Central Venous Pressure

Its Clinical Use and Role in Cardiovascular Dynamics

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M.B., B.S., F.F.A.R.C.S.
Wellcome Research Fellow, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London

Butterworths

ENGLAND: BUTTERWORTH & CO. (PUBLISHERS) LTD.
LONDON: 88 Kingsway, WC2B 6AB

AUSTRALIA: BUTTERWORTHS PTY. LTD.
SYDNEY: 586 Pacific Highway, 2067
MELBOURNE: 343 Little Collins Street, 3000
BRISBANE: 240 Queen Street, 4000

CANADA: BUTTERWORTH & CO. (CANADA) LTD.
TORONTO: 14 Curity Avenue, 374

NEW ZEALAND: BUTTERWORTHS OF NEW ZEALAND LTD.
WELLINGTON: 26–28 Waring Taylor Street, 1

SOUTH AFRICA: BUTTERWORTH & CO. (SOUTH AFRICA) (PTY.) LTD.
DURBAN: 152–154 Gale Street

Suggested U.D.C. Number: 612.144

Suggested Additional Numbers: 616.12–008.341

ISBN 0 407 13270 8

Text set in 11 pt. Photon Times, printed by photolithography, and bound in Great Britain at The Pitman Press, Bath

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Preface

This monograph is not a report of original experimental work but an explanation of central venous pressure for clinicians. It has four objectives: to explain the part played by the central venous pressure in cardiovascular dynamics; to discuss the clinical need to measure central venous pressure; to describe the apparatus and its use; and to discuss the interpretation of the measurements. This, I hope, will provide a guide to the management of patients with cardiovascular instability.

I wish to thank Professor J. G. Robson, Professor M. K. Sykes and my colleagues at the Royal Postgraduate Medical School and Hammersmith Hospital for their encouragement and suggestions during the writing of this monograph. I am also very grateful to my wife for much of the typing and preparation of the manuscript.

The kind permission of Professor A. C. Guyton, the American Journal of Physiology, Professor M. K. Sykes, the Annals of the Royal College of Surgeons of England, Professor G. S. Moss, the Annals of Surgery, Dr. T. Boulton and St. Bartholomew's Hospital Journal is acknowledged for the use of their illustrations.

W.J.R.

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Introduction

The first man to measure central venous pressure was Stephen Hales, in the 1st decade of the 18th century, although the exact date of his first experiment is uncertain. This measurement may have been made, while they were both at Cambridge, in co-operation with his friend William Stuckley, who was studying medicine there. In this first experiment they probably used a dog. Hales' better known observations on the venous pressure of mares were made later when he was vicar at Teddington (Clark-Kennedy, 1929). His years at Cambridge had given him a clear understanding of hydrostatics and so he was careful to refer his pressure observations to the level of the left ventricle. This set an excellent example for those who were to follow but unfortunately, even today, venous pressures are sometimes quoted without the reference level being stated. Hales not only measured the pressure at the internal jugular vein during his experiments, but he also observed that the pressure rose when the mare struggled.

These observations remained isolated for about 170 years. Then, in the later part of the 19th century, it was noted that venous pressure altered with changes in blood volume (Cohnheim and Lichtheim, 1877) and that it influenced the work of the heart (Howell and Donaldson, 1884).

During the past 50 years our understanding of the physiology of the heart and of the venous return has steadily

improved. With this better insight we have been more able to appreciate the significance of the central venous pressure and to see how it results from the interaction of the venous return and the cardiac function. However, central venous pressure is but one element in the juggling act of cardiovascular dynamics and its significance can be appreciated only when those dynamics are understood.

A convenient approach is to develop a model of the cardiovascular system. This model should not be too simple for it must adequately simulate the system, yet it must not be too complex or the behaviour of the model will not be understood and the vital insight into how the system works will be lost. When the dynamics are appreciated, variations in central venous pressure can be explained logically and the management of low output states can be approached rationally.

