SYSTEMATIC ANALYSIS OF THE CW DOPPLER TRACE

The patient appears to be in a regular atrial rhythm (possibly sinus rhythm) on evaluation of the simultaneous ECG recording. The cursor evaluating the transmitral flow appears to be well aligned within the colour Doppler flow demonstrated across the mitral valve. Forward flow across the valve is high, approaching 2 metres per second (2m/sec) and an envelope of flow can also be appreciated below the CW baseline. This envelope does not have the typical parabolic shape commonly seen with mitral regurgitation and its density is equal to that of the forward flow across the valve. From a complete evaluation of this spectral Doppler trace, it is possible to arrive at a correct diagnosis.

FURTHER DISCUSSION

Is it possible that this woman, who suffered a recent acute coronary syndrome with transient infero-lateral ST-elevation, also has mitral stenosis that has now led to her current admission in pulmonary oedema? Support for this might be the high forward flow velocity across the mitral valve (MV) approaching 2m/sec. However, further evaluation of the forward flow on the CW trace reveals a rapid downslope of the E-wave that would be unexpected in severe MS. It is possible to explain this observation in the presence of significant MS if the LVEDP was significantly elevated possibly secondary to the recent MI in this case. This may cause a rapid diastolic rise in LV pressures during early LV filling and therefore rapid equalisation of LA and LV pressures that would steepen the E-wave deceleration slope and therefore shorten the pressure half time (PHT). Tracing the forward flow envelope across the MV reveals peak and mean pressure gradients of approximately 10mmHg and 4mmHg respectively as calculated using the modified Bernoulli equation. This is not in keeping with severe mitral stenosis especially at this rapid heart rate that would tend to significantly increase the mean gradient in the presence of significant MS. Finally, mitral stenosis as an answer does not explain the CW envelope below the line and is therefore not the best answer. Answers (a) and (b) are therefore not good answers.

Let us now shift our focus to the CW envelope below the baseline. Is this a jet of mitral regurgitation or could it be an aortic stenosis (AS) jet? It is of value to note that anteriorly directed jets of MR can masquerade as AS and vice versa when evaluated by CW Doppler on a standard apical four- or five-chamber view. CW flow below the baseline (away from the probe as assessed in a 4C or 5C view) can represent MR but could equally be AS due to partial alignment of the cursor through the aortic valve whilst evaluating transmitral Doppler flow. The fact that the forward flow is typical of transmitral flow does not prove that the envelope below the baseline originates from the mitral valve. This is because the echo probe also picks up Doppler signals in the immediate vicinity around the Doppler curser and not only along the thin cursor line. The CW envelope below the line could therefore conceivably be a jet of aortic stenosis picked up adjacent to an anteriorly directed jet of MR. In support of this is the triangular jet shape more commonly seen with flow across the aortic valve and in fact aortic stenosis may be severe despite low gradients if the gradient across the aortic valve is reduced due to impaired LV function secondary to the recent MI. However, the triangular, early peaking nature of this jet, although seen in aortic flow argues against severe aortic stenosis which is typically a late peaking parabolic jet as the ventricle takes time to overcome the severe outflow obstruction. This shape is also typically maintained in the presence of conditions such as LV impairment that reduces the AV gradients and remains a clue to severe AS despite low gradients. Most importantly though we can identify the jet below the baseline as being definitely mitral regurgitation and not aortic in origin because of the absence of isovolumetric contraction and relaxation periods on either side of the transmitral forward flow which must be present if this is an AS jet (Figure 2). It is clear that the jet below the line starts immediately as forward flow ceases which effectively rules this jet out as an AS jet (Figure 3). Answer c is therefore incorrect and aortic stenosis would also not explain the high forward flow recorded across this patient's mitral valve.

The envelope below the line has now been identified as mitral regurgitation. The mitral regurgitation jet in our case has some

