Hemodynamics That Can Make For a Lifesaving Decision in the Cath Lab

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As our lab staff are in the process of hemodynamic case reviews, I wanted to discuss arterial hypotension and its causes to prevent potentially avoidable disasters.

Every day in the cath lab we see important and potentially life-changing or life-threatening scenarios involving patients with coronary artery disease, valvular heart disease, or cardiomyopathy. There are several critical hemodynamic measurements that may be lifesaving: arterial pressure (blood pressure [BP]), left ventricular end-diastolic pressure (LVEDP), pulmonary capillary wedge (PCW) pressure and right atrial pressure (RA). Low arterial pressure can be serious and an early warning sign of trouble to come. Hypotension may require emergency action in addition use of other hemodynamics (e.g. LVEDP, PCW, RA) in order to make a rapid diagnosis leading to the correct treatment. As our lab staff are in the process of hemodynamic case reviews, I wanted to discuss arterial hypotension and its causes to prevent potentially avoidable disasters. Other variables like LVEDP, PCW, and RA will be addressed in future editor's pages.

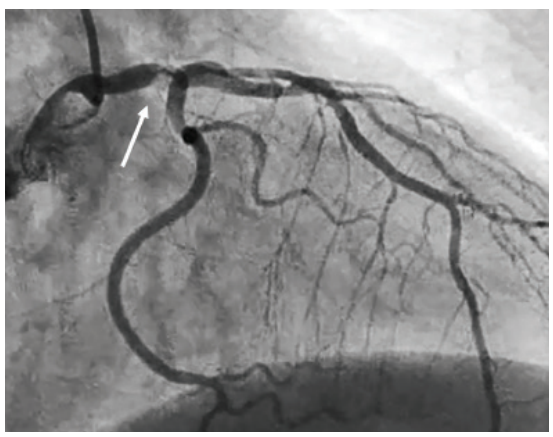

Figure 1. Cineangiographic frame with distal left main stenosis at the bifurcation of the left anterior descending coronary artery and circumflex.

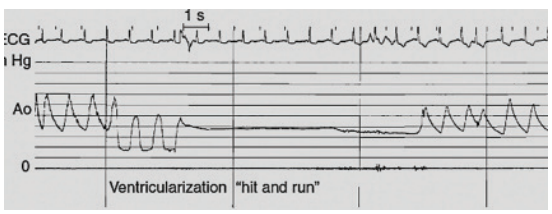

Figure 2. Aortic (Ao) pressure from the coronary angiographic catheter tip showing damping with "ventricularization", which requires quick injection and removal of the catheter, a "hit-and-run" maneuver. To reduce the chances of a coronary dissection, one should realign the catheter in a more coaxial position, if possible.
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Arterial Pressure

Normally, arterial blood pressure is 120/80 mmHg. As we perform a cath, we should be constantly observing the BP and should be alarmed to a degree when we see BP <90/60. In concert with observing the BP, we should look at the heart rate as well. Low BP with low heart rate may indicate a vagal reaction, while low BP with new tachycardia carries more serious connotations, as it is one of the signs of bleeding. Hypotension usually occurs in the cath lab for easily identifiable causes (vasovagal reaction, transient ischemia, or arrhythmia). At other times, the immediate cause is unknown, prompting activation of the team to identify and treat the patient's condition(s).

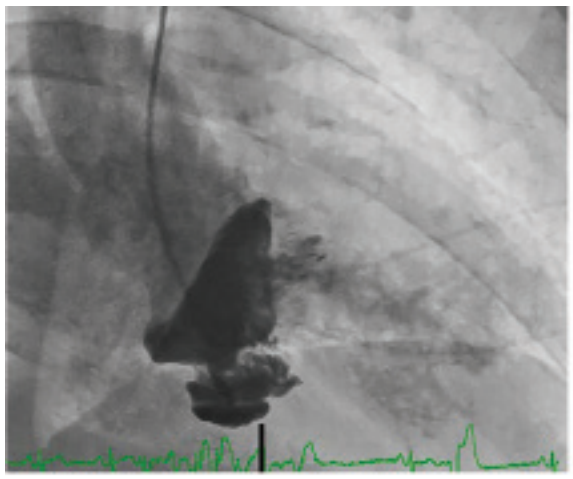

Figure 3. Frames from cine-ventriculography in right anterior oblique (RAO) projection (left image) and left anterior oblique (LAO) (right image) showing extravasation of radiographic contrast media into the left ventricular myocardium.
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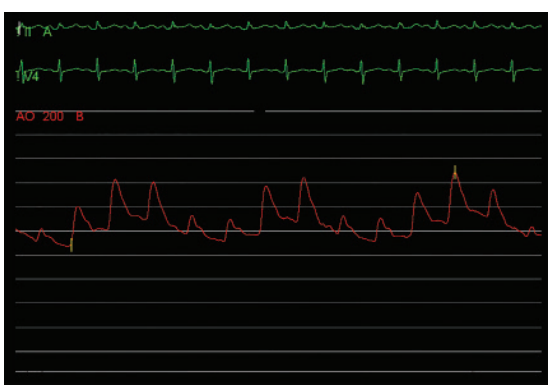

Figure 4. Hypotension after ventriculography. Note the reduced pulse pressure, inspiratory decline in systolic pressure (pulsus paradoxus), and tachycardia consistent with cardiac tamponade.