The cardiovascular system is a closed loop and a change in any part must have repercussions throughout the system. Normally, changes are perceived by specific receptors and counteracted through the autonomic nervous system. The chain of repercussions can be demonstrated by following the effect of infusing additional blood into the systemic veins. When blood is infused intravenously, the systemic volume is increased and the resistance of the venous side of systemic circulation diminishes. There is also a small rise in local venous pressure. Both these effects enhance the flow of blood back to the heart and this improved flow increases the pressure in the right atrium, the output of the right ventricle and pulmonary artery pressure. The increased pressure in the pulmonary artery increases flow through the pulmonary circulation which in turn increases the pulmonary venous pressure and the pressure in the left atrium. This atrial pressure change enhances the flow of blood into the left ventricle and thus increases the systemic arterial pressure. The systemic arterial pressure affects the capillary flow and the systemic venous flow. Thus, in time, a disturbance is felt all round the cardiovascular loop.

Although the vascular system is closed it is not rigid. It is sensitive to changes in pressure mainly because the ventricles,

which pump the blood through the circulation, are sensitive to their filling pressure. Any pressure changes—particularly a change on the venous side of the heart—alter the performance of the ventricles. Thus the heart is a pressure-sensitive pump driving blood around the body.

For many purposes an adequate model of the system can be made if we assume that the right ventricular output effectively governs the left ventricular output and the pulmonary circulation can be ignored. This 'single pump' simplification means that only a two-part model is required; a heart and a systemic circulation. Much valuable insight into the function of the cardiovascular system can be gained from this simpler model.

The heart

Many studies have been made of mammalian cardiac function, both with isolated hearts and in intact animals. Each approach has its own special difficulties but a common result can be expressed briefly: increased atrial pressure produces increased ventricular output. This is sometimes called Starling's law of the heart (Starling, 1918). It has been studied mainly in animals but has been shown to occur also in man (Braunwald and Ross, 1964).

We can understand this effect if we assume each ventricle has two properties: (1) that it will pump onwards whatever volume fills it—that is, for a given rate and resistance the ventricular volume at the end of systole is always the same regardless of the volume at the end of diastole; (2) that in diastole the ventricle is a compliant chamber, the filling of which is governed by the pressure gradient from just within the atrioventricular valve to just outside the ventricular wall in the pericardial sac (Berglund, 1954). This filling pressure is illustrated in *Figure 1*. Normally, the pressure just outside the ventricle is the intrathoracic pressure. In normal circumstances, therefore, the filling pressure for the ventricle is closely approximated by the pressure difference between the atrium and the pleural space. The importance of the pressure

just inside the atrioventricular valve is shown by the observation (Guyton and Greganti, 1956) that the pressure just inside the tricuspid valve was the best reference for ventricular filling and remained almost unchanged with changes in posture.

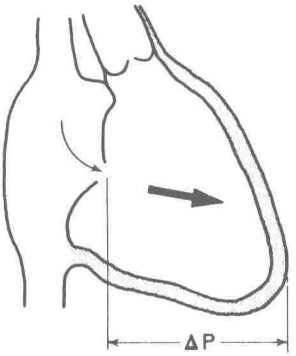

Figure 1. Diagram of the pressure gradient for ventricular filling. As the ventricle is a compliant chamber, it will fill until there is no pressure gradient between its interior and the atrium. The pressure across the ventricular wall is then balanced by the tension within the wall. In the dynamic situation some of the pressure between the atrioventricular valve and the pericardial space is taken up with the flow of blood into the distending ventricle. However, the statement that ventricular filling depends on the pressure gradient still remains true, although the relationship may not be a simple one

Any increase in pressure just outside the ventricle diminishes the pressure gradient. For example, fluid in the pericardium increases the pressure outside the ventricles and hinders ventricular filling (Spodick, 1967). If the pressure immediately outside the ventricle in the pericardial sac is constant, any increase in atrial pressure increases the pressure

gradient and hence increases the ventricular filling. Thus increased atrial pressure increases the end-diastolic volume, the stroke volume and the cardiac output.

