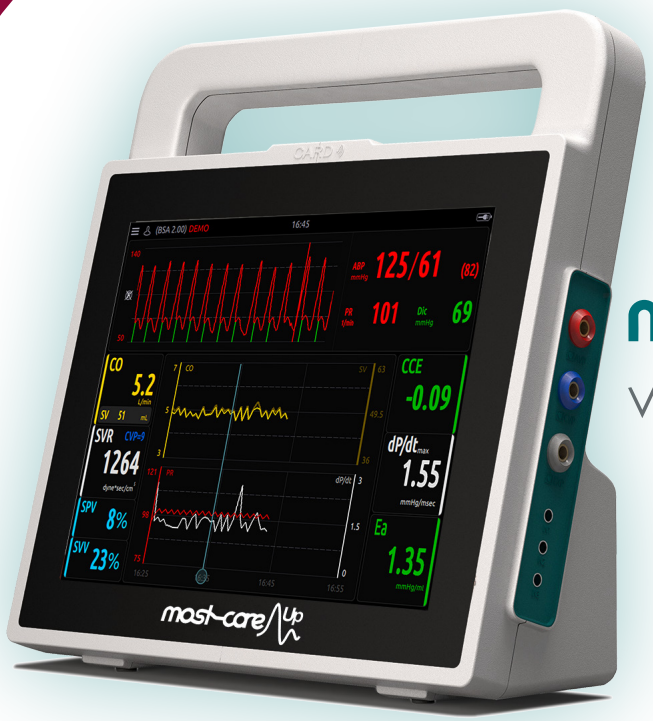
# MEDICAL EQUIPMENT

## Haemodynamic monitoring

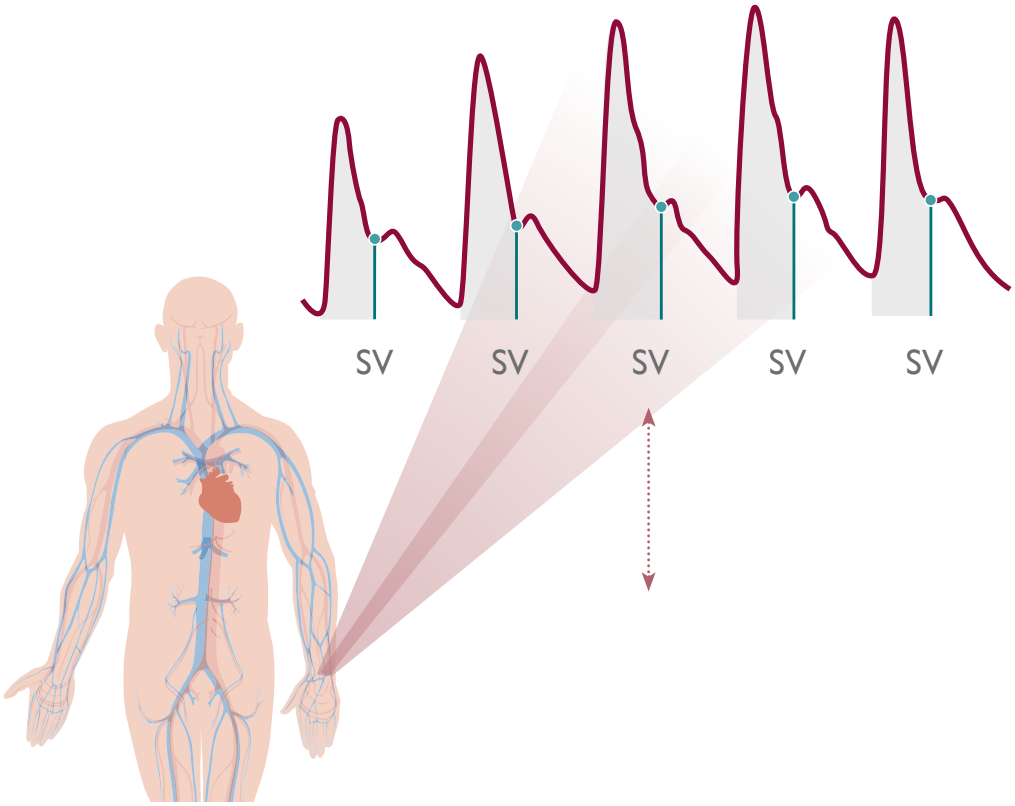



mostcare Up

variables

VYGON

Value Life




**CO**

| Pulse rate | Preload | Contractility | Afterload |
|---|---|---|---|
| PR | SVV, PPV *response to fluids* | $dP/dt_{max}$ *systolic function* | SVR, Ea |
| **chronotropic** | **volume** | **inotropes** | **vasoactives** |

CPO

**Efficiency**

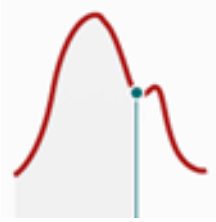
CCE

# STANDARD HAEMODYNAMIC VARIABLES

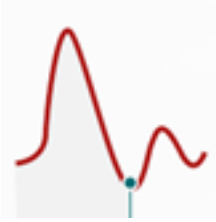
## Pressure and pulse rate

### ABP – Arterial Blood Pressure


PRAM measures systolic pressure as the maximum pressure value during the left ventricular ejection phase and diastolic pressure as the true end-diastolic pressure (not always corresponding to the lowest pressure value in cardiac cycle). Mean arterial pressure is calculated with standard formulas.



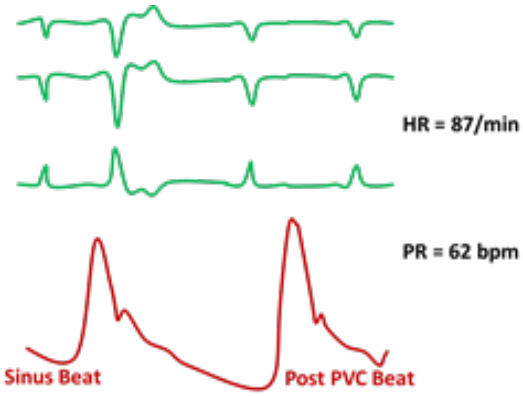
*A. Standard wave*



*B; Dicrotic pressure lower than diastolic pressure*

### PR - Pulse rate


wPulse rate is the number of effective heart beats per minute detected from the arterial blood pressure signal.
MostCareUp actually gives the number of mechanical pulses, independently from the electrical rate of contraction (e.g. ineffective ectopic beats).




