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CASE REPORT

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# Massive injury to the heart after attempted active compression-decompression cardiopulmonary resuscitation

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**Abstract** An 84-year-old woman was unsuccessfully resuscitated for 3 min using standard cardiopulmonary resuscitation (CPR), followed by 15 min of active compression-decompression (ACD). The autopsy revealed that death was due to myocardial infarction complicated by rupture of the infarcted area and pericardial tamponade was diagnosed. Furthermore, a series of rib fractures, a transverse fracture of the sternum, rupture of the pericardial sac, the right ventricle, both atria and lacerations of the ascending aorta, were found with no signs of a vital reaction. To our knowledge, such extensive cardiac injury after CPR has not been previously reported. It is suggested that the pre-existing pericardial tamponade, the age of the patient and the application of the ACD-device to incorrect areas of the chest contributed to the extent of the cardiac injury. This case further adds to the suspicion of an increased risk of cardiac injuries when using an ACD device for cardiac massage.

**Key words** Cardiopulmonary resuscitation · Active compression-decompression · Complications · Iatrogenic injury · Myocardial rupture

## Introduction

Active compression-decompression (ACD) is an approach used to improve the hemodynamics achievable at cardiopulmonary resuscitation (CPR) [1]. An increased overall survival rate compared to standard CPR (Std CPR) was reported [2]. The ACD device (Ambu Cardio pump) consists of a modified silicone rubber suction cup and a handle containing a force gauge (Fig. 1). At CPR the suction cup of the device is applied to the thorax which, contrary to Std CPR, is not only compressed (force: 300–500 N), but also decompressed (force: 100–150 N) (Ambu Cardio Pump Instruction Manual). Complications of Std CPR such as rib fractures, sternal fractures, pneumothorax, and rare cases of cardiac, gastric, or hepatic trauma have been documented [3–6]. Recently, a study reported an increased risk of resuscitation injuries when using ACD-CRP [7]. In addition to a higher incidence of rib fractures, cardiac injuries were found in 2 out of 31 patients reported. In the present paper a case of massive rupture of all cavities of the heart and the ascending aorta after the use of ACD-CPR is presented.

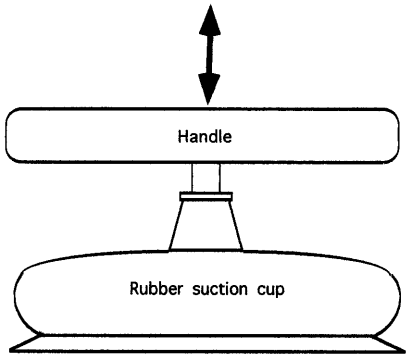


**Fig. 1** ACD device (Ambu Cardio Pump) consisting of a modified silicone rubber cup 6.5 cm in height and 13.5 cm in diameter. The device weighs 700 g and is operated by means of a circular handle containing a force gauge

## Case history

An 84-year-old woman collapsed in the street in front of her house. A medical doctor, who arrived within 5 min, diagnosed cardiac arrest and started Std CPR for 3 min followed by ACD-CPR. The patient was unsuccessfully defibrillated, intubated and 4 mg adrenaline was administered intratracheally. Despite these attempts no blood pressure was measurable. Therefore resuscitation was abandoned after 15 min of ACD-CPR. As the patient had no previous

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medical history, an autopsy was performed to establish the cause of death.

External examination

The body was that of a slim elderly female with abrasions caused by Std CPR and ACD-CPR on the skin at the lower third of the sternum, 5 cm in diameter, surrounded by a reddish ring corresponding to the rubber suction cup of the Cardiopump. Furthermore small abrasions were found at the right knee and at the left forehead, but no other signs of trauma were found.

Internal examination

The brain showed moderate signs of edema. Examination of the thoracic cage revealed bilateral fractures of ribs 2–8 at the sternal border and in the anterior axillary line with surrounding hemorrhages. The sternum was fractured transversely between ribs 3 and 4 without local hemorrhage. A triangular bony thorn measuring 3 mm was found at the upper fragment of the sternum. The right pleural space was empty, whereas the left space contained 900 ml fluid blood and 50 ml coagulated blood. Both lungs showed signs of senile emphysema, the left weighing 650 g, the right 680 g. Superficial hematomas corresponding to the vertebral column were also noted.

