The pulse

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The pulse has been studied for centuries
Informations gained:
1. frequency, regularity
2. patency of peripheral arteries
3. characteristics of the arterial pressure pulse wave
Palpation and other techniques use local compression
Jan Sten
The Sick Lady
Mid 17th cent
Rijksmuseum
George Washington
In his last illness.
cc.1800
Frans van Miers, the Elder, The doctors visit 1657, Kunsthistorisches Museum, Wien
Picasso 1897,
Ciencia y Caridad
Museo Picasso, Barcelona
The arterial pulse contour changes to the periphery:
damping
different rate of transmission of components
distorsion by reflected waves
conversion of kinetic to potential energy
1. resistance: viscosity, vessel geometry opposes flow, HR independent

2. inertia: mass opposes rate of change of flow, HR dependent

3. compliance: distensibility opposes changes of blood volume, HR-dependent
The anacrotic shoulder disappears to the periphery, the upstroke becomes steeper, starts later the “incisura” is characteristic for the carotid pulse contour it is gradually replaced by a later dicrotic notch and positive dicrotic wave
During palpation the auscultation of the heart serves for reference

Factors influencing the pulse:
- stroke volume
- rate of ejection
- distensibility of peripheral arteries
- peripheral resistance
- pulse rate
- pulse pressure
- size of the vessel
- distance from the heart
Figure 3  Carotid pressure waveform is recorded by applanation tonometry. The height of the late systolic peak (P1) above the inflection (P2) defines the augmentation pressure, and the ratio of augmentation pressure to PP defines the Alx (in percent).
Figure 1  Measurement of carotid-femoral PWV with the foot to foot method.
The abnormal arterial pulse

**Hypokinetic pulse** - small, weak
low stroke volume, narrow pulse
pressure, increased peripheral resistance
left ventricular failure
Ao valvular stenosis: pulsus parvus et
tardus
characteristic anacrotic notch
Pulsus tardus et parvus
The abnormal arterial pulse

**Hyperkinetic pulse** – strong, rapid upstroke “water hammer”
rapid runoff peripheral shunts
occasionally “thrill” on the carotid artery
hyperkinetic circulation: anxiety, exercise, fever, hyperthyroidism
Hyperkinetic pulse

Aortic regurgitation “pulsus celer et altus”

“systolic collapse” of the pulse

Peripheral shunts: rapid runoff of blood from the arterial system

Bradycardia
Specific abnormalities

The twice beating pulse

Dicrotic pulse: a second pulse wave is palpable during diastole, following S2

peripheral resistance

diastolic BP

fever, moderate AI
EXAMINATION OF THE ARTERIES AND THEIR PULSATION

bisferiens
HOCM
The twice beating pulse

**Anacrotic pulse**: a palpable double pulse both in systole, before S2

anacrotic notch on the upstroke

**Bisferiens pulse**: in HOCM

a very rapid initial upstroke the “percussion wave” is followed by a “dip” (the obstruction decelerates the ejection), this is followed by a second positive wave “tidal wave”
Pulsus alternans
Regular pulse with an alternating height of the pressure pulse + often S3: a sign of heart (LV) failure
Bigeminal pulse
The pulse size alternates from beat to beat caused by bigeminal ventricular ectopy
**Pulsus paradoxus**

In normal persons systolic BP $\downarrow$ by 3-10 mmHg during inspiration $\rightarrow$ pooling of blood in the pulmonary vasculature if this is more than 10 mmHg $\rightarrow$ p. p.

*cardiac tamponade*

*constrictive pericarditis*
CARDIAC TAMponade

MECHANISM OF PULSUS PARADOXUS
LV close to the surface  
LV swinging away from the surface  
(Mayo Foundation for Medicac Education)
Mayo
FIGURE 15-73 Typical pulsed-wave Doppler pattern of tamponade recorded with a nasal respirometer. A, Mitral inflow velocity decreases (single arrowhead) after inspiration (Insp) and increases (double arrowheads) after expiration (Exp). B, Tricuspid inflow velocity has the opposite changes. E velocity increases (double arrowheads) after inspiration and decreases (single arrowhead) after expiration. (Modified from Oh JK, Hatle LK, Mulvagh SL, Tajik AJ: Transient constrictive pericarditis: Diagnosis by two-dimensional Doppler echocardiography. Mayo Clin Proc 68:1158, 1993. Used with permission of Mayo Foundation for Medical Education and Research.)
Examination of the veins and their pulsations

