR is caused either by papillary muscle dysfunction/rupture or rupture of chordae. Papillary muscle rupture may occur from a few hours to 14 days after acute myocardial infarction, and its incidence is about 1% [12] and increases exponentially in fatal AMI %.

37.4.1 Etiology

Majority of patients presents inferoposterior AMI; the posteromedial papillary muscle is involved more frequently than anterolateral papillary muscle, this is in about 75% of cases. This depends on the different blood supply: the posteromedial muscle is perfused by the posterior descending artery, while the anterolateral muscle is perfused by both the left anterior descending and left circumflex coronary arteries. For this reason, posteromedial papillary muscle rupture occurs more frequently with relatively small infarcts [13].

37.4.2 Symptoms

Acute mitral regurgitation is usually characterized by sudden pulmonary edema, hypotension, and cardiogenic shock.

Diagnosis. The diagnosis is often difficult to make; echocardiography, transthoracic and transesophageal (Figs. 37.13 and 37.14), is essential also in differentiating between papillary muscle rupture and left ventricular dysfunction. Chest X-ray shows pulmonary congestion, an enlarged heart, and often bilateral pleural effusion. Right heart catheterization, positioning a Swan-Ganz catheter, will show a prominent "v wave," and it is important to exclude a left-to-right shunt (differential diagnosis with VSR).

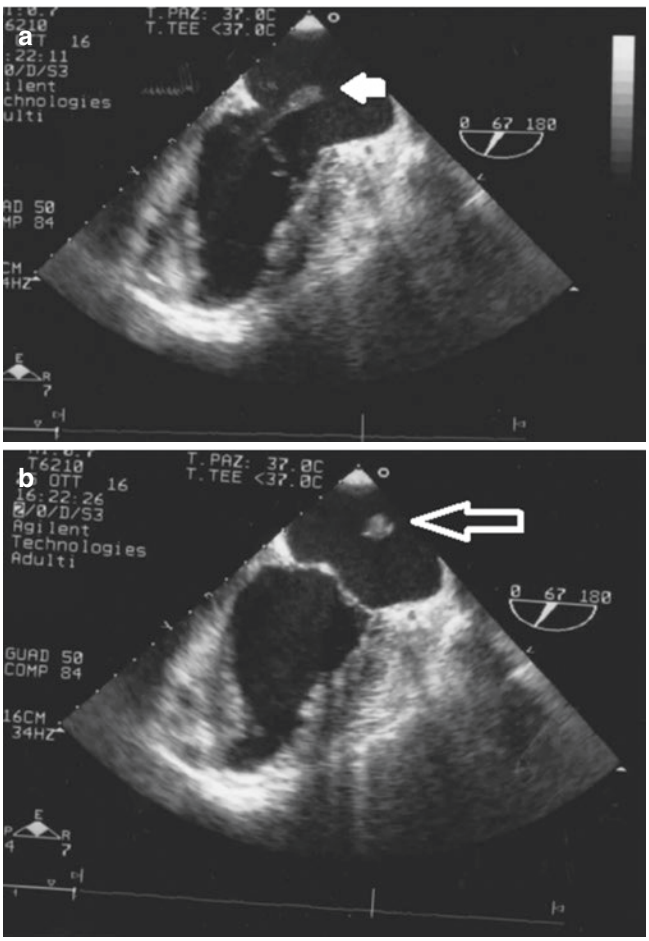


Fig. 37.13 In panel A and B, transesophageal echocardiogram showing a free-floating appendage, attached to the mitral valve, in the left atrium (white arrows). The prolapsing appendage is the ruptured portion of the papillary muscle

