The collapsing pulse

In modern medicine, reliance on clinical signs is rapidly diminishing. Although the cornerstone of valvular heart disease assessment is the history and echocardiographic findings, physical examination remains an important part of the diagnostic work-up to identify patients who require further investigation. Assessment of the pulse remains a fundamental skill for all doctors. This article reviews how to elicit and interpret a 'collapsing' pulse, and hopefully allay some fear in those sitting exams.

Historical perspective

The word pulse comes from the Latin 'pulsare' and means to throb or beat. Pulse examination has long been an important part of cardiac assessment and 'sphygmology' (study of the pulse) was an ancient medical specialty in its own right. The rapidly rising and falling pulse of aortic regurgitation has been recognized for centuries. Eponymous credit goes to British pathologist, Sir Dominic Corrigan, who as a young doctor in 1832 reported the visible pulsation in the carotid arteries of patients with chronic aortic regurgitation accompanied by a diastolic murmur or 'bruit de soufflet' (French for blowing noise) in the ascending aorta.

The description of the palpable nature of this pulse came from the English physician, Sir Thomas Watson, about a decade later (Dickey, 1969). He likened the feeling of the peripheral pulse to a water hammer, a 19th century toy that consisted of mercury within a glass vacuum tube which, when inverted, created a tapping or hammer sensation at the fingertips. Today, Watson's, Corrigan's and water hammer pulse are synonymous terms, used interchangeably to describe the collapsing character of a pulse.

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Definition

A 'collapsing pulse' describes a pulse with rapid upstroke and descent. It is typically associated with aortic regurgitation (Bonow et al, 2006).

Pathophysiology

The character of a pulse is described by its volume and waveform. While pulse volume provides a crude indication of systolic blood pressure, the waveform is more useful in diagnosis (*Figure 1*). The shape of the pulse is affected by several factors:

- Force of cardiac ejection
- Stroke volume
- Peripheral resistance
- Outflow tract obstruction (e.g. aortic stenosis)
- Compliance (or stiffness) of arterial wall
- Reflection of pulse waves from the periphery (the pulse wave arrives before the actual flow of blood).

A collapsing pulse occurs where there is increased stroke volume from the left ventricle and decreased peripheral resistance. The classic scenario is aortic regurgitation: a volume of blood is vigorously ejected and the pressure in the arteries goes up very quickly, but it then falls or 'collapses' almost immediately owing to backflow through the aortic valve. The arteries are emptier than usual at the end of diastole giving a low diastolic blood pressure.

In order to maintain a normal mean arterial pressure, the heart increases its force of contraction, generating a greater pressure during systole and hence a higher systolic blood pressure. This leads to a widening of the pulse pressure (systolic blood pressure minus diastolic blood pressure). The larger the stroke volume, the faster the rate of change will be (i.e. the steepness of the pulse waveform). A similar phenomenon occurs with backflow through a patent ductus arteriosus or arteriovenous malformation.

A collapsing pulse is also found with a hyperdynamic circulation, but the mechanism is different. Here the effective circulating volume is reduced, either as a result of anaemia or systemic vasodilatation. In the latter case this causes a fall in diastolic blood pressure. There is a compensatory increase in cardiac output, which explains the rise in systolic blood pressure and accompanying sinus tachycardia. These haemodynamic changes are seen in a number of conditions (*Table 1*) and the sinus tachycardia and clinical setting should help distinguish these from the true collapsing pulse of aortic regurgitation.

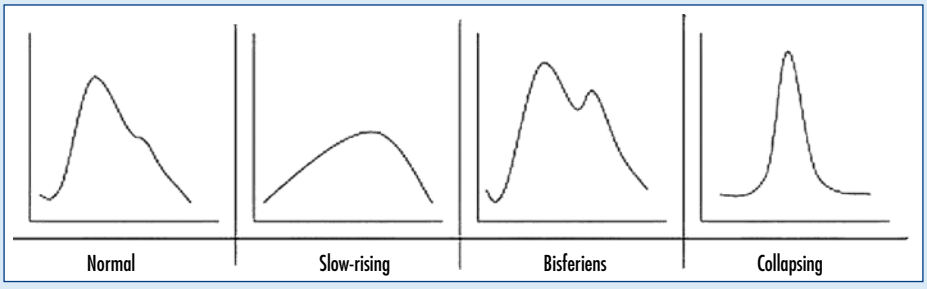
How to detect a collapsing pulse

In most examination routines clinicians usually start with the hands. In this scenario, the central pulses are assessed before

Table 1. Causes of a collapsing pulse

True collapsing pulse	Chronic aortic regurgitation
	Patent ductus arteriosus
	Arteriovenous shunt
Hyperdynamic circulation	After exercise
	Thyrotoxicosis
	Sepsis
	Severe anaemia
	Liver cirrhosis
	Pregnancy

Figure 1. Graphical representation of different pulse waveforms. Compare the slow-rising pulse of aortic stenosis with the collapsing pulse of aortic regurgitation. A bisferiens pulse (biphasic) is seen in mixed aortic valve disease or severe aortic regurgitation.



returning to the radial pulse for detailed assessment. Follow the basic principles of clinical examination:

Inspect

Look for arterial pulsations in the neck. The right carotid artery is anatomically closest to the heart and least subject to damping. Look here for the prominent pulsations Corrigan described. These are more obvious when the patient is in a sitting or standing position. Be sure to distinguish carotid pulsations from the jugular venous pressure, which has a double pulsation, draws inwards rather than outwards, and varies with position and respiration.

