Data Interpretation in Anesthesia

A Clinical Guide

Tilak D. Raj
Editor

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I would like to dedicate this book to my late parents, Arthur and Prema and also to: Catherine, Vijay, Anushka, Kieran, and Roshan

Foreword

In their daily practice, anesthesiologists are faced with a tremendous amount of data during the clinical management of their patients. This textbook, *Data Interpretation in Anesthesia: A Clinical Guide*, focuses on the interpretation of data commonly available to an anesthesiologist.

The book is divided into five parts, including monitoring, laboratory testing, imaging, physiologic studies, and conceptual images. It consists of 83 chapters starting with a presentation of a data point, followed by relevant questions and answers with discussion. Pertinent references are provided in each chapter.

Another textbook in this format is not currently available for the discipline of anesthesiology. There are a variety of reviews that are vignette driven, or are discussions of general topics, but none that focus concisely on the individual data points that an anesthesiologist must quickly and astutely interpret for patient care. This format allows the consultant to efficiently reference areas of review.

The editor of this much needed book, Tilak D. Raj, MD, is a cardiothoracic and vascular anesthesia fellowship-trained, board-certified anesthesiologist (both in the UK and the USA), who has been involved in clinical practice and academic medicine for 20 years.

Contributors include an excellent selection of anesthesiologists, cardiologists, and an interventional neurologist.

Anesthesiology as a specialty is seeing amazing advancements in patient care. More and more advanced clinical algorithms emerge every day to help anesthesiologists understand data points, interpret results, and make decisions. This book will be very useful for all anesthesiologists, anesthesia residents, and practitioners involved in Maintenance of Certification in Anesthesiology (MOCA). It is not a book that just sits on a shelf collecting dust. It is a must read. I hope you enjoy it!

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Preface

It is a pleasure to finally bring to fruition an idea I have had for a couple of years. As anesthesiologists, we come across a vast amount of clinical and investigative data during the perioperative care of our patients. This book should serve as a reference, providing information about the data we encounter in our daily practice. The current edition has 83 chapters and the list covers most of the data that we encounter. There is a basic layout for the chapters which start with a data point followed by discussion in a question and answer format. I chose this format to stimulate analytic thought and facilitate learning.

The chapters in the book are grouped into five parts. The "Conceptual Images" part has topics which are not strictly data but more topics of exam interest. They share the same format and provide additional knowledge in those areas.

This text should help residents and anesthesiologists striving to become board-certified anesthesiologists in practice working toward Maintenance of Certification in Anesthesiology (MOCA).

The project could not have been completed without the expert and valuable contribution by authors from different specialties both from America and England, to whom I am extremely grateful. Editing and contributing to this book has provided a great learning experience for me, not just in medicine and anesthesiology but also in life and human nature.

Physicians should be passionate lifelong learners to provide good patient care, and physicians in academic settings should do the same not just for patient care but also to teach and act as good role models for students and residents. I shall close with the apt and inspiring quote by John Cotton Dana.

"Who dares to teach must never cease to learn."

Edmond, OK — Tilak D. Raj

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I would like to thank my colleagues in helping me with compiling the list of chapters and parts; Dwight Reynolds, MD, for the wonderful X-rays we used in Chap. 46 (CXR—CIED); Scott Tatum, R.N., B.S.I.T., for providing some ECGs used in the "ECG Quiz" chapter; and the "expert" Dan Mason from Haemonetics for his invaluable help in two chapters on TEG.

I am also greatly indebted to Carin Hagberg, MD, for kindly agreeing to provide a foreword for this book; my precious artist Gail Gwin, who provided the drawings for many chapters which she tirelessly worked on, outside of her busy work schedule; to Vijay Raj for his help with some images and graphs; to all the residents who provided valuable feedback; and last but not the least my wife Catherine who kept me focused and on track and to my children Vijay, Anushka, Kieran, and Roshan—"it can be done and you can do it!"

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Part I
Monitoring

Chapter 1
CVP

Teodora Nicolescu

The below pressure waveform is obtained from an IV in the neck of a patient being monitored.

1. Identify the components labeled 1–5. Explain what they signify.
2. What information can be deduced from the central venous pressure measurements?
3. What determines the central venous pressure?
4. What factors influence the reading of central venous pressure?
5. What are the indications and contraindications of central venous catheter insertion?
6. Give some examples of CVP waveforms in pathological states.