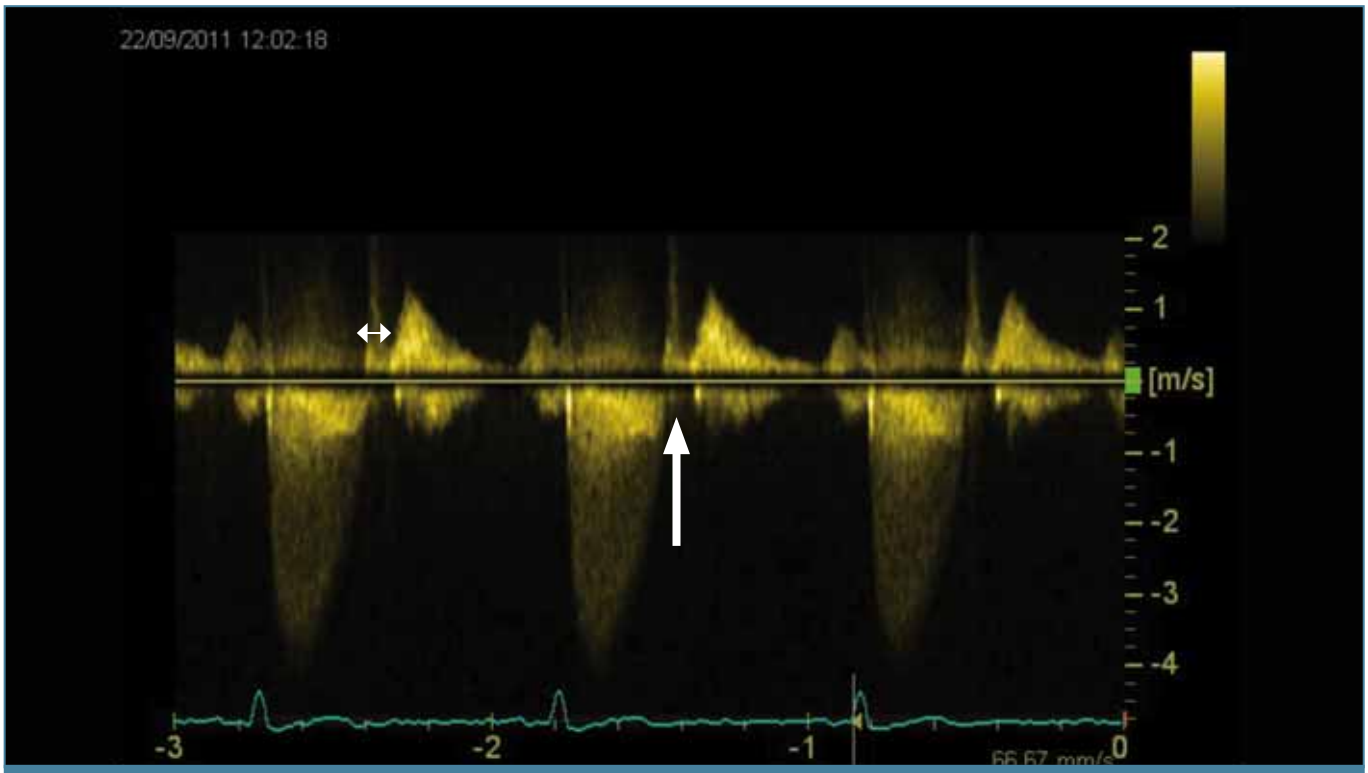

FIGURE 2: An example of transmitral CW Doppler in a patient with aortic stenosis (AS). The white arrow represents an isovolemic relaxation period between the forward mitral flow and the jet below the baseline identifying this jet as an AS jet.