Palpate

Next, palpate the radial, brachial and carotid pulse. The character of the pulse is best examined in the larger arteries, which are close to the heart. A collapsing pulse is bounding and forceful. Like so many clinical signs it is a subjective sensation that becomes easier with experience. Digital pulses can also be detected in moderate to severe aortic regurgitation.

Move

Finally, examine the radial pulse (*Figure 2a–d* and video on www.bjhm.co.uk). This manoeuvre is performed to affirm your earlier suspicions:

1. Place your palm over the flexor aspect of the patient's right wrist, just proximal to where you would feel the radial pulse
2. Check the patient does not have pain in his/her shoulder or arm
3. Raise the patient's arm vertically upwards above the level of his/her heart
4. Feel the radial pulse against your fingers.

A collapsing pulse is felt as a tapping impulse or slapping sensation. The rate of decline of the pulse is fastest at the beginning of diastole – when the left ventricular pressure is lowest. Raising the arm accentuates this feeling. This is not because 'the blood falls faster because of gravity', but because mean arterial pressure falls and pulse pressure narrows. The lower mean pressure makes the arterial wall more compliant, producing a greater excursion with each pulse (Warnes et al, 1983).

Clinical implications

After correctly eliciting the collapsing pulse, the next step must be to determine its aetiology and formulate a management plan.

Aetiology

In addition to aortic regurgitation, the presence of a collapsing pulse should also raise suspicion of an abnormal arteriovenous connection. The commonest arteriovenous shunt is a patent ductus arteriosus which accounts for 10% of congenital heart disease. The ductus fails to obliterate at birth, leaving a communication between the aorta and pulmonary artery. Usually these defects are identified and closed during infancy, but in some cases they remain undetected until adulthood. Large extracardiac shunts (e.g. pulmonary arteriovenous malformation) may produce similar signs. The quality and location of a murmur should help distinguish where the problem lies.

The cardinal sign of aortic regurgitation is an early diastolic decrescendo murmur. Cardiologists are better than non-cardiologists at detecting this and accuracy improves with experience (Choudhry and Etchells, 1999). There are numerous causes of chronic aortic regurgitation, which can broadly be separated into two groups: valvular or aortic root dilatation (*Table 2*).

Acute *vs* chronic aortic regurgitation

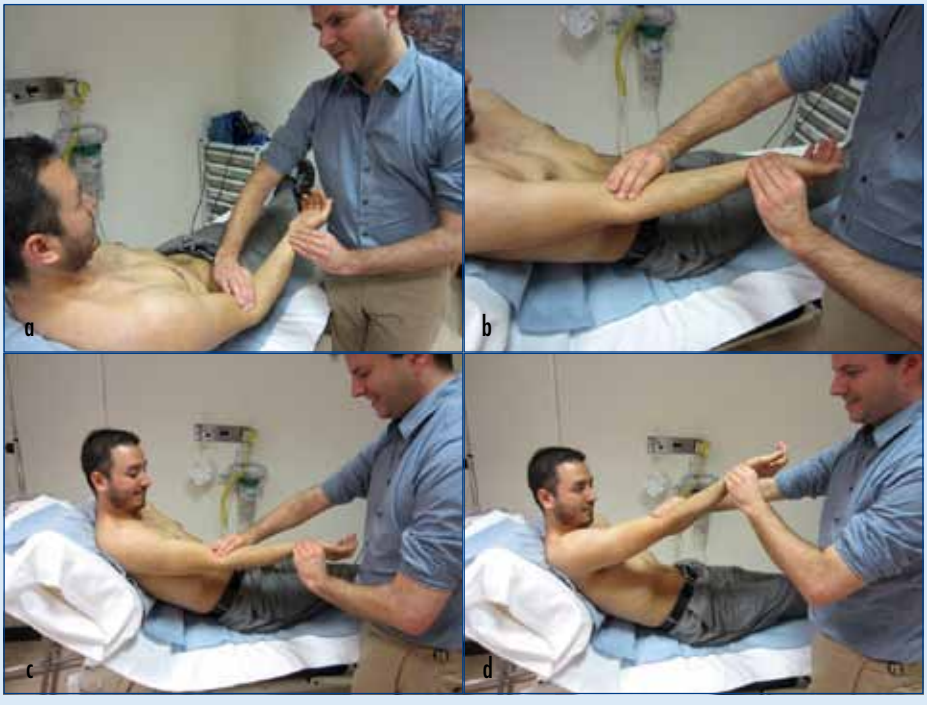
A collapsing pulse is typically seen in chronic, not acute aortic regurgitation. In acute aortic regurgitation the left ventricle has not had time to enlarge to accommodate the sudden increase in regurgitant volume. The forward flow is actually reduced, and although the heart rate increases to compensate it is not usually enough to maintain cardiac output. These patients can deteriorate quickly with pulmonary oedema and cardiogenic shock. Those with a hypertrophied, stiff ventricle (e.g. aortic dissection with hypertensive heart disease) probably fare the worst (Bonow et al, 2006).