### PP - Pulse Pressure


Pulse pressure is the difference between the systolic and diastolic pressure.
PP is a surrogate marker of arterial stiffness, it represents the relationship between left ventricle ejection and vascular status.

An increase in PP (for instance > 40 mmHg), when not related to aortic valve insufficiency, may correspond to a reduction of diastolic pressure (reduced vascular tone/resistance) and/or to an increase in systolic pressure related to arterial stiffness or to an increase in the volume ejected (stroke volume).


1. Dart AM, Kingwell BA. Pulse pressure: a review of mechanisms and clinical relevance. J Am Coll Cardiol. 2001 Mar 15;37(4):975-84.
2. Nawrot TS, Staessen JA, Thijs L, Fagard RH, Tikhonoff V, Wang JG, Franklin SS. Should pulse pressure become part of the Framingham risk score? J Hum Hypertens. 2004 Apr;18(4):279-86.
3. Franklin SS. Beyond blood pressure: Arterial stiffness as a new biomarker of cardiovascular disease. J Am Soc Hypertens 2008 May-Jun;2(3):140-51.
4. Safar ME, Plante GE, Mimran A. Arterial stiffness, pulse pressure, and the kidney. Am J Hypertens. 2015 May;28(5):561-9. doi: 10.1093/ajh/hpu206.
5. Al-Khalisy H, Nikiforov I, Jhajj M, Kodali N, Cheriyath P. A widened pulse pressure: a potential valuable prognostic indicator of mortality in patients with sepsis. J Community Hosp Intern Med Perspect. 2015 Dec 11;5(6):29426

