NORMAL ARTERIAL LINE WAVEFORMS

The arterial pressure wave (which is what you see there) is a shockwave; it travels much faster than the actual blood which is ejected. It represents the impulse of left ventricular contraction, conducted though the aortic valve and vessels along a fluid column (of blood), then up a catheter, then up another fluid column (of hard tubing) and finally into your Wheatstone bridge transducer.

Systolic upstroke:

**This is the ventricular ejection.**

The slope of this segment has some vague relationship with the rate of flow
through the aortic valve (probably more so when measured in the actual aorta). When its slope is slurred, there may be aortic stenosis.

Peak systolic pressure:

**This is the maximum pressure generated during the systolic ejection.** Added to it is the reflected pressure from the rest of the vascular tree. If the rest of the vascular tree is hardened and atheromatous, its poor compliance causes a powerful reflected wave, which when added to the systolic effort of the ventricle makes for a high peak systolic pressure.

**The peak systolic pressure is what you bleed with.** This is the pressure that blows the hemostatic thrombus plugs off the vessels you have so carefully cauterised, and stresses the wall of the fragile aneurysm.

Systolic decline

**This is the rapid decline** in arterial pressure as the ventricular contraction comes to an end. This decline is even more rapid when there is a left ventricular outflow tract obstruction (and systole comes to an abrupt halt before the left ventricle is finished with the ejection).

Mean Arterial Pressure (MAP)

MAP is roughly equivalent to the area under the arterial pressure curve, divided by the duration of the beat and averaged over several beats. The MAP is what perfuses your organs, and what commands their blood flow autoregulation.

Dicrotic notch:

In perfect circumstances, when measured in the aorta, this notch is very sharp and it actually does represent the closing of the aortic valve.

As mentioned below, the dicrotic notch position varies with the position of the arterial line. **A suspiciously low dicrotic notch could mean very poor vascular resistance,** eg. in a situation like severe septic shock.
Diastolic runoff:

This is the rapid decline in arterial pressure as the ventricular contraction comes to an end.

End-diastolic pressure:

This is the pressure exerted by the vascular tree back upon the aortic valve.

Hardened non-compliant vessels will cause this pressure to be raised. Soft vasoplegic vessels of a septic patient will offer little resistance, and the diastolic pressure will be lower.

A regurgitant aortic valve will cause this pressure to be lower than normal, because instead of meeting the aortic valve the pressure wave travels all the way through to the ventricle via the regurgitant jet.

The diastolic pressure is what fills your coronary arteries, and should not be ignored.

Pulse pressure:

A very widened pulse pressure suggests aortic regurgitation (as in diastole, the arterial pressure drops to fill the left ventricle though the regurgitating aortic valve)

A very narrow pulse pressure suggests cardiac tamponade, or any other sort of low output state (eg. severe cardiogenic shock, massive pulmonary embolism or tension pneumothorax)
Difference in waveforms according to site of insertion

The further you get from the aorta,
But, the MAP doesn't change very much.

This is because, from the aorta to the radial artery, there is little change in the resistance to flow.

- MAP only really begins to change once you hit the arterioles. The taller the systolic peak (i.e. a higher systolic pressure)
- The further the dicrotic notch
- The lower the end-diastolic pressure (i.e. the wider the pulse pressure)
- The later the arrival of the pulse (its 60msec delayed in the radial artery)

This is called **Distal systolic pulse amplification**:

**The systolic peak is steeper the further down the arterial tree you travel because of “reflected waves”**.

That is to say, the narrowing and bifurcation of blood vessels reflects some of the pulse back at the aortic valve. As the resistance of the branching arterial
tree increases, the pressure wave is reflected. The more resistant the tree (i.e. the more atheromatous the arteries) the more reflection there will be.

**Thus the systolic blood pressure can increase by as much as 20mmHg by the time you get to the radial artery** (as compared to the aorta).

In young people, this is a positive feature, as their relatively elastic vessels recoil slowly and the reflection wave is delayed, arriving to the aortic valve after it closes, and nourishing the coronary arteries.

In the elderly, the reflection wave arrives early, during systole - adding to afterload and thus to the myocardial workload; while in diastole there may be no reflection wave, which means the coronary arteries miss out on its benefit.
Anatomy of the CVP waveform

The peaks and troughs of the CVP waveform represent pressure changes in the right atrium.

(a) wave
- a is for atrium… this is the right atrial contraction.
- It correlates with the P wave on the ECG.
- It disappears with atrial fibrillation

(c) wave
- c is for cusp… this is the cusp of the tricuspid valve, protruding backwards through the atrium, as the right ventricle begins to contract.
- It correlates with the end of the QRS complex on the ECG

(x) descent
- This is the movement of the right ventricle, which descends as it contracts
- The downward movement decreases the pressure in the right atrium. At this stage, there is also atrial
diastolic relaxation, which further decreases the right atrial pressure.
- It happens before the T wave on the ECG

(v) wave
- As blood fills the right atrium, it hits the tricuspid valve and this is the back-pressure wave
- It happens after the T wave on the ECG
- It also gives an impression of tricuspid competence.
- A huge V wave is suggestive of tricuspid regurgitation, as it represents blood flowing back out of the contracting right ventricle; in this situation the V wave would be the most prominent wave, and would reach right ventricular systolic pressure (~ 30mmHg)

(y) descent
- This is a pressure decrease caused by the tricuspid valve opening in early ventricular diastole.
- This happens before the P wave of the ECG
- A loss of y-descent suggests tamponade; it means there is restriction to right ventricular filling.
ABNORMAL CENTRAL VENOUS PRESSURE WAVEFORM PATTERNS

There are distinct CVP waveform patterns associated with atrial fibrillation, junctional rhythms, tricuspid valve disease and reduced right ventricular compliance.