The pericardial sac and all four chambers of the heart which weighed 350 g were ruptured. The right ventricle and both atria were almost totally separated from the left ventricle and only parts of the posterior walls were still connected to the left ventricle. This laceration measured 15 cm and showed no signs of hemorrhage (Fig. 2). The trabecular muscles were flattened. A 12 mm long laceration was found in the left ventricular wall (Fig. 3) and the myocardium around the laceration was yellowish and surrounded by hemorrhaging. There was extensive arteriosclerosis in the coronary arteries which were severely narrowed due to calcified atherosclerotic plaques, but no complete occlusions were found. In the ascending aorta several calcified atherosclerotic plaques up to 1 cm in diameter were found, and two located in the posterior part were ruptured. These lacerations were strictly confined to the calcified area and the surrounding healthy areas were unaffected with no

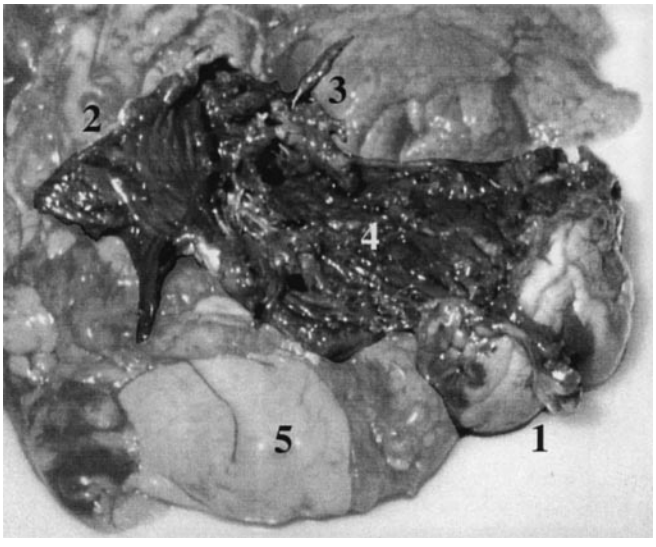


**Fig. 2** Ruptured heart of the deceased. 1) anterior wall of the left ventricle 2) inner aspect of the ruptured right ventricle which is subtotally separated from the left ventricle 3) tricuspid valve and torn out papillary muscle 4) posterior wall of the ruptured right ventricle 5) pericardium

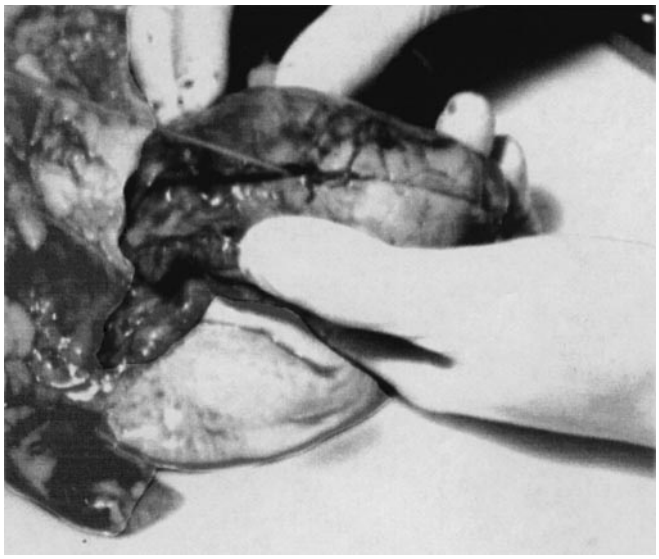

**Fig. 3** Single laceration of the left ventricle surrounded by bleeding into the subepicardial fatty tissue with a probe inserted