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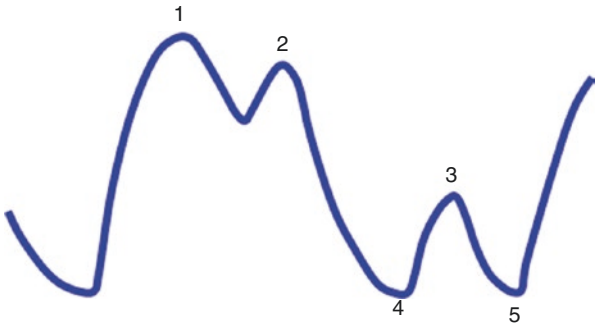

Fig. 1.1 Central venous pressure waveform

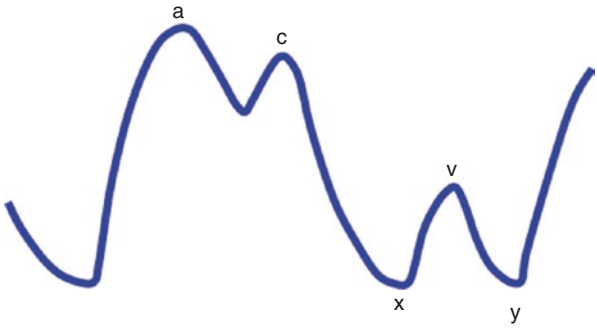

Fig. 1.2 Normal CVP

Answers

1. 1, a wave; 2, c wave; 3, v wave; 4, x descent; and 5, y descent.

   The **a wave** of the central venous pressure represents the **atrial contraction**. The right atrial pressure is at the highest value. It is mirrored by the PR interval on the ECG tracing. Notably, the a waves are absent in atrial fibrillation and are exaggerated in junctional rhythms and heart blocks (cannon waves). It is also enlarged in tricuspid and pulmonary stenosis as well as pulmonary hypertension.

   The **c wave** is due to the bulging of the tricuspid valve into the right atrium during early ventricular contraction (**ventricular systole**), while the **v wave** is due to the **rise in the atrial pressure** that occurs before the opening of the tricuspid valve. The v waves are prominent in tricuspid regurgitation.

   There are also two descents noted in the central venous pressure waveform.

   The **x descent** is due to the **atrial relaxation** or possibly by the tricuspid annular downward displacement during systole [1].

   The **y descent** represents the tricuspid valve displacement during diastole, as **atria start emptying** [2].

2. Central venous pressure measures right atrial pressure, which is a major determinant of right ventricular end-diastolic volume. It is used to assess (right) ventricular volume, filling, and therefore fluid status. It does however have limitations, mostly related to **ventricular compliance** which can be affected by a variety of

factors (**e.g., impaired relaxation**, **ischemia,** and **pharmacologic manipulation**). Of note, even in healthy patients, there is a wide variability in cardiac compliance. Although a very low CVP measurement may indicate volume depletion, a high value may be due to volume overload or poor ventricular compliance. Isolated central venous pressure measurements are not useful; instead, the trend of measurements over a given time and the response to a fluid challenge may provide useful information on the intravascular fluid status of a patient.

It is also important to keep in mind that filling pressure estimation is unreliable in predicting fluid responsiveness, particularly in septic patients. CVP measurement should be considered in the context of other parameters of a patient's volume status like heart rate, blood pressure, and urine output. In healthy hearts, right and left ventricular performances are parallel, therefore left ventricular filling can be approximated by the central venous pressure.

3. Determinants of the central venous pressure are as follows:
   (a) **Right ventricular function**
   (b) **Venous return** that in turn is determined by total blood volume, venous tone, cardiac output, right ventricular contractility, and intrathoracic pressure [3].

   It has to be understood that the central venous pressure can be overestimated, mainly due to fluctuations with respiration of the mean central venous pressure. Proper placement of the catheter just outside of the right atrium may insure more accurate readings. The pressure at the base of the c wave represents the right atrial pressure at the start of the right ventricular systole, making it the best estimate of right ventricular preload. Central venous measurements should be taken at end exhalation (lowest negative intrathoracic pressure) [5].