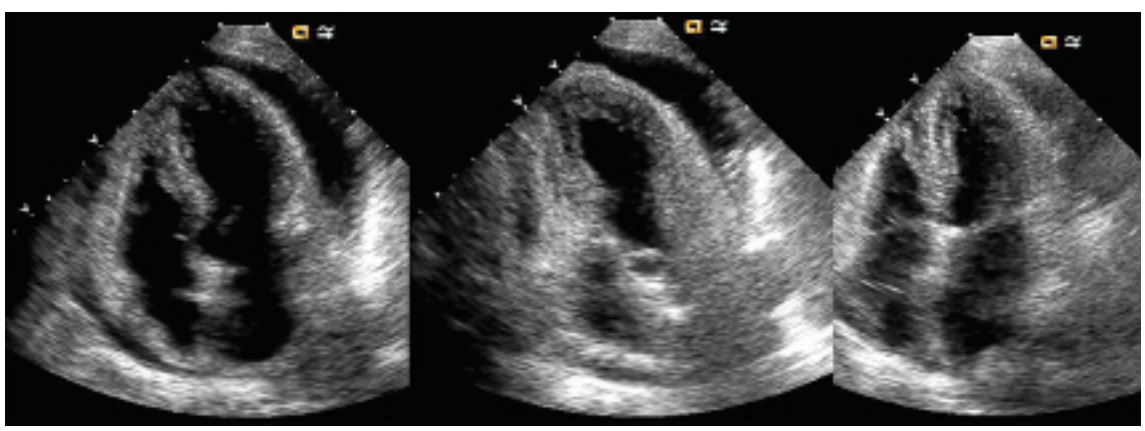

Figure 5. Echocardiographic frames showing pre-pericardiocentesis effusion (left), pericardiocentesis with introduction of echo bubble contrast (middle panel), and post pericardiocentesis after the effusion is removed (right).

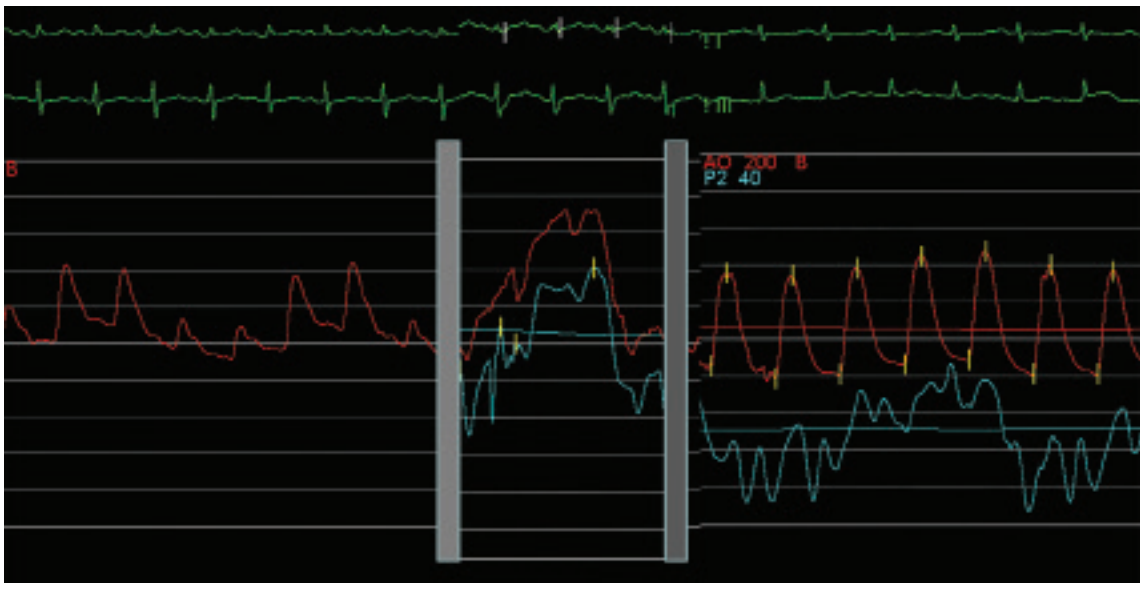

Figure 6. Arterial pressure before pericardiocentesis (left) showing typical pulsus paradoxus. Right atrial (RA) (red) and pericardial pressure (blue) (middle panel) shows pressure elevation with blunting of phasic waveforms of both tracings. After pericardiocentesis (right panel), arterial pressure shows resolution of pulsus paradoxus and reduction of RA pressure to about 12 mmHg (blue tracing).

Hypotension after a coronary contrast injection is an early warning sign of trouble.

