The Jugular Venous Pulse

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<**Opening credits**>

<**Paul Wood narrates over portrait of Mackenzie and pages from his seminal book on the pulse; followed by demonstrations of polygraphs**>

In 1902, Sir James Mackenzie published his classical work entitled *The Study of the Pulse: Arterial, Venous and Hepatic, and the Movements of the Heart*. This superb book can still be obtained and makes exciting reading. His investigations were made chiefly by means of the clinical polygraph.

A cup-shaped receiver was placed over the pulsating vein and connected pneumatically by way of a rubber tube to a timbre. To the membrane of this, a lever was attached. In Mackenzie's original instrument, the lever marked smoked paper covering a revolving drum. But in this instrument, modified by Sir Thomas Lewis, the lever carries a pen which writes on smooth white paper.

The polygraph has several drawbacks including time lag due to air transmission, overshooting due to the mechanical inertia of amplifying levers and the primitive method of recording.

In modern instruments, developed from the polygraph, the receiver is usually a carbon microphone. Electrical transmission eliminates time lag. Thermionic valves provide suitable amplification and the recording systems are similar to those used for electrocardiography. The phlebogram may be timed against the simultaneous arteriogram and electrocardiogram as shown here on the Sanborn Polyviso. As a matter of fact a phonocardiogram probably makes a better reference tracing.

<**Wood over diagrams explaining readings from the jugular pulse**>

The jugular pulse consists essentially of four main waves: A, X, V and Y; A and V being crests, registering high pressure; X and Y being troughs or periods of low pressure. The A wave corresponds to right atrial systole. A fifth positive wave, C, may interrupt the X descent. The X descent begins with atrial relaxation and is augmented during the earlier part of ventricular systole by displacement of the floor of the atrial ventricular septum towards the apex of the heart creating a negative pressure within the atria.

The venous pressure rises later during ventricular systole, to form the V wave because at this time, outflow from the atria is temporarily obstructed. The Y descent or the down slope of V begins as soon as the tricuspid valve opens when right atrial and ventricular pressures equal rapidly and fall together to the trough Y, after which they rise again before the onset of the next atrial contraction.

The nature of the C wave proper is controversial. The large C wave recorded in the jugular phlebogram, however, is the carotid pulse itself, as shown by Mackenzie. No wave of similar amplitude and timing is seen in right atrial pressure tracings.

<**Wood over shots of clinical examination of jugular pulse in patients and further explanatory diagrams**>

While taking full advantage of modern instrumental techniques, it must be emphasised that accurate analysis of the waveform of the jugular pulse and

reasonably precise measurement of the amplitude of each individual wave is usually possible at the bedside.

The patient should be placed supine if the pressure is below sternal angle level or propped up if the pressure is raised. The correct position being that which favours maximum jugular pulsation. The most readily inspected pulsation is that of the internal jugular vein.

With normal rhythm there are two main waves – the A wave of atrial systole, the V wave of ventricular systole; and two troughs, X and Y, per cardiac cycle. The movement is soft, diffuse, undulant and normally impalpable. When timed against the carotid pulse, only the first pulse, X, appears to coincide with systole; the A wave precedes it, the V peak is appreciably later and Y is clearly diastolic. You can see the arterial pulse here clearly by the light reflection on the red spot. The venous pressure rises on expiration and falls with inspiration, passively following the changes in intrathoracic pressure.

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The amplitude of all the waves increases during inspiration. Cervical venous pulsation ceases when the jugular veins are compressed at the root of the neck. Light pressure of the finger against the root of the external jugular vein distends the upper part of the vessel. On removing the finger, the vein collapses to the level of the mean jugular venous pressure.

Sir Thomas Lewis pointed out that the most satisfactory reference point on which to measure the venous pressure was the sternal triangle, because this was about 5cm above the centre of the right atrium in both horizontal and vertical positions. With reference to the sternal angle, the venous pressure swings <*jump in soundtrack*> a mean level of about -2cm in the horizontal position, but the range is considerable. Indeed, in this normal subject, the maximum systolic level, which happens to be A, is +3.5cm.

<**Intertitle**>

Abnormalities of the Venous Pulse

Let us now look at some abnormalities of the venous pulse.

