Hemodynamic Management During Closed Circuit Percutaneous Cardiopulmonary Bypass

George A. Justison, BS, CCP and William Pelley, CCP

William Beaumont Hospital, Royal Oak, MI
and PSICOR, Inc., San Diego, CA

Key words: Femoral-femoral bypass, hemodynamic management, emergency bypass.

ABSTRACT

The technological development of portable percutaneous cardiopulmonary bypass systems has expanded the use outside of the operating room. Two of the major indications have been for resuscitation and supported angioplasty. The use of a decision-making algorithm will decrease the learning curve for the perfusionist and cardiologist when dealing with a closed circuit CPB system.

INTRODUCTION

The use of cardiopulmonary bypass in an emergent setting dates back to 1958 when Stuckey used the then experimental technology to resuscitate and support patients suffering from myocardial infarction that were unresponsive to conventional medical treatment.[1] The technology of extracorporeal circulation has advanced dramatically since those early days. Improvement in techniques, reliability of equipment, and training of personnel has reduced the morbidity and mortality associated with CPB. Although the early demands for CPB were directed towards emergent resuscitative applications, the primary focus today is elective cardiopulmonary support for open heart surgical candidates.

Several constraints limited the use of CPB outside the controlled environment of the surgical arena. Early CPB systems required significant time to assemble and prime, and presented many logistical problems when transporting while attached to a patient.[1] Providing access to the patient's circulatory system required surgical exposure of the desired cannulation vessel, delaying the initiation of CPB.[2] Phillips in 1983 described a technique of percutaneous cannulation decreasing the time delay to initiate emergent CPB, however, the extracorporeal circuit (ECC) employed still depended on gravity venous return and was severely flow restrictive.[3]

Recently these problems have been addressed with the Bard Cardiopulmonary Support System (CPS).[a] CPS provides the operator with rapid setup and priming through a pre-assembled perfusion circuit that may easily be primed in less than five minutes. The problems of adequate vascular access reported by previous investigations has been eliminated through the development of arterial and venous cannula that will accommodate cardiopulmonary support and may be inserted through a modified Seldinger technique. Although percutaneous insertion places limitations on cannula size, venous return problems have been absolved by utilizing a closed circuit direct aspiration ECC rather than relying on the limitations of gravity drainage. Additionally, the entire system has been designed to allow intrahospital transport and supports a self-contained DC power system and oxygen source.

The development of a reliable self-contained portable cardiopulmonary bypass system has advanced the application of extracorporeal support to areas outside the operating room and traditional ECMO environment. One logical application for CPB support has been high risk Percutaneous Transluminal Coronary Angioplasty (PTCA) procedures where the patient is at risk for hemodynamic collapse. This may include such patients as those with left main coronary lesions and unprotected left main equivalents. In addition elective cardiopulmonary support of patients with compromised ventricular function may benefit from prophylatic CPB support. Application of the CPS system to this patient population has coined a new category of Supported Angioplasty in the PTCA realm. Prior to the availability of portable CPB equipment these patients may not have been considered for PTCA, thus opening a new patient population for the interventional cardiologist. Elective support of the high risk PTCA patient allows longer inflation times and the opportunity for the cardiologist to make decisions in a controlled environment with the comfort of knowing the patients systemic metabolic and respiratory needs are being maintained.

It is the purpose of this investigation to discuss the hemodynamic management differences for the perfusionist when dealing with a closed CPB system. Unlike traditional extracorporeal circuits commonly found in the operating room, the Bard CPS system provides no venous reservoir and depends on direct aspiration from the patients venous system to supply the CPB circuit. The inability to control the patient volume status through the Extracorporeal Circuit (ECC) presents new challenges to traditional patient management. Two additional factors found in the supported angioplasty setting, conscious patients and noncross clamped/non-vented hearts, influence the

Address correspondence to: George A. Justison, PSICOR, Inc. 16818 Via del Campo Court, San Diego, CA 92127

a. C.R. Bard, Inc., Billerica, MA

ECC management decisions of the perfusionist.

It is the purpose of the presentation to illustrate some of the hemodynamic management changes that occur during closed circuit percutaneous bypass as compared to conventional CPB, and present diagnosis and management options for the perfusionist providing ECC support during Supported Angioplasty.

