The internal jugular vein (IJV) connects to the right atrium without any intervening valves. The pulsation of the right atrium therefore causes the column of blood in the IJV to rise and fall - this is called the jugular venous pulse (JVP).

The jugular venous pressure (also called the JVP and often used interchangeably with the jugular venous pulse) is the height of this pulsation above the atrium.

The jugular venous pulse therefore provides an estimate of the central venous pressure (CVP) and hence the patient’s volume status and heart function.

Although an important part of the cardiovascular examination, clinical assessment of CVP using the JVP has poor sensitivity.

The usual JVP waveform looks like this:

See below for descriptions of the waveform
Question 2.

How do you measure the JVP?

To measure the JVP, position the patient at 45 degrees. Ask them to turn their head to the left and extend their neck gently backwards. It may be helpful to adjust the patient’s head into the correct position.

First measure the height of the JVP’s highest point in centimetres. This is the vertical height above the sternal angle at which a pulsation is observed in the internal jugular vein. Look for the JVP along the course of the vein which travels from the earlobe, down the neck and into the chest, between the two heads of sternocleidomastoid.

JVP-positioning

A JVP of greater than 4cm above the sternal angle is said to be elevated.
The JVP has a subtle double pulsation which follows the pattern below:

The waves and descents of the JVP are:

A wave: right Atrial contraction [presystolic]
X descent: right atrial relaxation
C wave (not seen Clinically): bulging of the tricuspid valve into the right atrium [beginning of systole]
V wave: maximum Venous return [late systole]
Y descent: Right ventricular filling [diastole]
Question 4.

How can you differentiate the JVP from the carotid pulse?
The JVP rises with pressure on the liver (hepatojugular reflux)
The JVP is easily occludable
The JVP is not strongly pulsatile
The JVP has a double waveform, the carotid pulse is single
Question 5.

What is Kussmaul’s sign?
Kussmaul’s sign is a paradoxical increase in the JVP with inspiration. It can occur in any condition where right ventricular filling is restricted such as constrictive pericarditis or cardiac tamponade.
Question 6.

What is the usual position of the apex beat?
The apex beat should be situated in the fifth intercostal space along the midclavicular line.
If the apex beat is impalpable here, move inferiorly and laterally.
Displacement of the apex in this direction suggests cardiomegaly.
Question 7.

Why might the apex beat be impalpable?
Hyperexpanded lungs: obstructive lung disease (e.g. COPD)
Reduced impulse: tamponade or restrictive pericarditis
Obesity
Dextrocardia (try palpating on the right)
Question 8.

What can you to to emphasise the apex beat? If you are unable to feel the apex try rolling the patient further to the left to bring the heart closer to the chest wall. If there is still no beat palpable, try feeling on the right for dextrocardia.
Question 10.

How can you describe murmurs in a cardiovascular examination?
Murmurs should be described in terms of timing, site of greatest intensity, character, loudness, and radiation.
The intensity of a murmur does not necessarily help in assessing the severity of the valve lesion, but a change in intensity can be important.
Question 12.

What is a third heart sound and what causes it?
A third sound is caused by passive filling of ventricles in systole
It happens in any condition where the artia are more full than they should be:
Volume overload
Mitral regurgitation
**Aortic regurgitation
**NB. An opening snap or myxoma polyp can be confused for a third s

**The doctor say this 2 points is wrong
What does the splitting of heart sounds mean?
The second heart sound (S2) is composed of aortic and pulmonary valve closure (A2 and P)
A2 usually occurs just before P2 as the aortic pressure is higher than pulmonary pressure so the valve snaps closed quicker. This difference in timings is referred to as splitting.
S2 is therefore physiologically slightly split
A2 gets closer to P2 during expiration
The loudness of P2 is a measure of pulmonary vascular resistance
Question 13.

What is a fourth heart sound?
The fourth heart sound is caused by atrial contraction against a stiff left ventricle. Causes include:
Vent. Hyper trophy
Uncontrolled Hypertension
Aortic stenosis
HOCM
Question 12.

What is a third heart sound and what causes it?
A third sound is caused by passive filling of ventricles in systole (diastole is the correct// systole is incorrect)
It happens in any condition where the artia are more full than they should be:
Volume overload
Mitral regurgitation
Normal in children
Question 15.

What is wide or pathological splitting of heart sounds
Pathological splitting is when the physiological splitting (A2 before P2) is exaggerated. It can happen either if A2 occurs early or if P2 occurs late.
A2 earlier: (i.e. if blood leaves the left ventricle by other means, giving a very quick reduction in LV pressure)
Mitral regurgitation
VSD
P2 later: (i.e. high volume load or delay for mechanical reasons)
RBBB
Pulmonary stenosis
ASD
Question 16.

