HAEMODYNAMIC CHANGES DUE TO CLAMPING OF THE ABDOMINAL AORTA

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SERIOUS PROBLEMS arise frequently during anaesthesia for major vascular surgery associated with clamping of the aorta. Haemodynamic changes at the moment of recirculation are readily observed and have been the subject of numerous publications.¹⁻⁴ The hypotension, at times severe, that follows the unclamping of the aorta is not without danger for these arteriosclerotic and hypertensive patients. It is obvious that hypotension, even of short duration, can reduce cerebral, coronary or renal perfusion and jeopardize an otherwise successful operation.

While the period of unclamping has been thoroughly examined, the events associated with clamping have not received much attention except for Perry's animal experiments, which showed decrease in cardiac output.⁵ The proximal occlusion of the aorta during operation for thoracic aneurysm can induce alarming increases in blood pressure and experienced anaesthetists will look for signs of left ventricular failure. The use of nitroprusside is often mandatory to relieve the acute afterload while the surgeon prepares a temporary bypass. It is not uncommon to see pulsus alternans, indicating ventricular failure probably due to discrepancy in regional perfusion of an already diseased myocardium.

The clamping of the distal aorta also induces haemodynamic changes, the most obvious response again being the increase in systolic pressure due to an afterload which could be of clinical significance for these patients. With that in mind we decided to investigate the haemodynamic changes due to cross-clamping of the abdominal aorta. We also attempted to evaluate renal function during and following operation and to correlate changes, if any, to haemodynamic alterations. The latter will be the subject of a future publication.

MATERIAL AND METHODOLOGY

Eighteen patients requiring surgery of the abdominal aorta were included in this study. None presented clinical evidence of cardiac insufficiency or dysrhythmia before operation. They were distributed at random in two groups involving nine Leriche's and nine aneurysms of the infrarenal aorta. Anthropometric data and type of surgery are summarized in Table I.

After premedication with diazepam 5 mg intramuscularly, induction of anaesthesia was carried out with thiopentone 5 mg/kg, and pancuronium 0.15 mg/kg for tracheal intubation and muscular relaxation. Maintenance was achieved with

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TABLE I
ANTHROPOMETRIC DATA AND TYPE OF SURGERY

No.	Diagnosis*	Age	Sex	Height (M)	Weight (Kg)	BSA (M ²)
1	L	70	M	1.66	66	1.73
2	A	78	M	1.71	64	1.75
3	L	44	F	1.60	40	1.37
4	L	57	M	1.73	63	1.75
5	A	69	M	1.75	58	1.70
6	L	65	F	1.60	64	1.67
7	A	68	M	1.68	70	1.79
8	A	65	M	1.68	47	1.52
9	A	70	M	1.60	52	1.52
10	A	68	M	1.63	56	1.59
11	L	53	F	1.63	55	1.58
12	L	50	M	1.70	73	1.84
13	L	52	F	1.58	67	1.68
14	A	61	M	1.68	83	1.94
15	A	56	M	1.78	68	1.85
16	A	76	M	1.65	73	1.79
17	L	70	F	1.53	46	1.40
18	L	52	M	1.68	86	1.96

*L = Leriche: age 57 (SD $\pm$ 9.2). P < 0.02.

A = Aneurysm: age 68 (SD $\pm$ 6.7). P < 0.02.

nitrous-oxygen and fentanyl according to analgesic requirements. Droperidol 0.1 mg/kg was also given before the surgical incision.

The left radial artery was cannulated under general anaesthesia after testing for adequacy of cubital artery compensatory function. A catheter was placed in the right atrium through the internal jugular vein; its position was ascertained by threading to the right ventricle and withdrawal into the atrium, the characteristic pressure signal being displayed on an oscilloscope.

All patients were placed on controlled ventilation with a tidal volume of 10 ml/kg at a frequency of 12/min. A control arterial blood gas analysis was performed routinely at the start of the procedure. Necessary adjustments to maintain PaCO₂ and PaO₂ within normal limits were rapidly applied. Fluid intake was adjusted to 20 ml/kg/hr before aortic clamping, half the volume consisting of 5 per cent glucose in water and the other half 5 per cent glucose with 0.45 per cent NaCl. A rather liberal fluid intake was administered prior to clamping in order to ensure that hypovolaemia would not explain the haemodynamic changes. Furthermore, in patients with over twelve hours of water restriction, it is important to prevent an augmentation in serum osmolality with subsequent release of antidiuretic hormone. Following occlusion of the aorta, fluid intake was reduced to 6 ml/kg/hr. Blood loss was replaced as accurately as possible.