# Flow and perfusion

## SV - Stroke Volume

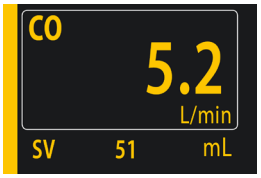

Stroke volume is the amount of blood ejected by the left ventricle in one beat.
PRAM facilitates the measurement of SV on a beat by beat basis. SV can be displayed as single beat value or average of n. beats.
Physiological range at rest: 60 to 100 mL.
The use of stroke volume monitoring and optimisation is recommended for perioperative Goal-Directed Therapy (GDT) to improve patient outcomes.

*Available also:*

| | | |
|---|---|---|
| SVI = SV/BSA | stroke volume index | from 35 to 45 mL/m$^2$ |
| $SV_{kg}$ = SV/weight | weighted stroke volume | (pediatric use) |

1. European Society of Anaesthesiology (ESA, 2016) Perioperative Goal-Directed Therapy Protocol Summary.
2. Oscier C, Cecconi M. Goal-directed therapy to maintain haemostasis. Best Pract Res Clin Anaesthesiol. 2016 Jun;30(2):217-28.

## CO - Cardiac Output




Cardiac output is the volume of blood ejected by the left ventricle in one minute.

CO = SV x PR

Physiological range at rest: 4 - 8 L/min.

CO determines the blood flow distribution to tissue and organs. Its value is computed from the SV on a beat by beat basis, tracking in real-time the rapid haemodynamic changes.

*Available also:*

| | | |
|---|---|---|
| CI = CO/BSA | cardiac output index | from 2.6 to 3.8 L/min/m$^2$ |

## $DO_2$ - Oxygen Delivery


Oxygen delivery is the amount of oxygen delivered to the tissue over time unit.

$DO_2 = CO \cdot CaO_2$ with $CaO_2 = Hb \cdot 1{,}34 \cdot SaO2$ (neglecting dissolved $O_2$)

$CaO_2$ is computed from a value of haemoglobin concentration (Hb) and arterial oxygen saturation ($SaO_2$), manually entered by the operator in the monitor.
Physiological range at rest: 600-900 mLO2/min.
Oxygen Delivery Index optimisation is recommended for perioperative Goal-Directed Therapy (GDT) to improve patient outcomes.

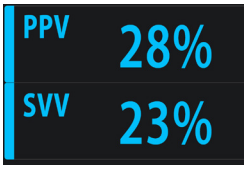
*Available also:*

| | | |
|---|---|---|
| $DO2I = CO/BSA \cdot CaO_2$ | Oxygen delivery index | from 400 to 600 mL/min/m2 |

1. Vincent JL, Pelosi P, Pearse R, Payen D, Perel A, Hoeft A, Romagnoli S, Ranieri VM, Ichai C, Forget P, Della Rocca G, Rhodes A. Perioperative cardiovascular monitoring of high-risk patients: a consensus of 12. Crit Care. 2015 May 8;19:224.
2. European Society of Anaesthesiology (ESA, 2016) Perioperative Goal-Directed Therapy Protocol Summary.
3. Saugel B, Vincent JL, Wagner JY. Personalized hemodynamic management. Curr Opin Crit Care. 2017 Aug;23(4):334-341.

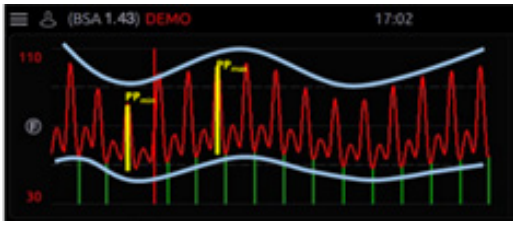
# Fluid Responsiveness

## Dynamic fluid responsiveness variables:




- **PPV - Pulse Pressure Variation**
- **SVV - Stroke Volume Variation**
- **SPV - Systolic Pressure Variation**

