

PHYSICAL EXAMINATION

Pulsus paradoxus: an underused tool

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A case of recurrent chest pain with syncope

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A 69-year-old man was brought to the emergency unit at 3 a.m. because of severe chest pain. He had no history of heart disease, chest pain, shortness of breath, palpitations, diabetes, hypertension or hyperlipidaemia, and there was no family history of heart disease. Chest pain was still present on admission. There were no electrocardiographic (ECG) changes diagnostic of acute cardiac ischaemia. Blood pressure (BP) was 170/100 mmHg in both arms, pulse 72 bpm and regular, no fever, and the respiratory rate was 16 breaths/min. Arterial blood gas analysis was not suggestive of pulmonary embolism, and the chest X-ray study was normal. While the patient was in the emergency unit, he had a transient loss of consciousness with BP 80/50 mmHg; the ECG morphology remained unchanged with a sinus rhythm at 74 bpm. Within 3 min the patient's BP spontaneously rose to 140/70 mmHg, and he became fully alert. The peripheral pulses were 2+ in the upper and lower extremities bilaterally. At cardiac examination, the heart tones were distant with no murmur, rubs or gallups. At echocardiography, the left ventricular chamber size was normal with an adequate left ventricular performance. Another possible explanation for the chest pain was aortic dissection; however, the aortic root was normal (36 mm) although an anterior and posterior slight pericardial effusion was present. The patient was then sent to the medical ward for further assessment.

The initial cardiac enzymes became available at 6 a.m., and were all in the normal range. At 7 a.m. the patient again complained of severe chest pain, followed by syncope with loss of the sphincter tones. The BP at this time

was 80/40 mmHg. The electrocardiogram showed sinus rhythm (90 bpm) with no signs of myocardial ischaemia. Consciousness returned within few seconds, and full consciousness was achieved after 3 min. The BP recovered over time. A computed tomography scan was then performed to investigate the thoracic aorta. Slight pericardial and bilateral pleural effusions were observed with a normal aorta and no signs of pulmonary thromboembolic disease.

At 11 a.m. a new crisis occurred with syncope, dyspnoea and diaphoresis with no chest pain. The pulse was rhythmic with a tachycardia (119 bpm) and the systolic BP was 85 mmHg. Consciousness returned fully within 2–3 min. At recovery systolic BP was 130 mmHg, but an additional 40 mmHg of pulsus paradoxus (PP) was found. Transthoracic echocardiography was unchanged. Transoesophageal echocardiography did not reveal any sign evocative of aortic rupture. Nevertheless, owing to the presence of the PP, a possible diagnosis of cardiac tamponade was made together with the decision to alert the cardiac surgery ward, and to operate immediately.

While the patient was being prepared for transfer to the cardiovascular surgery ward, haemodynamic parameters rapidly deteriorated. The jugular veins became swollen, cardiac sounds became soft with a sudden onset of shock. The patient was intubated and ventilated. Blood and bloody clots were removed with pericardiocentesis. At 1.00 p.m. a cardiac arrest occurred. Cardiopulmonary resuscitation was begun. At 2.10 p.m. the patient was in asystole.

Autopsy demonstrated blood in the pericardium (450 ml), with blood clots. An aortic dissection inside the pericardium was found, beginning at 0.5 cm from the aortic valve and extending 7 cm.

Comment

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This case has relevant implications on the role of the physical examination in clinical decision making because the attending physicians decided to perform exploratory surgery on the basis of pulsus paradoxus (PP), despite the negative results of two specific examinations (echocardiography and computed tomography scan).

Although diagnosis *in vivo* is made in as low as 15% of the cases, acute dissection represents the most common life-threatening aortic disease in clinical practice [1]. When the ascending aorta is involved, Stanford type A, the prognosis is very poor without prompt surgical treatment, because if left untreated, the mortality is 50% within the first 48 h [1, 2] with a mortality rate as high as 1–2%/h [3]. Such a short time window requires continuous re-evaluation of clinical signs,

physical findings and other investigations useful to build a Bayesian probability of diagnosis avoiding delays in obtaining surgery. However, in this patient the “negative” or “inconclusive” results of imaging studies left the clinician with a diagnostic puzzle to solve.

The patient had no history of ischaemic heart disease, hypertension or abdominal aortic aneurysm. The lack of tachycardia, the spontaneous regression of symptoms, as well as the normal left ventricular chamber size and the adequate left ventricular performance enabled the physicians to exclude a true cardiogenic shock. On the other hand the symmetrical blood pressure (BP) reduction in both arms enabled only exclusion of an involvement of a subclavian artery in acute aortic dissection. During hypotension, which was preceded by no warning, no changes in heart rate were recorded so that a neurally mediated (reflex) syncope, precipitated by severe aortic pain, with a prevalent contribution of vasodilatation rather than bradycardia, might be hypothesised. These findings make unlikely the possibility of an acute myocardial infarction but do not allow one to exclude an aortic dissection.

An acute aortic dissection was not confirmed by transoesophageal echocardiography and aortic spiral computed tomography scan. However, the clinicians were uncomfortable in discarding the diagnosis of aortic dissection because neither test was truly normal. Rather, both studies showed the presence of pericardial and pleural effusions that may accompany aortic dissection. The haemodynamic weight of pericardial effusion and the presence of cardiac tamponade was finally recognised after the third episode of syncope when PP was appreciated. This finding prompted the clinician to proceed to surgery.