Haemodynamic data (E.K.G., arterial and right atrial pressure curves and dilution curve) were inscribed on a polygraph recorder (Mingograph EMT 81 - Elema-Schonander). Pressures were continuously recorded from the amplified signals of Statham P23dB transducers. Mechanical and electrical calibrations were obtained and checked regularly during the investigation.

Cardiac output was determined by injection of indocyanine green dye into the right atrium and detection of the resulting dye dilution curve in the arterial blood by a Waters D-400 densitometer. The surface under the curve was calculated by

computer from ordinates taken at 1-second intervals. The recirculation was eliminated by extrapolating the exponential portion of the curve using a least square method. A special program was written which permitted us to obtain results as well as statistics from the computer thus eliminating human bias (varian 73). Derived variables were obtained as follows:

$$\text{SVR} = \frac{\overline{\text{AP}} - \overline{\text{RAP}}}{\text{C.O.}} \times 79.92 \text{ dynes.sec.cm}^{-5}$$

$$\text{SV} = \frac{\text{C.O.}}{\text{HR}} = \text{ml}$$

$$\text{C.I.} = \text{C.O.}/\text{m}^2$$

$$\text{LVSWI} = \overline{\text{AP}} (\text{SI}) 1.36 \times 10^{-2} = \text{g.m.m}^{-2}$$

$$\text{PTI} = \text{HR} \times \text{syst. AP} \times 10^{-3}$$

Where SVR is the systemic vascular resistance expressed in dynes.sec.cm⁻⁵; SV is the stroke volume in ml; LVSWI the left ventricular stroke-work index in gram-metre and PTI, the pressure time index.

In both groups the study was conducted as follows: pressure signals and standard Lead II of the EKG were recorded continuously; three control cardiac output determinations were obtained just before the clamping of the aorta and every two minutes for the first ten minutes after aortic occlusion. The same data were also collected five minutes before unclamping; the three last cardiac output curves being obtained at the end of the operation after stabilization of vital signs. Statistical analysis of the data applied two-tailed T-tests for comparison within groups and unpaired T-tests for comparison between groups. Unless otherwise mentioned, only statistically significant variations will be presented and discussed.

RESULTS

The results are tabulated using the mean of the readings in all patients with the standard error in brackets. Significant variations within the group ($p < 0.05$) as related to the control are followed by an asterisk and the statistically valid variations between the two groups ($p < 0.05$) are indicated by a cross.

Tables II to VIII show the variations of the measured parameters in the 18 patients. In the first ten minutes after clamping it can be seen that the heart rate, right atrial pressure, systolic work of the left ventricle, and the pressure time index do not differ from the control values at zero time (Figures 1-2). On the other hand, immediately after two minutes of aortic occlusion there is a significant increase in systolic arterial pressure and in systemic vascular resistance and a significant decrease in cardiac index (Tables IV, V, VI), at the expense of the stroke volume (Table VII). The maximum changes take place after eight minutes of clamping, at which time the systolic pressure has increased by 13 per cent, the vascular resistance by 37 per cent and the cardiac output has decreased by 18 per cent. After 50 minutes of aortic occlusion, while the systolic and right atrial pressures

TABLE II
CHANGES IN RIGHT ATRIAL PRESSURE AND
HEART RATE DUE TO AORTIC OCCLUSION
(MEANS $\pm$ SE) N = 18

Time	RAP Torr	HR
0	6.6 (0.8)	81 (3.7)
2'	6.2 (0.9)	82 (3.5)
4'	6.2 (0.8)	81 (3.7)
6'	6.2 (0.8)	80 (3.9)
8'	6.2 (0.8)	82 (4.0)
10'	6.1 (0.8)	82 (4.4)

Patients were studied before (T = 0) and after clamping (T = 2' . . .).