Adequate venous return to the heart is essential to support optimal stroke volume and hence Cardiac Output. Hypovolaemia may be harmful to patients and excessive fluid administration may also be harmful. The traditional static measures of preload (CVP, PAOP) are ineffective in predicting fluid responsiveness (i.e. whether the patient will develop an increase in CO in response to fluid administration).
PPV, SVV, and SPV are dynamic variables which are based on heart lung interactions during mechanical ventilation and have been shown to be accurate predictors of fluid responsiveness in mechanically ventilated patients under general anaesthesia or deep sedation. The increase in intrathoracic pressure induced by a positive pressure breath from the ventilator induces a change in pulse pressure, stroke volume and systolic pressure. PPV, SVV and SPV measure the percentage variations of these variables, in response to ventilation, within a fixed interval of time (15 seconds as a default in MostCareUp). The length of the time interval considered for the calculation of the dynamic variables can be adapted (from 10 to 30 seconds) to the duration of the respiratory cycle, along with a more flexible formula to calculate the values of the dynamic variables.




$$PPV = \frac{PPmax-PPmin}{(PPmax+PPmin)/2} * 100$$

$$SVV = \frac{SVmax-SVmin}{(SVmax+SVmin)/2} * 100$$

$$SPV = \frac{SPmax-SPmin}{(SPmax+SPmin)/2} * 100$$

PPV is often preferred to SVV because it is measured and not calculated. In addition, PPV demonstrated higher specificity and sensitivity values for predicting fluid responsiveness compared to other dynamic variables.
When PPV value is less than 9% administration of fluid is unlikely to lead to an increase in cardiac output (i.e. not fluid responsive).
When the PPV is greater than 13% administration of fluid is likely to lead to an increase in CO. (i.e. patient is fluid responsive). When PPV is in the grey zone (9-13%) response to fluid administration cannot be predicted.

It is important to appreciate that PPV, SVV and SPV are only validated for use as predictors of fluid responsiveness in specific conditions, including mechanically ventilated patients with no spontaneous breathing and who are in normal sinus rhythm.

The use of PPV or SVV monitoring and optimisation are recommended for perioperative Goal-Directed Therapy (GDT) to improve patients' outcomes.

1. Pinsky MR, Payen D. Functional hemodynamic monitoring. Crit Care. 2005.
2. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. Crit Care Med. 2009 Sep;37(9):2642-7.
3. Cannesson M, Le Manach Y, Hofer CK, Goarin JP, Lehot JJ, Vallet B, Tavernier B. Assessing the diagnostic accuracy of pulse pressure variations for the prediction of fluid responsiveness: a «gray zone» approach. Anesthesiology. 2011 Aug;115(2):231-41.
4. Vincent JL, Rhodes A, Perel A, Martin GS, Della Rocca G, Vallet B, Pinsky MR, Hofer CK, Teboul JL, de Boode WP, Scolletta S, Vieillard-Baron A, De Backer D, Walley KR, Maggiorini M, Singer M. Clinical review: Update on hemodynamic monitoring--a consensus of 16. Crit Care. 2011 Aug 18;15(4):229.
5. Carsetti A, Cecconi M, Rhodes A. Fluid bolus therapy: monitoring and predicting fluid responsiveness. Curr Opin Crit Care. 2015 Oct;21(5):388-94.
6. European Society of Anaesthesiology (ESA, 2016) Perioperative Goal-Directed Therapy Protocol Summary.
7. Monnet X, Marik PE, Teboul JL. Prediction of fluid responsiveness: an update. Ann Intensive Care. 2016 Dec;6(1):111.
8. Toscani L, Aya HD, Antonakaki D, Bastoni D, Watson X, Arulkumaran N, Rhodes A, Cecconi M. What is the impact of the fluid challenge technique on diagnosis of fluid responsiveness? A systematic review and meta-analysis. Crit Care. 2017;21(1):207.

# Afterload

## SVR - Systemic Vascular Resistance


Systemic Vascular Resistance is one of the determinants of the left ventricle afterload.
SVR = (MAP-CVP) · 80 /CO
In MostCareUp the value of central venous pressure (CVP), mandatory for the calculation of SVR, can be entered manually or can be continuously monitored via a central venous line and transducer.
Physiological range at rest: 800 – 1400 dyne·sec/cm5
As a component of arterial load, SVR will vary with changes in the haemodynamic status of the patient (e.g. septic shock, cardiac failure, hypovolemia).

