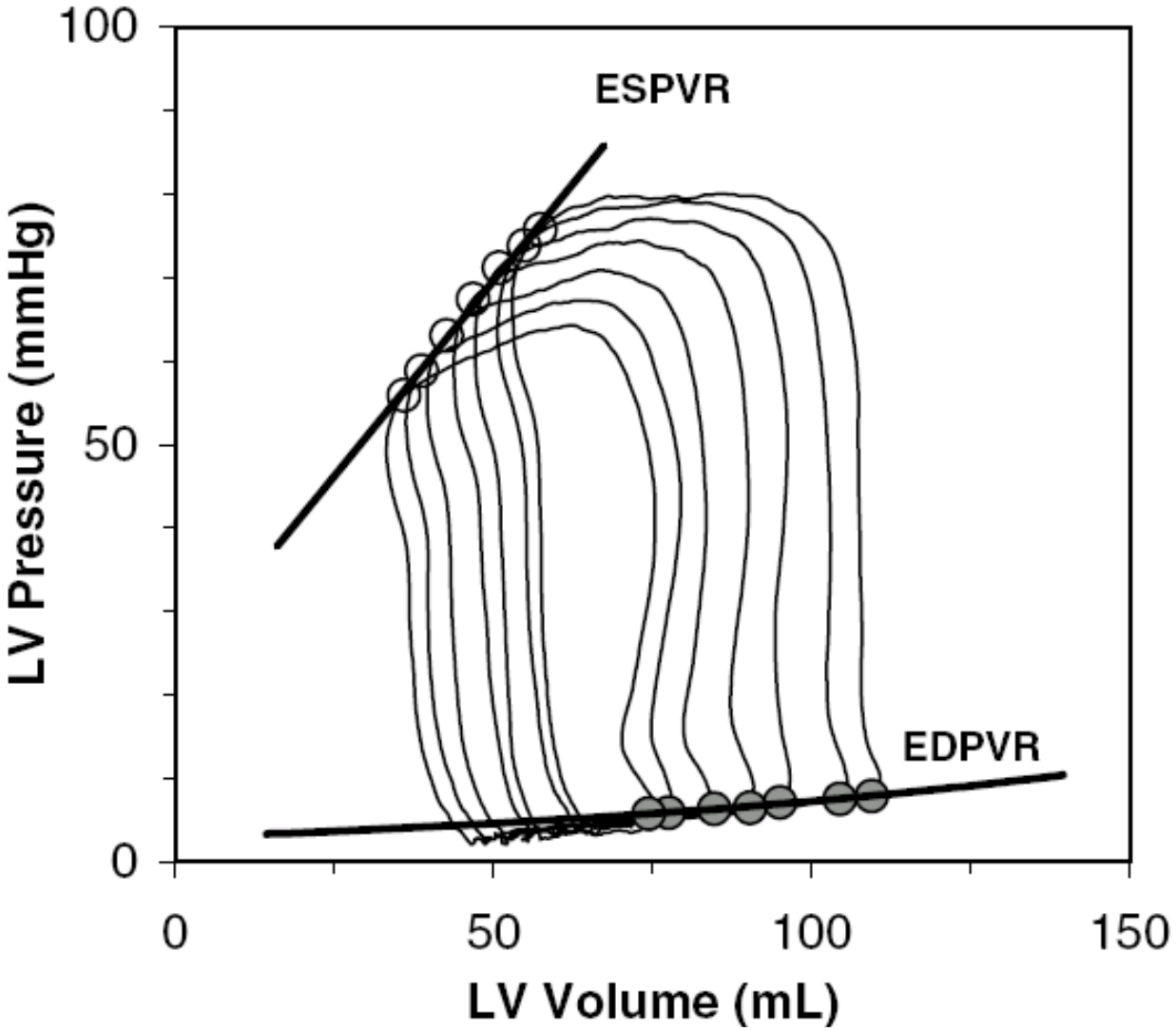
