BALLOON PUMP–INDUCED PULSATILE PERFUSION DURING CARDIOPULMONARY BYPASS DOES NOT IMPROVE BRAIN OXYGENATION

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Background: **Whether pulsatile flow offers substantial advantages for brain protection during cardiopulmonary bypass is controversial. The purpose of this study is to determine whether differences exist between pulsatile and nonpulsatile bypass concerning the effects on internal jugular venous saturation and on the state of regional cerebral oxygenation during normothermia.** ***Methods:*** **Twenty-two patients undergoing elective coronary artery bypass grafting were randomly divided into 2 groups: group 1 (n = 11) received nonpulsatile perfusion during cardiopulmonary bypass and group 2 (n = 11) received pulsatile perfusion during bypass. We used an intra-aortic balloon pump to generate pulsatility. A spectrophotometric probe (INVOS 3100R, Somanetics, Troy, Mich) was used to assess the state of regional cerebral oxygenation. A 4F fiberoptic oximetry oxygen saturation catheter was inserted into the right jugular bulb to monitor jugular venous oxygen saturation. Hemodynamic variables, arterial and jugular venous blood gases, and regional cerebral oxygenation were measured at 7 times points.** ***Results:*** **In both groups, jugular venous oxygen saturation decreased at the early stage of the cardiopulmonary bypass (*P* = .03). Five patients in group 1 and 6 in group 2 had a jugular venous oxygen saturation of less than 50%. In both groups, the regional cerebral oxygenation value decreased during cardiopulmonary bypass (*P* = .04).** ***Conclusions:*** **The present results showed that pulsatility generated through the use of intra-aortic balloon pumping did not produce any beneficial effects on jugular venous oxygen saturation and regional cerebral oxygenation at normothermia. (J Thorac Cardiovasc Surg 1999;118:361-6)**

Whether pulsatile flow offers substantial advantages over nonpulsatile flow for organ protection, especially of the brain, during cardiopulmonary bypass (CPB) is controversial.[1] Studies have reported the benefits of pulsatile perfusion, even in instances of poor-quality pulsatility.[2,3] In contrast, other studies performed by Hindman and associates[4,5] reported no differences in cerebral blood flow or metabolism between pulsatile and nonpulsatile perfusion under either normothermic or hypothermic conditions, even at a very high quality of pulsatility.

Internal jugular venous oxygen saturation ($SjvO_2$), thought to be an index of the global balance of cerebral blood flow and cerebral metabolic rate, has been widely used to assess the adequacy of flow/metabolism coupling in the brain during the operation and in the intensive care unit.[6]

In 1994, Cook and associates[7] reported that a state of cerebral desaturation (defined as an $SjvO_2$ value less than 50%) was more often observed in normothermic groups than in hypothermic groups. In addition, Croughwell and colleagues[8] reported that a state of cerebral desaturation was closely associated with postoperative neurologic disorders. Mutch and coworkers[9] reported that a state of cerebral desaturation, which was often observed during CPB, was markedly reduced through the use of pulsatile perfusion in the porcine model. Thus it is important to assess the effects of pulsatile perfusion on $SjvO_2$ during CPB in human beings.

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However, to date few studies have described the effects of pulsatile perfusion on $SjvO_2$ in human subjects.[10]

Near-infrared spectroscopy (NIRS) is a noninvasive technique that enables physicians to continuously monitor alterations in regional cerebral tissue oxygenation.[11] Recently, NIRS has been used in clinical practice to detect brain ischemia in patients with head injuries.[12] The effects of pulsatile perfusion on the state of regional cerebral oxygenation (rSO_2) have not been examined with the use of NIRS in human beings.

To date, many methods have been used to generate pulse pressure during CPB.[12] However, no general definition nor any criteria have been reported for pulsatile perfusion.[13] In the present study, we elected to use intra-aortic balloon pumping (IABP) to generate pulse pressure, given that IABP allows an approximation of physiologic heart rate, stroke volume, and rate of pressure rise (dP/dt).[14] Mulay and associates[15] recommended the use of IABP, because this method is a simple and reliable way to obtain pulsatile flow during CPB.

The present study attempts to determine whether the effect of CPB pulsatile perfusion, generated through the use of IABP, on $SjvO_2$ and rSO_2 at normothermia is better than that of nonpulsatile perfusion.