What is reverse splitting of the heart sounds?
Reverse splitting is when the aortic sound occurs AFTER the pulmonary sound. It can happen either if A2 occurs late or if P2 occurs early:
A2 later (delay LV contraction)
Aortic stenosis
HOCM
LBBB
RV pacing
P2 early (occurs if blood leaves the right ventricle by other means, giving a very quick reduction in RV pressure)
TR
PDA
Note that in modern medicine, splitting of heart sounds is rarely clinically relevant. Sadly it still appears in exams.
What are the cardiac causes of clubbing?
Congenital cyanotic heart disease
Atrial myxoma
Infective endocarditis
Question 18.

What are the risk factors for endocarditis?
- Previous cardiac valve surgery
- Previous infective endocarditis
- Mitral valve prolapse with valve leakage
- Abnormal valves (e.g. rheumatic fever and degenerative conditions)
- Congenital heart disease
# 6.15 Causes of a fast or slow pulse

<table>
<thead>
<tr>
<th>Heart rate</th>
<th>Sinus rhythm</th>
<th>Arrhythmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast (tachycardia, &gt;100 bpm)</td>
<td>Exercise, Pain, Excitement/anxiety, Fever, Hyperthyroidism, Medication: Sympathomimetics, e.g. salbutamol, Vasodilators</td>
<td>Atrial fibrillation, Atrial flutter, Supraventricular tachycardia, Ventricular tachycardia</td>
</tr>
<tr>
<td>Slow (bradycardia, &lt;60 bpm)</td>
<td>Sleep, Athletic training, Hypothyroidism, Medication: Beta-blockers, Digoxin, Verapamil, diltiazem</td>
<td>Carotid sinus hypersensitivity, Sick sinus syndrome, Second-degree heart block, Complete heart block</td>
</tr>
</tbody>
</table>
### Some pathological causes of sinus bradycardia and tachycardia

<table>
<thead>
<tr>
<th>Sinus bradycardia</th>
<th>Sinus tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI</td>
<td>Thyrotoxicosis</td>
</tr>
<tr>
<td>Sinus node disease (sick sinus syndrome)</td>
<td>Phaeochromocytoma</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Drugs, e.g. β-agonists (bronchodilators)</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Drugs, e.g. β-blockers, digoxin, verapamil</td>
</tr>
</tbody>
</table>

### Sinus tachycardia

- Anxiety
- Fever
- Anaemia
- Heart failure
Cardiovascular: History

Chest pain, discomfort
Dyspnoea
Palpitations
Fatigue
Syncope, dizziness
Intermittent claudication
Ankle swelling
Past medical, surgical history
Family, social, drug history
Systems
Chest pain, discomfort
SOCRATES.
Onset: recent pattern changes.
Character: dull discomfort (angina)
Timing: at rest vs. exertion, meals (GERD)
Exacerbating: inspiration, movement.
Alleviating: sublingual nitrates, leaning forward.
Dyspnoea
Exertional: how far can you walk?
Orthopnoea: sleep with pillows?
Paroxysmal nocturnal dyspnoea: gasp at night?
Palpitations
Ever unusually aware of heartbeat?
SOCRATES.
Onset: sudden vs. gradual.
Timing: slow vs. fast.
Timing: regular vs. irregular.
Missed beat sensations.
Fatigue
Change in fatigue levels. Syncope, dizziness
SOCRATES.
Onset: saw blood (vasovagal)
Onset: stood up (HTN drugs, angina drugs)
Onset: felt it coming on.
Conscious vs. unconscious.
Intermittent claudication
Distance walked before leg pain.
Ankle swelling
Symmetrical vs. asymmetrical.
How far up leg.
Past medical, surgical history
MI, angina.
Rheumatic fever.
HTN, HTN of pregnancy.
Congenital heart problems.
STDs, infections.
Dental work (S. viridans.)
Investigations: angiogram.
Surgery: CABG, transplant, valve replacement, angioplasty.
Family history

Condition in a relative, what age.
MI.
Angina.
Congenital heart dz.
Mitral valve prolapse.
Marfan's.
DM.
HTN.
Social history
Smoking: ever smoked, how many per day, for how long, type [cigarette, pipe, chew.]
Alcohol (hypercholesteremia risk.)
Occupation: stress, work interruption.
Activity levels.
High cholesterol diet.
See Atherosclerosis Risk Factors Reference.
Describe your home: stairs, etc.
Who is with you there at home [help if MI, help with tasks.]
Drug history
HRT.
Thyroid drugs.
Diabetic drugs.
Prostate dz drugs.
Steroids.
Vasodilator (cause of ankle edema)
Allergies.
Allergies to drugs, dyes.
Systems
Pulmonary (dyspnea cause)
Alimentary GERD (chest pain cause)
Nervous (syncope cause)
Cardiovascular Exam«