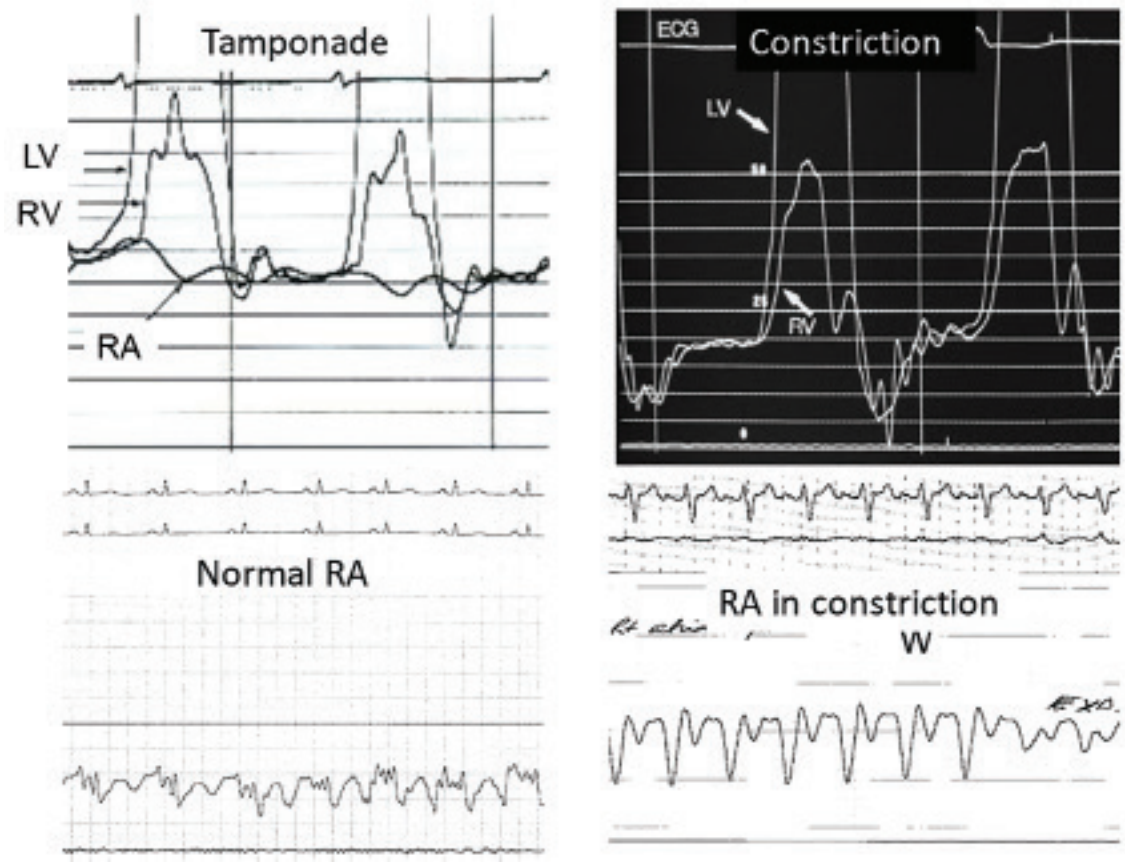

Figure 7. Review of waveforms comparing tamponade vs constrictive physiology. (Top left) Tamponade with diastolic equalization of left ventricular (LV) and right ventricular (RV) pressures (0-40 mmHg scale) with blunted right atrial 'x' and 'y' descents. (Lower left) Normal right atrial waveforms for comparison. (Top right) Constriction with diastolic equalization of LV and RV pressures; however, Y descent is preserved, if not exaggerated, producing the 'dip' followed by the diastolic pressure plateau, with no further ventricular filling until ejection. (Bottom right) Right atrial (RA) pressure wave with steep 'y' descent and blunted 'x' descent, producing a 'W' configuration.

The causes of life-threatening hypotension include left main coronary stenosis, acute myocardial infarction, cardiac tamponade, retroperitoneal bleeding, and anaphylactic contrast reaction. Non-life-threatening hypotension may be due to a vasovagal reaction, sensitivity to pre-medications, or may be artefactual due to loose pressure tubing connections or transducer system errors. For example, a loose tubing connection can register a low blood pressure.

Arterial hypertension (pressure >200/100), while not immediately life-threatening, requires urgent attention to reduce the chance of a stroke or heart failure. Sudden hypertension in the cath lab has been known to be inadvertently induced by the administration of a vasoconstrictor medication unsuspectedly taken from the back table (that's why we label our meds.)