MATERIALS AND METHODS

Ten adult patients were selected for elective supported angioplasty. Selection criteria included the presence of a left main lesion, or unprotected left main equivalents. A left main equivalent may be considered to be a patient with a high grade stenosis in the Left Anterior Descending (LAD) or Left Circumflex (LCx) that supplied a majority of left ventricular myocardium and was unprotected by collateral circulation. In addition, patients with significant (greater than 60%) Right Coronary Artery (RCA) lesions which were the dominant vessel supplying collateral circulation were candidates. All patients had ejection fractions less than 25%. The risks and benefits of Supported Angioplasty were explained to all patients and each agreed to the procedure under informed consent.

The ECC consisted of the Bard CPS system provides a self-contained portable ECC that can be rapidly instituted anywhere in the hospital. The disposable perfusion circuit consists of a Bio-Medicus constrained vortex pump head, water based heat exchanger, and Bard HF4000 Hollow Fiber Membrane Bundle. Disposable components are interconnected with 3/8" polyvinylchloride tubing incorporating a rapid infusion line proximal to the pump head, Bio-Probe electromagnetic flow probe and blood temperature connector. The entire circuit is pre-assembled and tie banded at each connection. CPS hardware include either a Bio-Medicus 520D or 540 Pump Controller,[b] DC power source, and Normothermia Heater System all placed in a portable cart with IV pole and mounting bracket for an E size oxygen tank (not provided).

After inspection of the CPS disposable circuit for sterility and integrity, the circuit was hung in the respective holders on the CPS cart. Water supply lines from the normothermia unit were connected to the heat exchanger and water flow was started at 37°C to ensure integrity of the heat exchanger. The bio-Probe transducer and temperature probe were connected in the appropriate fashion. The unit was primed with 1,400 milliliters of Plasmalyte A,[c] 12.5g Mannitol, and 5000 units of beef lung heparin. Recirculation and debubbling was completed following the manufacturer's instructions. Gas supply was provided by a Sechrist[d] blender connected to wall oxygen and compressed room air supplies at 50 psi.

Once the patient was placed on the cardiac catheterization table, ventilation was maintained with nasal oxygen at five liters per minute (LPM). Left and right groins as well as right anticubital and right external jugular areas were prepped and aseptically draped. After heparinization with 300 units/Kg of beef lung heparin, adequacy of anticoagulation was determined by an activated clotting time greater than 400 seconds. Patients one through four received a surgical cutdown on the right femoral artery and vein and cannulation under direct visualization. Patients 5-10 received percutaneous cannulation using the Bard 20 French Percutaneous arterial and venous CPS cannula using a modified Seldinger technique. Left femoral vein was cannulated with an 8 French catheter and a transvenous temporary pacemaker was placed in the right ventricle. Left femoral artery was also cannulated with an 8 French catheter and served as the access site for PTCA guiding catheter and PTCA balloon catheter. A Swan Ganz catheter was placed in the right anticubital vein and advanced to provide pulmonary artery and central venous pressure monitoring. An S-tip multipurpose catheter was placed in the coronary sinus for monitoring of coronary sinus percent oxygen saturation and lactate levels.

The CPS circuit was connected to the CPS cannula ensuring that all air was removed from the arterial and venous junction. Baseline hemodynamic values, coronary sinus lactate and hemoglobin oxygen saturation, aortic lactate and hemoglobin saturation values were recorded. CPB was instituted to a cardiac index of 2.4 liters/minute/meter square body surface area as the patient tolerated to ensure no drop in mean arterial pressure. Neosynephrine (10mg/250ml 0.9 NaCl) was administered in 3cc bolus increments to augment arterial pressure to maintain a minimum MAP of 70 mm Hg. Circulating blood volume was increased by the transfusion of Plasmalyte A as the CVP approached zero in order to achieve maximum blood flow. Once the patient was stabilized a full CPS support hemodynamic, lactate, and hemoglobin saturation values were repeated. ACT's and blood gas data were evaluated every 20 minutes. Anticoagulation was maintained by bolus injections of heparin to maintain ACT's greater than 400 seconds.

Full CPS support was maintained throughout the PTCA procedure. The patient's Systemic Vascular Resistance (SVR) was manipulated using either neosynephrine or IV nitroglycerin injection to keep the MAP>70mmHg and SVR between 800-1500 dynes*sec*cm-5. Volume transfusions were controlled to ensure adequate circulating blood volumes necessary to support full CPS support. At the conclusion of the CPS procedure, bypass was terminated in a usual fashion of decreasing ECC blood flows while transferring volume to the patient's native circulation. All blood was salvaged from the CPS circuit and administered to the patient as post-PTCA volume replacement dictated. Heparin was not reversed in this patient population and the arterial and venous cannulas were left in place until the body metabolized the circulating heparin and ACT's returned to baseline. Statistical analysis between pre-bypass and CPS bypass data was performed using a Student's paired t-test.