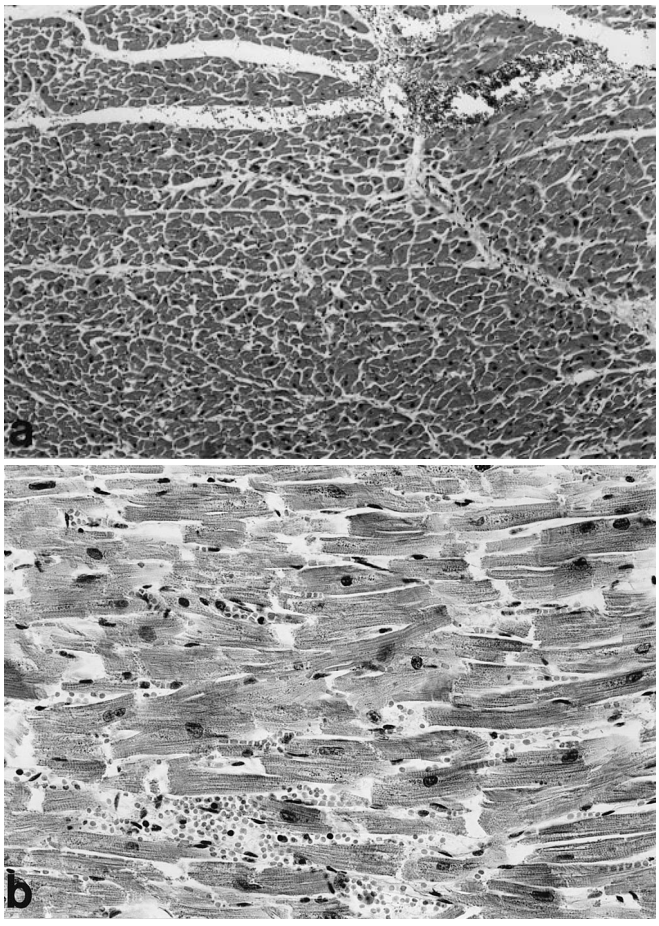


**Fig. 4 a, b** Areas of an anemic infarction in the left ventricular myocardium with coagulation necroses characterized by deeply eosinophilic staining of myocardial fibers, loss of cross-striation, and loss of hematoxylin-stained myocardial nuclei. In the surroundings emigrated granulocytes have aligned in a densely cellular rim of demarkation to the vital residual myocardium. Hematoxylin-eosin (HE) **a** 188 ×, **b** 375 ×

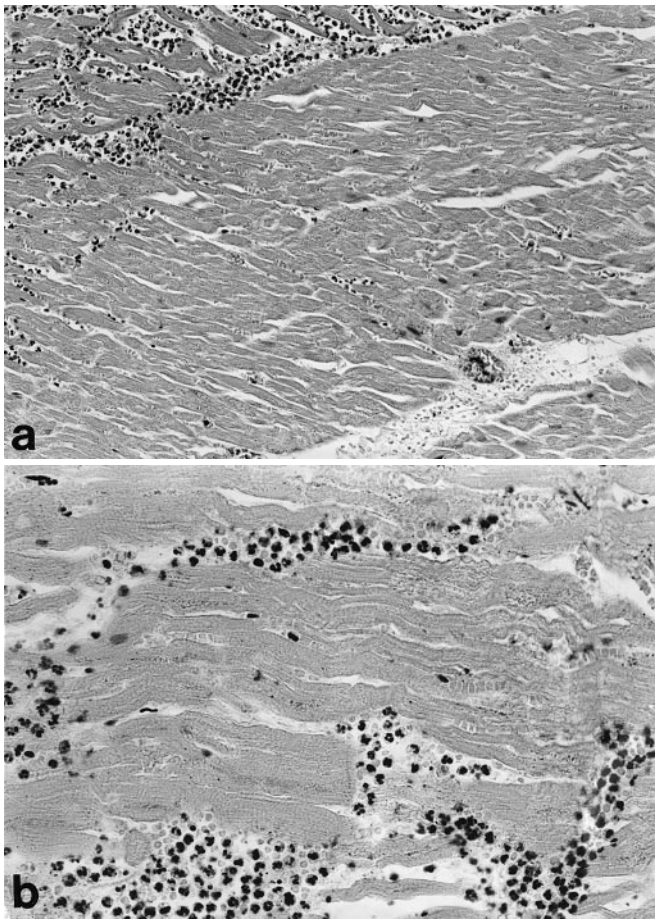


**Fig. 5 a, b** Specimens from areas around the disrupted right ventricular wall present myocardium with vital staining reactions. However, microhemorrhages can be seen interstitially between partially disconnected but vital muscle fibers. Hematoxylin-eosin (HE) **a** 94 ×, **b** 248 ×

signs of vitality. The abdominal organs were unremarkable except for left adrenal hyperplasia. The vertebral spine showed moderate signs of spondylarthrosis without prominent osteophytes.