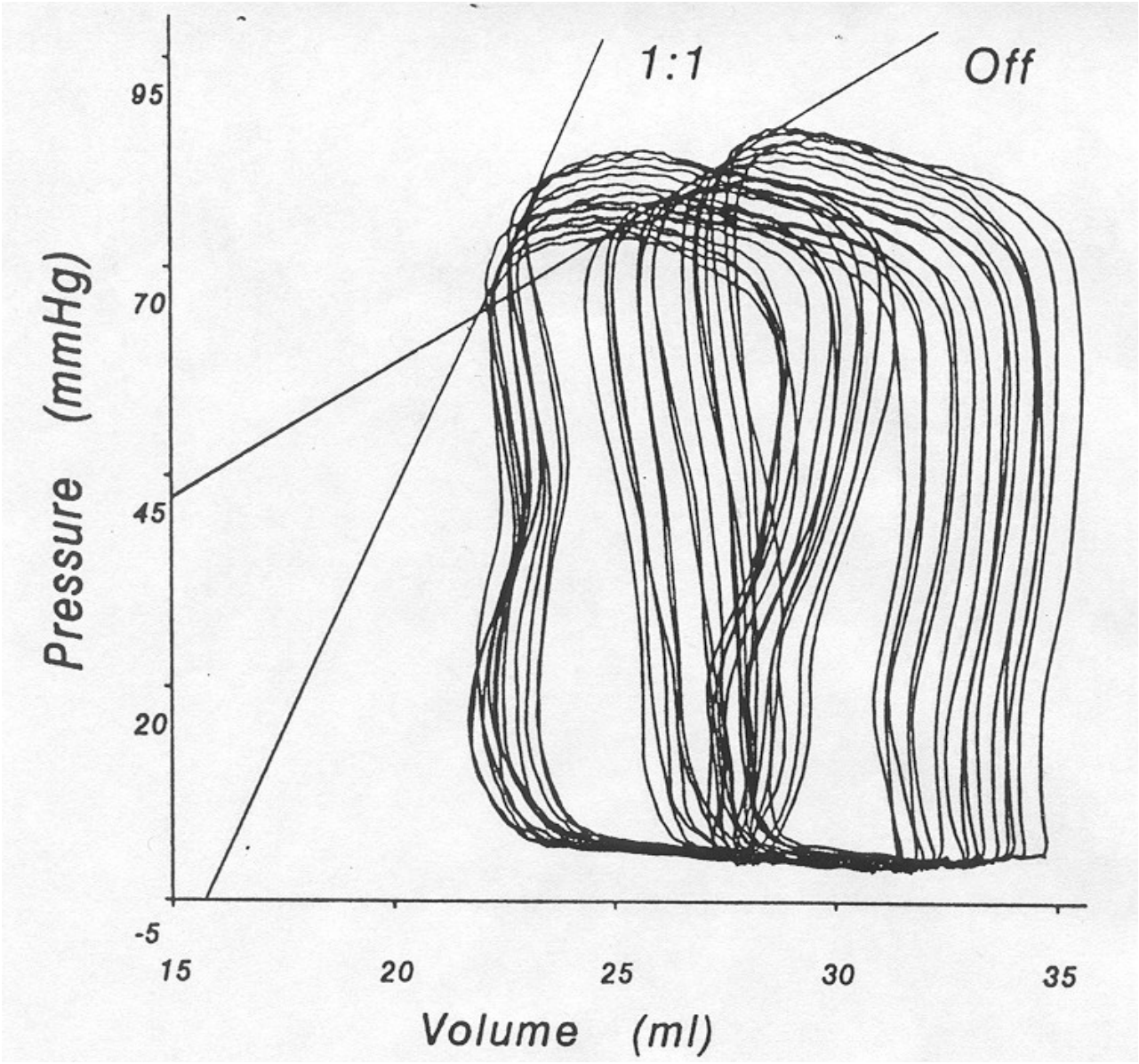