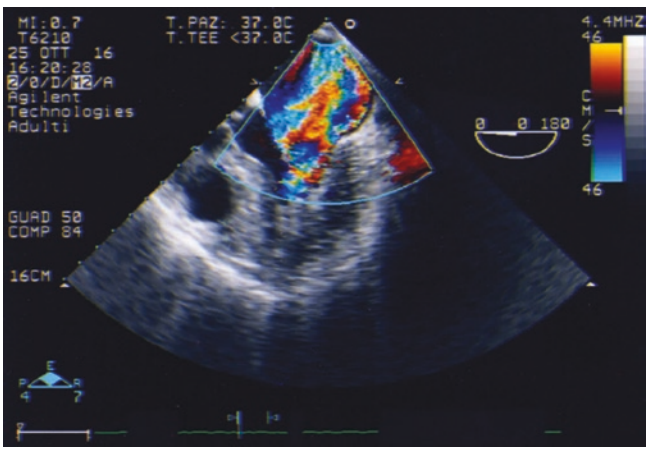


Fig. 37.14 Doppler color flow shows severe mitral regurgitation

37.4.3 Surgery

It is mandatory to try to stabilize the patient positioning intra-aortic balloon pump. Surgery is performed on an emergency basis immediately after diagnosis. In Figs. 37.13, 37.14, 37.15, 37.16, 37.17, and 37.18 we report the clinical case of a 66-year-old man who developed IMR 4 days after posterior AMI. He had sudden pulmonary edema and congestive heart failure; echocardiography showed massive mitral regurgitation (Fig. 37.14), and intra-aortic balloon pump was immediately positioned. Coronary angiography revealed circumflex coronary artery occlusion and LAD proximal critical disease. At surgery a saphenous sequential coronary bypass to obtuse marginal and LAD coronaries was first performed; then the mitral valve was exposed, the ruptured posteromedial papillary muscle was excised (Figs. 37.17 and 37.18), and the mitral valve was replaced with a mechanical prosthesis.

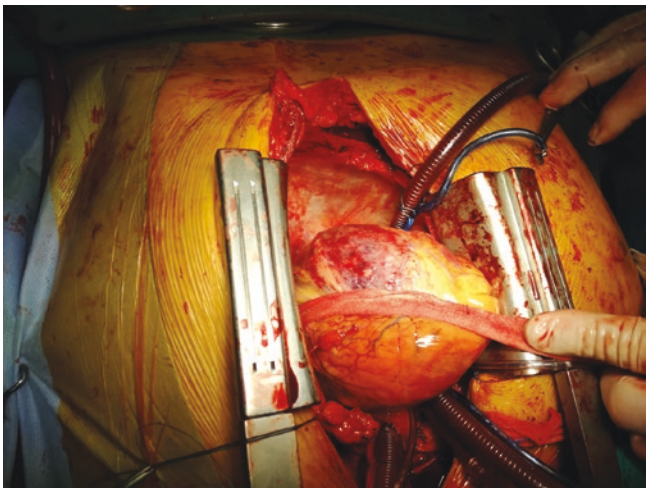


Fig. 37.15 Inferoposterior myocardial infarction

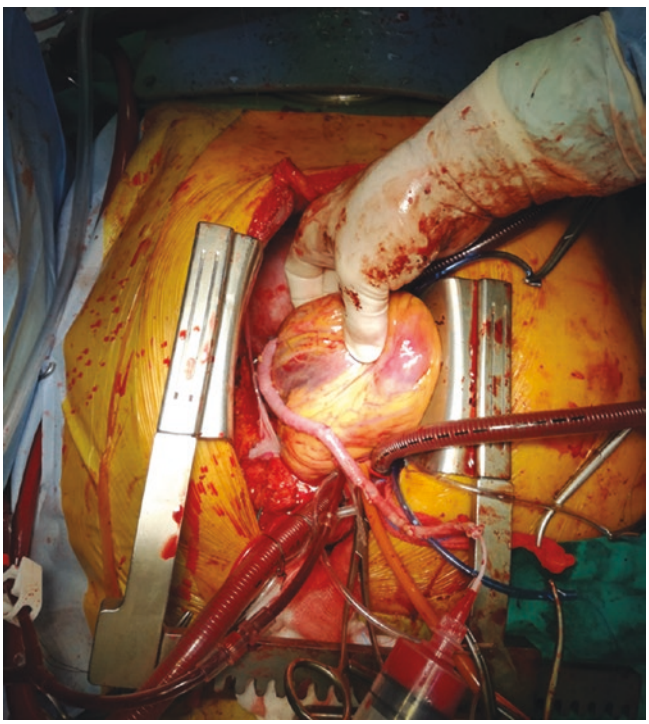


Fig. 37.16 Saphenous vein graft to obtuse marginal and left anterior descending arteries in the sequential fashion