The usual definition of PP is an inspiratory fall in systolic BP exceeding 10 mmHg. Kussmaul [4] first described the finding as the diminution or complete absence of the radial pulse during inspiration in 3 patients with pericardial disease. It was felt to be a paradox because despite the absence of radial pulses, heart sounds could be auscultated. The term “paradox” is unfortunate because the finding is nothing more than an exaggeration of normal physiologic change (6 ± 3 mmHg in normal persons) [5]. Therefore, when checking for PP the clinician has to have the patient breathe quietly and regularly, because even normal persons can induce a PP with vigorous respirations.

PP is detected by palpating the pulse or by using BP measurement (cuff sphygmomanometer). Systolic BP during inspiration and expiration has to be measured. The difference between the two recorded pressures is the PP value. The PP value that appears reproducible after at least three attempts is the one to be considered. Only paradoxical pulses exceeding 15–20 mmHg are palpable [6] so that the use of BP measurement is preferred by most clinicians [7], with the added advantage of quantifying the finding. The use of other non-invasive devices useful to measure PP has been tested [8, 9]. In monitored patients, PP can also be noted in the pulse oximetry tracing appearing as respiratory movement of the

tracing’s baseline [10]. The height of the oscillation correlates with the severity of PP [10].

The clinical significance of PP is high in cardiac tamponade (defined as improvement in cardiac output of 20% or more following pericardiocentesis) and acute asthma. PP is detectable in 98% of patients with cardiac tamponade, where it is one of three key findings, the other being elevated neck veins (sensitivity 100%) and tachycardia (sensitivity 77%–100%). A compilation of common and not so common physical findings seen in 56 patients with cardiac tamponade diagnosed at the bedside by Guberman et al. [11] is reported in Table 1. The finding of a PP > 12 mmHg discriminates patients with tamponade with a sensitivity of 98%, specificity of 83%, positive likelihood ratio of 5.9 and negative likelihood ratio of 0.03 [11]. In only 2% of patients with tamponade, a PP is absent due to one of five associated disorders: (1) atrial septal defect, (2) severe left ventricular dysfunction, (3) regional tamponade (tamponade affecting only one or two heart chambers, a complication of cardiac surgery), (4) severe hypotension and (5) aortic regurgitation. Knowing that aortic regurgitation may eliminate PP is especially significant because patients with proximal type (type A) aortic dissection and haemopericardium may lack the paradoxical pulse, despite significant tamponade, when the aortic valve is involved. The unaware clinician may thus exclude the possibility of tamponade to the harm of the patient. Indeed the patient of Bandinelli and Lagi had no signs of aortic regurgitation at both physical examination and echocardiography.

When the pressure of fluid inside the pericardial space exceeds the diastolic pressure in the heart chambers, tamponade develops. The diastolic BP in cardiac chambers, reflected in the neck veins, then becomes a measurement of the forces acting to compress the heart. The four chambers, now smaller in size, begin to compete for space, and an increase in size of one comes at the expense of another. Inspiration increases the filling of the right side and shifts the interventricular septum to the left thus obliterating the left ventricular chamber. Therefore, PP is absent in cardiac tamponade associated with an atrial septal defect (when the right ventricle does not fill more during inspiration), and in aortic regurgitation (when the left ventricle fills from a different

Table 1 Physical findings identified in 56 patients with cardiac tamponade diagnosed at the bedside [11]

	%
Elevated jugular vein pressure	100
Pulsus paradoxus	98
Tachypnoea	80
Tachycardia	77
Systolic blood pressure <100 mmHg	36
Decreased heart sounds	34
Rub	29
Rapidly falling blood pressure	25

Table 2 Aetiologies of pulsus paradoxus other than pericardial effusion and cardiac tamponade

Large pulmonary embolus
Severe COPD exacerbation
Bronchial asthma
Tension pneumothorax
Large pleural effusions

source than the left atrium). A regional tamponade, as probably initially occurred in the patient of Bandinelli and Lagi, compresses only one or two chambers, may limit cardiac output, but is too confined to cause the heart chamber to compete for space and to generate a PP. PP is also absent in conditions with fixed cardiac output such as supra-avalvular, valvular and subvalvular aortic stenosis, mitral valve stenosis, and in conditions with severely impaired left ventricular systolic function such as severe cardiomyopathy or acute myocardial infarction.

PP is not 100% specific for the diagnosis of pericardial effusion and cardiac tamponade. A list of other pathologic conditions in which PP can be seen is presented in Table 2. In particular, conditions characterised by laboured respiration (asthma) may present with PP. Indeed the initial National Heart, Lung and Blood Institute guidelines for the diagnosis and management of asthma specified that patients with a PP > 12 mmHg deserve hospital admission [12]. Present guidelines recommend PP measurement although do not identify PP thresholds [7]. Traditional teaching in medical schools includes the expectation that physicians have the ability to measure PP with a BP cuff as a useful adjunct to physical examination. However, PP assessment is often considered time-consuming and only rarely are students instructed to practise their skill in determining PP, which thus remains largely underused in the clinical setting.

In conclusion, although the gold standard for the diagnosis of a pericardial effusion is echocardiography, the diagnosis of cardiac tamponade can be based on physical examination. There is a *continuum* of patients with pericardial effusion, those with a compensated haemodynamic status and those with a decompensated tamponade status. The point at which the compensatory mechanisms fail and tamponade

ensues may not be readily identified by echo, but usually can be readily identified by physical examination.

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