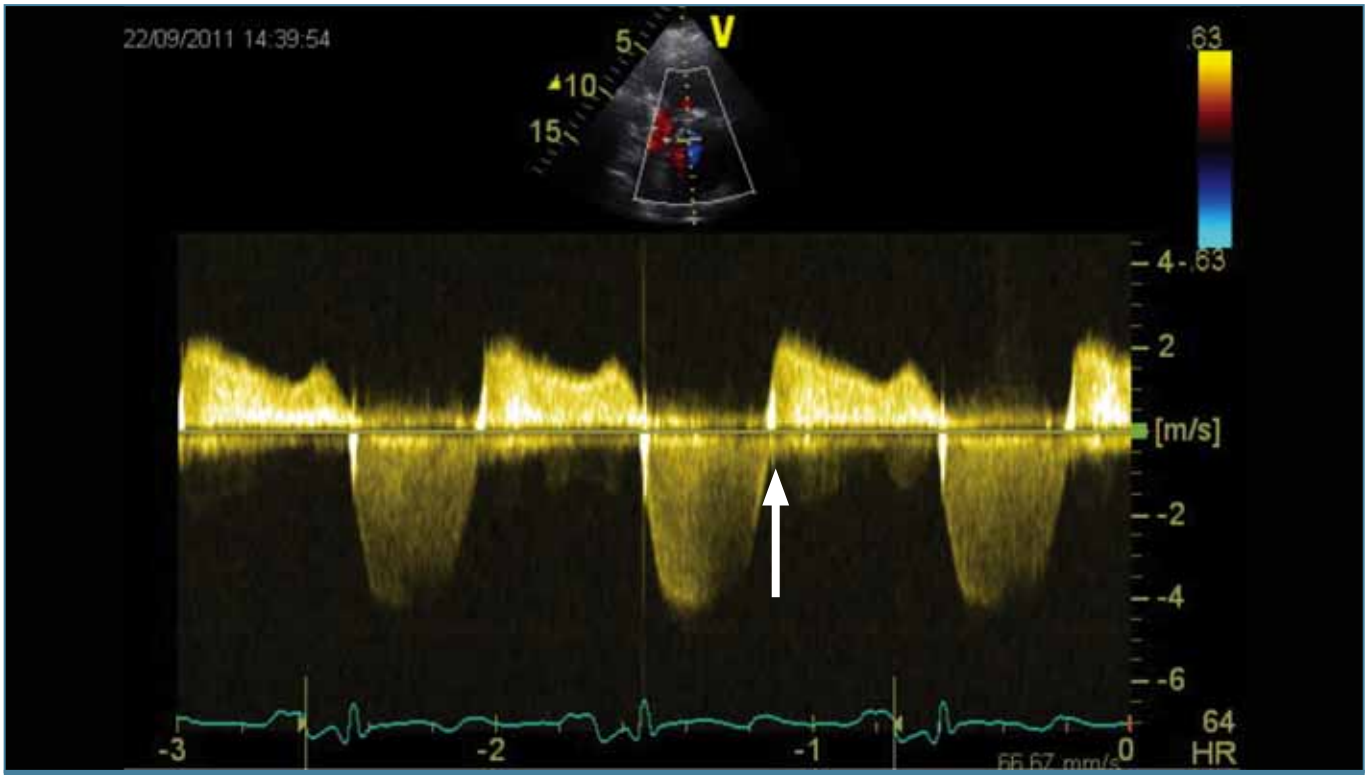

FIGURE 3: An example of transmitral CW Doppler in a patient with mitral regurgitation (MR). The absence of either an isovolumetric contraction or relaxation period between transmitral forward flow and flow below the baseline identifies the jet below the baseline as an MR jet.