4. Several factors will influence the accuracy of the central venous pressure reading:
   (a) Changes in intrathoracic pressure (PEEP, ascites).
   (b) Cardiac rhythms disturbances.
   (c) Tricuspid valve disease.
   (d) Myocardial compliance changes (pericardial disease, tamponade). In tamponade there is equalization of diastolic pressures (in the absence of left ventricular dysfunction).

$$\text{RAP} = \text{RVEDP} = \text{LAP} = \text{LVEDP}$$

   Of note, the limited ventricular filling abolishes the y descent. In return the x descent (atrial relaxation) is accentuated or normal [4].

5. **Indications:**
   (a) Fluid management (particularly hypovolemia and shock)
   (b) Infusion of vasoactive drugs
   (c) Hyperalimentation
   (d) Insertion of pacemaker wires
   (e) In surgeries with air embolism potential
   (f) Difficult IV access

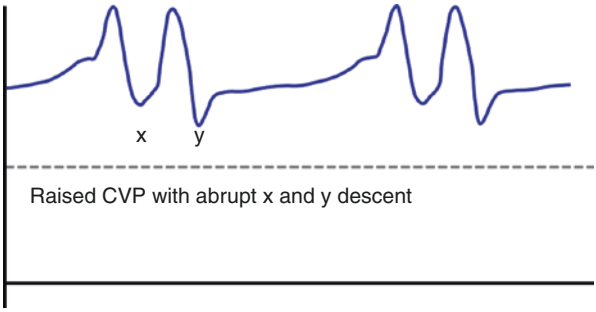

Fig. 1.3 Abnormal CVP—steep x and y descent (constrictive pericarditis)

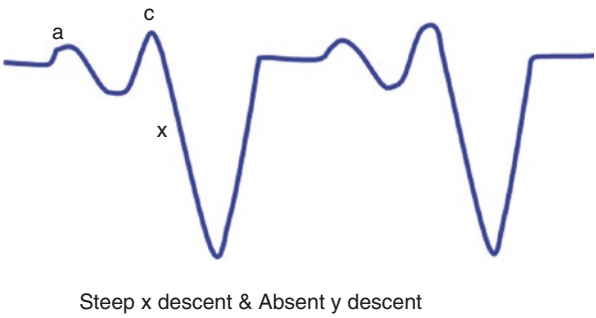

Fig. 1.4 Abnormal CVP—steep x descent (pericardial tamponade)

Contraindications:

(a) Right atrial tumor extension (renal cell carcinoma)
(b) Endocarditis (fungating valve vegetations)
(c) Relative presence of ipsilateral carotid endarterectomy

6. Waveform analysis

Large a waves	Pulmonary hypertension, tricuspid, and pulmonic stenosis
Cannon a waves	Irregular—complete heart block
	Regular—AV dissociation
Large v waves	Tricuspid regurgitation
Exaggerated x descent	Pericardial tamponade, constrictive pericarditis
Sharp y descent	Severe tricuspid regurgitation, constrictive pericarditis

References

1. Johnson B. Decrease in central venous pressure by lower body negative pressor or blood loss elicits similar responses. J Appl Physiol (1985). 2014;117(2):131–41.
2. Butterworth JF. Atlas of procedures in anesthesia and critical care D. Berlin-Starling curves and central venous pressure. Crit Care. 2015;19(1):55.
3. Avcil M. Comparison of estimating central venous pressure. Int J Clin Exp Med. 2015;8(7): 10586–94.
4. Carmona P, Mateo E, et al. Management of cardiac tamponade after cardiac surgery. J Cardiothorac Vasc Anesth. 2012;26(2):302–11.
5. Pittman JAL, Ping JS, et al. Arterial and central venous pressure monitoring. Int Anesthesiol Clin. 2004;42(1):13–30.