Hypotension After Coronary Injection

Hypotension after a coronary contrast injection is an early warning sign of trouble. For the right coronary artery (RCA), a vagal response (low BP, low heart rate) would be common and is transient. For the left coronary artery, hypotension can herald danger, with the most immediate life-threatening event being left main (LM) obstruction due to dissection, thrombus, or spasm (Figure 1). A LM ostial stenosis is detected by pressure damping on the coronary angiogram, which takes on a ventricular-like wave pattern (Figure 2). Removal or readjustment of the catheter is important to discriminate between non-coaxial malalignment of the catheter tip and true LM narrowing. In patients with LM disease, forceful injections or catheter manipulations with plaque disturbance may cause acute myocardial ischemia due to closure of the vessel. Hypotension during coronary angiography associated with ST segment changes is indeed a life-threatening emergency that requires emergent evaluation, and possibly angioplasty or bypass surgery.

Hypotension After Ventriculography

Rarely, hypotension after ventriculography may occur due a contrast reaction, arrhythmia (ventricular tachycardia [VT]/ventricular fibrillation [VF]) or left ventricular (LV) perforation. We have previously discussed the dangers of end-hole catheter ventriculography[1] by showing a patient with extensive myocardial contrast staining following LV injection using a universal radial Jacky catheter (Terumo) (Figure 3).

In another case illustrating tamponade after an LV gram or during a percutaneous coronary intervention (PCI) with wire perforation, the arterial pressure tracing (Figure 4) shows a narrowed pulse pressure, tachycardia, and large differences in systolic pressure variation between inspiration and expiration (pulsus paradoxus), typical hemodynamic findings for pericardial tamponade. Noting such a pressure, the operators should prepare to

Table 1. Complications of TAVR and associated hemodynamic indicators.

Complication	Hemodynamic Waveform
• Residual aortic stenosis	LV-Ao gradient
• Subvalvular hypercontractile, HCM-like obstruction	LV-Ao gradient, subvalvular
• Acute aortic regurgitation	LV rapid diastolic filling, end-diastolic Ao-LV, normal Ao pp
• Aortic dissection, ascending	If perivalvular leak, same as aortic regurgitation
• Aortic dissection, root with MR	Low BP, high LVEDP and PCW pressure w/ large V wave
• Coronary occlusion	Low BP with ST depression
• LV perforation and tamponade	Low BP, pulsus paradoxus, tachycardia
• Retroperitoneal bleeding	Low BP, Low LVEDP, low RA pressure, tachycardia

LV=left ventricle, Ao=aortic, HCM=hypertrophic cardiomyopathy, pp=pulse pressure, MR=mitral regurgitation, BP=blood pressure, LVEDP=left ventricular end-diastolic pressure, PCW=pulmonary capillary wedge, RA=right atrial.

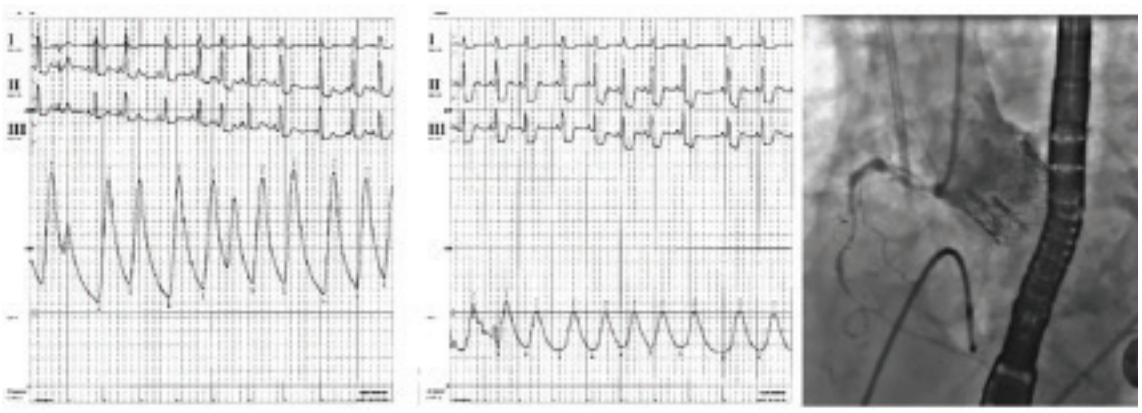

Figure 8. Arterial pressure before (left) and after (middle) transcatheter aortic valve replacement (TAVR) showing arterial hypotension with marked new ST segment depression. Right panel, frame from coronary angiogram of occluded right coronary artery (RCA).
Reprinted with permission from Jilaihawi H, Kar S, Doctor N, et al. Contemporary application of cardiovascular hemodynamics: transcatheter aortic valve interventions. Cardiol Clin. 2011 May; 29(2): 211-222. doi: 10.1016/j.ccl.2011.01.002.

