

# Scisense PV Technical Note

## Right Ventricle Hemodynamic Assessment

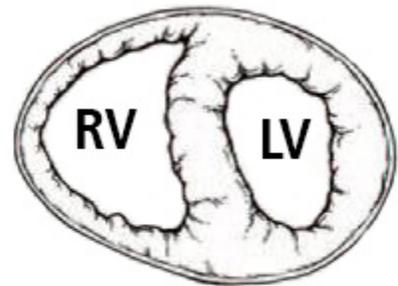
As hemodynamic assessment techniques progress, RV pathology can be better characterized including assessing changes from a healthy to a compensated (hypertrophied) condition and then decompensated condition. Cardiac catheterization remains the best method to diagnose pulmonary hypertension, assess disease severity, and determine prognosis and response to therapies. By directly measuring pressures and volumes, right heart catheterization allows determination of prognostic systolic indices, diastolic indices, pulmonary vascular indices and the coupling ratio. For more information about using right ventricular PV loops to study pulmonary artery hypertension see technical note: "Pulmonary Artery Hypertension & RV PV Loops (RPV-13-tn)."

Primarily, RV function may be impaired due to:

- Primary right sided heart disease
- Secondary LV issues
- Heart valve disease (RV or LV)
- Post-reparative changes from congenital heart disease (e.g. Fallot tetralogy, pulmonary stenosis, insufficiency)
- Post-correction of hypoplastic left heart syndrome
- Arterio-caval shunt (increased afterload)

RV dysfunction may affect LV function by:

- Limiting LV preload
- Systolic and diastolic interaction through intra-ventricular septum
- Pericardium ventricular interdependence by constraints of heart pericardium post-inflammation



**The anatomical and physiological characteristics of a typical right ventricle is complex. Compared to the ellipsoidal LV shape, the RV appears triangular when viewed from the side and crescent shaped when viewed in cross section (above). The RV shape is also influenced by the position of the inter-ventricular septum. Under normal loading and electrical conditions, the septum is concave toward the LV in both systole and diastole. RV has different genetic composition as compared to the LV, making it vulnerable when hypertrophied due to its septomarginal muscular band, which can divide the ventricle into 2 chambers (double-chambered RV). Additionally, the volume of the RV is larger than the volume of the LV, whereas RV mass is approximately one sixth that of the LV. This allows better accommodation of volume but not pressure. A sudden increase in RV pressure is deadly, as compared to a slow afterload increase since RV adaptation occurs at a slower pace.**

## Right Ventricle Hemodynamics Cont.

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### METHODS FOR ASSESSING RV HEMODYNAMICS

#### Non-Invasive Methods:

- Echocardiography (2D or 3D)
- Tissue Doppler imaging (TDI)
- Magnetic resonance imaging (MRI)
- Micro-computed tomography (microCT)

#### Invasive Methods:

- Virtual pressure-volume loops (MRI and Pressure)
- Catheter measurement of PA and pulmonary capillary wedge pressure (PCWP)
- Pressure Catheter combined with Perivascular Flowprobe
- Right ventricle pressure volume (RV-PV) loops (Pressure-volume catheter)

Each method has its advantages and limitations so it is important to consider what parameters are of interest, study methodology requirements, equipment requirements and cost, measurement time, difficulty and repeatability. For a comparison between Echocardiography, PV Catheterization, CT and MRI see "Comparing Cardiac Volume Measurement Modalities (RPV-10-tn)."

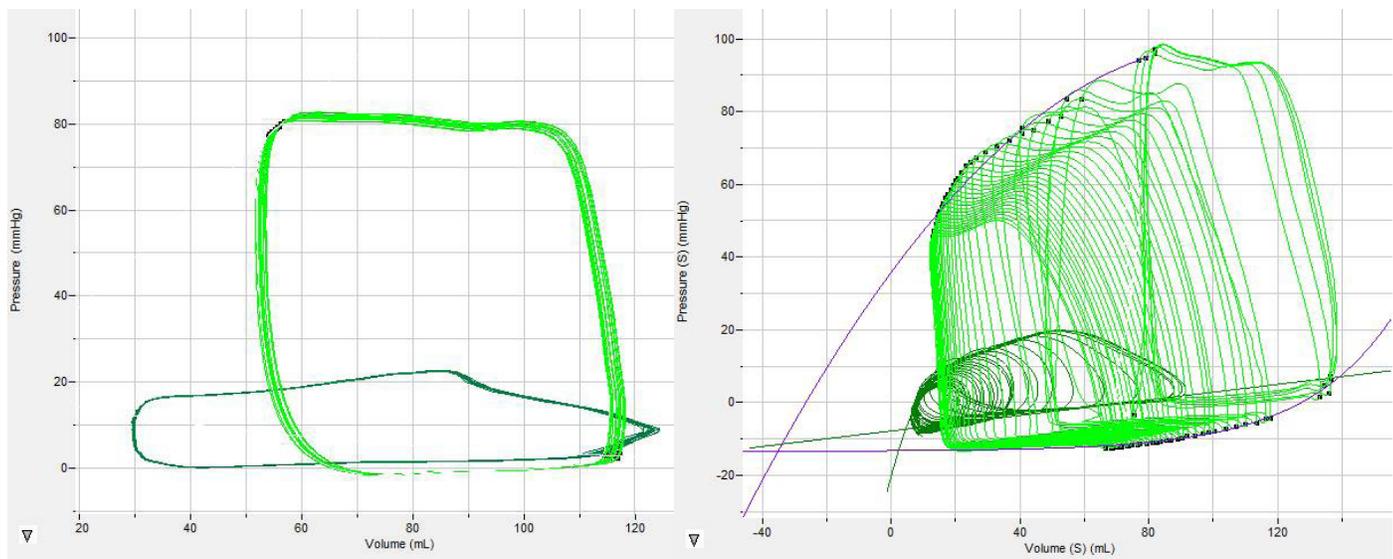
Note: Major difficulties of basic RV 2D echocardiography as compared to the basic LV 2D echo include:

