## Heart sounds, blood pressure and the cardiac cycle



**Diastolic BP** 

Wigger's diagram: This seemingly complex diagram (Don't Panic!) is actually a very useful tool to learn the components of the cardiac cycle and how they relate to cardiac exam findings, which I have indicated in blue. To start, identify the aortic, LV and LA pressure lines and talk yourself through the sequence of events, starting with contraction of the LV (red line):

<u>LV contracts</u>: pressure begins to rise in the LV and mitral valve closes (First heart sound – S1 – follow vertical line down to heart sounds)

<u>Pressure rises in LV</u> until it exceeds the pressure in the aorta – aortic valve opens.

Pressure in the LV and aorta peak (systolic BP), then diastolic relaxation begins and pressure begins to fall. LV pressure falls below the "back pressure" in the aorta, aortic valve closes (Second heart sound -S2) Aortic pressure falls a bit further as blood runs off into peripheral vessels but then stabilizes as the diastolic pressure you measure with your BP cuff. Pressure in the LV continues to fall; when it drops below the pressure in the LA, the MV opens, and the LV begins to fill from the LA. In pathological states, when the LV is enlarged and the muscle function is abnormal, the initial rapid inflow of blood from LA to LV is associated with an abnormal "thud", or third heart sound (S3)

At end diastole, the LA contracts into the LV, causing the small "blip" in pressure you see just before the onset of ventricular contraction (go back to beginning of diagram). In pathological states, if the LV is abnormally stiff and non-compliant, LA contraction is associated with an abnormal fourth heart sound – S4.

After LA contraction, the LV begins to contract, LV pressure rises above LA pressure, the MV closes and the sequence starts again. <u>"Splitting" of the second heart sound</u>: All the events described above are occurring in the LV, LA and aorta (Ao); they are simultaneously occurring in the RV, RA and pulmonary artery (PA) respectively, associated with similar "right sided" heart sounds that occur more or less simultaneously with the "left sided" heart sounds. HOWEVER: there is a phasic respiratory variation in the timing of pulmonic valve closure which results in an audible "split" between the aortic closure sound (A2) and the pulmonic closure sound (P2). The sequence of events is as follows:

With inspiration, negative pressure develops in the intrathoracic cavity...this is what causes the lungs to fill with air. It also causes more venous blood to flow into the great veins (SVC and IVC) and to return to the RA and then to the RV. This increased RV diastolic volume takes slightly longer to eject, so pulmonic valve closure (P2) is slightly delayed relative to aortic valve closure (A2), i.e., there is an audible gap between the aortic and pulmonic components of S2 (sounds like "lub da-dup" instead of "lub dup"). With expiration, return of blood to the right heart diminishes, RV ejection is NOT delayed and there is no significant delay between A2 and P2.

<u>Jugular venous waveform</u>: Now look at the venous waveform (yellow arrow). The venous wave form is caused by fluctuations in pressure in the right atrium reflecting up into the jugular vein, which you can see as you examine the neck. Similar pressure changes in the left atrium cause pressure fluctuations in the pulmonary veins that can be measures with special catheters (more about this next year). The sequence of events is as follows:

The right atrium contracts causing an initial rise in jugular venous pressure called the a wave...this occurs just before ventricular systole so it will be seen in the neck just before the pulse is felt. RV contraction forces the tricuspid valve closed...the abrupt closure is associated with a brief pressure wave....but this is small and usually not visible. During ventricular systole, the TV remains closed so pressure in the RV is not reflected into the RA. However, during this time, the RA is filling from venous inflow and this causes a slow gradual rise in RA pressure, the v wave. The venous wave pattern in the jugular veis is thus seen as two fluctuations, the a and v wave, for each arterial pulsewave pulse (carotid, brachial or radial, whatever) that you palpate. Examples of two abnormalities you might see: In certain disease states, the RV may be thick-walled, stiff and non-compliant...when the RA contracts into it there will be an abnormally prominent a wave (sound familiar? .... which abnormal heart sound might you hear?....an S4). If there is tricuspid valve disease and the valve fails to close normally when the RV contracts. blood will leak or "regurgitate" backwards into the RA and there will be an abnormally large v wave. (What abnormality might you hear on auscultation of the heart?....a systolic murmur.)

Now move to the ECG (green arrow) and try to correlate the ECG event with the hemodynamic events and physical findings. **P** wave...atrial systole....a wave of the jugular venous waveform....maybe an S4. **QRS**....onset ventricular contraction....mitral valve closure...S1