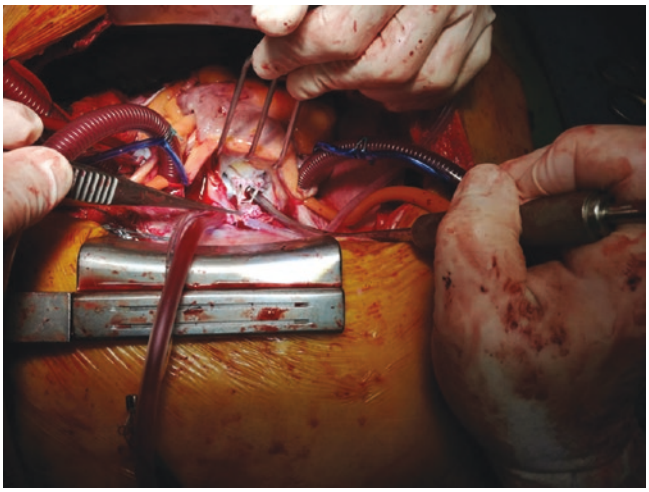


Fig. 37.17 The posteromedial papillary muscle is completely resected

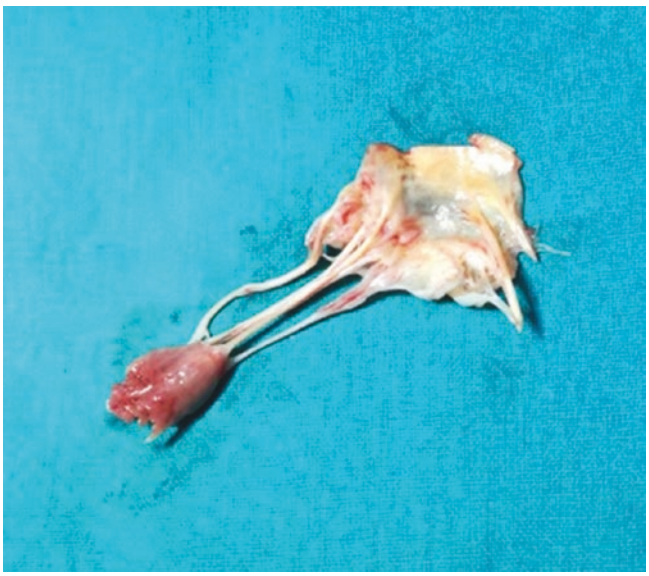


Fig. 37.18 The ruptured apical portion of the posteromedial papillary muscle after surgical excision

Mitral valve repair is often performed in IMR; anyway it is interesting to observe the results of "the mitral valve repair versus replacement for severe ischemic mitral regurgitation trial" [14] where 251 patients with severe ischemic MR were randomized to mitral valve repair or chordal-sparing mitral valve replacement. The primary end point was the left

ventricular end-systolic volume index (LVESVI) at 12 months and secondary endpoints were major adverse cardiac and cerebrovascular events, mortality, degree of residual MR, functional status, and quality of life. It is remarkable to note that recurrence of moderate or severe MR at 1 year was 32.6% in the repair group and only 2.3% in the replacement group ($P < 0.001$). In patients that had valve repair, the LVESVI was 64.1 ± 23.9 mL/m^2 in those with recurrent MR versus 47.3 ± 23.0 mL/m^2 in those without recurrent MR ($P < 0.001$). These results suggest that valve repair can dramatically improve LVESVI, but a multidisciplinary team should analyze all the preoperative findings that can indicate and predict a long-lasting repair. Otherwise it is advisable to perform a mitral valve replacement procedure.

37.5 When the Diagnosis Is Missed

You will find hereafter some clinical cases where the missed diagnosis determined the patient's death.

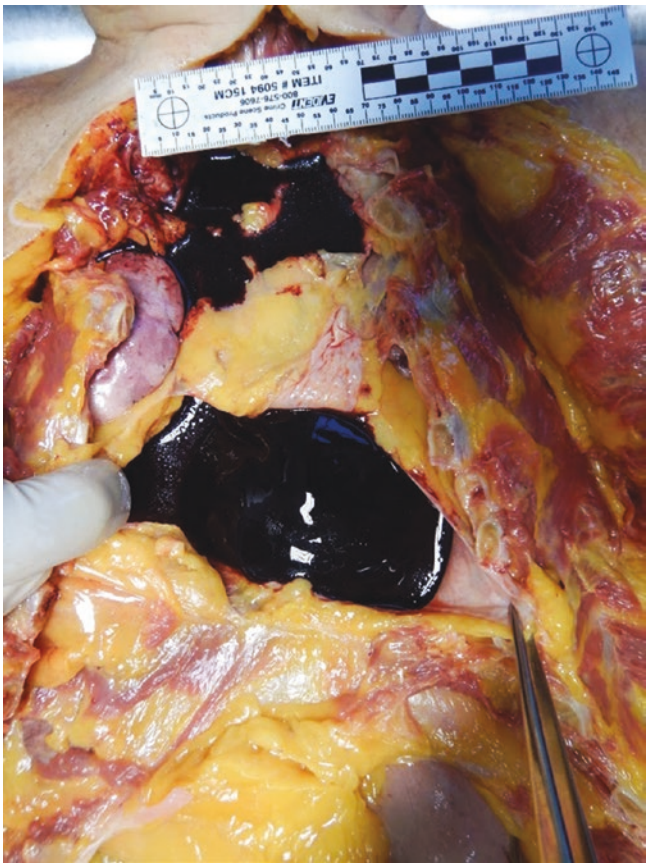


Fig. 37.19 Clinical case 1. Upon removal of the sternal plastron, the lungs were expanded, without adhesions in the pleural cables; the pericardium was intact and contained about 200 mL of blood, partially coagulated