Chapter 2
Pulmonary Artery Catheters

Teodora Nicolescu

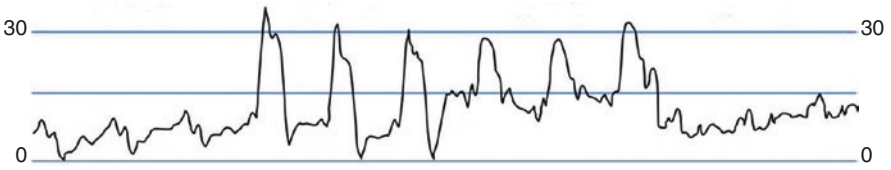

Fig. 2.1 Pulmonary artery catheter normal waveform

Questions

1. The above sequence of waveforms was encountered during a line placement in a patient. Describe what you see.
2. What information does the PA catheter provide?
3. How does ventilation management affect the accuracy of data from a PA catheter?
4. When is the pulmonary artery occlusion pressure (PAOP), also referred to as pulmonary capillary wedge pressure (PCWP), different from the left ventricular end-diastolic pressure (LVEDP)?
5. What do large v waves on the PA catheter tracing mean?
6. How can you accurately interpret mixed venous oxygen saturation?
7. What are the indications, complications, and evidence for PAC use?

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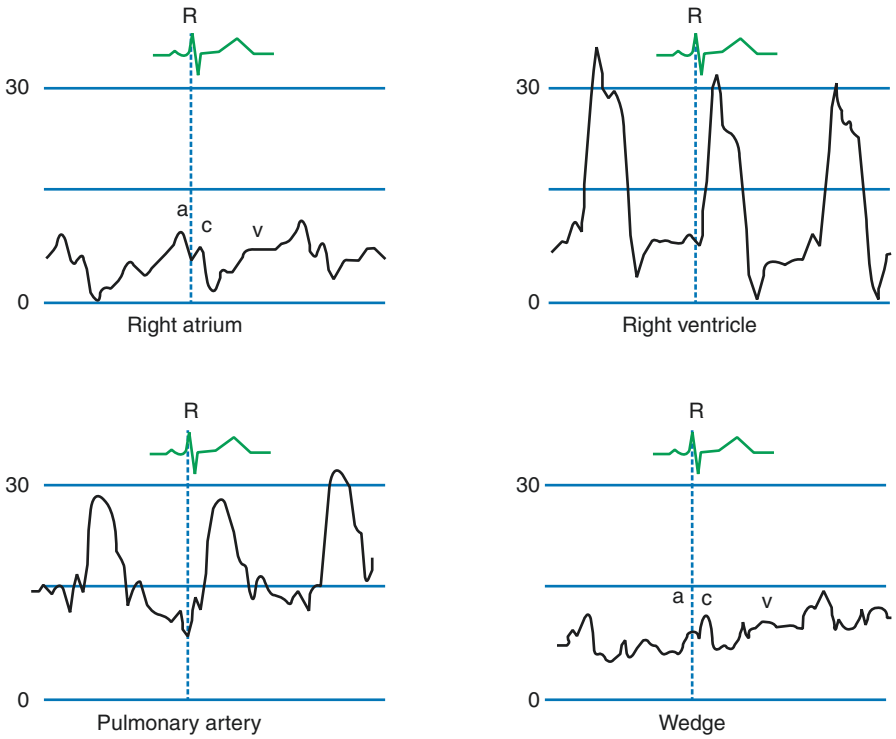

Fig. 2.2 Waveforms encountered during PAC advancement

Answers

1. When inserted, the PA catheter is first advanced through the sheath and at approximately 15–20 cm, the balloon is inflated. Along this path the catheter will pass through the (1) **right atrium**, (2) **the right ventricle**, and (3) **the pulmonary artery**, at which point, with slight advancement into a small arterial branch, it can obtain (4) the **pulmonary artery occlusion pressure**.

   The **right atrial pressures** (values 0–5 mmHg) will be similar to a **central venous tracing** that varies with respiration.

   A **sudden systolic pressure increase** (values 15–30 mmHg) confirms entrance into the **right ventricle**.

   Advancement into the **pulmonary artery** will result in a sudden **increase in diastolic pressures** (values 8–15 mmHg) confirming entrance into the pulmonary artery.

   The **pulmonary capillary wedge pressure** (values 8–12 mmHg) will rapidly fall once the balloon is inflated and reveal a left atrial pressure waveform with a, c, and v waves, just like a central venous tracing except the waves appear later.

2. The PA catheter provides a more precise left ventricular diastolic pressure estimation.

The right ventricular pressures do not correlate with pulmonary artery pressures distal to the occlusion point. However, this is not true for the relationship PAOP, LAP, and LVEDP which correlate.