Fig. 4 Pressure–volume loops during preload reduction by vena cava occlusion. Systolic and diastolic function indices are derived from the curves fitted to the end-systolic and end-diastolic pressure–volume points, respectively. From Steendijk et al., 2004, Eur. Hrt. J. Supl. D, D35-42


1:1
Off
95
70
45
20
-5
Pressure (mmHg)
15
20
25
30
35
Volume (ml)

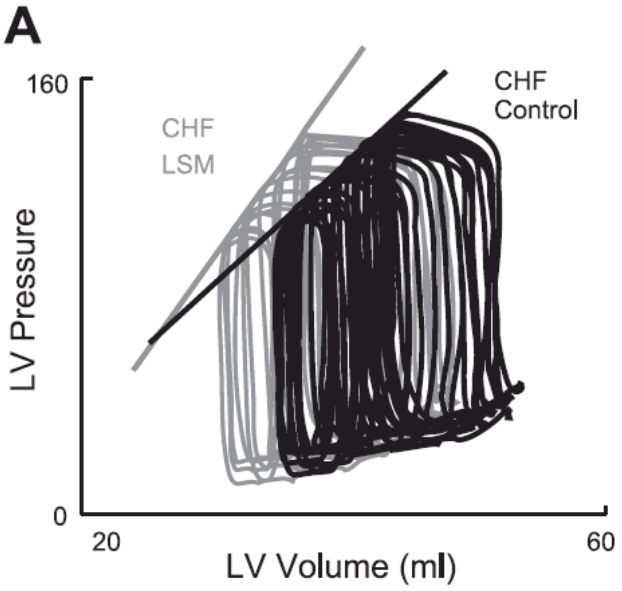
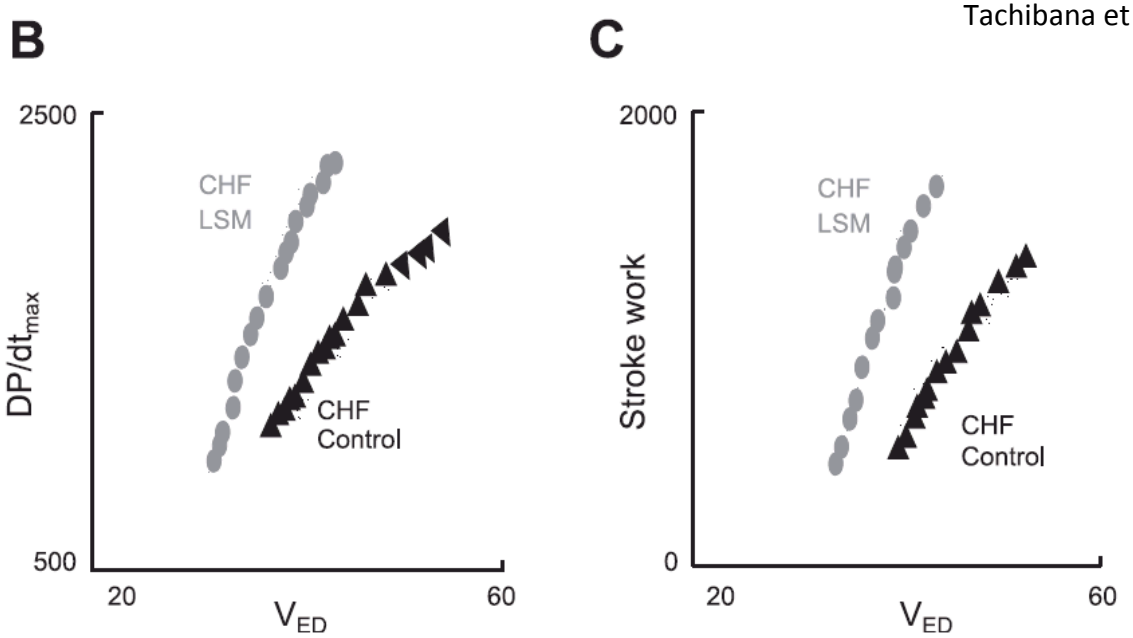


