Advanced Techniques in Resuscitation

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For Cherie Bank, Patti Gernerd, and Lydia Roberts

Preface

In 1985, resuscitation is an art that goes far beyond the simple performance of basic cardiopulmonary resuscitation. New concepts must be addressed not only by the researcher in the laboratory, but also by the emergency physician providing front-line care for critically ill or injured patients. This book provides an outline of some of the areas that the practicing emergency physician must come to terms with if the goal of increasing resuscitation is constantly changing. Drug dosages, routes of administration, and treatment modalities are continually being refined.

The goal of this book is to apprise the providers of emergency care of some of these changes and hopefully to stimulate thought for the generation of more change. A book dealing with resuscitation quickly can become out of date due to the prodigious amounts of research being done today. If this work stimulates even a one-minute piece of research to speed the obsolescence of information herein, we will consider ourselves to have been successful.

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Contents

CHAPTER 1

Paradoxes of CPR Physiology and Their Practical Implications

CHARLES F. BABBS, M.D., Ph.D.

A great deal seems to be said and written about cardiac resuscitation on the basis of established principles of physiology and pharmacology that have been derived from experiments in normal animals. Virtually all such classical knowledge is based upon observations made while the heart is beating rather than upon observations made during cardiopulmonary arrest and resuscitation. The relatively small number of studies made during actual CPR indicate a variety of paradoxes and anomalies which are at first surprising from the standpoint of classical physiology. Erroneous extrapolations from classical physiology to the circumstances of CPR can have direct therapeutic impact, leading to habitual practices that are probably to the detriment of patients already in the dire predicament of cardiopulmonary arrest. In this chapter I shall discuss some of these apparent paradoxes and point out their significance in practical resuscitation. Table 1.1 gives a listing of the issues to be addressed.

VENTILATION PRESSURE

Critical care physicians are routinely aware of the deleterious effects upon cardiac output of positive pressure ventilation at high airway pressures. It is easy to demonstrate in the laboratory or at the bedside that a decrease in blood pressure and, if one cares to measure it, a decrease in blood flow results from an increase in ventilation pressure above, say, 20 cm H_2O. If ventilation pressure is increased suddenly, acute observers will notice first a transient increase in arterial pressure as blood is squeezed from the pulmonary vasculature, followed by a sustained decrease in blood pressure as venous return to the chest is impeded by higher intrathoracic pressures. When the heart is beating normally the dominant effect of excessive positive airway pressure is inhibition of venous return.

However, the Johns Hopkins group has demonstrated increased carotid artery blood flow during CPR with simultaneous compres-

Table 1.1
Important Differences between Classical Physiology and CPR Physiology

Classical Physiology	CPR Physiology
High ventilation pressure decreases cardiac output by impeding venous return.	High ventilation pressure often increases blood flow by enhancing the thoracic pump mechanism.
Arteries pulsate; veins don't.	Arterial and venous pulses are roughly equal, except in the jugular veins.
Jugular venous pressure is a good indicator of right atrial pressure.	Functional venous valves at the thoracic inlet often block transmission of pressure pulses to the jugular vein.
In a given patient pulse amplitude indirectly indicates stroke volume.	Pulse amplitude relates poorly to blood flow and in the field may relate inversely to blood flow.
Right carotid artery flow is roughly equal to left carotid flow.	Right and left carotid flows may be grossly unequal.
The intravenous route gives the fastest onset of drug action.	The endopulmonary route provides faster onset of action than peripheral or central ivs.
Isoproterenol increases cardiac output in low output states and improves the chances of survival.	Isoproterenol decreases cardiac output during CPR, shunts blood away from vital organs, and minimizes the chances of survival.

sion and ventilation at exceedingly high airway pressures (75–110 cm H_2O). This experimental technique is termed SCV-CPR to denote simultaneous compression and ventilation. Our group has measured slightly increased cardiac output during standard CPR with high ventilation pressure as well as approximately 50% greater cardiac output during SCV-CPR with 75 cm H_2O ventilation pressure in three of four animal models studied. It now seems well established that during CPR high ventilation pressures tend to increase, not decrease, cardiac output.