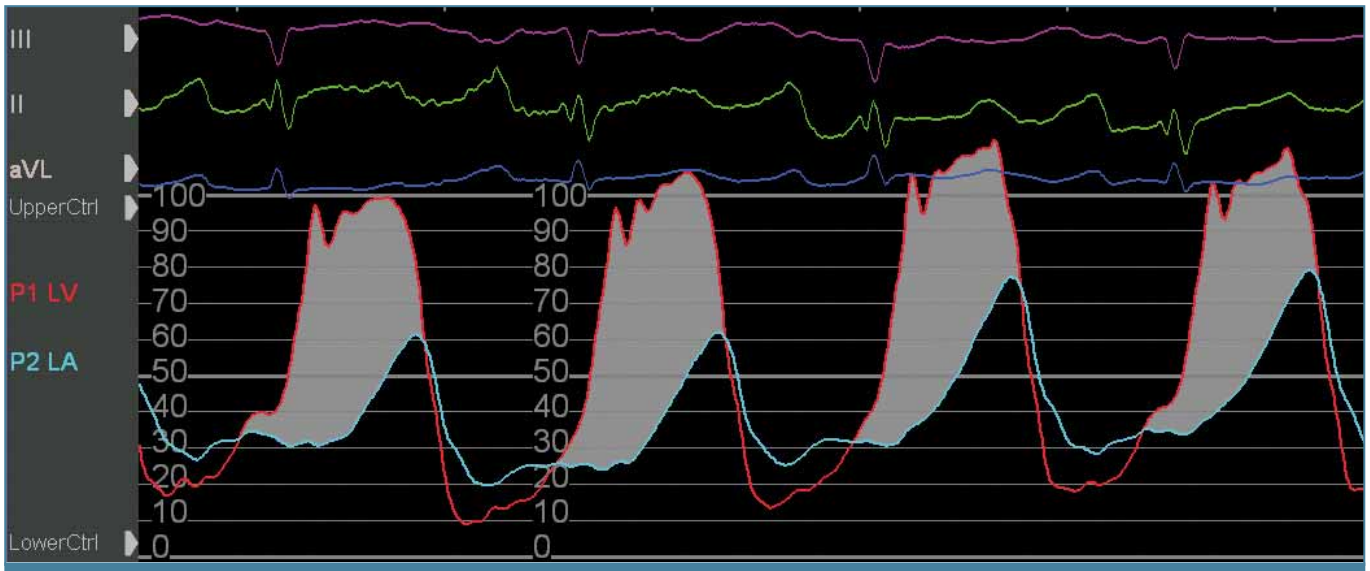

FIGURE 4: Simultaneous haemodynamic tracing of LV (red) and LA (blue) pressures in the setting of acute severe MR. Note the large atrial V-wave. The shaded area represents the effect that the large V-wave has on degrading the systolic LV-LA pressure gradient leading to V-wave cut-off on the transmitral Doppler trace. Compare this to Figure 5 in a patient without acute severe MR where the LV-LA systolic gradient is maintained throughout systole.

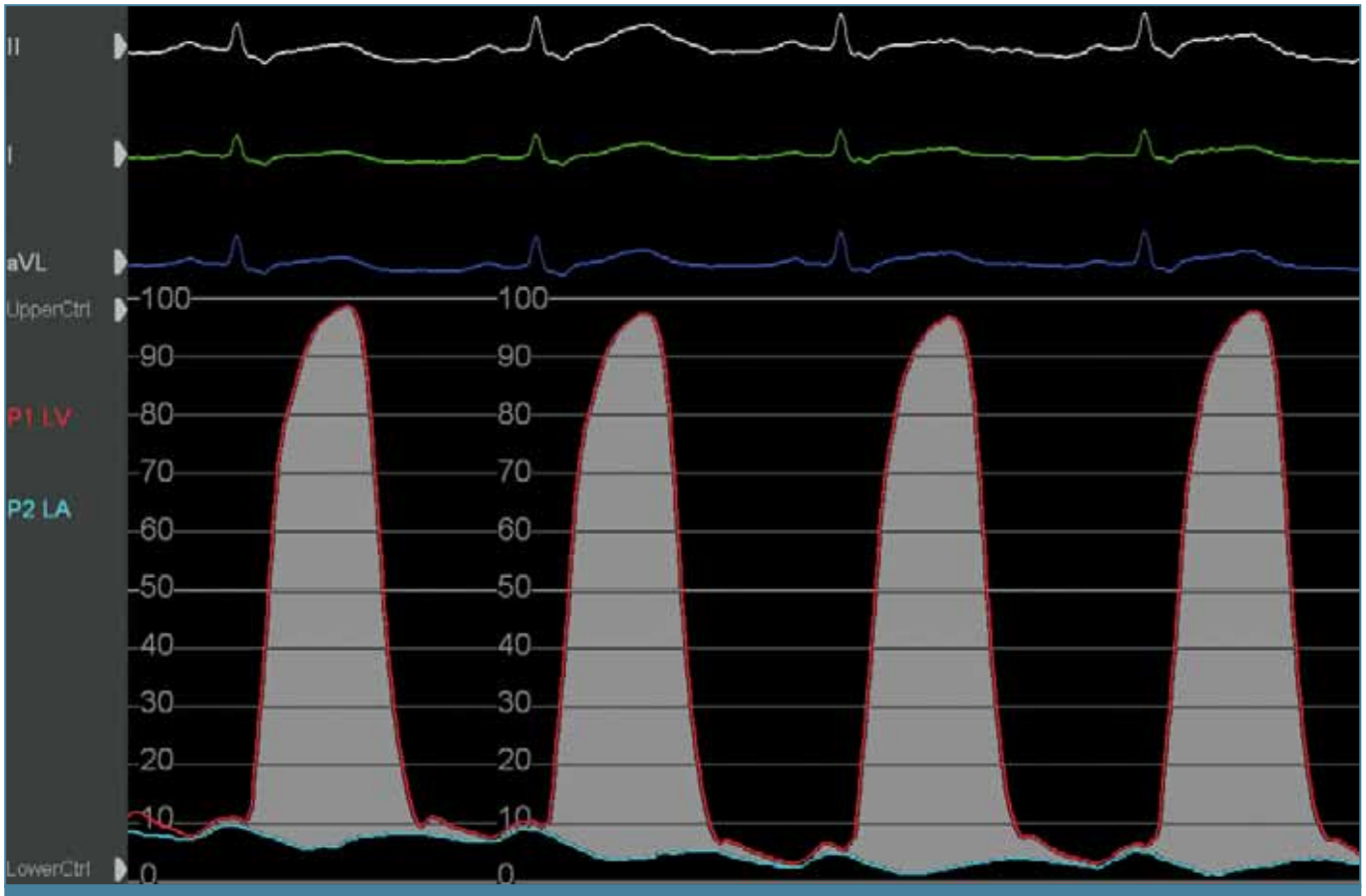

FIGURE 5: A patient without acute severe MR where the LV-LA systolic gradient is maintained throughout systole.