RESULTS

Table 1 presents the demographic data for this patient population. The mean age was 69.5 years with a range of 51 to 78 years. A total of 13 vessels received PTCA in the 10 patients requiring a mean bypass time of 58 minutes, range 35 to 89 minutes. Of the 13 coronary arteries dilated, four were LAD's,

b. Bio-Medicus, Eden Prairie, MN
c. Travenol Laboratories, Inc., Deerfield, IL
d. Sechrist Industries, Inc., Anaheim, CA

four LCx, two RCA and three LM. A total of 68 dilatations were performed with a mean dilatation time of 4.7 minutes (range 1.5 to 10 minutes).

Figure 1 represents the comparison of expected or target blood flow based on a cardiac index of 2.4 l/min/M^2 and the actual peak blood flow maintained during CPS. Statistical analysis shows no statistically significant difference between the two groups at $p<0.001$ ($n=10$).

Table 2 illustrates the differences between aortic pulse pressure and MAP pre bypass and during CPS. A statistically significant, $p<0.001$, difference exists between the pulse pressure groups. No statistical difference is found in the MAP group ($p<0.001$) with a 99.9% confidence limit.

Figure 2 depicts the relationship between total input volume during the CPS procedure and the urine output. **Figure 3** is perfusion management decision making algorithm that may be used by the perfusionist to help distinguish between patient volume, ECC blood flow, and SVR management decisions during the course of closed circuit percutaneous bypass. **Figure 3** presents the pre-bypass and bypass levels for pulse pressure, $ScVO_2$, and lactate. No statistically significant difference were found between the pre-bypass groups for saturation or lactate.

DISCUSSION

The development of percutaneous portable cardiopulmonary bypass systems has allowed the perfusionist to expand his/her professional knowledge and services outside of the operating room and into the interventional cardiology market. Two valuable uses for emergency femoral-femoral bypass has been in the applications of emergency resuscitation and supported angioplasty.[4] The primary benefit of emergency femoral-femoral bypass during cardiac arrest is the ability to return the entire basal cardiac output to the systemic organs and myocardium within a few minutes regardless of whether electro-mechanical function has been restored. During supported angioplasty the perfusionist can ensure the cardiologist that systemic metabolic needs are being met and provide a controlled environment for PTCA or mechanical arthrectomy. Three objectives exist for the perfusionist during supported angioplasty, support systemic organ metabolic requirements, decrease myocardial oxygen demands, and preserve myocardial function.

Several differences exist in the perfusion management scheme when dealing with closed circuit percutaneous bypass as compared with the more familiar open CPB systems employed in the cardiac surgical arena. During open chest cardiac surgery, the perfusionist incorporates a venous reservoir (either a soft shell bag or hard shell reservoir) for control of the total circulating blood volume. This allows exsanguination of part or all of the patients circulating blood volume and a continuous estimation of the circulating volume. Venous return is typically controlled by gravity drainage and directly affects the ECC blood flow. In the closed circuit system, the equation becomes much more complicated. Venous return in direct aspiration systems, such as the CPS circuit, depends on the negative pressure developed by the vortex pump, cannula design, venous capacitance, and venous volume status of the patient. Since the CPS circuit is a fixed volume, the patient serves as the venous reservoir.

Upon the initiation of femoral-femoral bypass blood is diverted from the right atrium through the venous cannula positioned at the junction of the inferior vena cava and the right atrium. At this point the filling pressure to the right ventricle is decreased reducing the output to the pulmonary circulation and in return decreasing the end diastolic volume of the left ventricle. This is illustrated in **Table 2** by a reduction in the aortic pulse pressure. Optimal closed circuit CPB for supported angioplasty is depicted by a maximal reduction in the aortic pulse pressure while maintaining a pre-bypass MAP. Optimization of this scenario represents maximum decompression of the left ventricle while maintaining the driving force for systemic and myocardial perfusion, the MAP.

Decision Making Algorithm

Figure 3 represents a decision making algorithm for the perfusion manager to use during closed circuit CPB. The originating diagnostic question begins with, "Is the MAP within the desired range?" If the MAP is acceptable then progression through the algorithm stops. If the MAP is high further diagnosis and suggested corrective actions may be found by taking the "High" MAP pathway. With the MAP below the desired limits further diagnosis and support is found in the "Low" MAP pathway. The next prompt is common to both pathways, "Is the Trace Pulsatile?" If the aortic pressure tracing is still pulsatile an assumption may be made that we have not diverted all the blood flow to the femoral venous cannula. The patient is circulating the remaining volume through the native circulation causing the pulsatility in the aortic tracing.