Theoretically at least, at end diastole no pressure gradient should occur, making end diastole the best time for pressures correlation.

The values obtained from the PA catheter are as follows [2]:

(a) Cardiac output (CO)—the only value measured (all the rest are calculated values).

The cardiac output measurements are obtained by the **thermodilution method,** the basic principle being that the difference in temperature between the cold injectate and body temperature is inversely proportional to the pulmonary blood flow (cardiac output).

Accuracy of measurements is directly dependent on the speed of injection and precise quantification of injectate volume and temperature.

Once the average value of three measurements is obtained, calculations can provide the rest of the data derived from the PA catheter.

(b) Cardiac index(CO/BSA) where CO represents cardiac output and BSA is body surface area

(c) Systemic and pulmonary vascular resistance:

$$SVR\left(\text{systemic vascular resistance}\right) = \frac{\left(MAP - CVP\right)\times 80}{CO}$$

Normal: 900–1600 dynes.sec.cm^{-5}

where MAP represents mean arterial pressure, CVP central venous pressure, and CO cardiac output.

$$PVR\left(\text{pulmonary vascular resistance}\right) = \frac{\left(MPA - LAP\left\{\text{PAOP}\right\}\right)\times 80}{\text{Pulmonary flow}\left(CO\right)}$$

where MPA represents mean pulmonary artery pressures, LAP left atrial pressure (PAOP—pulmonary artery occlusion pressure), and CO cardiac output.

Normal: 20–130 dynes.sec.cm^{-5}

(d) Stroke volume and index:

$$\text{Stroke volume} = \frac{CO\times 1000}{HR}$$

$$\text{Stroke index} = \frac{\text{Stroke volume}}{BSA}$$

3. PA catheter data may be unreliable due **to intrathoracic pressure variations**. Balloon inflation will not occlude capillaries unless it is placed in West lung zone III (arterial pressure exceeds venous, which exceeds alveolar pressure), where

the capillaries can remain open. Placement in zone I or II can obstruct blood flow rendering the readings inaccurate, reflecting alveolar rather the pulmonary occlusion pressures.

Thus, it is important to remember that intravascular volume depletion or PEEP, for example, may convert a lung zone III to a zone II (alveolar pressure exceeds arterial pressure), thereby affecting the readings. This may also occur during any ventilation management in which there is insufficient expiratory time (air trapping or inverse ratio ventilation).

Pressures are evaluated at end expiration to minimize the effect of pleural pressures on intracardiac pressures.

4. There are conditions when PAOP **overestimates** or **underestimates** the LVEDP.

Overestimation:

(a) Tachycardia (shortened diastolic filling time). At rates greater than 115/min, the pulmonary artery end-diastolic pressure (PAEDP) is greater than the PAOP.
(b) Increase in pulmonary vascular resistance (sepsis, pulmonary disease, obstruction to venous drainage).
(c) Mitral stenosis, atrial myxoma.
(d) Increased intrathoracic pressures (mediastinal tumors).
(e) Conditions associated with large PA v waves (large v waves may obscure catheter wedging with pulmonary artery rupture being a real danger). The normal PA waveform has an arterial waveform with an upward slope, a downward slope, and a dicrotic notch associated with the pulmonic valve closure. While the peak systolic wave on the PA tracing corresponds to the electrographic T wave, by contrast, the large v waves occur after the electrocardiographic T wave. Large v waves on the PAC are seen in mitral regurgitation, VSD, and CHF.

Underestimation:

(a) Aortic regurgitation.
(b) Non-compliant left ventricle—transmyocardial filling pressure and LVEDP have a curvilinear relationship, therefore changes in left ventricular end-diastolic volume (LVEDV) will result in changes in the LVEDP based on the location on the curve. Of note, ventricular compliance is affected by vasoactive drugs, and beta-blockers.
(c) Pulmonary embolism.
(d) Right bundle branch block (delay in right ventricular systole).
(e) Pulmonary edema.

5. Large v waves are seen in (1) myocardial ischemia, (2) mitral regurgitation, (3) decreased atrial compliance, (4) or increased SVR. The diastolic PAOP offers the best approximation for the LVEDP when large v waves are present.