Fig. 2. LV P-V loops and P-V relations determined from a conscious dog after CHF and before and after administration of levosimendan (LSM). Treatment with LSM produced leftward shifts of the LV end-systolic pressure (ESP)-end-systolic volume (ESV; *A*), dP/d*t*max-end-diastolic volume (VED; *B*), and stroke work-end-diastolic volume (*C*) relations with increased slopes. This indicates that LSM increased LV contractility after CHF. From Tachibana et al. (2005) AJP-Heart 288:914



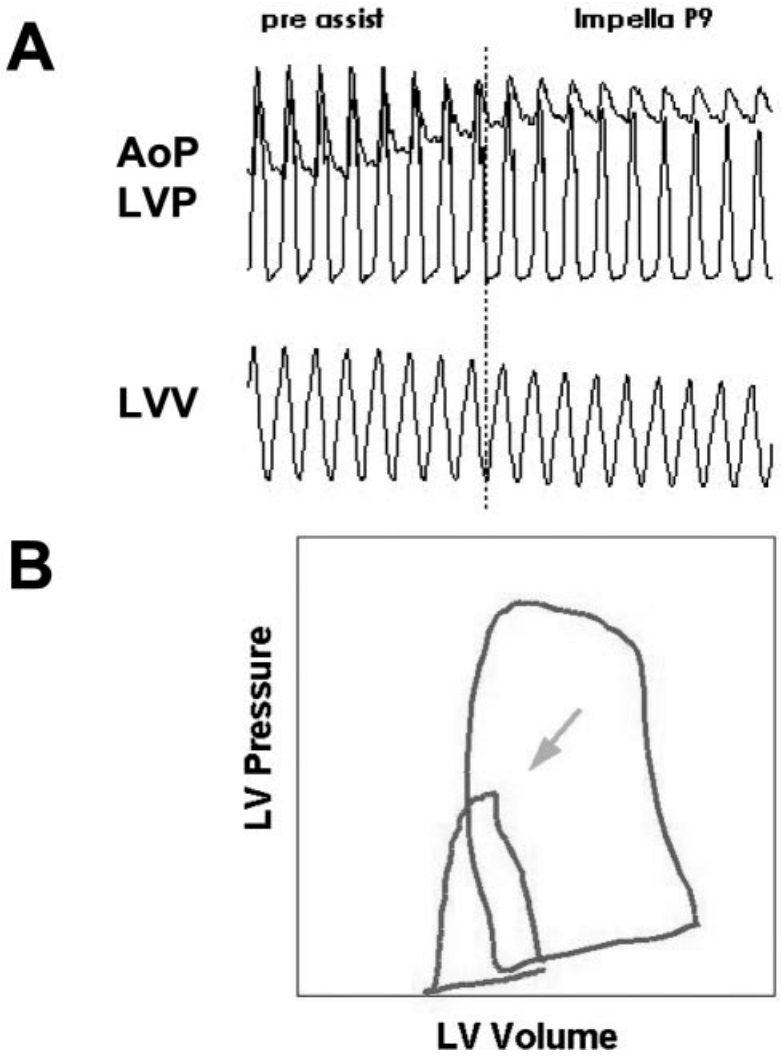


Figure 5. A, Aortic (AoP) and left ventricular pressure (LVP; top) and left ventricular volume (bottom) obtained in animal before and with initiation of hemodynamic support with catheter-based axial flow pump (Impella). This continuous flow pump reduces systolic arterial pressure with little effect on diastolic pressure; left ventricular volume is markedly reduced. B, Left ventricular pressure-volume loops corresponding to tracing in A. Loops demonstrate pressure and volume unloading provided by pump that is associated with markedly reduced oxygen demands of left ventricle.
Tracing provided courtesy of Dr F.H. van der Veen, PhD, Department of Cardiothoracic Surgery, Maastricht, the Netherlands. From Mancini & Burkhoff, 2005 Circ. 112:438.