If the MAP "High" pathway is chosen, two etiologies can exist for this situation either improper blood flow selection or a high SVR. If the trace is pulsatile the perfusionist must determine if the ECC blood flow is below the ideal limit for this patient. This may be accomplished by monitoring the mixed venous oxygen saturation of hemoglobin to maintain saturations greater than 65%. If the actual blood flow is less than ideal, administration of a vasodilator will lower the SVR and MAP allowing an increase in ECC blood flow. If blood flow is adequate, administration of a vasodilator will return the MAP to the desired range. If the aortic trace is not pulsatile and the MAP is high, then an adjustment in the patient's SVR is necessary. Either systemic vasoconstriction is present or the perfusionist is hyperperfusing the patient. Monitoring of the SvO_2 along with SVR will aid in the diagnosing of this situation. It should be remembered that calculation of the SVR is only dependable during nonpulsatile CPB due to the inability to determine the percentage of the total cardiac output being provided by the patient in a pulsatile situation.

If the "MAP Low" pathway is selected four potential underlying reasons for this clinical situation could exist. Either the SVR is low and needs to be corrected with a vasoconstrictor, the ECC blood flow selected is inadequate for the patient, the patient is volume depleted, or a mechanical obstruction exists at the venous cannula limiting blood flow to the femoral-femoral ECC. Answering the prompts in the "Low MAP" pathway of the

decision making algorithm will help the perfusionist diagnose and correct the underlying cause of low MAP. As in the high MAP pathway, SvO_2 and SVR determinations should be made when determining adequacy of ECC blood flow selection.

Significance of Hemodynamic Management Decisions

The significance of choosing the correct hemodynamic management scheme can be explained by discussing intracardiac volume changes and resulting wall tensions during closed circuit cardiopulmonary bypass. As stated earlier our goal for hemodynamic management during supported angioplasty is to minimize the pulse pressure generated by the left ventricle while maintaining the pre-bypass MAP. This should result in a decrease in myocardial oxygen consumption by reducing left ventricular work while ensuring myocardial oxygen supply. MAP may be considered the major determinate in myocardial blood flow assuming the coronary vascular resistance remains constant. However, incorrect perfusion management decisions may actually increase the myocardial oxygen consumption (MVO_2) during critical periods of PTCA dilatation.

Table 2 illustrates the reduction in pulse pressure associated with the initiation of CPB and the corresponding changes in MAP. Statistically significant changes occurred in pulse pressure for every patient with the initiation of CPB. MAP did not change significantly but isolated changes did occur on select patients, especially those who presented with high MAP pre-bypass. The "ideal" CPB management may be identified by those cases where the pre- and post-CPS MAP lines intersect. Here we find the scenario of pulse pressure reduction and maintenance of MAP. In **Figure 2** we can compare the CPS volume status we can note the input/output status of the corresponding case numbers. In CPS procedures one through four volume input significantly exceeds urine output ($p<0.001$) during the CPS procedure. In these patients volume additions were made to augment the patients circulatory volume in an attempt to increase the stroke volume of the heart to prevent a reduction in the MAP. In patient two a total of 4.2 liters was transfused to maintain the MAP. Although graphically this appears to satisfy the criteria for "ideal" management, expansion of cardiac volume can have negative effects on an already compromised myocardium.

At the initiation of our supported angioplasty program, it was assumed that a reduction in the aortic pulse pressure corresponded with a reduction in left ventricular work due to decreased stroke volume and ultimately decreased MVO_2. However, this is only true under select perfusion management schemes. The volume-elasticity coefficient (Ev) is indicative of the effect of changing systemic volume and distensibility on the amount of pulse pressure generated by the heart under conditions of constant contractility. The Ev may be represented by the equation:[5]

$$Ev = (P/v) * V * 100$$

where:

Ev = Volume-Elasticity Coefficient
P = Pulse Pressure
v = Stroke Volume
V = Arterial Reservoir Volume

Rearranging this equation we can see that the stroke volume becomes a function of the pulse pressure and arterial reservoir volume divided by the Ev.

$$v = (P*V)/Ev$$

If no circulating volume changes occur, indicating that the perfusionist has not transfused any volume and negligible urine output has occurred resulting in a net zero fluid balance, the arterial reservoir volume (V) may be ignored. Therefore, the equation reduces to:

$$v = P/Ev$$

The volume-elasticity coefficient is representative of the resistance to blood flow generated by the radius of the arterioles. This can be estimated during CPB by the Geometric Component (GC) which is the resistance to blood flow in the vasculature minus the viscous effects on blood flow resistance.