Methods

A total of 22 patients undergoing elective coronary artery bypass graft surgery were selected after obtaining the approval of the ethics committee of our institution and getting written informed consent from the patients. We divided the series of 22 consecutive patients randomly into 2 groups: one group who received IABP before induction of anesthesia and one who did not. Group 1 received nonpulsatile perfusion during CPB, and group 2 received pulsatile perfusion during CPB.

No patients had pulmonary, renal, or hepatic disease. In addition, no patients had a neurologic disease or any cerebral vascular disorders, as confirmed by preoperative brain computed tomography, ultrasonography, and magnetic resonance imaging.

Anesthesia was induced by intravenous injections of 30 μg · kg^{-1} of fentanyl, 0.2 mg · kg^{-1} of midazolam, and 0.2 mg · kg^{-1} of vecuronium, and the trachea was then intubated. After induction of anesthesia, a pulmonary artery catheter (Vigilance Swan-Ganz CCO Thermodilution Catheter; Baxter Healthcare Corp, Irvine, Calif) was inserted through the right internal jugular vein. $SjvO_2$ was monitored with a 4F fiberoptic oximetry oxygen saturation catheter (dual-lumen oximetry catheter; Baxter) inserted retrogradely into the right jugular bulb. The position of the catheter was verified radiographically. The catheter was connected to an Explorer system (Baxter), which was calibrated in vivo by drawing a blood sample from the catheter. The partial pressure of the arterial, mixed venous, and jugular venous blood gases was analyzed with a Stat Profile Ultima device (Nova Biomedical, Waltham, Mass). All patients' lungs were ventilated with 100% oxygen, and the end-tidal carbon dioxide was monitored (Ultima, Datex, Helsinki, Finland) and maintained between 35 and 40 mm Hg. After induction of anesthesia, infusion of propofol 4 mg · kg^{-1} · hr^{-1} was begun with the use of a syringe pump and continued until the patients were brought into the intensive care unit. No volatile anesthetics were administrated. Tympanic membrane temperature was monitored with a Mon-a-Therm thermometer (Mallinckrodt Co, St Louis, Mo).

All patients had a dual-lumen intra-aortic balloon catheter with 40-mL balloon volumes placed percutaneously through the femoral artery before the induction of anesthesia. We positioned the distal tip of the balloon catheter in the descending thoracic aorta 2 cm distal to the origin of the left subclavian artery. The catheter position was confirmed by a chest radiograph. Balloon inflation was triggered by the R wave of the electrocardiogram. The balloon was inflated just before the dicrotic notch of the arterial pressure waveform and deflated before the ventricular systole. The trigger ratio of IABP was 1:1 at 100% balloon augmentation during the operation. The IABP was turned off when clamping or unclamping the aorta or while cardioplegia cannulas were inserted or removed. The frequency of the setting was 80 times a minute during CPB.

To identify the IABP pressure waveforms in the cerebral artery during the CPB period, we measured the flow velocity at the middle cerebral artery (MCA), as described previously.[16]

To estimate the state of rSO_2, a spectrophotometer probe (INVOS 3100; Somanetics, Troy, Mich; distances between the light source and the two receivers were 3 cm and 4 cm) was attached to the mid forehead with adhesive and a rubber strap, with rSO_2 being recorded throughout the procedure.

CPB was primed with a crystalloid, nonglucose-containing solution, and the pump flow rate was maintained at 2.2 to 2.5 L · min^{-1} · m^{-2}. A membrane oxygenator and a 40-μm arterial line filter were used, and arterial carbon dioxide tension, which was not corrected for temperature, was adjusted to normocapnic levels (35-40 mm Hg) by varying the fresh gas flow to the membrane oxygenator (alpha-stat regulation).

The target nasopharyngeal temperature was 35°C in the normothermic condition.

Hematocrit was maintained at approximately 0.20 on CPB, and blood was infused as needed. Phenylephrine infusions were used during CPB to maintain the mean arterial pressure at 50 to 90 mm Hg.

Intermittently, antegrade blood cardioplegia was administrated at 37°C. Distal and proximal coronary anastomoses were performed during a single period of aortic crossclamping.

Hemodynamic variables, arterial and jugular venous blood gases, and rSO_2 were measured at the following times: (1) after the induction of anesthesia and before the start of the operation, (2) after sternotomy, (3) 20 minutes after CPB, (4) 40 minutes after CPB, (5) 60 minutes after CPB, (6) 30 minutes after the cessation of CPB, and (7) at the end of the operation.

