Henry Ford Hospital Medical Journal

Volume 34 | Number 2

Article 5

6-1986

Hemodynamic Response to Fluid Challenge: A Means of Assessing Volume Status in the Critically III

H. Mathilda Horst

Farouck N. Obeid

Follow this and additional works at: https://scholarlycommons.henryford.com/hfhmedjournal Part of the Life Sciences Commons, Medical Specialties Commons, and the Public Health Commons

Recommended Citation

Horst, H. Mathilda and Obeid, Farouck N. (1986) "Hemodynamic Response to Fluid Challenge: A Means of Assessing Volume Status in the Critically III," *Henry Ford Hospital Medical Journal* : Vol. 34 : No. 2, 90-94. Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol34/iss2/5

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons.

Hemodynamic Response to Fluid Challenge: A Means of Assessing Volume Status in the Critically Ill

H. Mathilda Horst, MD,* and Farouck N. Obeid, MD*

Cardiovascular and oxygen transport variables were studied during fluid challenges in 50 critically ill patients. The results show three distinct patterns of response: hypovolemic, normovolemic, and hypervolemic. Colloid fluid challenge is a rapid, effective diagnostic modality for determination of cardiovascular function in the surgical critical care patient. (Henry Ford Hosp Med J 1986;34:90-4)

E valuation of intravascular volume status in the critically ill is difficult (1). These patients may have already undergone vigorous fluid resuscitation for shock or dehydration and also may have varying degrees of impairment of cardiac, pulmonary, and renal function. These factors may cause the basic parameters of volume status (blood pressure, pulse, and urine output) as well as some invasive measurements (central venous and pulmonary capillary wedge pressures) to be misleading (2). The intravascular volume in critically ill patients may be normal, increased, or decreased. Appropriate fluid therapy, which may be crucial for sustaining organ function and ultimately survival, depends on rapid and accurate assessment of the intravascular volume status in these patients (3).

One method of evaluating intravascular volume utilizes measurement of hemodynamic response to a fluid challenge. A fluid challenge is the infusion of a defined amount of fluid over a defined period of time. The response to the infusion may be monitored by clinical parameters such as blood pressure, pulse rate, or urine output or by invasive measurements such as central venous pressure or pulmonary wedge pressure. The volume status of the patient in response to the fluid challenge may then be assessed by the criteria of Weil (3) or Gill and Long (4) (Table 1).

The present study was performed to determine the physiological patterns of response to rapid infusion of 250 mL of a colloid solution (Plasmanate, Cutter Biologicals) in critically ill surgical patients.

Materials and Methods

A 250 mL colloid fluid challenge was performed in 50 patients in the Surgical Intensive Care Unit at Henry Ford Hospital. All patients were monitored with pulmonary artery catheters and arterial lines. No blood transfusions and respiratory or medication changes were implemented during the study period. Baseline cardiovascular and oxygen transport variables were obtained (T_0). The 250 mL colloid solution was infused rapidly over five minutes. Cardiovascular and oxygen transport parameters were measured immediately after the infusion (T_5) and repeated after five minutes (T_{10}). The following parameters were measured in each patient at T_0 , T_5 , and T_{10} : blood pressure, heart rate, pulmonary artery pressure, pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), cardiac output (CO), hemoglobin, inspired oxygen concentration, mixed venous and arterial gases, and body surface area. The following variables were devised using standard formula: mean arterial pressure (MAP), mean pulmonary artery pressure (MPAP), stroke volume and index, cardiac index, right and left stroke work index (RVSWI and LVSWI), systemic vascular resistance, pulmonary vascular resistance, arterial venous oxygen content difference, oxygen delivery, oxygen consumption, and shunt. Hemodynamic patterns in response to the fluid challenge were identified based on a 10% change from baseline values.