The explanation for this apparent paradox is as follows. During CPR the act of chest compression produces high thoracic pressure pulses which impede venous inflow during chest compression at least as effectively as would high positive pressure ventilation. Consequently, venous inflow during the compression phase is already zero, and filling of the pump mechanism occurs during

chest recoil. The addition of high pressure ventilation simultaneous with chest compression, therefore, does not make net venous return any worse. On the other hand, in the circumstances of CPR pulmonary veins do become engorged with blood, and high ventilation pressures tend to squeeze the engorged pulmonary veins to produce enhanced forward flow by the thoracic pump mechanism (Fig. 1.1). In the case of standard CPR the addition of interposed

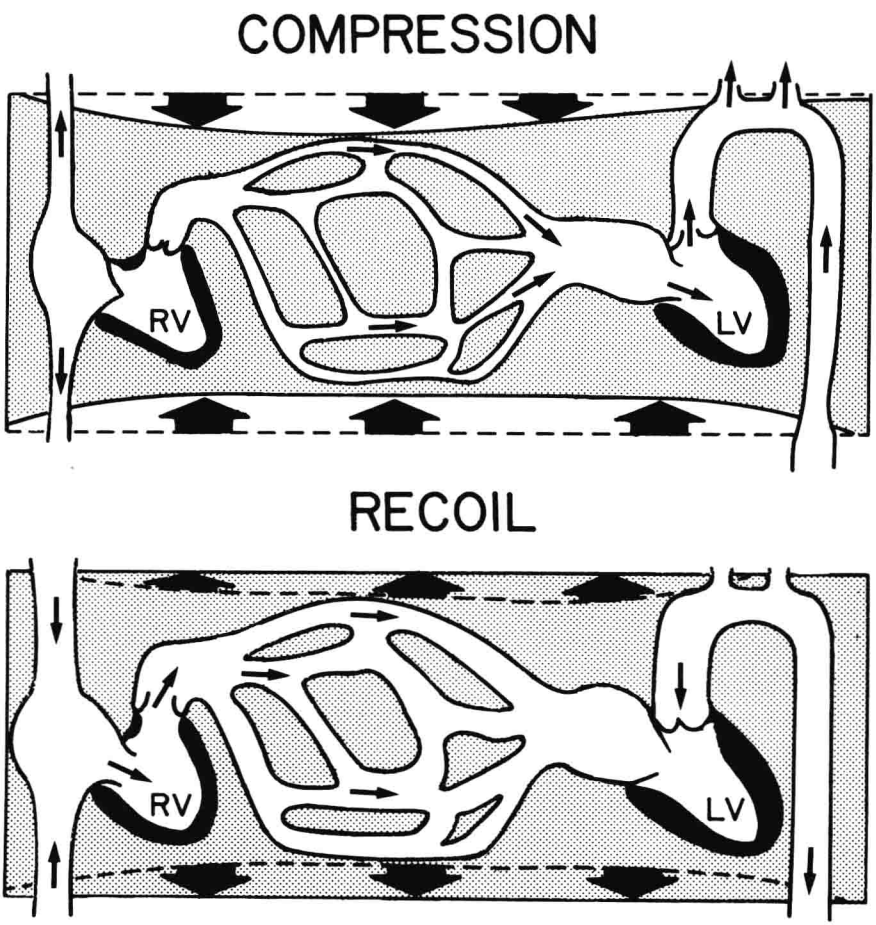

Figure 1.1. Thoracic pump mechanism for blood flow during CPR, redrawn from Niemann and co-workers (1980). During chest compression intrathoracic contents are abruptly subjected to pressures approaching 100 mm Hg. Blood is propelled forward from collapsible pulmonary vasculature through the left ventricle and into the periphery. Right heart valves and venous valves prevent backflow. Filling occurs during chest recoil.

high pressure ventilation after every fifth chest compression would not impede venous return during four of five cycles, and often tends to augment stroke volume during the compression cycle immediately following ventilation.