*Available also:*

| | | |
|---|---|---|
| SVRI = (MAP-CVP) / CI | SVR index | 1600 – 2400 dyne·sec·m2/cm5 |

# ADVANCED VARIABLES

## Afterload

### Ea – Arterial Elastance


The left ventricle ejects blood into the aorta and arterial system. The arterial load represents the force against which the left ventricle must eject. This "arterial load" contains the resistances to flow and the elasticity of arterial system. Effective arterial elastance (Ea) is mainly related to the elasticity of the arterial system.
The elasticity of the large arteries allows these vessels to expand during the ejection phase accommodating more blood and then, because of the systolic stored of elastic energy relapsed during diastole, assures diastolic blood flow in periphery. This arterial property allows to convert pulsatile flow in more continuous one according to the organ vascular resistances. The more rigid the vessels, the lower the volume (i.e. higher Ea), the less rigid the vessels, the higher the volume (i.e. lower Ea).

Arterial elastance is physiologically determined by the ratio of end systolic pressure and stroke volume (ESP/SV, mmHg/mL). ESP corresponds to aortic valve closure which is the dicrotic pressure (Dic) on the pressure wave. In MostCareUp ESP is replaced by the dicrotic pressure (Dic).

Ea = Dic / SV

Physiological range at rest: from 1.10 to 1.40 mmHg/mL

Arterial elastance can change according to different drug administered (e.g. Ca++ antagonist, B-blocker, nitrates, volume-induced vasodilation, etc…).

1. Nichols WW, O'Rourke MF. Vascular impedance. In: McDonald's Blood Flow in Arteries: Theoretical, Experimental and Clinical Principles. 4th ed. London, UK: Edward Arnold; 1998:54–97, 243–283, 347–395.
2. Chantler PD, Lakatta EG, Najjar SS. Arterial-ventricular coupling: mechanistic insights into cardiovascular performance at rest and during exercise. J Appl Physiol (1985). 2008 Oct;105(4):1342-51.
3. Scolletta S, Bodson L, Donadello K, Taccone FS, Devigili A, Vincent JL, De Backer D. Assessment of left ventricular function by pulse wave analysis in critically ill patients. Intensive Care Med 2013;39: 1025-33.
4. Morelli A, Singer M, Ranieri VM, D'Egidio A, Mascia L, Orecchioni A, Piscioneri F, Guarracino F, Greco E, Peruzzi M, Biondi-Zoccai G, Frati G, Romano SM. Heart rate reduction with esmolol is associated with improved arterial elastance in patients with septic shock: a prospective observational study. Intensive Care Med 2016.
5. Messina A, Romano SM, Bonicolini E, Colombo D, Cammarota G, Chiostri M, Della Corte F, Navalesi P, Payen D, Romagnoli S. Cardiac cycle efficiency and dicrotic pressure variations: new parameters for fluid therapy: A pilot observational study. Eur J Anaesthesiol 2017; 34:1–9.
6. Monge Garcia MI, Jian Z, Settels JJ, Hatib F, Cecconi M Pinsky MR. Reliability of effective arterial elastance using peripheral arterial pressure as surrogate for left ventricular end-systolic pressure. J Clin Monit Comput. 2018 Dec 14.

### PPV/SVV - Dynamic Elastance


Dynamic elastance, is the ratio between PPV and SVV. It has been suggested as a variable that represents the dynamic changes of arterial load and tone in mechanically ventilated patients.
MostCareUp calculates the averaged value of PPV/SVV in a window of 15 seconds (default).

Physiological range at rest: from 0.5 to 1.5 units.

Dynamic elastance can change according to different clinical settings (e.g. hypotensive patients, vasodilation) and may be useful for the prediction of blood pressure response to fluid challenge or vasoactive drugs administration.