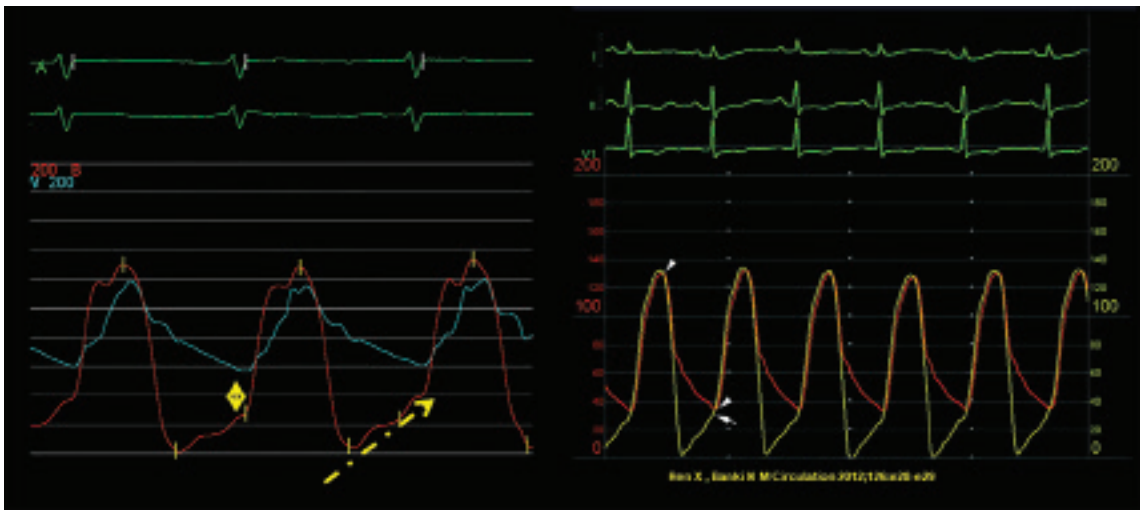

Figure 9. Hemodynamic tracings showing acute aortic regurgitation (left) with elevated and rapid upslope of left ventricular (LV) diastolic pressure (yellow dotted arrow), preserved aortic pulse pressure (double-headed arrow). Subacute aortic regurgitation (right) is characterized by widened aortic pulse pressure (arrowheads), and near equalization of LV and aortic end-diastolic pressures.
Reprinted with permission from Ren X, Banki NM. Classic hemodynamic findings of severe aortic regurgitation. Circulation. 2012; 126(3): e28-e29. doi: 10.1161/CIRCULATIONAHA.112.103937.

perform immediate pericardiocentesis, begin fluid resuscitation, and notify the operating room as the situation requires. An in-lab echo is very helpful in identifying the fluid and assisting in the pericardiocentesis. Figure 5 shows echocardiographic frames of the effusion, bubble contrast during pericardiocentesis image, and a post-cath evacuation of the pericardial fluid.

The hemodynamics before pericardiocentesis show arterial pressure pulsus paradoxus with a mean arterial pressure of 110 mmHg (Figure 6). The middle frame shows elevated right atrial and pericardial pressures. In this case, although RA/pericardial pressures are not identical, the blunted pressure waveforms and near-mean equalization at around 22 mmHg are consistent with the echo findings. After pericardiocentesis, arterial pulse pressure and respiratory variations have resolved with an increase in mean arterial pressure and decrease in RA pressure. The final RA pressure at 12 mmHg suggests some effusive-constrictive pericardiocentesis remains.

Hypotension after TAVR can be caused by the acute coronary occlusion and is associated with the marked ST segment changes of coronary occlusion. The left main occlusion, of course, is the most life-threatening, but also hypotension can occur from occlusion of the right coronary artery (Figure 8).

Hemodynamic Waveforms of Tamponade Versus Constricted Physiology

For a quick review, recall that tamponade compresses the heart and elevates all chambers with near end-diastolic equilibration.[2] Tamponade blunts the X and Y descents of atrial phasic waveforms (Figure 7, left). Compare the findings in the left image of Figure 7 with those of constrictive pericarditis (Figure 7, right). Pericardial constraint permits early rapid LV filling with a sudden cessation of further filling over diastasis, i.e. the early 'dip', followed by a diastolic plateau in both the RV and LV diastolic filling pattern. The right atrial pressure waveform has a steep X and Y descent and W configuration. There is some overlap of hemodynamic filling patterns during early and mid phases of tamponade.

Hypotension After TAVR

The complications of transcatheter aotic valve replacement (TAVR) have been discussed[3] and

Table 2. Differential of hypotension in the cath lab.

Hypotension	Hemodynamics	Associated conditions
LM dissection, occlusion	Low BP	Ischemic ECG changes, shock
Acute MI	Low BP, low CO	Ischemic ECG changes
Tamponade	Low BP, pulsus paradoxus, high HR, narrow pulse pressure	Increased RA pressure, LVEDP, heart sounds, +echo
Retroperitoneal bleed	Low BP	Low LVEDP, abdominal complaints
Anaphylaxis	Low BP, high CO, high HR	Low LVEDP
Artifact of system	Low BP out of proportion to patient's appearance	Patient is comfortable and speaking. Low BP w/o HR changes or other findings.
Vasovagal reaction	Low BP, low HR	Pain, full bladder, visual stimuli
Medication sensitivity	Low BP, normal or increased HR (depending on effect)	Probably premedication effects; consider reversal agents.