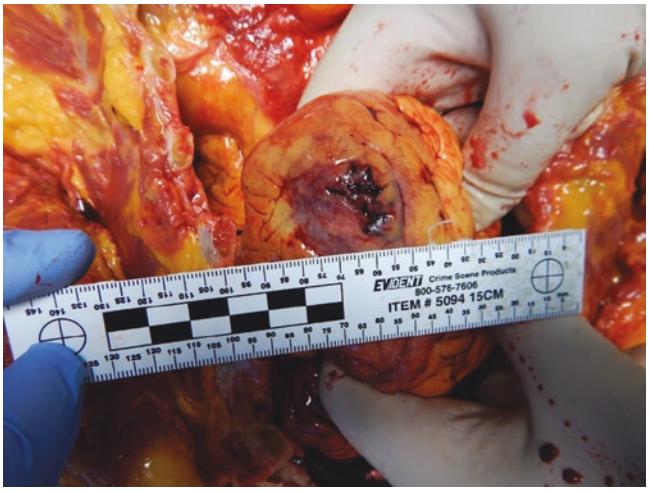


Fig. 37.20 A continuous solution, 3 × 4 cm, of the full thickness of the free wall was found in the left ventricle apex

Case 1 (Figs. 37.19, 37.20, and 37.21) shows the case of an 83-year-old woman, with a history of systemic arterial hypertension, that while on vacation has been complaining of nausea, vomiting, slight epigastric pain, and paresthesias in her left arm over the past several days. Her physician found orthostatic hypotension and rib pain when pressing the left side of her ribcage; her doctor doubted her symptoms, AMI diagnosis missed, and the woman was found dead the same day. Upon removal of the sternal plastron, the lungs were expanded, without adhesions in the pleural cables; the pericardium was intact and contained about 200 mL of blood, partially coagulated. A continuous solution, 3 × 4 cm, of the full thickness of the free wall was found in the left ventricle apex (Fig. 37.21).

Case 2 (Figs. 37.22, 37.23, and 37.24). An asymptomatic 67-year-old man, heavy smoker and with diabetes 2, was found dead at home. At autopsy cardiac tamponade was found; the pericardium contained 420 mL of blood; there were extensive adhesions between the pericardium and inferoposterior portion of the left ventricle, the heart weighed 495 g. The left ventricular posterior wall showed an old infarcted area causing a scar tissue, containing all the myocardial layers, and a pseudoaneurysm with a diameter of 4.5 × 5 cm, its wall thickness being <1 mm. Histopathologic examination showed that the structure contained only pericardial and fibrous elements in its wall; myocardial tissue couldn't be observed. In the apical portion of the pseudoaneurysm, there was an oval perforation, 1.5 × 1 cm. Left anterior descending artery had a 50% stenosis, while right coronary artery and left circumflex artery were hardened by severe atherosclerosis and calcification.

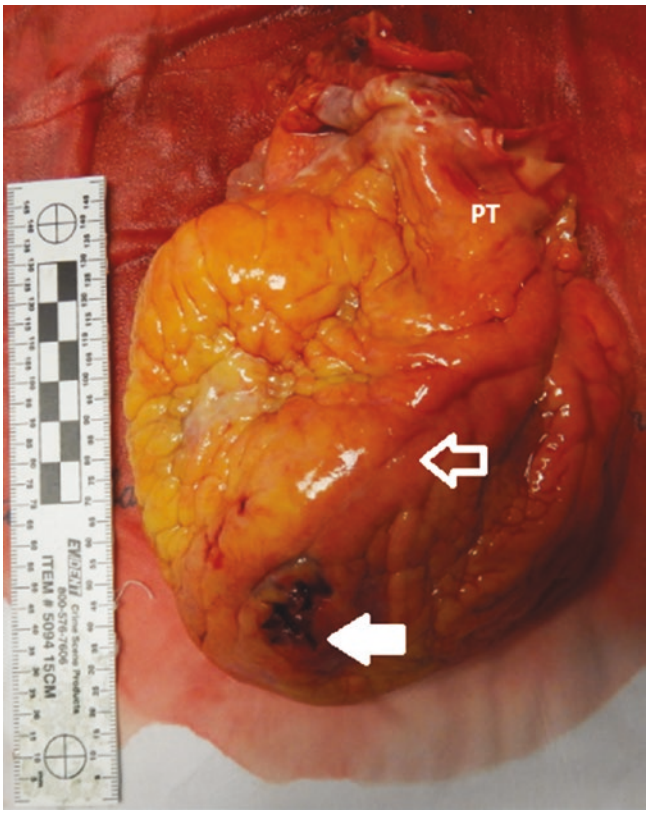


Fig. 37.21 Same case in Fig. 37.20. *PT* pulmonary trunk, hollow arrow left anterior descending artery, solid arrow free wall rupture