Results

Ages of the 50 study group patients ranged from 16 to 84 years with an average of 47 years. There were 40 males and ten females. Of the 50 patients, 42 patients (84%) had undergone surgical procedures. Thirty patients sustained blunt or penetrating trauma. Thirteen patients were septic from peritonitis, soft tissue infections, or meningitis. The remaining seven patients included three patients with carcinoma, two patients who had undergone craniotomy, one patient with hemorrhagic pancreatitis, and one patient with a gastrointestinal bleed. The severity of illness was apparent from the mortality; 20 of the 50 patients (40%) died during their hospitalization.

Baseline (T_0) cardiovascular and oxygen transport data for the 50 patients are summarized in Tables 2 and 3. A wide range of values existed for each parameter. Only three (6%) of the 50 patients experienced shock with an arterial pressure less than 100 mm Hg: one patient with a fractured femur from blunt trauma and a hemoglobin of 6.3 g%, one with a gunshot wound to the abdomen requiring a pancreatico-duodenectomy with a hemoglobin of 11.4 g%, and another with peritonitis with a hemoglobin of 9.4 g%.

With the fluid challenge in these 50 patients, a slight increase occurred in the mean values for MAP, MPAP, CVP, PCWP, and CO while a slight decrease occurred in heart rate (Table 2). No change occurred in the calculated parameters (Table 3). Al-

Submitted for publication: May 16, 1986.

Accepted for publication: June 2, 1986.

^{*}Department of Trauma Surgery, Henry Ford Hospital.

Address correspondence to Dr Horst, Department of Trauma Surgery, Henry Ford Hospir tal, 2799 W Grand Blvd, Detroit, MI 48202.

ıg

n, mixed ollowing

n arterial (MPAP), ft stroke sistance, n content nd shunt. nge were s.

o 84 years s and ten ndergone penetratnitis, soft n patients who had ic pancrene severity 50 patients

lata for the e range of the 50 pas than 100 unt trauma ound to the th a hemoth a hemo-

ht increase PCWP, and able 2). No ble 3). Al-

nry Ford Hospi-

tus—Horst et al

Table 1 **Standard Volume Challenges**

Gill and Long's Volume Challenge Rapid Infusion 200 mL Colloid (15 minutes)			Weil's Fluid Challenge	
CVP*	HEART RATE	MEANING	CVP < 8 Give 200 mL/10 min	Rise in CVP = 5 Stop
Low or decrease	Increase	Hypovolemia	CVP < 14 Give 100 mL/10 min	Rise in CVP 2-5 Wait 10 minutes Repeat CVP
Low	Same	Correcting hypovolemia		
Increase to normal	Same or decrease	Correcting hypovolemia		
Increase to normal	Increase	Hypervolemia, cardiac failure	$CVP \ge 14$ Give 50 mL/10 min	Rise in CVP < 2 Repeat challenge
Increase	Same	Hypervolemia, cardiac failure	PCWP [†] < 12 Give 200 mL/10 min	Rise in PCWP > 7 Stop
Increase	Increase	Hypervolemia, cardiac failure, cardiac tamponade	PCWP < 16 Give 100 mL/10 min	Rise in PCWP 3-7 Wait 10 minutes Repeat PCWP
			PCWP > 16 Give 50 mL/10 min	Rise in PCWP ≤ 3 Repeat challenge

*CVP = central venous pressure. †PCWP = pulmonary capillary wedge pressure.