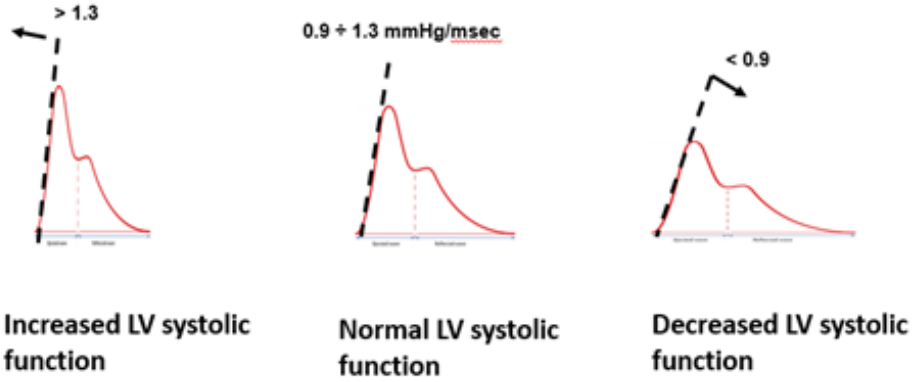
1. Pinsky MR (2002) Functional hemodynamic monitoring: applied physiology at the bedside. Springer, Berlin, pp 537–552 book.
2. Pinsky MR. Probing the limits of arterial pulse contour analysis to predict preload responsiveness. Anesth Analg. 2003 May;96(5):1245-7.
3. Monge García MI, Gil Cano A, Gracia Romero M. Dynamic arterial elastance to predict arterial pressure response to volume loading in preload-dependent patients. Crit Care. 2011;15(1):R15.
4. García MI, Romero MG, Cano AG, Aya HD, Rhodes A, Grounds RM, Cecconi M. Dynamic arterial elastance as a predictor of arterial pressure response to fluid administration: a validation study. Crit Care. 2014 Nov 19;18(6):626.
5. Guinot PG, Bernard E, Levrard M, Dupont H, Lorne E. Dynamic arterial elastance predicts mean arterial pressure decrease associated with decreasing norepinephrine dosage in septic shock. Crit Care. 2015 Jan 19;19:14.

# Left ventricular systolic function

## $dP/dt_{max}$ - Maximum slope of the systolic upstroke


$dP/dt_{max}$ is the maximum slope of arterial blood pressure during systolic upstroke.
Peripheral $dP/dt_{max}$ has been suggested as a feasible surrogate for the left ventricle $dP/dt_{max}$, which is classically considered as a marker of left ventricle inotropic state (contractility).
The steeper the slope, the better the left ventricular systolic function. The high sampling frequency of 1000 samples per second allows PRAM method to measure $dP/dt_{max}$ with high precision and at each cardiac cycle.

Physiological range at rest: 0.90 to 1.30 mmHg/msec




Because MostCareUp measures $dP/dt_{max}$ at peripheral sites, changes in arterial elastance and systemic vascular resistance may influence $dP/dt_{max}$ value.

$dP/dt_{max}$ can change according to different clinical settings (e.g. congestive heart failure, haemorrhagic shock, deep sedation and vasodilation) and may be useful for assessing the response of left ventricle to inotropes and vasoactive medications.

$dP/dt_{max}$ values higher than 1.7 mmHg/msec is unphysiological and might suggest that the arterial waveform is underdamped.