$$GC = SVR/viscosity$$
$$Viscosity = e\ (.2345 \times HCT + B)$$
$$B = e\ (-.04827 \times T + .9213)$$

where:

e = Exponential e (2.718)

HCT = Hematocrit of Circulating Blood Volume
T = Temperature of Circulating Blood Volume

In essence the GC allows use to evaluate the effect of arteriole radius on the resistance to blood flow without the effects of viscous drag imparted by the blood. Vasoconstriction increases the GC and conversely vasodilatation decreases the GC.[6] Substituting the GC for Ev the equation reads:

$$v = P/GC$$

The relationship between P and Gc demonstrate that P alone is not a good indicator of v and resulting myocardial work. One must take a look at the function of La Place.

LaPlace relates the amount of myocardial wall tension to intraventricular pressure. Total myocardial wall tension (Ttw) is representative of sum of the tension developed by all the muscle fibers to maintain the integrity of the myocardial wall during diastole and systole.[5] It is represented by the equation:
where:

Pv = Intraventricular Pressure (dynes per square centimeter)
r = radius of the ventricular (assumes a spherical model)

If we accept the spherical model for the ventricle we know the radius by:

$$V = 4/3* *r\ ^3$$

In a normal size and functioning myocardium, the wall tension actually decreases during systole due to the large decrease in the radius during contraction as the result of the decreasing cardiac volume. This significant reduction in radius is enough to overshadow the increase in pressure caused by the contractile element. However, in cardiac failure or during PTCA (when the mechanical efficiency of the myocardium is disturbed due to the lack of blood flow to a significant portion of the

myocardium from the PTCA catheter), a small change in the radius occurs in respect to the diastolic radius (and diastolic volume) so a net increase in total wall tension occurs. An examination of total wall tension and left ventricular volume curves for a normal and dilated heart illustrate the effect of increasing cardiac volume on myocardial work and oxygen consumption. In order to eject the same stroke volume in a dilated heart a significant increase in wall tension is required. Myocardial work and oxygen consumption can be related to the area inside the curve.

It becomes apparent then that we cannot look at pulse pressure reduction alone as an indicator of proper hemodynamic management and adequate decompression of the heart during closed circuit CPB.

A typical case scenario may be after initiation of CPS a 50% reduction in pulse pressure occurred while the MAP was maintained at 80 mmHg. The first dilatation of the LCx was planned to be for five minutes. After one minute dilatation, the aortic pulse pressure began to decrease as a result of decreasing myocardial efficiency related to compromised blood flow to the left ventricle. Since the systolic pressure has a greater effect on the MAP than the diastolic pressure the MAP falls to 60mmHg. The physician instructs you to transfuse the patient to increase the stroke volume and in return raise the pulse pressure and MAP. After transfusing 800 cc volume the pulse pressure and MAP return to normal. In effect, we have increased the cardiac volume and forced the left ventricle to increase the stroke volume with an exponential increase in total wall tension. This clinical rationale jeopardizes already compromised myocardium by mechanically induced ischemia. A wiser choice would have been to increase the GC to augment MAP in light of natural reduction in pulse pressure.

Volume enhanced perfusion management can be illustrated in patient's one through four where a significant imbalance exists in the input/output status. As we progressed in the learning curve on patients five through ten the less volume management was used in light of GC manipulation. Use of the decision making algorithm shortens the learning curve for closed circuit hemodynamic decision making and presents the patient with fewer situations of artifically increased cardiac volume. Examination of CPS volume status and CPS pulse pressure/MAP data shows that fewer volume administrations were required to meet the ideal criteria of decreased pulse pressure and equilibrium of MAP.

Observation of the SCO_2 and lactate levels indicate that mycardial metabolism was maintained in homeostatic state during supported angioplasty. Coronary sinus oxygen saturation values increased and coronary sinus lactate levels decreased in this patient population. Significant anerobic metabolism did not occur in patients with this presenting pathology and compromised coronary reserves as would be suspected.

The future of supported angioplasty will depend on the development of improved perfusion management techniques. Currently research is under way at this institution to relate the correlation between perfusion management strategies and cardiac volumes using transesophageal echocardiography. More development on percutaneous ventricular vent catheters must be completed so the cardiac volume may be directly controlled in order to prevent possible deleterious myocardial damage during supported angioplasty.