	Table 2		
Hemodynamic Parameters	Measured	During Fluid	Challenge

Measured Parameters	Total Group (50 Patients) $M \pm SD^*$	Hypovolemic (30 Patients) M ± SD	Normovolemic (15 Patients) M ± SD	Hypervolemic (5 Patients) M ± SD
T ₀ †				
Mean arterial pressure	96 ± 18	97 ± 18	97 ± 18	88 ± 19
Heart rate	105 ± 21	107 ± 20	106 ± 26	95 ± 7
Mean pulmonary artery pressure	20 ± 8	20 ± 9	20 ± 6	19 ± 7
Central venous pressure	7.7 ± 3.9	7.6 ± 3.5	7.5 ± 4	9 ± 6
Pulmonary capillary wedge pressure	9.3 ± 6	9.3 ± 6.6	9 ± 6	10 ± 6
Cardiac output	7 ± 2	7 ± 2	8 ± 3	9 ± 4.5
Hemoglobin	11 ± 2.4	10.7 ± 2	11.4 ± 3	12.1 ± 2.7
Inspired oxygen concentration	0.47 ± 0.19	0.50 ± 0.22	0.42 ± 0.9	0.4 ± 0.1
Mixed venous saturation	0.69 ± 0.91	0.69 ± 0.8	0.68 ± 0.1	0.69 ± 0.1
Arterial saturation	0.97 ± 0.2	0.97 ± 0.3	0.97 ± 0.3	0.97 ± 0.1
Γ ₅ ‡				
Mean arterial pressure	99 ± 16	98 ± 16	101 ± 16	97 ± 21
Heart rate	102 ± 19	103 ± 17	105 ± 25	92 ± 7
Mean pulmonary artery pressure	23 ± 9	23 ± 9	22 ± 7	26 ± 11
Central venous pressure	9.5 ± 5	9.4 ± 6	9 ± 5	12 ± 6
Pulmonary capillary wedge pressure	12 ± 6	12 ± 6	11 ± 4	14 ± 8
Cardiac output	8 ± 2.6	8 ± 2.5	8.5 ± 3	7 ± 3
Mixed venous saturation	0.70 ± 0.8	0.70 ± 0.6	0.70 ± 0.1	0.66 ± 0.1
Arterial saturation	0.97 ± 0.2	0.97 ± 0.2	0.97 ± 0.2	0.98 ± 0.1
Γ ₁₀ §				
Mean arterial pressure	99 ± 19	100 ± 21	99 ± 16	89 ± 19
Heart rate	103 ± 19	103 ± 17	106 ± 24	98 ± 13
Mean pulmonary arterial pressure	23 ± 9	24 ± 11	22 ± 6	25 ± 10
Central venous pressure	9 ± 5	8.5 ± 5	9 ± 5	13 ± 7
Pulmonary capillary wedge pressure	12 ± 5	11 ± 5	12 ± 4	13 ± 7
Cardiac output	8 ± 2.6	8 ± 2.4	8.8 ± 3	7 ± 2.4
Mixed venous saturation	0.69 ± 0.8	0.70 ± 0.7	0.69 ± 0.94	0.66 ± 0.1
Arterial saturation	0.97 ± 0.2	0.97 ± 0.3	0.96 ± 0.3	0.98 ± 0.6

*Mean \pm standard deviation.

[†]Baseline.

‡Postinfusion.

§Five minutes postinfusion.

Assessing Volume Status—Horst et al 91

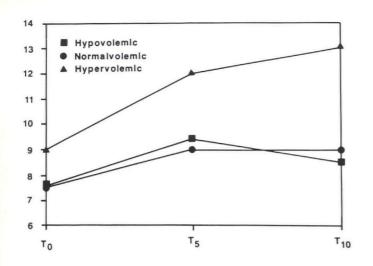


Fig 1—Mean central venous pressure (mm Hg) plotted over time for three response patterns to fluid challenge.

though changes in parameters were observed with each individual patient, comparison of the mean values of all patients showed little change because of the wide range of values for each parameter.

Three response patterns were seen when CVP, PCWP, CO, LVSWI, and oxygen delivery were graphed or plotted against time (Figs 1 through 5). The cardiac output either increased, decreased, or remained the same. The mean values (Tables 2 and 3) of 30 (60%) of the 50 study group patients for CVP, PCWP, CO, LVSWI, and oxygen delivery in response to fluid challenge are represented by the squares in Figs 1 through 5. The response pattern of cardiac output, left ventricular stroke work index, and oxygen delivery seen in these patients was termed hypovolemic, ie, fluid should improve the cardiovascular and oxygen-dependent variables.