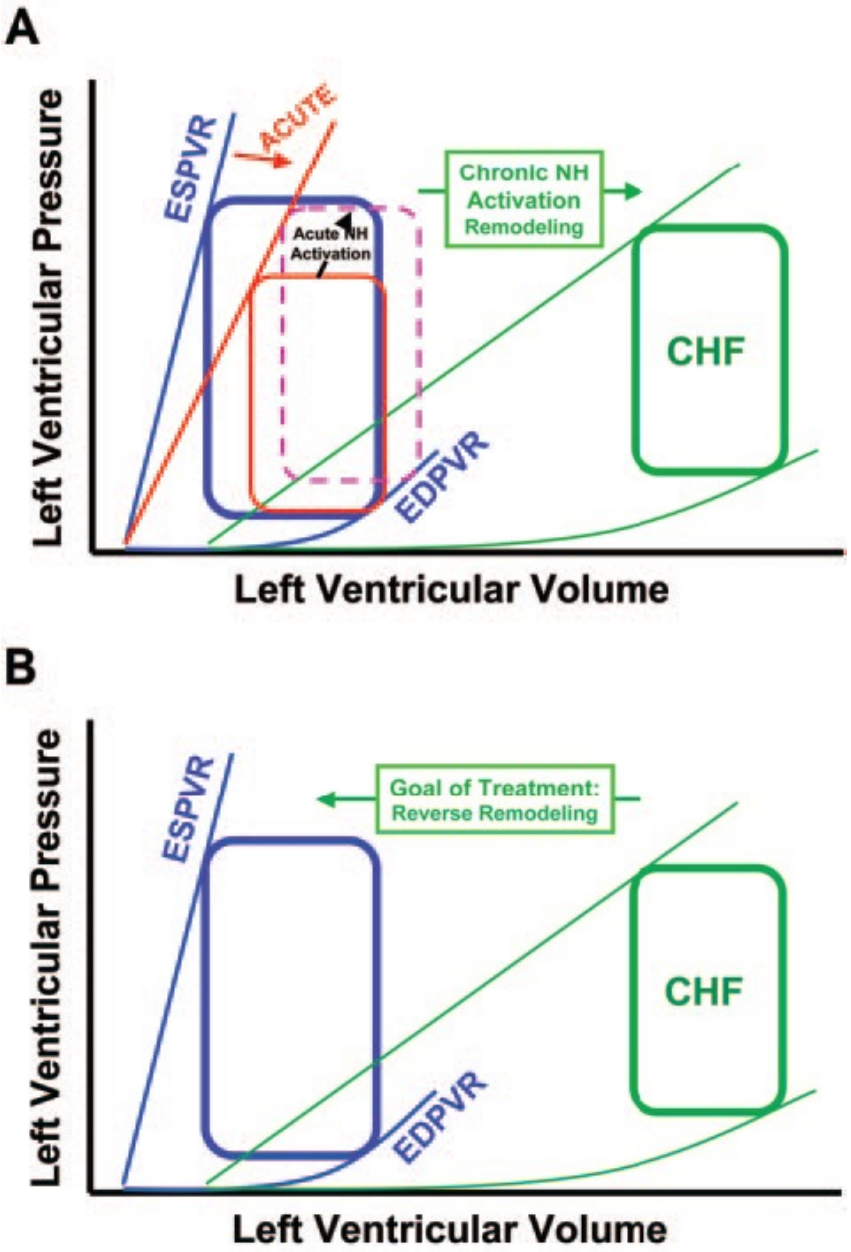


Figure 2. A, Ventricular remodeling represented on pressure-volume diagram. Systolic and diastolic properties shown by end-systolic and end-diastolic pressure-volume relations (ESPVR and EDPVR, respectively) with normal pressure-volume loop (blue). Acute decrease in contractility causes shift of ESPVR with reduced stroke volume and blood pressure (red solid line). Acute neurohormonal (NH) activation increases heart rate and constricts arteries and veins, which increases arterial, venous, and ventricular end-diastolic pressures (dashed purple line). When compensation is incomplete and neurohormonal activation persists, remodeling manifests as progressive shifts toward larger volumes of both ESPVR and the EDPVR (green). B. Goal of many drug- and device-based treatments for heart failure is to prevent, slow, or reverse remodeling so that ESPVR and EDPVR return toward normal. From Mancini & Burkhoff, 2005 Circ. 112:438.

END

Video 3, Module 7