Clinically, there is only one reason to avoid high ventilation pressures during CPR: fear of gastric insufflation and consequent vomiting and aspiration, the latter being a major complication of practical CPR. Arterial alkalemia does occur with high pressure ventilation during CPR, but does not seem to produce cardiac arrhythmias, impaired cardiac function, or reduced survival, as has been demonstrated following rapid CO_2 depletion in dogs with beating hearts. This result itself is a paradox, which is probably explained by the very low flows during CPR which do not permit tissue alkalosis and hypocarbia even though arterial blood is quite depleted of CO_2. Pulmonary rupture with high pressure ventilation during CPR has simply not been reported.

So, as long as the airway is secured by an endotracheal tube, there is good reason to apply high pressure ventilation. Ventilation pressures lower than 20 cm H_2O are known to produce unsatisfactory blood oxygenation even when the breathing gas is 80–100% oxygen. The cause for the intrapulmonic shunt fraction is evident in experimental studies at post mortem: atelectasis of substantial portions of the lung due to chest compression. Such atelectasis can largely be reversed by ventilation at higher pressures. In standard CPR with interposed ventilations at 20–40 cm H_2O oxygenation will be improved and cardiac output will not be diminished. During experimental SCV-CPR with ventilation pressures over 70 cm H_2O both blood oxygenation and blood flow will be improved.

VENOUS PULSATION

In surgery one quickly learns to distinguish arteries from veins by palpation as well as by inspection. Arteries pulsate, veins generally don't. During CPR, however, the high intrathoracic pressure pulses are transmitted into peripheral veins as well as peripheral arteries (Fig. 1.2). The one known exception to this rule is the jugular vein, which is next to be discussed. In the 1960s Birch and co-workers noted the prominent venous pulsations during experimental CPR in dogs and in baboons. Thinking them artifactual, Birch and co-workers advocated measurement of pe-