- Inadequate formula to characterize RV volume based on the RV shape. Current formulas specific to the LV.
- As the RV has thinner walls and chamber has more trabeculations as compared to the LV, it is more complex to echocardiographically characterize it.
- An acoustic barrier is created by its retrosternal position.

## Right Ventricle Hemodynamics Cont.

### RIGHT VENTRICLE PRESSURE-VOLUME LOOP CHARACTERISTICS AS COMPARED TO THE LEFT VENTRICLE

- EDPVR of the RV is characterized by its high compliance (Compliance is increased) as compared to LV.
- At any given end-diastolic volume (EDV) the RV end-diastolic pressure (EDP) is lower, making the final EDPVR shallower as compared to the LV.
- Ejection of blood into the highly compliant, low-resistance pulmonary circulation results in better coupling ( $E_{es}/E_a$ ).
- Normal RV generates less than 20% of the stroke work (SW) of the LV while moving the same volume of blood. Compared to LV, a much lower proportion of RV SW goes to pressure generation as compared to LV.
- The RV accommodates dramatic variations in venous return resulting from changes in volume status, position, and respiration while maintaining more or less constant cardiac output (CO). In part this is because the thin RV is easily distensible, but to a larger extent it is a direct result of RV geometry. Like the LV, the RV utilize the Frank-Starling mechanism to increase SW as a consequence of an increase in RV stretch, but much larger changes in RV volume are needed before Frank-Starling mechanism is engaged.
- The RV coronary perfusion pattern significantly differs from that of the LV. Because tissue pressure in the LV rises during systole to systemic levels, coronary perfusion of the LV is largely confined to the diastolic interval. Tissue pressure in the RV does not normally exceed aortic root systolic pressure, permitting continued coronary flow throughout the cardiac cycle; thus, under typical hemodynamic conditions, coronary flow to the RV is roughly balanced between systolic and diastolic time periods.



**Pressure-Volume loops during steady state recording. Images are taken from the same sheep heart. Dark green PV loops represent RV steady state, while light green are LV PV loops.**

**Pressure-Volume loops during IVC occlusion. Images are taken from the same swine heart. Dark green PV loops represent RV, while light green are LV PV loops post-IVC occlusion.**

## Right Ventricle Hemodynamics Cont.

RV INDICES IN MICE (Tabima et. al. 2010)			
INDICES	HEALTHY RV	HYPERTROPHIED RV	FORMULA
Heart rate (HR)	611 ± 31 bpm	636 ± 31 bpm	
RV Systolic pressure	27 ± 3 mmHg	45 ± 17 mmHg	
RV Diastolic pressure	1.4 ± 0.9 mmHg	2.7 ± 1.4 mmHg	
RV Stroke work (SW)	386 ± 76 mmHg* $\mu$ L	926 ± 265 mmHg* $\mu$ L	SV*MAP
RV Cardiac output (CO)	8.5 ± 2.3 ml/min	5.2 ± 2.6 ml/min	SV*HR
RV Stroke volume (SV)	13.9 $\mu$ L	8.2 $\mu$ L	CO/HR (EDV-ESV)
Ejection Fraction (EF)	51 ± 11%	28 ± 13%	SV/EDV
Mean Arterial pressure (MAP)	27.8 mmHg	112.9 mmHg	SW/SV
dP/dt <sub>max</sub>	2522 ± 660 mmHg/s	3164 ± 826 mmHg	
dP/dt <sub>max</sub> - end-diastolic volume	84 ± 17 mmHg/s/ $\mu$ L	177 ± 93 mmHg/s/ $\mu$ L	dp/dt <sub>max</sub> vs. EDV
dP/dt <sub>min</sub>	-1971 ± 499 mmHg/s	-3009 ± 1120 mmHg/s	
Preload recruitable stroke work (PRSW)	20.9 ± 5.6 mmHg	33.9 ± 5.9 mmHg	SW vs. EDV
Ventricular end-systolic elastance (Ees)	1.8 ± 0.5 mmHg/ $\mu$ L	2.4 ± 0.2 mmHg/ $\mu$ L	ESP/ESV
Effective arterial elastance (Ea)	2.7 ± 1.2 mmHg/ $\mu$ L	16.4 ± 2.5 mmHg/ $\mu$ L	ESP/SV
Coupling ratio: Ees/Ea	0.71 ± 0.27 Optimal > 0.5	0.35 ± 0.17 Uncoupled < 0.5	SV/ESV

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