In contrast, the ventricle in chronic aortic regurgitation gradually dilates, becoming more compliant, such that ventricular filling pressures are normal and forward flow is maintained.

Severity of aortic regurgitation

The collapsing pulse is one of numerous peripheral signs associated with severe aortic regurgitation. These rather obscure signs all result from wide pulse pressure and large stroke volume. However, they tend to be over-represented in textbooks (*Table 3*) and all have low sensitivity and specificity (Babu et al, 2003).

Figure 2. Examination of the radial pulse. a. Locate the radial and brachial pulses. b. Place fingers flat over the radial pulse. c. Ensure the patient is comfortable before moving his/her arm. d. Raise the patient's arm above the level of the heart.



Although emphasis is often placed on these eponymous signs, the most useful physical findings to determine the severity of chronic aortic regurgitation are:

- Wide pulse pressure – the lower the diastolic pressure, the more severe the aortic regurgitation
- Laterally displaced hyperdynamic apex beat
- Prolonged diastolic murmur, best heard at the lower left sternal border
- Presence of an Austin Flint murmur (a low-pitched mid-diastolic murmur best heard at the apex) caused by apparent reverberations of the mitral valve leaflet as blood flow from the left atrium mixes with aortic regurgitant flow. It was originally described by Flint (1862) as 'pre-systolic blabbering'
- Presence of a systolic 'flow' murmur (an early crescendo-decrescendo sound caused by the large volume of blood flowing across the aortic valve).

Assessment for heart failure

The prognosis of chronic aortic regurgitation is closely related to left ventricular systolic dysfunction. Patients may not develop symptoms until a very late stage. Angina often precedes breathlessness and occurs because of reduced coronary diastolic flow (as a result of low diastolic aortic pressure and increased left ventricular end diastolic pressure). It is therefore not sufficient to just take a history and examine these patients. Serial echocardiography is required to monitor left ventricular size and ejection fraction. Aortic valve replacement is recommended in patients with severe aortic regurgitation who are:

1. Symptomatic with normal left ventricular function (ejection fraction >50%)
2. Asymptomatic with mild to moderate left ventricular dysfunction (ejection fraction 25–49%)
3. Undergoing coronary artery bypass grafting or other valvular surgery.

Patients with severe left ventricular systolic impairment (ejection fraction <25%) have a poor prognosis and surgery is often considered too high risk (Bonow et al, 2006). Early diagnosis and careful monitoring is therefore paramount.

Table 2. Aetiology of chronic aortic regurgitation

Subtype	Cause	Exam notes (MRCS or PACES)
Valvular	Bicuspid aortic valve	Only echocardiography will distinguish the exact underlying valve lesion. Listen carefully for other murmurs (e.g. mixed aortic valve disease) and look for signs of endocarditis
	Degenerative	
	Aortic stenosis	
	Rheumatic	
	Infective endocarditis	
Aortic root dilatation	Severe hypertension	Examine for retinopathy
	Marfan syndrome and other connective tissue diseases)	Tall stature, skeletal deformities, hypermobile joints and vision problems may be present
	Sero-negative arthritides	Consider ankylosing spondylitis and psoriatic arthritis
	Rheumatoid arthritis	Look for small joint deformities and possible lung fibrosis
	Syphilis	Aortic aneurysm is a result of tertiary syphilis and occurs decades after initial infection. Rare

Table 3. Peripheral signs of severe aortic regurgitation

Hill's sign	A marked increase in systolic blood pressure in lower *vs* upper limbs (brachial–popliteal pulse gradient)
Traube's sign	Pistol shot sound over the femoral artery
Quincke's sign	Pulsation of the nail bed
Duroziez's sign	Systolic and diastolic murmurs with compression at the femoral artery
De Musset's sign	Head nodding in time with the heart beat

KEY POINTS

- Pulse character is determined by several physiological and anatomical factors.
- The collapsing pulse is typically seen in aortic regurgitation.
- Corrigan's pulse describes the visible nature of the pulse.
- Water hammer describes the palpable nature of the pulse.
- Severe aortic regurgitation is associated with wide pulse pressure and prolonged diastolic murmur.
- Aortic valve replacement should be performed before left ventricular function deteriorates.

Conclusions

Although the pulse is a rather antiquated method of diagnosis, it is a low-cost test that can often provide important clinical information. The presence of a collapsing pulse should always raise suspicion of chronic aortic regurgitation and point the physician towards looking for other markers of severity. **BJHM**

The authors are grateful to Dr Luis Lopes and Dr Joel Salazar-Mendiguchia for their assistance with producing Figure 2 *and the video clip.*
Conflict of interest: none.

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