REFERENCES

1. Litzie K, and Roberts CP.: Emergency femoral-femoral cardiopulmonary bypass. *Proceed Am Acad Cardiovasc Perf*, Vol 8, September 1987.
2. Phillips SJ, Ballentine B, Slonine D, et al.: Percutaneous initiation of cardiopulmonary bypass. *Ann Thorac Surg*, Vol. 36, 223-225, 1983.
3. Riley JB, Litzie K, et al.: Supported angioplasty: A new contribution for extra-corporeal technology. *JECT*, Vol 20, No 4, Winter, 1988.
4. Sabatelli P, Hill AG.: Resuscitation with cardiopulmonary bypass and intra-aortic balloon pump. *Proceed Am Acad Cardiovasc Perf*, Vol. 5, Jan 1984.
5. Little RC.: Physiology of the heart. *Circulation*, Chicago, 1981, ppl80-262.
6. Riley JB and Zaiden JR.: The immediate hemodynamic and metabolic effects of bolus injections of pharmacologic agents during cardiopulmonary bypass. *JECT*, Vol 15, NO. 3, May/June 1983.

LEGENDS

Table One: Patient demographics for supported angioplasty patients.

Figure One: Comparison of expected CPS blood flow based on cardiac index of 2.4 and actual blood flow maintained during PTCA

Table Two: Relationship of pre-bypass and bypass pulse pressure and mean aortic pressure.

Figure Two: Relationship of total volume input and urine output during supported angioplasty.

Figure Three: Comparison of pre-bypass and bypass pulse pressure reduction, coronary hemoglobin saturation, and lactate levels.

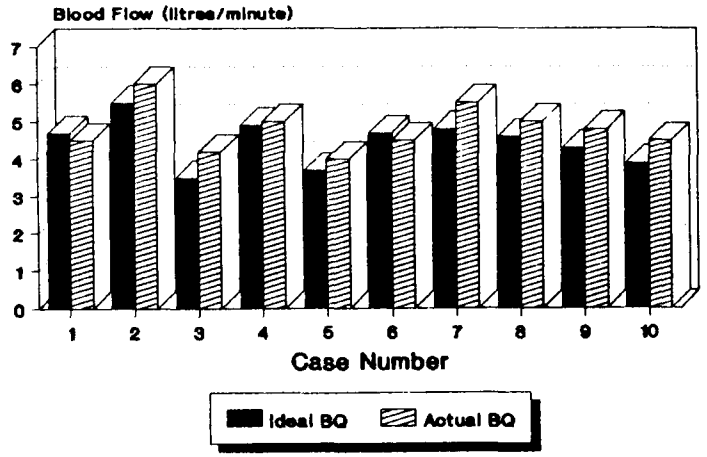
TABLE 1

CPS Patient Demographics

Parameter	Value	Range
Age	69.5	51-78
CPS Time	58 min	35-89
PTCA Vessels		
LAD	4	
LCx	4	
RCA	2	
LM	2	
Total Inflations	68	

FIGURE 1

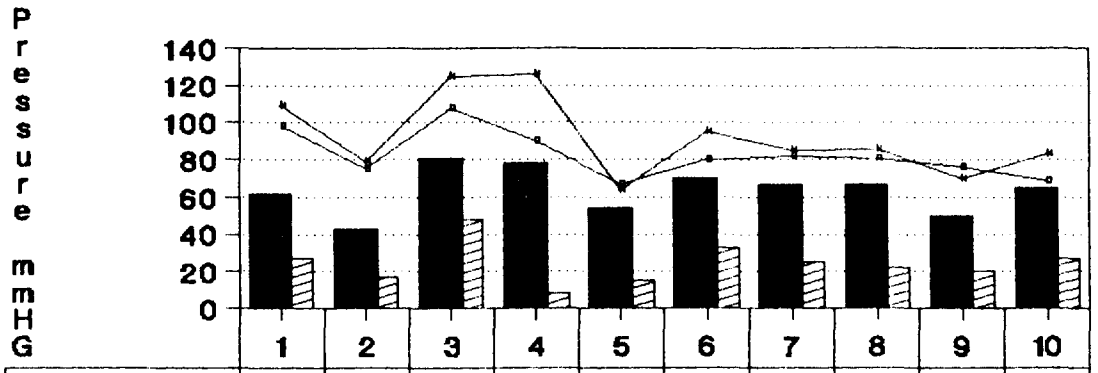
CPS BLOOD FLOW



Ideal vs actual BQ. NS at p<0.001

TABLE 2

CPS PULSE PRESSURE vs MAP



	1	2	3	4	5	6	7	8	9	10
Pre CPS P.P.	62	43	81	78	54	70	67	67	50	65
Post CPS P.P.	27	17	48	8	15	33	25	22	20	27
Pre CPS MAP	109	79	125	126	64	95	85	86	70	83
Post CPS MAP	98	75	108	90	67	80	82	81	76	69