TABLE III
CHANGES IN LEFT VENTRICULAR SYSTOLIC WORK
INDEX BEFORE (T = 0) AND AFTER AORTIC
OCCLUSION (MEANS $\pm$ SE) N = 18

Time	LVSWI (g.m.m ⁻²)
0	50.1 (3.0)
2'	49.0 (3.7)
4'	48.6 (3.3)
6'	47.9 (3.8)
8'	44.7 (3.2)*
10'	45.0 (3.8)

*P < 0.02.

Patients were studied before (T = 0) and after aortic occlusion (T = 2' . . .).

TABLE IV
SYSTOLIC ARTERIAL PRESSURE CHANGES DUE TO
AORTIC OCCLUSION (MEANS $\pm$ SE) N = 18

Time	AP Torr
0	155 (5.7)
2'	171.1 (5.5)*
4'	178.6 (5.6)*
6'	173.4 (6.3)*
8'	175.6 (6.7)*
10'	174.6 (6.4)*

Patients were studied before (T = 0) and every 2' after the clamping of infrarenal aorta (T = 2' . . .).

*P < 0.01.

and heart rate are not different from the controls, the cardiac index has dropped by 29 per cent and the resistance is augmented by 50 per cent (Table VIII).

Table IX compares the systolic arterial pressure changes between Leriche (N = 9) and aneurysm (N = 9). There are no significant variations between groups except after four minutes of clamping. There is a tendency for the systolic pressures to be higher in the aneurysm group, though this does not reach a significant level.

TABLE V
CHANGES IN SYSTEMIC VASCULAR RESISTANCE DUE
TO AORTIC OCCLUSION (MEANS $\pm$ SE) N = 18

Time	R (dynes.sec.cm ⁻⁵)
0	1989 (201)
2'	2392 (232)*
4'	2542 (223)*
6'	2500 (224)*
8'	2732 (266)*
10'	2663 (253)*

Patients were studied before (T = 0) and after aortic occlusion (T = 2' . . .).

*P < 0.01.

TABLE VI
CHANGES IN CARDIAC INDEX INDUCED BY CLAMPING
THE INFRARENAL AORTA (MEANS $\pm$ SE) N = 18

Time	C.I. (l/min/m ²)
0	2.78 (0.21)
2'	2.54 (0.20)*
4'	2.41 (0.18)†
6'	2.40 (0.18)†
8'	2.26 (0.17)†
10'	2.27 (0.17)†

*P < 0.05, †P < 0.01.

Patients were studied before (T = 0) and after aortic occlusion (T = 2' . . .).

TABLE VII
CHANGES IN STROKE INDEX DUE TO AORTIC
OCCLUSION (MEANS $\pm$ SE) N = 18

Time	S.I. (ml/min/m ²)
0	35.9 (2.3) control
2'	31.5 (2.4)*
4'	30.8 (2.4)*
6'	30.8 (2.4)*
8'	28.3 (2.2)*
10'	28.8 (2.4)*

*P < 0.01.

Patients were studied before (T = 0) and after aortic occlusion (T = 2' . . .).

The variations in cardiac and stroke indices are shown in Table X. The decrease following aortic occlusion is not significantly different in the Leriche group as compared to the aneurysm group. The same is noted in Table XI, which indicates that the increase in systemic vascular resistance, though slightly more in the aneurysm group, does not reach statistical significance.

The overall picture of haemodynamic changes before, during and after aortic

N = 18

FIGURE 1. Neither the heart rate nor the right atrial pressure are modified by clamping the infrarenal aorta.

TABLE VIII
 AORTIC CLAMPING: HAEMODYNAMIC MODIFICATIONS

Time	HR syst.min ⁻¹	RAP Torr	LVSW gm.m ⁻²	PTI HR $\times$ SAP $\times 10^{-3}$	SAP $\times 10^{-3}$ Torr	C.I. l.min ⁻¹ .m ⁻²	SVR dynes.sec.cm ⁻⁵
0	82	7	50.1	12.6	155	2.78	1989
2'	81	6	49.0	13.4	171*	2.54*	2392*
4'	80	6	48.6	13.7	178*	2.41*	2541*
6'	82	6	47.9	13.3	173*	2.40*	2500*
8'	82	6	44.7	13.8	175*	2.26*	2732*
10'	82	6	45.0	13.2	174*	2.27*	2662*
50'	81	6	35.7	13.0	161	1.95*	2996*
End	82	6	44.1	12.8	156	2.57	2142

*P < 0.05.