The response patterns seen in 15 (30%) of the 50 study group patients are represented by the circles in Figs 1 through 5. Baseline values for blood pressure, pulse, hemoglobin, and cardiac output were not significantly different from those in the hypo-



volemic j sponse to shows litt dex, and euvolemi Values challenge

fluid chal Figs 1 th since can with flui sponse to

The in

cess in (suscitati either fr fluid los overhyd or may tem. Ur in these occur if Adva and pul measur rameter to docu measur pressur for the The (4) foll to dete cedure terms mance affect

Henry Fo

Table 3	
Hemodynamic Parameters Calculated During Flui	d Challenge

Calculated Parameters	Total Group (50 Patients) M ± SD*	Hypovolemic (30 Patients) M ± SD	Normovolemic (15 Patients) M ± SD	Hypervolemic (5 Patients) M ± SD
T_0^{\dagger}				
Stroke volume index	42 ± 14	35 ± 12	43 ± 14	52 ± 22
Cardiac index	4.2 ± 1	3.6 ± 1	4.4 ± 1.3	5 ± 2
Right ventricular stroke work index	8 ± 4	6 ± 4.5	7 ± 4	6.7 ± 3
Left ventricular stroke work index	50 ± 20	42 ± 18	53 ± 20	55 ± 23
Systemic vascular resistance	1006 ± 610	1171 ± 651	909 ± 253	799 ± 282
Pulmonary vascular resistance	129 ± 88	146 ± 106	121 ± 88	98 ± 71
Oxygen content difference	4.4 ± 1.1	4 ± 0.9	4.5 ± 1.1	5.3 ± 2.5
Oxygen delivery index	651 ± 211	535 ± 166	692 ± 202	860 ± 484
Shunt	0.17 ± 0.10	0.17 ± 0.10	0.16 ± 0.01	0.17 ± 0.15
T ₅ ‡				
Stroke volume index	43 ± 14	43 ± 15	44 ± 13	44 ± 15
Cardiac index	4.3 ± 1.3	4.3 ± 1.3	4.4 ± 1.3	4 ± 1
Right ventricular stroke work index	7.8 ± 4	8 ± 4	7.9 ± 4	8 ± 5
Left ventricular stroke work index	52 ± 20	51 ± 21	54 ± 19	49 ± 16
Systemic vascular resistance	970 ± 407	974 ± 472	937 ± 272	1041 ± 388
Pulmonary vascular resistance	125 ± 83	129 ± 85	107 ± 61	152 ± 124
Oxygen content difference	4.4 ± 1.2	4.3 ± 1	4.3 ± 1.2	5.5 ± 2
Oxygen delivery index	655 ± 213	632 ± 195	687 ± 212	696 ± 335
Shunt	0.17 ± 0.10	0.17 ± 0.1	0.17 ± 0.01	0.13 ± 0.10
T ₁₀ §				
Stroke volume index	42 ± 14	41 ± 14	44 ± 13	41 ± 12
Cardiac index	4.2 ± 1.1	4.1 ± 1.1	4.6 ± 1.4	4 ± 1
Right ventricular stroke work index	8 ± 4.5	8 ± 4.5	8 ± 4	7 ± 3
Left ventricular stroke work index	50 ± 20	50 ± 20	54 ± 20	43 ± 18
Systemic vascular resistance	1006 ± 315	1071 ± 750	906 ± 299	917 ± 280
Pulmonary vascular resistance	129 ± 97	141 ± 96	99 ± 50	147 ± 113
Oxygen content difference	4.4 ± 1.2	4 ± 0.9	4 ± 1	5.4 ± 2
Oxygen delivery index	651 ± 240	620 ± 189	704 ± 220	682 ± 313
Shunt	0.18 ± 0.1	0.19 ± 0.10	0.17 ± 0.01	0.14 ± 0.11

*Mean ± standard deviation

†Baseline.

‡Postinfusion.

§Five minutes postinfusion.

patients for each

WP, CO, d against ased, dees 2 and c, PCWP, hallenge response dex, and volemic, n-depen-

dy group 5. Based cardiac he hypo-

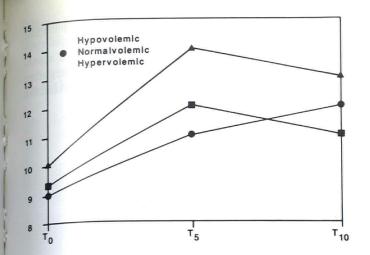


Fig 2—Mean pulmonary capillary wedge pressure (mm Hg) plotted over time for the three response patterns to fluid challenge.

volemic patients (Table 2). However, the cardiovascular response to fluid challenge in these 15 patients (Figs 1 through 5) shows little change in cardiac output, left ventricular stroke index, and oxygen delivery (Table 3). These patients were termed euvolemic or normovolemic.