### Histological examination

Histologically, in the blocks taken from the area surrounding the small rupture of the left ventricular myocardium (Fig. 4 a, b), an anemic infarct was found. The coagulation necrosis was characterized by a loss of nuclear staining and normal cross-striation in the deeply eosinophilic myocardial fibers. The areas of infarction were already well demarcated by a rim of granulocytes.

In numerous blocks of the right ventricular myocardium (Fig. 5 a, b) taken from the area of the disruption of the right cardiac wall no morphologically visible necrosis could be detected. However, multiple foci of hemorrhages could be found between the well stained, vital muscle fibers, which were sometimes disconnected due to dilatation. The extravasated erythrocytes were not accompanied by any cellular reaction such as leukocyte demarcation.

No other abnormalities were found in the heart and histological examination of liver, lungs, psoas, and a mediastinal lymph node revealed no signs of generalised vasculitis or connective tissue disorders.

## Discussion

Rupture of the heart and the aorta are common after severe trauma e.g. car accidents [8]. Nevertheless, in the present case no signs of trauma other than those inflicted during the CPR except minor abrasions were present. Moreover, we did not find any morphologically detectable ischemic alterations in the myocardium at the ruptured area of the right cardiac wall. Although it is generally known that changes cannot be seen under light microscopy after at least 4–8 h of ischemia, topographically an infarction is highly improbable since these events are very rare in the right ventricular and atrial myocardium. However, the area around the laceration of the left ventricle showed macroscopical bleeding into the muscle and histologically, signs of an approximately 3 days old infarction. Because of the fact that a small portion of the blood found in the left pleural space was coagulated, it can be assumed that the infarcted area had ruptured before the attempted resuscitation, leading to death from pericardiac tamponade, whereas the lacerations of the pericardium, the right ventricle, the atria, and the aorta were caused by the attempted resuscitation. The haemothorax, on the other hand, could be attributed to the rupture of the pericardium which already contained partially clotted blood at the time of CPR.

Cardiac injury is a rare complication from Std CPR with only a few cases reported [9–13]. Until recently, reports on complications associated with ACD-CPR, however, have been limited to skin abrasions and a few instances of rib fractures and pneumothorax and the incidence was reported to be below 5%. In particular, no cardiac lacerations were observed. [14–16]. In contradiction to these results Rabl et al. [7] recently reported an increased incidence of complications after ACD-CPR compared to Std CPR and in two patients, cardiac injuries occurred attributable to ACD-CPR. These injuries were similar to most of those reported after Std CPR with single lacerations measuring no more than 1–3 cm, whereas we report a 15 cm long rupture of the right ventricle and both atria and two small lacerations of the ascending aorta. As in those cases, both Std CPR and ACD-CPR were performed in the present case. The question as to which of these methods actually caused the injuries is thus difficult to assess. Nevertheless, as Std CPR was performed for only 3 min compared to 15 min of ACD-CPR, it seems to be more likely that the injuries were caused when using the Cardio Pump.

Baldwin and Edwards [13] reported two cases of ruptured right ventricles after Std CPR and assumed that the cardiac lesions were the result of an unusually high intracardiac pressure caused by trapping of blood in the right ventricle due to obstruction of the outflow tract and a closed tricuspid valve by the pressure applied. Thus the injuries to the right ventricle observed in the present case could be explained, but not those to the pericardium, the atria, and the aorta. Therefore we attributed the injuries found in the present case to direct penetration of the myo-

cardium by ends of fractured ribs or, more probably, by the fractured sternum as assumed for other cases [9, 12]. Several factors could be held responsible for the extent of the mediastinal injuries found in the patient. First of all, it could be speculated that the antemortem tamponade of the pericardial sac had not only compressed but also fixed the heart and thus focused the mechanical trauma on the base, atria, and right ventricular wall during resuscitation. Furthermore, the age of the patient might have been a relevant factor contributing to the amount of trauma in terms of the general degeneration of ageing. Finally, the doctor who performed the resuscitation stated that the chest of the patient was soiled with electrode gel from initial attempts at defibrillation which caused the rubber suction cup of the ACD device to slip away several times. It is possible that this slipping contributed to the extent of the injuries to the heart, as applying CPR to parts of the chest other than the lower third of the sternum increases the risk of injuries [4]. Meticulous cleaning of the soiled thorax before application of the ACD device should therefore be recommended.

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