Patients were studied before and after clamping of aorta.

LEFT VENTRICLE SYSTOLIC WORK INDEX ($\pm$ SE) N=18

FIGURE 2. The only significant change in left ventricular systolic work is noted at the eighth minute after clamping the aorta. This absence of response to a 30 per cent increase in vascular resistance explains the reduction of cardiac output.

occlusion is given in Table XII. It shows that apart from changes due to clamping already mentioned, at the end of surgery and once the vital signs have been stabilised, the cardiac output, vascular resistance and systolic pressure are not significantly different from control values.

DISCUSSION

It should be emphasized that these patients were free from cardiac diseases, that anaesthesia was conducted without undue myocardial depressant doses that P_{aCO_2} was maintained at normal levels and fluids administered liberally (20 ml/kg/hr before clamping). Meaningful information can be drawn from these results. Under balanced anaesthesia with droperidol-fentanyl-pancuronium, just before aortic cross-clamping, right atrial and arterial pressures as well as heart rate were normal. Nevertheless, the systemic vascular resistance was elevated and the cardiac and stroke indices were at the lower limits of normality. The clamping of the aorta did induce alarming haemodynamic changes (Figures 3, 4, 5). The sudden increase in aortic impedance was followed by a 20 per cent reduction in

TABLE IX
 SYSTOLIC ARTERIAL PRESSURE CHANGES DUE TO CLAMPING OF AORTA.
 COMPARISONS OF PATIENTS WITH LERICHE SYNDROME (N = 9) AND
 ANEURYSM (N = 9). (MEANS $\pm$ SE)

Time	Leriche AP Torr		Aneurysm AP Torr
0	148.6 (5.2)	NS	160.2 (9.5)
2'	160.5 (4.9)*	NS	181.7 (8.8)*
4'	167.5 (5.7)*	+	189.8 (8.3)*
6'	166.5 (7.3)*	NS	180.2 (9.9)*
8'	164.8 (6.4)*	NS	186.3 (10)*
10'	166.7 (7.1)*	NS	182.2 (10)*

*P < 0.01 within each group.

+P < 0.05 between the two series.

Patients were studied before (T = 0) and every two minutes after aortic occlusion (T = 2' . . .).

TABLE X
 CARDIAC AND STROKE INDICES CHANGES DUE TO CLAMPING OF AORTA.
 COMPARISONS OF PATIENTS WITH LERICHE (N = 9) AND PATIENTS
 WITH ANEURYSM (N = 9)

Time	Leriche		Aneurysm	
	C.I. l/min/m ²	S.I. ml/min/m ²	C.I. l/min/m ²	S.I. ml/min/m ²
0	2.77	35	2.79	36.7
2'	2.49	29†	2.59	33.5†
4'	2.49	31*	2.34†	30.6†
6'	2.41*	29†	2.39*	32.0*
8'	2.27†	27†	2.25*	28.5†
10'	2.27†	27†	2.26*	28.7†

There is no significant difference between the two series (+) while a significant variation is shown within each group (*P < 0.05, †P < 0.01).

Patients were studied before (T = 0) and after aortic occlusion (T = 2' . . .).

cardiac output at the expense of the stroke volume. The cardiac performance deteriorated because the derived left ventricular work did not change, though the latter could be questioned because the left ventricular filling pressure was not used in its determination. Adjustment does not appear to occur with time; on the contrary, owing to blood losses, evaporation and oozing, all difficult to appraise, and in spite of adequate replacement, after 50 minutes of aortic clamping the cardiac output is even lower. Five minutes before unclamping, right atrial and arterial pressures being at control values, heart rate at 81, cardiac index was lower than 2 litres per minute and vascular resistances almost reached 3000 dynes.

It was not possible to document a significant difference in the behaviour of patients with chronic obstruction of blood flow to lower extremities (Leriche) and those with a somewhat more acute disease (aneurysm). Nevertheless, it can be seen that the former have a tendency to show less marked haemodynamic changes. As the patients with aneurysm are older, one can surmise that they had a certain degree of iliac obstruction and that younger subjects with traumatic aneurysm would show a larger increase in resistance following aortic cross-clamping.