The ability of the ventricle to increase its output as the atrial pressure is increased can be demonstrated by a cardiac performance curve. The curve shows the response of the ventricle over a range of atrial pressures. The upper limit of the performance curve is only achieved by a high atrial pressure. A very low atrial pressure may produce almost no output. Thus the performance curve relates the ventricular filling pressure to the ventricular output, and in fact separate performance curves should apply to the right and left sides of the heart. Each curve (or pair) describes the heart under set conditions which are determined by the sympathetic and parasympathetic activity impinging on the heart and by the intrinsic quality of the ventricular muscle. The level of autonomic activity influences both heart rate and myocardial contractility and thus plays a major role in determining the ability of the heart to respond to the atrial pressure. Maximal sympathetic influence gives a high performance curve, while minimal sympathetic influence gives a low performance curve. A family of curves describe the possible performance of the heart under the widest range of conditions (*Figure 2*). Usually attention is focused on the highest performance curve as this is the one most deteriorated by disease. However, from the potential performance, the actual cardiac output is determined by the atrial pressure which fills the ventricle, and could be any amount between nothing and the upper limit of the performance curve.

The output of the heart depends upon the right atrial pressure and on the autonomic activity which is the main determinant of the cardiac performance curve. An increase in cardiac output could be achieved by an increase in right atrial pressure or an improvement in ventricular performance. Normally changes in cardiac output are achieved by adjustment of the autonomic nervous activity which changes the ventricular performance. These multiple levels of ventricular performance have been described as the Frank-Starling mechanism (Sarnoff, 1955; Fry, Braunwald and Cohen, 1960). This is

probably the natural mechanism for regulating cardiac output in health, while the atrial pressure/ventricular output mechanism maintains the precise balance between the ventricles. In *Figure 2* this effective ventricular filling pressure is expressed as right atrial pressure, assuming a constant mean pressure in the pericardial sac.

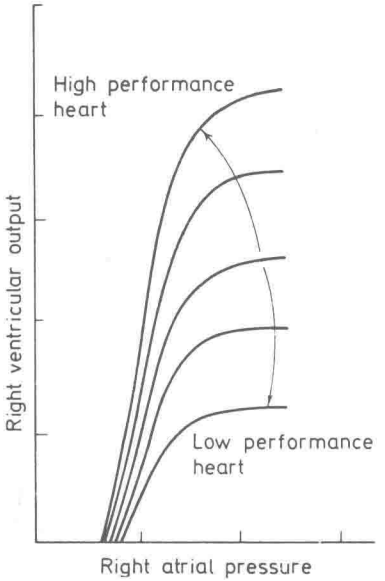

Figure 2. Diagram of Frank-Starling curves. The output increases as the right atrial pressure increases until a maximum is reached when further rises in right atrial pressure do not improve output (and may possibly diminish it). Increased sympathetic activity increases the sensitivity of the heart to right atrial pressure (high performance heart): there is a greater increase in output for the same right atrial pressure, and the maximum output is greater. Conversely, parasympathetic influences or myocardial damage reduce the sensitivity to atrial pressure and also cause a reduction in the maximum output

When the heart is beating slowly, the ventricle can fill to the atrial pressure well before atrial systole occurs; the volume in the right ventricle at the end of diastole is effectively governed by the right atrial pressure, and hence this pressure controls the stroke volume. As cardiac output is the product of the stroke volume and the heart rate, it would be determined by the heart rate alone if a given right atrial pressure produced a consistent stroke volume. A family of Frank–Starling curves would then be merely an expression of a succession of heart rates. A slow heart rate means the cardiac output would increase only modestly with an increase in atrial pressure and this could be expressed as a low performance curve (*Figure 2*). For example, at a rate of 60 beats/min a change in atrial pressure which produced a 10 ml increase in stroke volume would improve the cardiac output by 600 ml/min; at a rate of 120 beats/min, the same increase in stroke volume would improve the output by 1,200 ml/min.

In life, the situation is more complex but probably an increased cardiac output is achieved mainly by the change in rate, augmented in some circumstances by improved ventricular emptying (Rushmer, 1959). Certainly in man, increases in heart rate alone can enhance the velocity of ventricular contraction (Glick *et al.*, 1965). The variation between Frank–Starling curves represents a change in performance that is probably the result of a change in heart rate augmented to a slight extent by better emptying of the ventricles.