Loss of a-waves: atrial fibrillation

Because the atrial contraction is responsible for the a wave, loss of atrial contraction results in a missing awave. Very simple. The baseline in the picture is undulating to represent AF, but in reality this may not be visible because the atrium is contracting in such a feeble and disorganised manner that its activity may not produce any pressure waves whatsoever.

Cannon a-waves: junctional rhythm
In a junctional rhythm, the atrial contraction occurs at the same time as the ventricular contraction, which results in a fusion of the a and c waves.

Additionally, there are circumstances when the atrium contracts against a closed tricuspid valve, with the force of this contraction being reflected off the valve leaflets, forming a cannon a-wave.

This happens with retrograde conduction of ventricular action potentials. It can also be seen in complete heart block, where much of the time the atrium contracts against a closed or partially closed tricuspid valve. Lastly, ventricular tachycardias are typically events where the atrium depolarises by retrograde conduction, so any atrial activity happens during ventricular systole, and generates a cannon a-wave.

Thus the causes of cannon a-waves are:

- retrograde conduction of ventricular depolarisation:
  - ventricular tachycardia
  - junctional rhythm
- Asynchronous atrial activity
  - complete heart block

Regurgitant cv waves: tricuspid regurgitation

In tricuspid regurgitation, the backflow of blood out of the right ventricle obliterates the normal x descent. The c wave becomes accentuated and fuses with the v wave, as both are the results of right ventricular contraction (and the v wave peak pressure is often the same as the right ventricular peak systolic pressure).
Tricuspid stenosis produces a large a-wave because of increased resistance to flow from the atrium to the ventricle. The y wave is attenuated (i.e. of longer duration and of lower amplitude) because the right atrial filling is slow and lazy, without a pronounced change in pressure which would normally occur.

This can also happen in pericardial disease (or anything which results in decreased myocardial compliance), the result of which is a restriction of filling for all the chambers.

Of course, if tricuspid stenosis can cause these waves, then any loss of compliance ahead of the tricuspid valve can also do this. In this fashion one may have prominent a-waves in the presence of a reasonably normal tricuspid valve, with pulmonary stenosis or pulmonary hypertension.

Thus, **the causes of dominant a-waves are**

- Tricuspid stenosis
- Pulmonary stenosis
- Pulmonary hypertension
In pericardial constriction, the CVP will be raised, and the $x$ and $y$ descent is steep and abrupt. This contrasts with cardiac tamponade where the $y$-descent is prolonged.
Insertion of the Pulmonary Artery Catheter

How far to the right atrium?
As one shoves the catheter deeper and deeper, the distal lumen is transduced; the resulting pressure waveforms are one's guide to the catheter's position

- 10-15cm from the subclavian vein, 15-20cm from the jugular vein, 30-40cm from the femoral vein
- The right ventricle is another 10cm, and the pulmonary artery is another 10 cm after that.

In the Superior Vena Cava and the right atrium

The introducer sheath goes in first. Before the PA catheter is threaded in, the distal lumen is connected to a CVP transducer, so the pressure wave can be observed. The pressure here will be 1-6mmHg.

Atrial fibrillation may be encountered at this stage as the catheter tip tickles the atrium.
In the right ventricle

Once you are past the tricuspid valve, you suddenly get a nice pulsatile waveform, which is the right ventricular contraction.

The systolic pressure here should be between 15 and 30mmHg. The diastolic should be same as right atrial pressure, about 1-6mmHg (makes sense given that the right ventricle fills from the right atrium).

Not always is the waveform so pretty; mitral regurgitation may give a large v-wave, which may be confused for a pulmonary atrial wave. Additionally, a normal RV waveform may come and go, as the catheter tip flicks in and out of the ventricle. Its not a common place occurrence, but ventricular arrhythmias may be expected to happen here.

As soon as you are in the RV, and are seeing the pulse waveform, you can inflate the balloon with air. The volume is 1.5ml.
Past the pulmonary valve, one can now see the PA waveform, which resembles the waveform of any other artery. At this stage the diastolic pressure rises to about 6-12mmHg (due to flow resistance in the pulmonary arterial network). This is the fabled PA diastolic pressure, the PADP, which maintains a stable and reliable relationship with the PAWP.
Now, we wedge the catheter

The catheter with the inflated balloon is advanced further, until the PA waveform disappears, and a venous-looking waveform appears. This is the wedge waveform. It indicates that the pulmonary artery is occluded.

The pressure here should also be 6-12 mmHg, like the PA diastolic pressure. If it is different, it shouldn't be far off (about 5mmHg) and this relationship should persists for some hours, so you can just use the PADP (this way you don't have to wedge repeatedly). Once you have found this wedging point, deflate the balloon passively, and fix the catheter in position.

DO NOT KEEP IT INFLATED. Bad things will happen.

References

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