<**Intertitle**>

The Giant a Wave

<**Wood over shots of heart tracings interspersed with case studies of adults and children with various abnormalities in heartbeat whose jugular pulses are examined closely in detail**>

A giant A wave, abrupt and collapsing in quality, and measuring between 6 and 15mm of mercury above V, is usual in tricuspid stenosis and atresia but is equally characteristic of severe pulmonary hypertension and severe pulmonary stenosis. It is palpable and transmitted to the liver and tends to be increased by inspiration.

Here is a case of mitral stenosis with severe reactive pulmonary hypertension due to a high pulmonary vascular resistance. The giant A is well seen in the right jugular pulse and measures about 6cm above the sternal angle. On inspiration its amplitude increases, even though the mean right atrial pressure may fall.

And here is a man with severe pulmonary valve stenosis with normal aortic root. He has a conspicuous A wave. The cyanosis is due to reversed interatrial shunt, also the slight clubbing of the fingers. You see the venous pulse is moving the lobe of the ear. That the large venous wave is presystolic can be seen by timing it against the temporal arterial pulse which may be observed just in front of the ear. It is shown very clearly there. From this angle, the height of the A wave may be compared with the height of V, which can also be seen, just there in the supraclavicular fossa – you can see V just coming up.

The giant A wave also occurs in tricuspid atresia, particularly when associated with a foramen ovale rather than a large atrial septal defect. Such is the case in this cyanosed girl.

The degree of clubbing emphasises the low level of the arterial oxygen saturation. The giant A wave is well seen, timed against the carotid pulse it is obviously presystolic. The time relationship between the venous and arterial pulses may be analysed by inspection in this closer view – you can see the arterial pulse in that shadow there, just below the lobe of the ear. The powerful atrial contraction responsible for the giant A wave is due to increased resistance to right ventricular filling, acting over a long period of time. It serves to increase the contractile force of the right ventricle in accordance with Starling's Law which states that within certain limits the force of a contraction varies directly with the length of the muscle fibres at the length of diastole.

The giant A wave rarely occurs in pulmonary hypertension when there is free communication between the ventricles as in Fallot's tetralogy on the left there and Eisenmenger's complex on the right.

Here is the child with Fallot's tetralogy, and you see very little in the neck, just a very small A wave. Here is the patient with Eisenmenger's complex and in these circumstances, the right ventricular systolic pressure never rises above systemic level, and the right ventricle is able to adapt itself to this sealing with little help from its atrium.

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The brachial artery pressure, pulmonary artery pressure and right ventricular pressure are all around 100mm of mercury. The right atrial tracing, shown on the right, shows A only slightly higher than V, both around 0. The link of the P-R interval can be rapidly estimated by noting the time interval between the jugular A wave and the carotid pulse. With experience it is not difficult to decide clinically whether the A-C interval is more or less normal, around 0.16 seconds, or obviously prolonged, 0.24

seconds or more as in the tracing. The jugular phlebogram is the middle tracing shown.

A cannon wave is a particular form of giant A wave and occurs when all the energy released by right atrial contraction is translated into pressure because forward flow is impossible owing to simultaneous closure of the tricuspid valve. This happens regularly in nodal rhythm and partial heart block. Nodal rhythm in this case was probably caused by digitalis. There is very marked sagging of the S-T segment in lead v-4 in the electrocardiogram there.

Immediately following the slow arterial pulse there is a large systolic cannon wave in the jugular pulse – this might well be mistaken for tricuspid incompetence, but the rhythm is regular and the absence of a presystolic wave should prevent error. Independent A waves can also be seen in many cases of complete heart block. Any type of ectopic beat may cause a cannon wave if the necessary conditions are fulfilled. Regular cannon waves also occur in paroxysmal nodal tachycardia.

In this boy with paroxysmal tachycardia, rapid cannon waves are conspicuous suggesting that it is nodal in origin. In fact, it proved to be atrial with 2:1 atrioventricular block; alternate atrial contractions coinciding with ventricular systole. The speed here is about 180.

<**Intertitle**>

Atrial Fibrillation

The A wave disappears of course in atrial fibrillation, it should be noticed that the X descent is also inconspicuous. Since this is commonly so, it is logical to conclude that the X descent depends more on atrial relaxation than on descent of the base. The jugular pulse is characterised by a single systolic wave. This appearance of the X descent in tricuspid incompetence in atrial fibrillation is characterised by much larger V waves – all the more apparent because of the deep wide trough which follows.