LM=left main, BP=blood pressure, ECG=electrocardiogram, MI=myocardial infarction, CO=cardiac output, HR=heart rate, RA=right atrial, LVEDP=left ventricular end-diastolic pressure

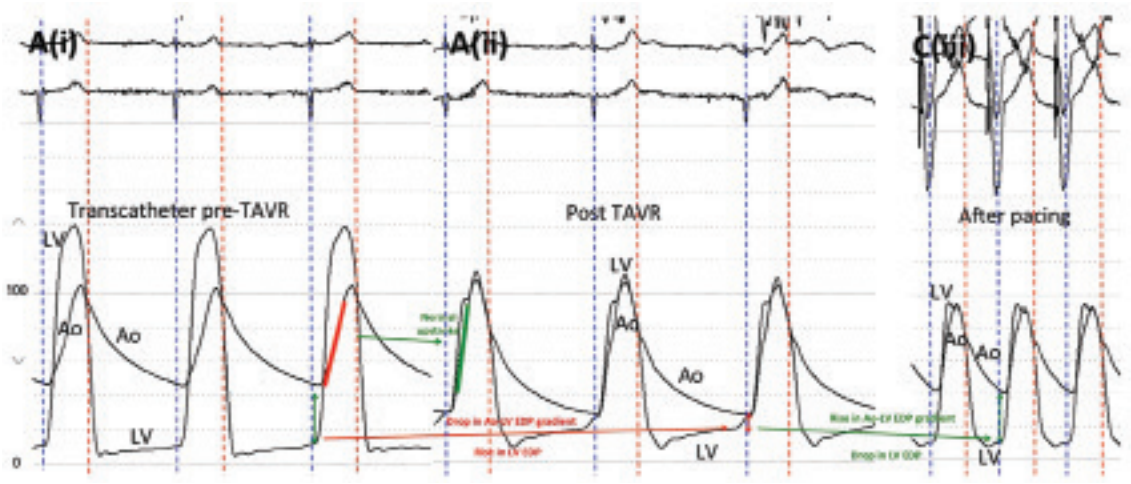

Figure 10. Aortic regurgitation with pacing. (A) Transcatheter hemodynamics before (i) and after (ii) TAVR (with heart-block related bradycardia) and after RV pacing post-TAVR (iii). Resolution of diastasis, increased diastolic pressure and end-diastolic LV-Ao gradient, and drop in LVEDP points to no significant aortic regurgitation. *Courtesy of Dr. Raj Makkar.*

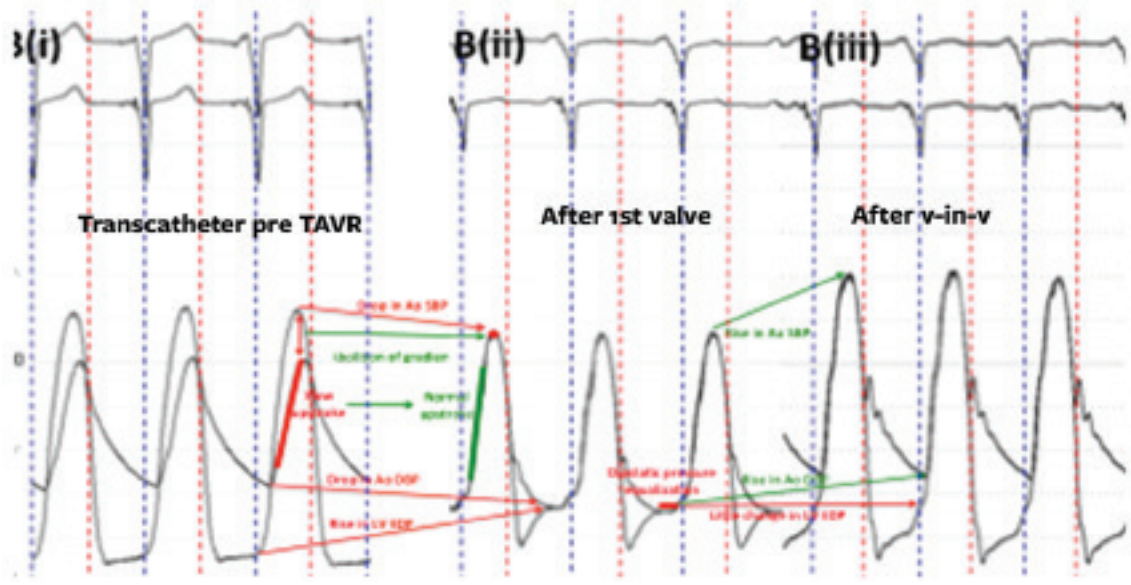

Figure 11. Severe aortic regurgitation post TAVR. (Bi) shows moderate aortic stenosis gradient. After valve implant (Bii), severe aortic regurgitation occurs immediately post TAVR, suggested by significant aortic and LV pressure matching, which ameliorates after valve-in-valve (far right panel). *Courtesy of Dr. Raj Makkar. From Kalra A, Makkar RR, Bhatt DL, et al. Transcatheter and Doppler waveform correlation in transcatheter aortic valve replacement. Open Heart. 2018; 5(1): e000728. doi: 10.1136/openhrt-2017-000728.*

are listed on Table 1. Hypotension after TAVR can be caused by the acute coronary occlusion and is associated with the marked ST segment changes of coronary occlusion. The left main occlusion, of course, is the most life-threatening, but also hypotension can occur from occlusion of the right coronary artery (Figure 8).