Values for the remaining five patients who underwent fluid challenge are shown in Tables 1 and 2. The response pattern to fluid challenge in these patients is represented by the triangles in Figs 1 through 5. These patients were termed hypervolemic since cardiovascular and oxygen delivery patterns decreased with fluid challenge, indicating a failing cardiovascular response to fluids.

Discussion

The intravascular volume status is especially difficult to access in critically ill surgical patients because of previous resuscitation and fluid loss. These patients may be hypovolemic either from their disease, insufficient resuscitation, excessive fluid loss, or hypothermia. However, the surgical patient may be overhydrated, ie, hypervolemic from aggressive resuscitation, or may appear overloaded due to a failing cardiovascular system. Unfortunately, the blood pressure, pulse, and urine output in these patients may be misleading (2). Errors in therapy may occur if treatment is based on these measurements.

Advances in invasive monitoring with central venous lines and pulmonary artery catheters have provided invasive pressure measurements. Clinical observation of the trends of these parameters over time as in response to therapy allows the physician to document cardiovascular function and fluid status. These measurements are used as a rough estimate of preload of filling pressure for the heart: the CVP for the right ventricle and PCWP for the left ventricle (5,6).

The fluid challenge proposed by Weil (3) and Gill and Long (4) follows the response of the CVP and PCWP measurements to determine intravascular volume status (Table 1). This procedure assumes that intravascular volume can be defined in terms of the relationship between preload and cardiac performance (Frank Starling law) and that changing the preload will affect cardiac performance (7).

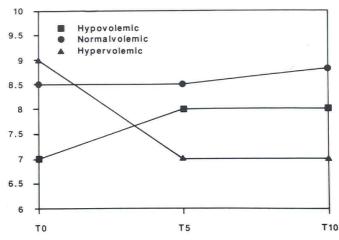


Fig 3—Response to cardiac output (L/min) during rapid fluid challenge.

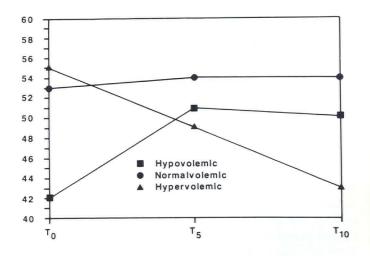


Fig 4—Three response patterns of mean left ventricular stroke work index $(g \cdot m/m^2)$ during fluid challenge.

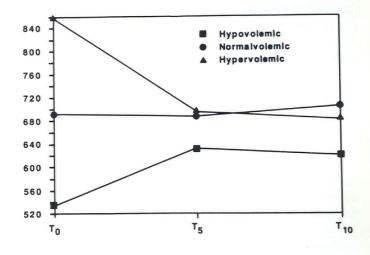


Fig 5—Oxygen delivery index $(mL/min/m^2)$ plotted over time during rapid fluid challenge.

Although changes in CVP and PCWP have been used to define preload, recent evidence suggests that this assumption is not always reliable (3-5). Both pressures are affected by cardiac function, pulmonary disease, body habitus, equipment problems, and increasing intrathoracic pressures (5,6). If these pressures do not adequately reflect preload, a different parameter or parameters must be defined to follow rapid fluid administration. In this study the actual volume status of the 50 patients was unknown prior to fluid challenge. Based on a 10% change in parameters, we observed three cardiovascular response patterns to rapid fluid challenge. Cardiac output and oxygen delivery in the 30 patients represented by the squares in Figs 1 through 5 did improve with fluids. The patients responded to the fluid challenge with increases in the cardiovascular and oxygen transport variables over a five-minute period which returned toward baseline levels by ten minutes. In this manner the patients behaved as if they were hypovolemic, and improvement in oxygen delivery could be expected with increasing fluid administration. The five patients represented by the circles in Figs 1 through 5 responded as if they were hypervolemic, ie, fluid overloaded, or had a failing cardiovascular system because the cardiac output and oxygen delivery decreased with the fluid challenge. In this group of patients ionotropic or vasodilator therapy or diuresis may help to improve oxygen transport. The response pattern represented by the triangles in Figs 1 through 5 is a minimal response group of 15 patients. Ionotropic agents may be useful in this group as well to improve cardiac output and oxygen delivery.