TABLE XI
VASCULAR RESISTANCE CHANGES DUE TO CLAMPING OF AORTA
OF PATIENTS WITH LERICHE (N = 9) AND ANEURYSM (N = 9)

Time	Leriche	Aneurysm
	SVR (dynes.sec.cm ⁻⁵)	SVR (dynes.sec.cm ⁻⁵)
0	2064 (363)	1915 (196)
2'	2364 (384)*	2421 (286)†
4'	2349 (337)*	2733 (296)†
6'	2430 (329)†	2570 (322)†
8'	2497 (284)†	2996 (464)†
10'	2564 (352)†	2762 (382)†

*P < 0.05, †P < 0.01.

Patients were studied before (T = 0) and after aortic occlusion (T = 2' . . .).

TABLE XII
HAEMODYNAMIC CHANGES BEFORE, DURING AND AFTER CLAMPING
OF AORTA (N = 18)

	HR	SAP Torr	C.I. l/min/m ²	SVR dynes.sec.cm ⁻⁵	RAP Torr
Control	82 (3.2)	155 (5.7)	2.78 (0.21)	1989 (201)	7 (0.8)
During clamping	81 (1.8)	174* (6.1)	2.37* (0.18)	2565* (239)	6 (0.8)
End of surgery	82 (1.9)	156 (5.4)	2.57 (0.17)	2142 (220)	6 (0.8)

*P < 0.05.

It is obvious that an increase in afterload can reduce the cardiac output. Nevertheless, in physiological conditions there are mechanisms which can maintain the stroke volume in spite of this load. First, the Starling mechanism. The increase in afterload leads to an increase in the amount of blood remaining in the ventricle at the end of the systole. This means that the next end diastolic volume will be larger, thence an elongation of the fibre will take place. Following this elongation, the mechanism described by Patterson, Piper and Starling will be put in action and the stroke volume will be maintained.⁶

A second mechanism can be called upon to maintain the stroke volume against the larger afterload; it is the positive inotropic effect due to systolic pressure increase.⁷ This reflex was demonstrated by Anrep in 1912, and Starling believed it to be the result of the enhancement of coronary perfusion by the increased aortic pressure.⁸ Nearly half a century later, Sarnoff⁹ demonstrated that the Anrep reflex did not call upon an increase in coronary flow, thus confirming the results of Rosenblueth and Taquini.^{10,11} For these authors the increase in activity of the myocardium induces an enhancement of contractility after a few beats; the ventricular work is augmented and the rate of rise of ventricular pressure is more rapid. This takes place without any change in end-diastolic pressure and thus without the intervention of the Starling mechanism. For Sarnoff this homeometric auto-regulation depends on the level of circulating catecholamines. This means that at

FIGURE 3. The clamping of aorta increases systolic arterial pressure by 15 per cent.

a high level of circulating catecholamines the heart can maintain a constant stroke volume against an increased afterload, without any change in end-diastolic pressure. On the other hand, a diseased myocardium with left ventricular end-diastolic pressure above 12 torr would not show this inotropic response and an increase in afterload would precipitate acute failure. Though the precise mechanism controlling this homeometric autoregulation is not known, Sarnoff proposed local modification of norepinephrine or facilitation of its use by the myocardium.¹²

This Anrep reflex has its importance. It implies that the ventricle can maintain a constant stroke volume against a range of resistances without an increase in end-diastolic pressure. Furthermore, the increase in the rate of rise of ventricular pressure will shorten the systolic time. This Bowditch effect forestalls the reduction in diastolic interval with increasing heart rate. Thus a complete ventricular relaxation is attained before the next systole and the increase in end-diastolic volume is prevented. If we remember that coronary perfusion takes place during diastole, and that subendocardial perfusion is counteracted by the end-diastolic volume, this mechanism is not without clinical significance.

FIGURE 4. The clamping of aorta induces a rapid and sustained increase in systemic vascular resistance (lower graph) which is paralleled by a decrease in cardiac output (upper graph).