Case Number

Pre CPS P.P. Post CPS P.P.
Pre CPS MAP Post CPS MAP

P.P. sign at p<0.001,MAP ns at p<0.001

FIGURE 2

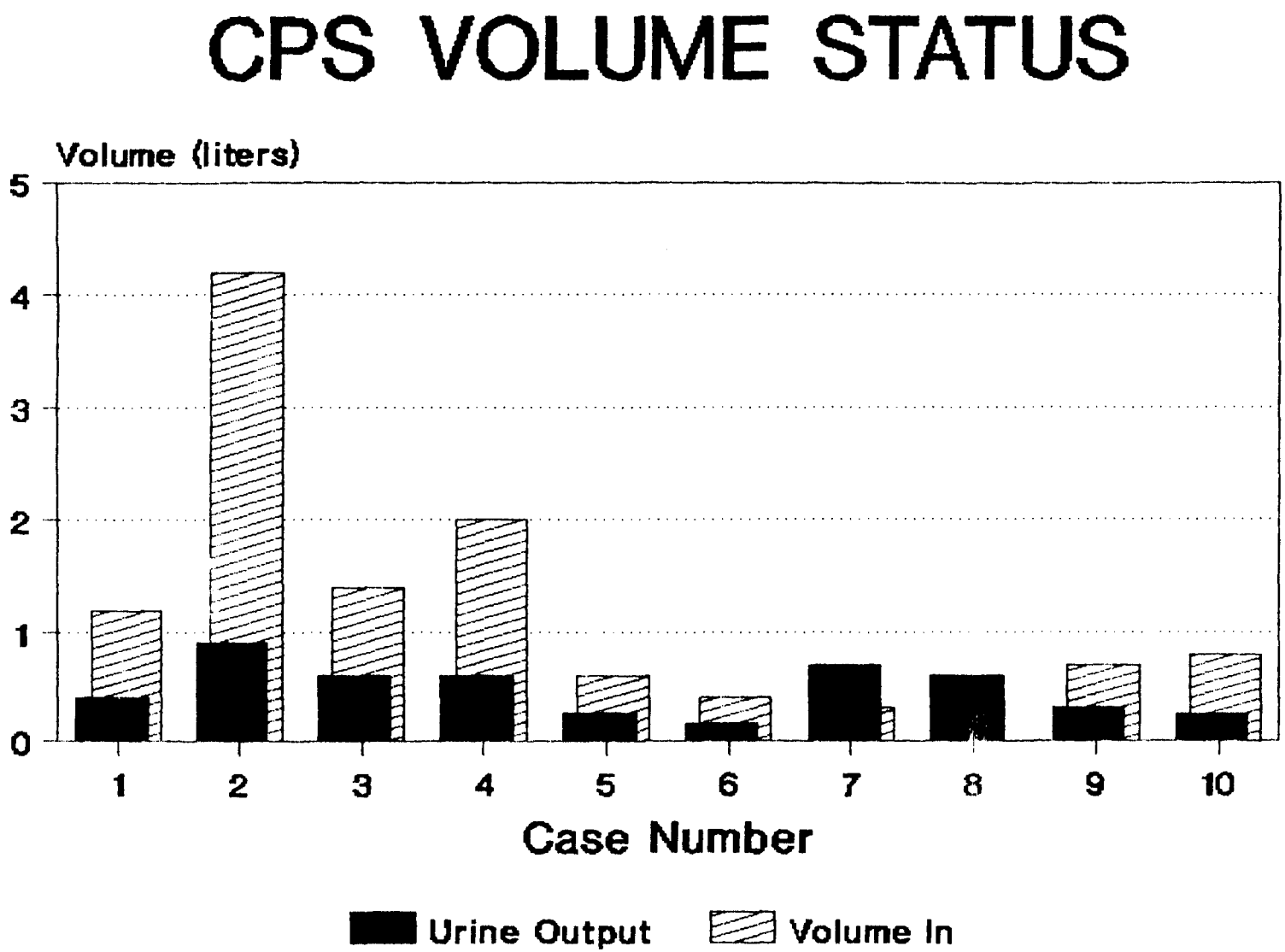
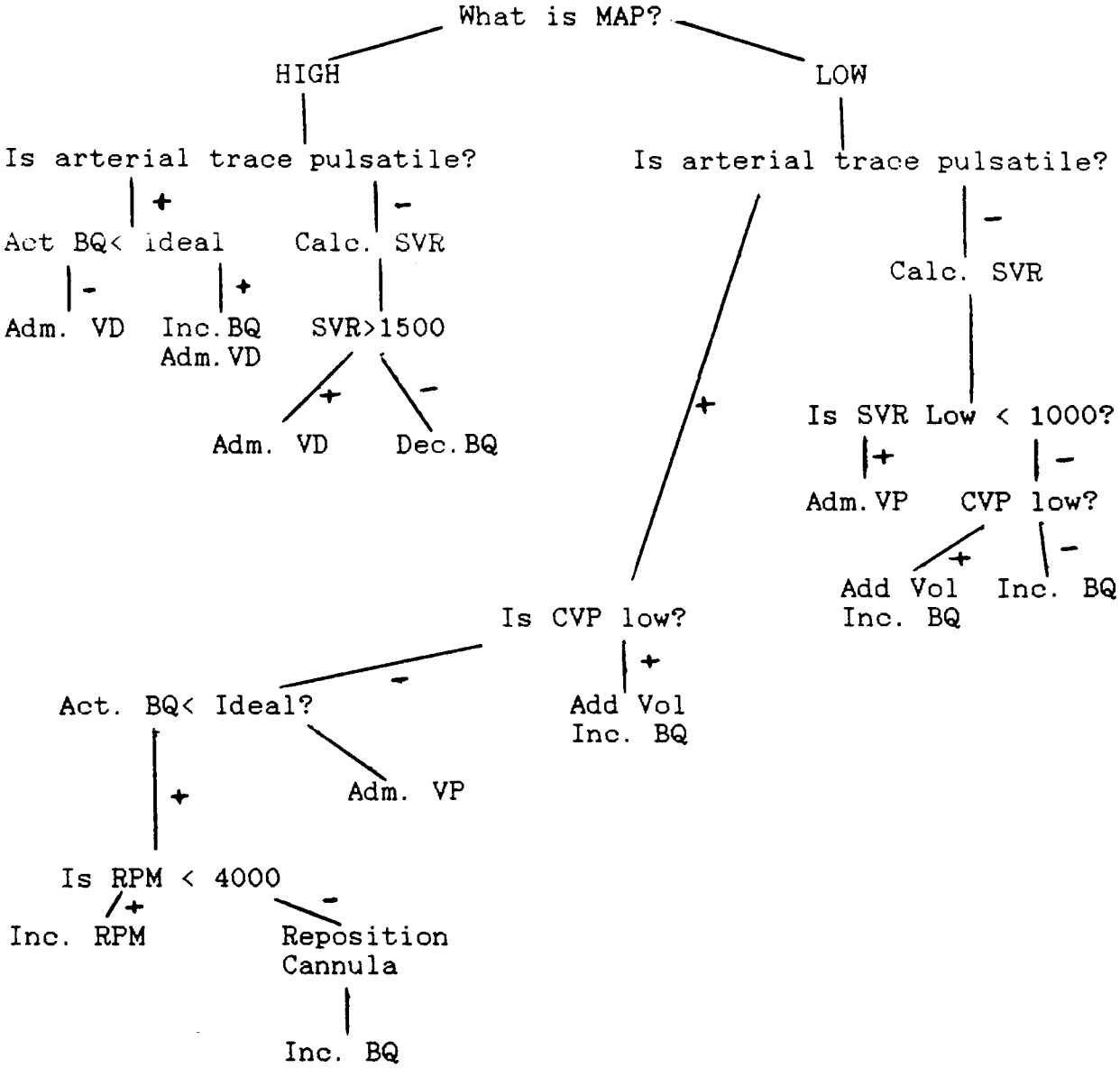

CPS VOLUME STATUS
Volume (liters)
5
4
3
2
1
0
1
2
3
4
5
6
7
8
9
10
Case Number
Urine Output
Volume In

FIGURE 3

DECISION MAKING ALGORITHMS FOR BARD CPS USERS



KEY

Act. BQ< Ideal= Is actual blood flow less tha ideal?

Adm. VD= Administer vasodilator (Nitroglycerin, Nipride)

Adm. VP= Administer vasopresser (Neosynephrine)

Add Vol= Administer volume (Plasmalyte A, Hespan, Plasmanate)

Inc. BQ= Increase Blood Flow

Dec. BQ= Decrease Blood Flow

Calc SVR= Calculate Systemic Vascular Resistance