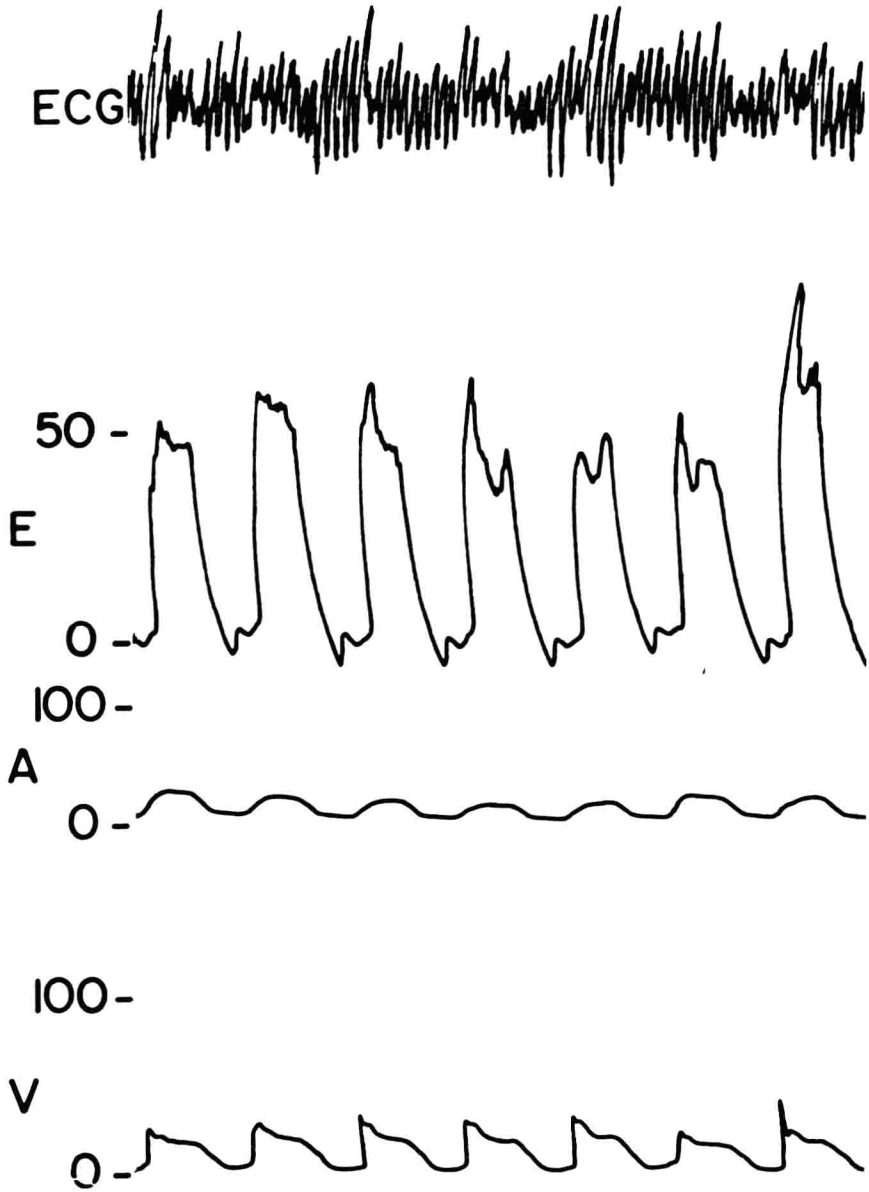

Figure 1.2. Femoral artery and vein pulses during CPR. The graphic record shows the electrocardiogram (ECG), esophageal pressure (E), femoral artery pressure (A), and femoral vein pressure (V) in a dog during ventricular fibrillation and manual CPR. In some cases venous pulsations can be equal in amplitude and more abrupt in onset than arterial pulses during CPR.

ripheral venous pressure with catheters pointed away from the heart. The pressure pulses are real, however, and, as shown in Figure 1.2, can be equal or greater in amplitude to arterial pulses during the compression phase—even during effective CPR. The often observed equality of systolic arterial and venous pressure pulses has been emphasized in discussions of the hemodynamic mechanisms of CPR.

The obvious clinical implication is that detection of a good peripheral pulse does not necessarily imply good CPR. In general, the pulse is a notoriously unreliable guide to perfusion during CPR, not only for this reason but also because quick jabbing compressions, quite ineffective in producing flow, are capable of generating palpable pulses. Indeed, the easiest way to produce a detectable pulse by chest compression is by quick jabbing sternal compressions rather than the properly recommended 50% duty cycle compressions described in CPR standards. When the production of a palpable pulse becomes the focus of CPR training, bad technique is unconsciously but systematically encouraged. The real goal, as I shall emphasize later, is to maximize not the peak amplitude of the pulse but rather the area under the blood pressure tracing displayed as a function of time.*

JUGULAR VENOUS VALVES

Physicians are taught that there are no functional valves between the right atrium and external jugular veins in the neck. Everyday physical examination confirms this impression. It is easy to observe height of the jugular venous column in patients and the classical jugular venous pulsations that seem to indicate an open connection with the right atrium. However, it is also easy to observe the jugular venous column during a Valsalva maneuver—an experiment you, the reader, can perform yourself while looking into a mirror. At the onset of the Valsalva maneuver the jugular veins do not fill from below, as one would expect if a direct communication transmitted the suddenly increased intrathoracic pressure. Instead, the veins fill gradually from above. This conclusion can be confirmed by occluding the vein with digital pressure

*The issue of compression duration and style has been resurrected in 1983–84 by researchers at Duke University, who advocate "high impact" CPR, in which 50% compression duration is maintained but at a much faster rate—120/min.

at the base of the neck just before beginning the Valsalva. The pattern of filling is just the same; so filling of the jugular vein during a Valsalva maneuver is not due to retrograde flow from the thorax. Some mechanism prevents transmission of high intrathoracic pressure pulses to the external jugular veins in the neck; this mechanism is not operative when thoracic pressures are low.

The explanation for lack of transmission of high intrathoracic pressures to the external jugular vein is the presence of the valves at the level of the clavicle in man which seem to close only in the presence of high intrathoracic pressures, such as are generated by cough, Valsalva—or CPR. Evidence of functioning of these valves during CPR has been reported by us, by Rudikoff, and by Niemann and co-workers, who have shown by a simple catheter advance/pullback maneuver that high intrathoracic pressure pulses suddenly disappear when the tip of the venous catheter passes the thoracic inlet both in animals and in man (Fig. 1.3). Niemann and Rosborough dissected the site of the transition in dogs and located anatomic valves, the existence of which had actually been reported decades earlier. Indeed, the valves are clearly noted in Gray's anatomy text. They probably function in normal life during straining at stool and during cough, when they may serve to prevent sudden increases in intracranial pressure.