In our patients these mechanisms either were silent or ineffective because the response to the increased afterload was a reduction in stroke volume and no change in systolic work. It is known that anaesthesia, old age and hypertension are accompanied by modifications of the integrative mechanism of circulatory reflexes. An example is shown in Figure 6 which indicates the inadequacy of baroreceptor

SYSTOLIC INDEX ($\pm$ SE) N = 18

FIGURE 5. The decrease in cardiac output is due to a reduction in the stroke volume, for the heart rate does not change.

reflexes in our patients. Compared to the response of conscious patients of the same age group, it is obvious that under anaesthesia the reflex slowing of heart rate following an increase in systolic pressure is somewhat sluggish. The haemodynamic modification following aortic cross-clamping could be explained in part by inadequate circulatory reflexes either at the level of baroreceptors, integrative centres, afferent pathways, or effectors. There was neither homeometric autoregulation nor slowing of the heart rate in response to increased afterload.

This absence of homeometric autoregulation is not without interest. If we consider the low values of cardiac output after the first ten minutes of aortic occlusion, before blood loss interferes with the Starling mechanism, the situation can be cause for concern. While our patients had normal cardiac function, we are often required to cope with arteriosclerotic hearts or with hypertensive subjects with left ventricular hypertrophy. Their ventricular function curve is already depressed

FIGURE 6. The RR intervals (on the y axis) in milliseconds, are augmented with increases in arterial pressure (on the x axis). The response of conscious subjects, adapted from Duke's data¹⁸ is shown on the upper regression line (A). In our anaesthetized patients, change in heart rate following the increase in blood pressure due to clamping of the aorta is almost non-existent (B). (Δ) represents systolic pressure before clamping and (○) after clamping.

and a further increase in impedance could precipitate failure. We have stressed that, even before aortic clamping, the high systolic pressure and large pulse pressure indicate the higher velocity that the heart must impart to the blood to eject against an aorta that has lost its elasticity. The diastolic pump is no more effective in extending cardiac work to the diastolic period. The increased kinetic energy implies a much larger work, for it is proportional to the square of velocity.

In our patients the systemic vascular resistance increased by nearly 40 per cent and the ventricle must generate a much higher pressure to maintain the same velocity.

We have seen that the necessary autoregulation and reflexes are not available and that cardiac performance decreases. It is obvious that anaesthesia must be conducted with great care. Hypertensive crisis and tachycardia must be particularly feared for they increase cardiac work while the time available for coronary perfusion is reduced. This can lead to cardiac failure due to subendocardial ischaemia. On the other hand, anaesthesia must spare sympathetic activity which presides over myocardial contractility and the homeometric autoregulation of the heart.

SUMMARY

Haemodynamic changes due to aortic cross-clamping were examined in 18 patients. It has been shown that the distal occlusion of the abdominal aorta induces an increased impedance to ejection. Because the mechanisms controlling the autoregulation of cardiac output and the circulatory reflexes are damped by anaesthesia, the augmentation of resistance is not followed by any increase in contractility or vasomotor tone and thus the cardiac output falls. Patients with chronic obstructive disease (Leriche) did not differ significantly from subjects with more acute obstruction (aneurysm), though the latter have a tendency to show more important changes in their haemodynamic response.

RÉSUMÉ

Nous avons étudié les changements hémodynamiques causés par l'occlusion de l'aorte abdominale chez 18 patients qui ne présentaient aucune évidence clinique de maladie cardiaque. L'étude a révélé que l'occlusion basse de l'aorte abdominale provoque une augmentation de l'impédance à l'éjection. Comme l'anesthésie amène une dépression des mécanismes d'autorégulation du débit cardiaque et des réflexes circulatoires, il n'y a pas d'amélioration de la contractilité myocardique ni du tonus vasomoteur, d'où baisse du débit cardiaque. Les malades présentant une maladie obstructive progressive (Leriche) ne diffèrent pas de façon significative de ceux qui ont une obstruction subite (anévrisme); quoique ces derniers présentent des changements plus marqués. Une attention toute spéciale doit être portée à la stabilité de l'anesthésie afin d'éviter les crises hypertensives et de tachycardie, et de conserver une certaine activité sympathique.

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ABBREVIATIONS

- AP = arterial pressure
 $\overline{AP}$ = mean arterial pressure
 CI = cardiac index
 CO = cardiac output
 HR = heart rate
 LVSWI = left ventricular stroke work index
 $\overline{RAP}$ = mean right atrial pressure
 SI = stroke index
 SVR = systemic vascular resistance
 SAP = systolic arterial pressure

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