interesting features. Firstly, it is important to understand that the CW jet density holds important clues as to the severity of the MR. The density of MR flow on CW is a reflection of scatter from red blood cells and hence reflects the volume of blood that regurgitates through the valve. The more red cells that regurgitate, the more scatter will take place and the denser the jet will be. It is also possible to increase any jet's density by simply dialling up the Doppler gain. It is therefore important not to look at the absolute density of the regurgitant flow but rather compare the density of the regurgitant flow to that of the forward flow. In mild MR, the CW curser often moves in and out of the thin MR jet giving a faint and typically incomplete regurgitant jet envelope. If a complete regurgitant jet envelope is seen throughout systole and its density is equal to that of the forward flow it most likely represents a large volume or severe MR such as is seen in our case. This then also offers a likely explanation for the high forward flow velocities seen across the MV, which is related to increased volume flow across the valve in the absence of valvular stenosis. Answer (d) is therefore incorrect.

This leaves either (e) or (f) as possible correct answers. Can we answer the remaining question of whether the MR is more likely to be chronic severe, with the patient decompensating due to a ventricular problem, or whether we are dealing with acute severe MR? This is where jet velocity and shape are useful.

The velocity of the jet measured below the baseline in our case is low for a typical mitral regurgitation jet at 2.5m/sec. This velocity reflects the pressure gradient between the LV and LA during systole, which is the driving force generating the MR velocity. In chronic severe MR the jet velocity typically is in the region of 5m/sec (range of 4m/s to 6m/s is usual) reflecting a systolic pressure gradient of around 100mmHg between LV and LA. The atrium has time to dilate as the MR progresses and it remains relatively compliant even in the presence of chronic severe MR. This typically limits the pressure rise in the LA during systole. The result of this is that the jet velocity in chronic severe MR remains relatively high and the shape of the MR jet remains parabolic reflecting the fact that the LV to LA pressure gradient is maintained throughout systole because the LA pressure does not increase precipitously. In the setting of acute severe MR, there is a rapid rise in left atrial pressure due to the large regurgitant volume that has to be accommodated by a non-compliant and non-dilated left atrium. This rapid pressure rise is reflected by a large V-wave in the left atrium as seen at cardiac catheterisation. The large V-wave degrades the pressure gradient between the LV and LA in the second half of systole reducing the driving pressure of the regurgitant velocity and therefore changing the shape of the regurgitant envelope seen at CW Doppler. The typical parabolic shape of MR thus becomes triangular in shape (the V-wave cutting the terminal portion of the parabolic jet off, so-called V-wave cut-off) as in the Doppler tracing of our patient and reduces the peak regurgitant velocity measured. This makes answer (e), acute severe MR the most likely answer and very nicely accounts for the very low peak regurgitant velocity measured in our patient, as well as the triangular jet shape recorded.

Figures 4 and 5 make use of haemodynamic tracings of simultaneous LV and LA pressures to explain the principle of V-wave cut-off in acute severe MR. Further findings that would support a diagnosis of acute severe MR include a non-dilated LV in the presence of severe MR and the demonstration of a clear mechanism known to cause acute MR such as chordal rupture, papillary muscle rupture, a destroyed leaflet in the setting of endocarditis, a mechanical prosthesis leaflet stuck in the open position or dehiscence of a prosthetic valve.

Our patient had suffered a recent infero-posterior infarction and the mechanism for her acute severe MR was demonstrated to be rupture of the posteromedial papillary muscle. She required both invasive ventilatory and inotropic support for her cardiogenic shock followed by emergency mitral valve replacement. At surgery, almost the entire posteromedial papillary muscle was found to be lying within the left atrium. The mitral valve was excised together with the ruptured papillary muscle and replaced with a tissue prosthesis.

The anterolateral papillary muscle receives dual blood supply from both the left anterior descending and left circumflex coronary arteries, whilst the posteromedial papillary muscle receives a single supply via the posterior descending coronary artery. The posteromedial papillary muscle is therefore less protected from rupture as a complication of acute myocardial infarction with often devastating consequences.

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