These valves also have functional significance in CPR. First, their existence ensures the validity of the practice of palpation of the carotid pulse rather than femoral or other pulses, as a guide to field CPR. Because of these valves, acting together with internal jugular venous valves, a pulse felt in the neck is likely to be arterial rather than venous in origin. Second, by preventing high jugular venous pressure they increase the carotid-jugular pressure difference. In my opinion they account, at least in part, for the preferential perfusion of the brain during experimental CPR that we and others have observed.

Niemann's valves also explain why increased carotid flow is consistently obtained by experimental maneuvers such as simultaneous ventilation and compression that create pulses of generally raised intrathoracic pressure. Weale and Rothwell-Jackson and the Hopkins group, who have most intensively studied SCV-CPR consistently, report that systolic aortic and right atrial pressures are the same during standard external CPR or SCV-CPR, yet when the amplitude of these pulses is increased by simultaneous

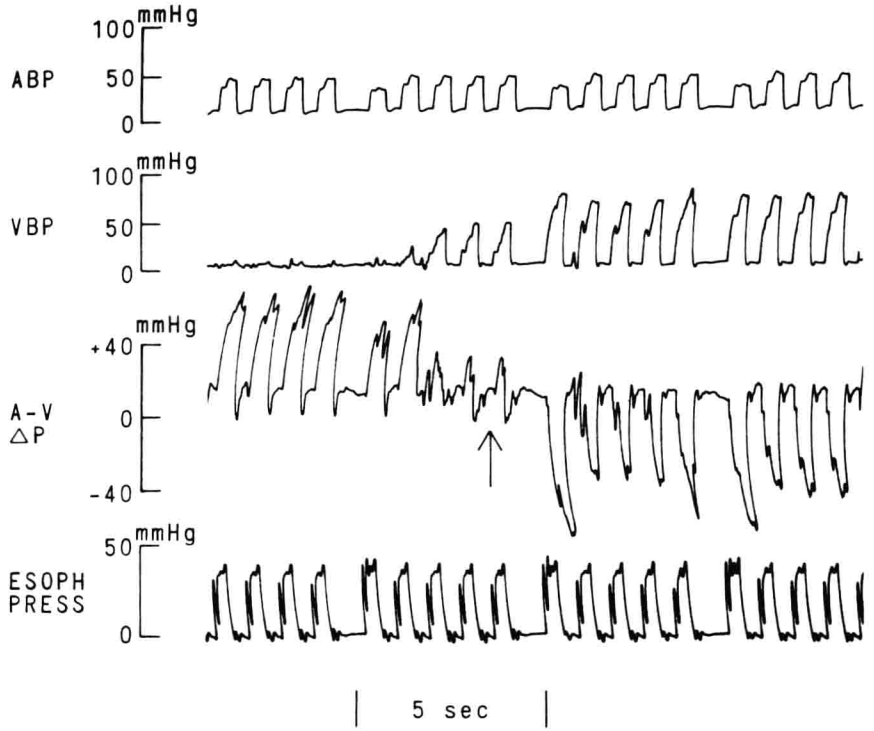

Figure 1.3. Arterial blood pressure (ABP), venous blood pressure (VBP), electronically derived arteriovenous pressure difference (A-V ΔP), and esophageal pressure (ESOPH PRESS) recorded during experimental CPR in a dog. The functional jugular venous valve is demonstrated by advancing the venous catheter tip from the jugular vein into the superior vena cava (arrow). The force of chest compression remains constant, as shown by the esophageal pressure trace.

ventilation and compression, carotid flow increases. This result is exactly what would be expected if valves protected the cranial circulation from high venous pressure pulses during the compression phase, for the same reason that the mitral and tricuspid valves of the heart ensure forward flow when the ventricles contract during normal sinus rhythm.

This privileged position of the cerebral circulation is further significant in that many investigators have relied chiefly on measurements of carotid artery flow to indicate perfusion during CPR. Such measurements may not be representative of flow to other body parts, especially the heart, which are not protected from high venous pressure pulses.