1. De Hert SG, Robert D, Cromheecke S, Michard F, Nijs J, Rodrigus IE. Evaluation of left ventricular function in anesthetized patients using femoral artery dP/dt(max). J Cardiothorac Vasc Anesth. 2006 Jun;20(3):325-30.
2. Tartiere JM, Logeart D, Beauvais F, Chavelas C, Kesri L, Tabet JY, et al. Noninvasive radial pulse wave assessment for the evaluation of left ventricular systolic performance in heart failure. Eur J Heart Fail. 2007;9(5):477–83
3. Tartière JM, Tabet JY, Logeart D, Tartière-Kesri L, Beauvais F, Chavelas C, Cohen Solal A. Noninvasively determined radial dP/dt is a predictor of mortality in patients with heart failure. Am Heart J. 2008 Apr;155(4):758-63.
4. Chew MS, Åneman A. Haemodynamic monitoring using arterial waveform analysis. Curr Opin Crit Care. 2013 Jun;19(3):234-41.
5. Scolletta S et al. Assessment of left ventricular function by pulse wave analysis in critically ill patients. Intensive Care Med 2013;39: 1025-33.
6. Monge Garcia MI, Jian Z, Settels JJ, Hunley C, Cecconi M, Hatib F, Pinsky MR. Performance comparison of ventricular and arterial $dP/dt_{max}$ for assessing left ventricular systolic function during different experimental loading and contractile conditions. Crit Care. 2018 Nov 29;22(1):325.

# V-A coupling

## Dic – Dicrotic pressure


Dicrotic pressure is the pressure at the end of systolic phase which correspond to the closure of aortic valve.
MostCareUp, based on a sophisticated algorithm working at 1000 samples per seconds, can detect beat by beat the value of dicrotic pressure, marking the position of dicrotic notch by a vertical green line.

Dicrotic pressure provides information on the relationship between systolic function and the characteristics of the arterial tree (V-A coupling) and its value is accepted to be physiologically close to the MAP value.

Dicrotic pressure is often estimated as the central systolic blood pressure multiplied by 0.9, while MostCareUp measures Dic real-time at each cardiac cycle.

Physiological range: 70 – 105 mmHg.

Dic may change according to different clinical settings (e.g. tachycardia, vasoconstriction, sepsis) and may be useful for evaluating the response of the cardiovascular system to cardio-vasoactive drugs.

1. Lewis T. The factors influencing the prominence of the dicrotic wave. J Physiol 1906; 34:414–429.
2. Smith D, Craige E. Mechanism of the dicrotic pulse. Br Heart J 1986; 56:531–534.
3. Nichols WW, O'Rourke MF 1990: McDonald's blood flow in arteries, third edition. Sevenoaks: Edward Arnold.
4. Chantler PD, Lakatta EG, Najjar SS. Arterial-ventricular coupling: mechanistic insights into cardiovascular performance at rest and during exercise. J Appl Physiol (1985). 2008 Oct;105(4):1342-51.
5. Pavoni V, Romagnoli S, Batignani G, Gianesello L, Horton A, Romano SM. Unsuspected Heart Failure: Usefulness of a Minimally Invasive Hemodynamic Monitoring System. J Anesth Clin Res 2012; 3(8).
6. Scolletta S et al. Assessment of left ventricular function by pulse wave analysis in critically ill patients. Intensive Care Med 2013;39: 1025-33.
7. Guarracino F, Ferro B, Morelli A, Bertini P, Baldassarri R, Pinsky MR. Ventriculoarterial decoupling in human septic shock. Crit Care. 2014 Apr 24;18(2):R80.
8. Messina A, Romano SM, Bonicolini E, Colombo D, Cammarota G, Chiostri M, Della Corte F, Navalesi P, Payen D, Romagnoli S. Cardiac cycle efficiency and dicrotic pressure variations: new parameters for fluid therapy: A pilot observational study. Eur J Anaesthesiol 2017; 34:1–9.
9. Ristalli F, Romano SM, Stolcova M, Meucci F, Squillantini G, Valente S, Di Mario C. Hemodynamic monitoring by pulse contour analysis during trans-catheter aortic valve replacement: A fast and easy method to optimize procedure results. Cardiovasc Revasc Med 2018 Jul 19.

## CCE - Cardiac Cycle Efficiency


Cardiac Cycle Efficiency describes haemodynamic performance in term of energy expenditure. Indeed, CCE depends on the energy required to generate a given SV, which depends on the interaction between pump function and arterial system (i.e. A-V coupling).

See more details in the Efficiency paragraph.