This method of fluid challenge differs from those described by Weil and Gill and Long (Table 1) in that it allows definition of the cardiovascular response and oxygen transport function rather than relying strictly on increases in CVP or PCWP. Theoretically, an actual observance of cardiovascular response and oxygen transport function gives more complete information about the patient's physiological status and therefore should be more reliable. With this additional information, therapy can be rapidly individualized.

In this study we utilized rapid fluid challenge and followed the response by monitoring changes in the cardiovascular and oxygen transport variables. Three response patterns were found: 1) a hypovolemic response pattern in which the patients responded with increased cardiovascular and oxygen transport variables over five minutes which returned toward baseline levels by ten minutes; 2) a normovolemic response in which variables were relatively unchanged; and 3) a pump failure response where both oxygen transport and cardiovascular parameters decreased with

the fluid challenge. We found this method of fluid challenge useful in our patient population because it provided a rapid evaluation of cardiovascular function and defined the fluid status. The additional information obtained allowed us to categorize our patients into three groups for rapid therapy and avoid the hazard of relying on a single pressure measurement.

Notation

Parameters	Units
Mean arterial pressure	mm Hg
Heart rate	beats/min
Mean pulmonary artery pressure	mm Hg
Central venous pressure	mm Hg
Pulmonary capillary wedge pressure	mm Hg
Cardiac output	L/min
Hemoglobin	g/100 mL
Inspired oxygen concentration	%
Mixed venous saturation	%
Arterial saturation	%
Stroke volume index	mL/m ²
Cardiac index	L/min/m ²
Right ventricular stroke work index	g·m/m ²
Left ventricular stroke work index	$g \cdot m/m^2$
Systemic vascular resistance	dyne·sec/cm·m ²
Pulmonary vascular resistance	dyne·sec/cm·m ²
Oxygen content difference	mL/100 mL
Oxygen delivery index	mL/min/m ²
Shunt	%

References

1. Shoemaker WC, Montgomery ES, Kaplan E, et al. Physiologic patterns in surviving and nonsurviving shock patients. Arch Surg 1973;106:630-6.

2. Lazrove S, Waxman K, Shippy C, Shoemaker WC. Hemodynamic, blood volume, and oxygen transport responses to albumin and hydroxyethyl starch infusions in critically ill postoperative patients. Crit Care Med 1980;8:302-6.

3. Weil MH. Patient evaluation, "vital signs," and initial care. Critical care: State of the art. Fullerton: Society of Critical Care Medicine, 1980;1(A):1.

4. Gill W, Long WB. Shock trauma manual. Baltimore: Williams & Wilkins Co, Vol 19, 1979.

5. Shoemaker WC, Thompson WL, Holbrook PR. Textbook of critical care. Philadelphia: W.B. Saunders Company, 1984.

6. Forrester JS, Diamond G, Chatterjee K, Swan HJC. Medical therapy of acute myocardial infarction by application of hemodynamic subsets. N Engl J Med 1976;295:1356-62.

7. Manny J, Grindlinger GA, Dennis RC, Weisel RD, Hechtman HB. Myocardial performance curves as guide to volume therapy. Surg Gynecol Obstet 1979;149:863-73.

Technic to we for nutriti them are weight pe cep skinfo mid-uppe of lean bo surement a better in Curren dards inc errors, a sites and for a reli

sive care

of anthro

body me tainable A rec throporr (1,2,4-1 for nutr conduct Hospita

In a sureme either r

The Fa

Judith J