At faster heart rates, the ventricles cannot fill passively so completely as at the slower rates. The intraventricular pressure fails to equal the atrial pressure, and the atrial contraction plays an increasingly important role in ventricular filling (Benchimol, 1969; Mitchell and Shapiro, 1969). The atrium is more compliant than the ventricle and so an increase in atrial pressure produces a greater increase in atrial volume than the same increase in ventricular pressure would produce in ventricular volume. This change in atrial volume means more blood is ejected during atrial systole. The greater atrial emptying enhances ventricular filling and maintains the relationship between right atrial pressure and the ventricular

stroke volume. Thus even at fast rates, when atrial contraction is important to ventricular filling, Starling's law remains relevant and the atrial pressure/cardiac output relationship is maintained.

The loss of ventricular compliance ultimately limits the stroke volume so that at maximum exercise when the cardiac output is limited by the ventricular stroke volume, an increase in atrial pressure does not improve the performance (Robinson *et al.*, 1966).

The output of the normal heart can vary widely. If the ability of the heart is expressed as the family of curves relating cardiac output to right atrial pressure, then the appropriate curve is selected mainly by the cardiac rate. The heart rate reflects the influence of sympathetic and parasympathetic activity and which curve is appropriate depends on the work demanded of the heart.

The work done is determined by the cardiac output and the arterial resistance (sometimes called the after-load).

There is a firm relationship between cardiac output and cardiac work, and so performance curves can be expressed in either way. The standard definition of work in physics is:

$$\text{FORCE} \times \text{DISTANCE} = \text{WORK}$$

An alternative expression (*see Figure 3*) is:

$$(\text{FORCE/AREA}) \times \text{DISTANCE} \times \text{AREA} = \text{WORK}$$

The introduction of the area across which the force acts does not alter the equation as area is both a divisor and a multiplier. However, force/area is pressure and moving the area

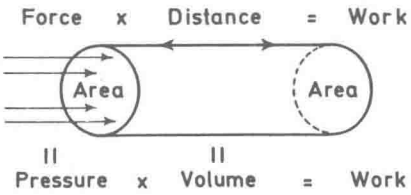

Figure 3.

some distance sweeps out a volume. Thus the work done can also be expressed as:

$$\text{PRESSURE} \times \text{VOLUME} = \text{WORK}$$

If we consider the useful work done per minute as the power of the heart. we have:

$$\underset{\text{(arterial pressure)}}{\text{PRESSURE}} \times \underset{\text{(cardiac output)}}{\text{VOLUME per min}}$$

$$= \underset{\text{(power of the heart)}}{\text{WORK per min}}$$

Thus performance curves which are expressed in units of cardiac work are referring to the power of the heart. If the arterial pressure is steady then the cardiac power alters with the output, and by assuming a steady arterial pressure we can express the cardiac performance curve simply in terms of cardiac output. In practice the output requirement dominates the performance curve but changes in arterial peripheral resistance do alter the work of the heart. Unless the performance curve of the heart changes, the cardiac output will be altered inversely as the change in resistance. However the normal left ventricle is influenced indirectly by stabilizing mechanisms such as the baroreceptors which make it less sensitive than it would be otherwise to increases in arterial resistance. Only a small decline in output occurs with increased arterial resistance (Guyton *et al.*, 1959; *Figure 4*); that is, the normal left ventricle increases its effective work. Conversely, a lower resistance only slightly alters the performance curve and increases the cardiac output. However, if the ventricle is so badly damaged by disease that its maximal performance curve is low, a useful improvement in cardiac output may be achieved by deliberate vasodilatation. The arterial pressure is lowered as much as is compatible with adequate perfusion and so the greatest cardiac output occurs with the least resistance and there is no increase in work. When cardiac work is limited, the lowest resistance achieves the best flow, for it is flow which provides the vital tissue oxygenation. Careful vasodilatation has been successful in selected patients with