# Efficiency

## CPO – Cardiac Power Output

Cardiac power output represents cardiac pumping ability to generate blood flow. It expresses the cardiac power reserves that can be recruited to increase the heart's pumping ability. The higher the CPO, the higher the recruitable reserves to increase pumping ability.
CPO is directly correlated to CO and MAP: CPO = MAP · CO/ k with k=451 as a conversion factor.
Physiological range at rest: from 0.80 to 1.20 Watt.
CPO has been demonstrated to be a good predictor for mortality in heart failure patients.
Available also:
CPI = MAP · CI/ k cardiac power index from 0.50 to 0.70 W/m2


1. Cotter G, Williams SG, Vered Z, Tan LB. Role of cardiac power in heart failure. Curr Opin Cardiol. 2003 May;18(3):215-22
2. Fincke R, Hochman JS, Lowe AM et al (2004) Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: a report from the SHOCK trial registry. J Am Coll Cardiol 44:340–348.
3. Mendoza DD, Cooper HA, Panza JA. Cardiac power output predicts mortality across a broad spectrum of patients with acute cardiac disease. Am Heart J. 2007 Mar;153(3):366-70.
4. Hothi SS, Tan LB, Cotter G. Resting cardiac power index and prediction of prognosis in heart failure. Eur J Heart Fail. 2015 Jul;17(7):642-4.
5. Nathania M, Hollingsworth KG, Bates M, Eggett C, Trenell MI, Velicki L, Seferovic PM, MacGowan GA, Turnbull DM, Jakovljevic DG. Impact of age on the association between cardiac high-energy phosphate metabolism and cardiac power in women. Heart. 2018 Jan;104(2):111-118.


## CCE – Cardia Cycle Efficiency


Cardiac Cycle Efficiency describes haemodynamic performance in term of energy expenditure. Indeed, CCE depends on the energy required to generate a given SV, which depends on the interaction between pump function and arterial system (i.e. A-V coupling).
Many factors may influence CCE, for example changes in left ventricular function, heart rate, preload, afterload, including arterial elastance and reflected pressure waves.

The value of CCE can range from +1 (ideal condition with no energy expenditure) to negative values: the greater the energy expenditure to generate a given SV, the lower the CCE value.

CCE is computed beat by beat as the ratio of the sum of systolic powers [W(t)sys] to the sum of all powers of entire cycle [W(t)beat].

Physiological range: -0.2 – 0.3 units.

CCE can change according to different clinical settings (e.g. bradycardia, tachycardia, poor myocardial contractility, increased or reduced venous return, changes of systemic vascular resistance) and may be useful for assessing the haemodynamic response to cardioactive and vasoactive drugs. Additionally, monitoring the trend of CCE can be of value in preventing unexpected haemodynamic impairments and may help in clinical decision-making.



1. Giglioli C, Landi D, Cecchi E, Chiostri M, Gensini GF, Valente S, Ciaccheri M, Castelli G, Romano SM. Effects of ULTRAfiltration vs DIureticS on clinical neurohormonal and hemodynamic variables in patients with deCOmpensated heart failure: the ULTRADISCO study. Eur J Heart Fail 2011;13(3): 337-46.
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8. Romano SM. Cardiac cycle efficiency: a new parameter able to fully evaluate the dynamic interplay of the cardiovascular system. Int J Cardiol 2012; 155:326-7
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10. Scolletta S et al. Assessment of left ventricular function by pulse wave analysis in critically ill patients. Intensive Care Med 2013;39: 1025-33.
11. Morelli A, Singer M, Ranieri VM, D'Egidio A, Mascia L, Orecchioni A, Piscioneri F, Guarracino F, Greco E, Peruzzi M, Biondi-Zoccai G, Frati G, Romano SM. Heart rate reduction with esmolol is associated with improved arterial elastance in patients with septic shock: a prospective observational study. Intensive Care Med 2016.
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13. Ristalli F, Romano SM, Stolcova M, Meucci F, Squillantini G, Valente S, Di Mario C. Hemodynamic monitoring by pulse contour analysis during trans-catheter aortic valve replacement: A fast and easy method to optimize procedure results. Cardiovasc Revasc Med 2018 Jul 19.

CRITICAL CARE

**For further information, please contact:** questions@vygon.com

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