Hypotension after TAVR may also occur due to acute aortic regurgitation due to perivalvular leak or leaflet failure. On examination of hemodynamic tracings immediately after TAVR implant in those patients becoming hypotensive, check for wide pulse pressure and look for other causes of aortic regurgitation as noted above. Hemodynamics can separate acute from sub-acute aortic regurgitation physiology. Acute aortic insufficiency is characterized by near-normal arterial pulse pressure and somewhat slower upstroke, but a very dramatic large upstroke of the LV diastolic pressure curve and slightly increased pulse pressure (Figure 9, left). Compare the left image of Figure 9 to the chronic or subacute aortic regurgitation image on the right, where the pulse pressure is markedly enlarged, with equilibration of left ventricular end-diastolic pressure with aortic diastolic pressure, and rapid rise in the LV diastolic pressure wave over diastole (Figure 9, right). Another clue to severe, acute aortic regurgitation is a marked increase in left ventricular end-diastolic pressure and left ventricular diastolic pressure with marked upslope of pressure. In addition, in severe aortic regurgitation, the aortic pressure matches left ventricular pressure. A periaortic regurgitant leak may mimic acute aortic regurgitation. The treatment for severe aortic regurgitation during TAVR is rapid atrial pacing (Figure 10). Rapid pacing shortens the diastolic filling period, reduces the aortic flow into the left ventricle, and stabilizes the patient until the perivalvular leak can be closed.

Severe aortic regurgitation post TAVR due to failed leaflet deployment may require a valve-in-valve insertion to restore normal hemodynamics (Figure 11). On rare occasions, hypotension after TAVR may be due to acute mitral regurgitation

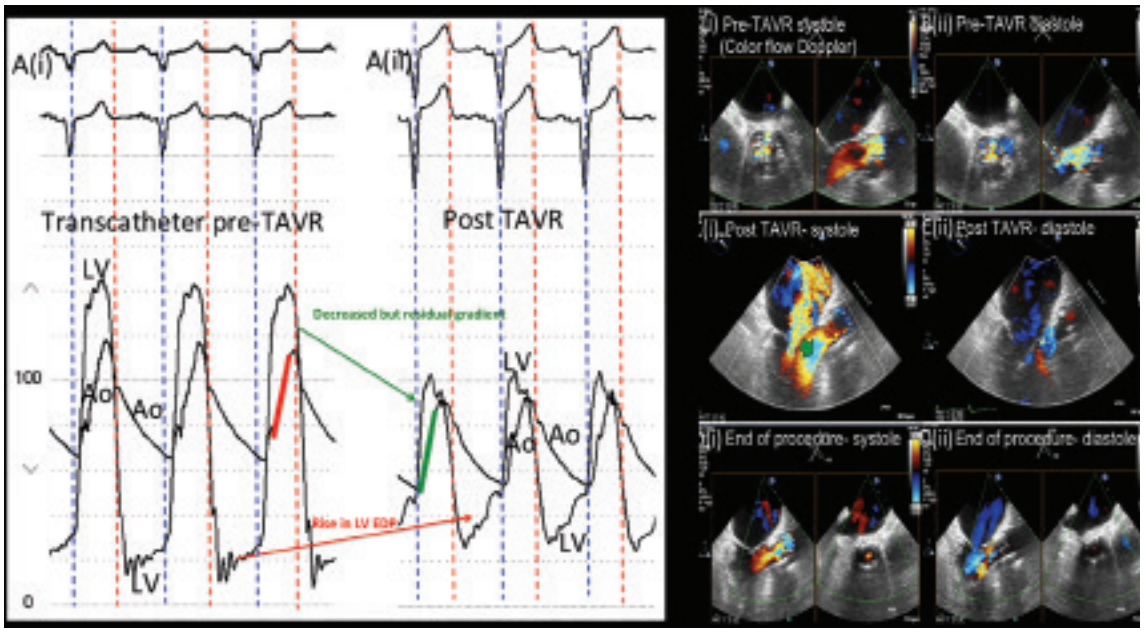

Figure 12. Hypotension after TAVR. Changes in aortic stenosis hemodynamics immediately after TAVR (left panel) show a reduced LV-aortic (Ao) gradient, but with rapid rise in LVEDP with equilibration of the Ao diastolic pressure. By the tracing alone, one cannot differentiate acute aortic regurgitation (AR) from possible mitral regurgitation (MR). Echocardiography (right panel) showed mitral regurgitation after TAVR by systolic anterior motion of the anterior leaflet (AMVL). Color flow Doppler findings at baseline top right, post TAVR (middle right), and at the end of the procedure, in systole (bottom right, i) and diastole (bottom right, ii). Relief of aortic valve stenosis increased contractility resulting in systolic anterior motion of the AMVL and severe mitral regurgitation. Fluid loading relieved the MR.
Courtesy of Dr. Raj Makkar. From Kalra A, Makkar RR, Bhatt DL, et al. Transcatheter and Doppler waveform correlation in transcatheter aortic valve replacement. Open Heart. 2018; 5(1): e000728. doi: 10.1136/openhrt-2017-000728.