The normal venous pulse

3 positive waves “a”, “c”, “v”

2 negative “x”, “y”
“a”: the retrograde transmission of RA systole, at atrial relaxation it descends
“c”: 1. Impact of the carotid artery
    2. Retrograde bulging of the tricuspid valve in RV systole
“x” descent: 1. Displacement of the base of ventricles during systole
2. Right atrial relaxation

“V” the tricuspid is closed blood is filling the venae cavae and right atrium in late ventricular systole
“y” descent: “diastolic collapse”,
the tricuspid opens
RA pressure rapidly
Rapid filling of RA, the “y” nadir may coincide with a S3
the ascending limb of “y” wave depends on the rate of venous return. Long diastole → plateau: “h” wave
Abnormalities

“a” wave: absent in atrial fibrillation
giant in tricuspid or pulm sten
“cannon” waves - the right atrium contracts while the tricuspid is closed

“x” wave: tricuspid regurgitation + “r”
Kata Tjuta in Ayers Rock, Australia
“y” wave: depends on the RV filling - compliance relation

A slow “y” descent  \( \rightarrow \) obstruction to RA emptying

A sharp “y” descent shortly after S2 in constrictive pericarditis followed by a rapid ascent and plateau:

“dip and plateau”
**Venous pressure**

Estimated at the bedside

1. Veins of the hand: passive elevation to and above the sternal angle emptying
2. External jugular: trunk elevated to 30-60° occlude the e. j. by pressing with finger above the clavicle, it fills within 15-40 s release and observe the fluid column
3. Paradox increase in venous distension during inspiration “Kussmaul sign” constrictive pericarditis
4. Hepatojugular reflux
FIGURE 12-3 Abnormal jugular venous waveforms. **A**, Large *a* waves associated with reduced RV compliance or elevated RV end-diastolic pressure. The phonocardiographic tracing (below) shows timing of the corresponding right-sided *S*_4*. **B**, Normal jugular venous waveform (bottom), mild TR (middle), and severe TR (top), with corresponding phonocardiogram. With severe TR, there is “ventricularization” of the jugular venous waveform, with a prominent *V* wave and rapid *Y* descent. The *X* descent is absent. **C**, Jugular venous waveform in constrictive pericarditis with a prominent *Y* descent. Note the timing of the pericardial knock (*K*) relative to *S*_2*. The abrupt rise in pressure after the nadir of the *Y* descent is caused by the rapid rise in venous pressure with ventricular filling. JVP = jugular venous pulse.

TABLE 14–3. THE CARDIAC CYCLE

**Left Ventricular Contraction**
- Isovolumic contraction (b)
- Maximal ejection (c)

**Left Ventricular Relaxation**
- Start of relaxation and reduced ejection (d)
- Isovolumic relaxation (e)
- LV filling: rapid phase (f)
- Slow LV filling (diastasis) (g)
- Atrial systole or booster (a)
FIGURE 14–22. The mechanical events in the cardiac cycle were first assembled by Lewis in 1920 but first conceived by Wiggers in 1915. Note that mitral valve closure occurs after the crossover point of atrial and ventricular pressures at the start of systole. The visual phases of the ventricular cycle in the bottom panel are modified from Shepherd and Vanhoutte (Shepherd JT, Vanhoutte PM: The Human Cardiovascular System. New York, Raven Press, 1979, p 68.) For explanation of phases a to g, see Table 14–3. ECG = electrocardiogram; JVP = jugular venous pressure; M1 = mitral component of first sound at time of mitral valve closure; T1 = tricuspid valve closure, second component of first heart sound; AO = aortic valve opening, normally inaudible; A2 = aortic valve closure, aortic component of second sound; P2 = pulmonary component of second sound, pulmonary valve closure; MO = mitral valve opening, which may be audible in mitral stenosis as the opening snap. S3 = third heart sound; S4 = fourth heart sound; a = wave produced by right atrial contraction; c = carotid wave artifact during rapid LV ejection phase; v = venous return wave that causes pressure to rise while tricuspid valve is closed. Cycle length of 800 milliseconds for 75 beats/min. (Modified from Opie LH: The Heart, Physiology, from Cell to Circulation. Philadelphia, Lippincott-Raven, 1998. Figure copyright L. H. Opie, © 2001.)
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