Giant V waves from tricuspid incompetence are well seen in this patient. That the venous pulse is systolic can be observed by recognising its time relationship to the carotid pulse which is lifting the pointer and the hand is moving up with it. In this case the venous valves have become incompetent and the giant V wave is transmitted to the extremities. The pulsation can be seen most easily when the legs are raised to a critical height; just as posture must be used to bring out maximum venous pulsation in the neck.

When there is normal rhythm in tricuspid incompetence a small X descent is often present but is overshadowed by the giant V.

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<**Intertitle**>

The y descent

The Y descent was first recognised in 1846 by Freidreich who drew attention to what he called ‘diastolic collapse of the venous pulse in chronic constrictive pericarditis.’ In other words, a sharp Y descent, a deep Y trough, followed by a sharp ascent as in this tracing from a typical case of Pick’s disease. This wave form signifies increased rapid ventricular filling followed by resistance to further filling. Here is the x-ray showing the calcific pericardium.

At the bedside, the venous pulse shows two crests and two troughs, the rhythm being normal. The Y descent is rapid and the Y trough conspicuous as in all cases of Pick’s disease. It may be noticed that the first trough, or X descent, is also unusually sharp; and this is frequently so in this condition, for it is easier for the ventricles to pull the base of the heart towards the apex than it is for them to pull in the calcified pericardium. The steep Y descent and deep Y trough, though characteristic of Pick’s disease, may be observed in any condition with a sufficiently high venous filling pressure provided there is no obstruction at the tricuspid orifice.

This is shown in this isolated case of myocarditis which had been previously diagnosed as Pick's disease. The chief wave here is a trough and represents a steep Y descent followed by a rapid return to the 0 point, the dominant movement being diastolic. Systole may be recognised by the movement of the thumb which has been placed over the carotid. The diagnosis in this case was proved a thoracotomy. Since a rapid Y descent and deep Y trough imply rapid ventricular filling such a wave form categorically denies tricuspid stenosis or any other obstruction at the tricuspid orifice.

In contrast, a slow Y descent, an absent Y trough, are virtually diagnostic of tricuspid stenosis provided the venous pressure is high. Both features are seen in this patient. The venous wave here is systolic and the irregularities due to atrial fibrillation. The Y descent is relatively slow and no trough can be seen. Some experience is necessary to estimate the speed of the Y descent, but the absent trough is not difficult to detect. The appearances are very different from those of Pick's disease and heart failure. They are not incompatible with a moderate degree of tricuspid incompetence as well but they do mean sufficient obstruction of the tricuspid orifice to prevent equalisation of right atrial and right ventricular pressures throughout the major part of diastole.

In differential diagnosis, some attention has been paid to the Kussmaul's sign, a paradoxical rise of venous pressure on inspiration, well seen in this tracing. This was first recognised in Pick's disease when it was attributed to an increase of pericardial tension caused by descent of the diaphragm and this explanation still holds good. Whilst it is agreed that Kussmaul's sign is characteristic of chronic constrictive pulmonary pericarditis, it is not at all uncommon in any form of heart failure, perhaps because descent of the diaphragm increases the tension of the pericardium when the heart is grossly distended.

Here, for example, is a case of primary pulmonary hypertension. The venous pressure is obviously raised and the giant A wave is well seen. On inspiration, not only is the amplitude of the A wave increased, but the venous pressure rises as a whole.

Perhaps we may conclude now, with a case for diagnosis. This patient was sent to the clinic with a case of aortic and mitral stenosis, complicated by heart failure. Looking at the neck, the high venous pressure is obvious; it will be seen that there is only one venous pulse wave per cardiac cycle and that the rhythm is irregular – certainly then, she has atrial fibrillation. When the single venous pulse is timed against the carotid, it is found to be a systolic event or if you like a large V wave. Is this simply heart failure or is it organic tricuspid incompetence?

Looking at the wave form more critically, the relatively slow Y descent is impressive, and if you look closely you will see that there is no visible Y trough in diastole; the Y descent subsiding to a flat plateau. This means that there must be some obstruction of the tricuspid valve. In fact she had organic tricuspid valve disease with both stenosis and incompetence. The stenosis has put a break on the speed of the Y descent and has abolished the Y trough. The incompetence has increased the amplitude of the V wave and caused the Y descent to be less sluggish than one might expect in severe stenosis.

<**End credits**>