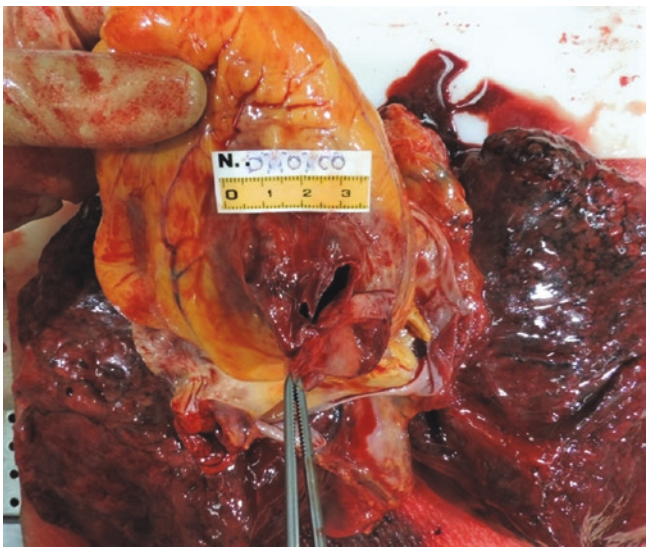


Fig. 37.22 Clinical case 2. At autopsy a pseudoaneurysm in the infero-posterior aspect of the left ventricle, diameter 4.5 × 5 cm, thickness at the same point <1 mm. In the apical portion of the pseudoaneurysm, there is an oval perforation, 1 × 1.5 cm

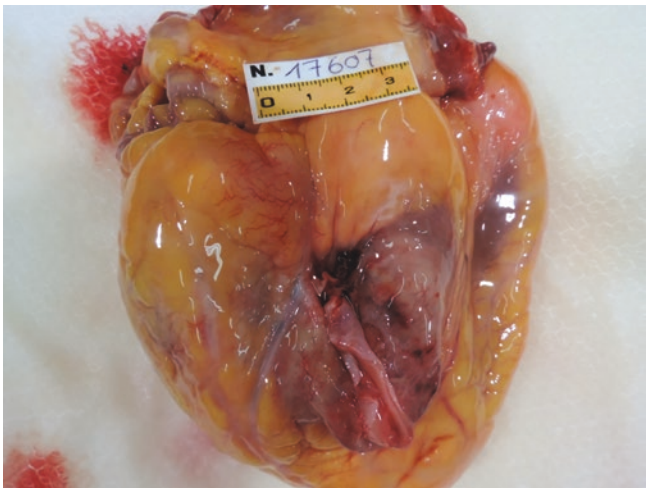


Fig. 37.23 Clinical case 2. Posterior aspect of the heart

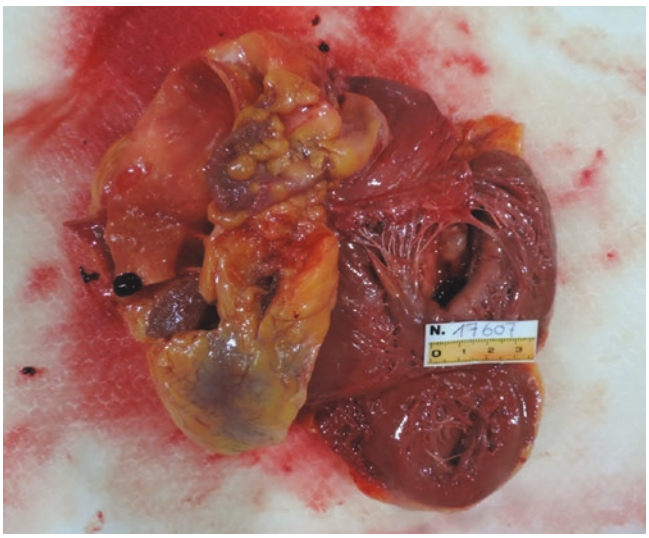


Fig. 37.24 Internal aspect of left ventricle: underneath the medial-posterior papillary muscle, a clot covering the ruptured pseudoaneurysm can be observed

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