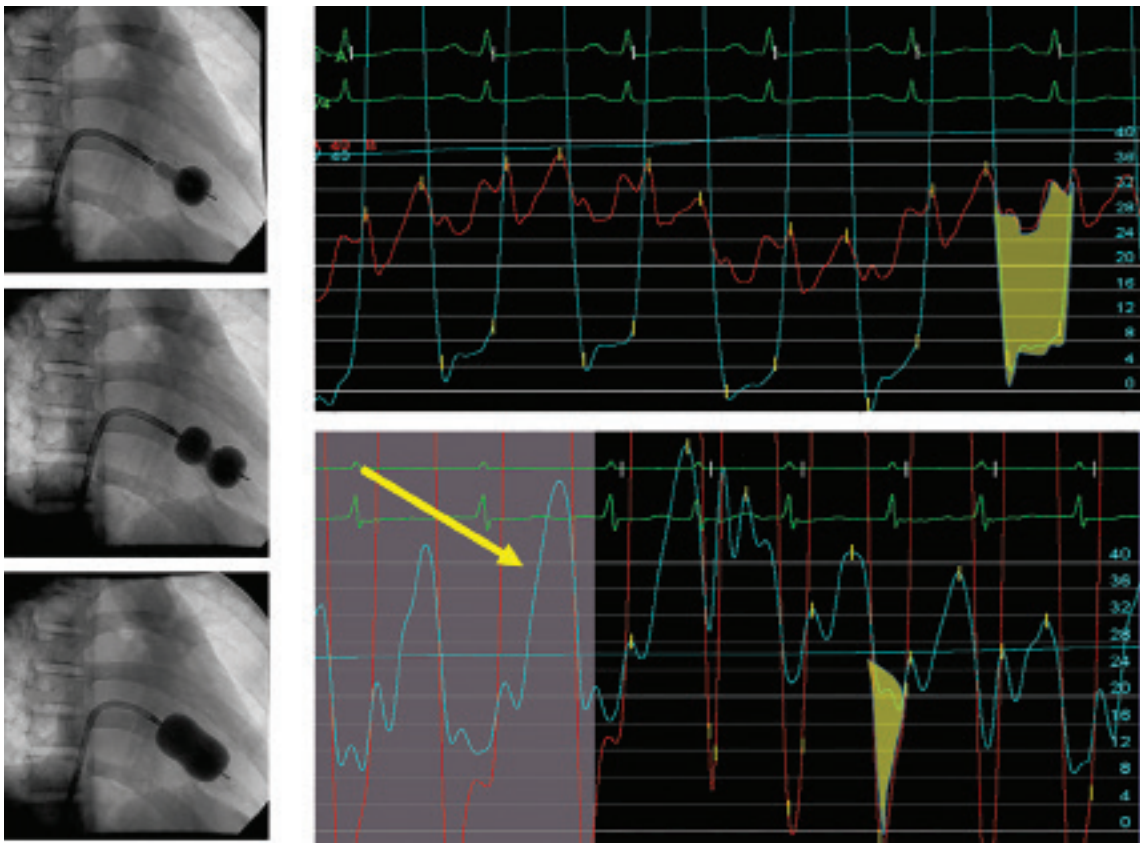

Figure 13. Hemodynamics after mitral balloon valvuloplasty (left panels) reduced the mitral stenosis gradient (top right and bottom right, yellow shaded areas) but produced a giant V wave on the left atrial (LA) pressure tracing (blue tracing) due to overstretched or torn mitral leaflets. Large V wave on lower panel (arrow) is typical for severe acute mitral regurgitation.

(Figure 12). The pulmonary capillary wedge or left atrial pressure shows a markedly elevated new V wave (Figure 13), characteristic of mitral regurgitation due to systolic flow into the atrium. The height of the V wave is determined by the compliance of the left atrium. Another setting where monitoring the PCW (or LA) pressures may be important is that of balloon percutaneous mitral valve valvotomy (Figure 13).

A differential list of causes of hypotension in cath lab can be found in Table 2 and a full review of pertinent hemodynamics can be found elsewhere.[4,5] I hope this brief hemodynamic review will remind us of the quick reactions that may be needed, and will help staff identify dangerous conditions and prevent problems in your lab. ■

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Disclosures: Dr. Morton Kern reports he is a consultant for Abiomed, Abbott Vascular, Philips Volcano, ACIST Medical, Opsens Inc., and Heartflow Inc.

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