Intraoperative epiaortic ultrasonography revealed no

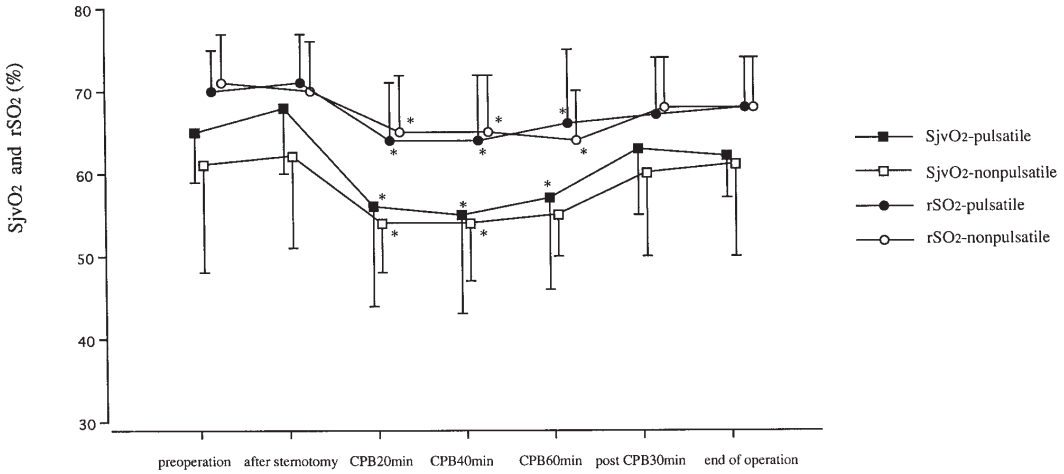

Fig 1. Time course changes in $SjvO_2$ and NIRS during the study. No significant differences in SjvO2 and NIRS were observed between the 2 groups. Data are expressed as mean ± SD.

moderate or severe atherosclerotic lesions in the ascending aorta.

Statistical analysis.

The 22 patients were equally divided into pulsatile and nonpulsatile groups by a block randomization method. After the study was completed, we examined the sample size. On the basis of the variance in previous measurements of $SjvO_2$,[17] the sample size provides 80% power to detect a 20% difference between groups with a 5% probability of α-type error.

All data are expressed as mean ± SD. Analysis of variance for repeated measurements was used to test for significant differences between and within groups. Post hoc data were analyzed by paired or unpaired *t* tests when appropriate, with Bonferroni corrections for multiple comparisons. All calculations were performed on a Macintosh computer with the SPSS (SPSS, Inc, Chicago, Ill) and StatView 4.0 software packages (Abacus Concepts, Inc, Berkeley, Calif).

Results

No patients showed postoperative neurologic dysfunction, such as cerebral infarction, agitation, or delirium.

Table I shows the demographic data of the 2 groups. No significant differences were observed in age, height, weight, left ventricular ejection fraction, aortic clamping time, and total CPB time between the 2 groups.

In both groups, the $SjvO_2$ value decreased at an early time point of CPB compared with period (1) ($P = .03$) (Fig 1). Five patients in group 1 and 6 in group 2 had an $SjvO_2$ value less than 50%. However, the $SjvO_2$ value did not differ significantly between groups at any time point.

In both groups, the rSO_2 value decreased during the CPB period ($P = .04$) (Fig 1). However, the rSO_2 value did not differ significantly between groups at any time point.

Table I. *Demographic data for the 2 groups*

	Group 1	*Group 2*	P *value*
Age (y)	66 ± 7	63 ± 5	.33
Height (cm)	158 ± 6	157 ± 7	.68
Weight (kg)	53 ± 5	55 ± 5	.55
LVEF (%)	64 ± 7	61 ± 6	.32
Total CPB time (min)	132 ± 13	148 ± 18	.26
Aortic clamping time (min)	101 ± 11	109 ± 12	.41

Values are means ± SD. *LVEF,* Left ventricular ejection fraction.

No significant differences were observed in tympanic membrane temperature, cardiac index, internal jugular vein pressure, or arterial carbon dioxide tension at any point in the study (Table II).

During three 20-minute periods of CPB, no significant differences were observed in the mean MCA flow velocity of the 2 groups (group 1, 20 ± 4 cm · sec^{-1}; group 2, 24 ± 4 cm · sec^{-1}) (Table III). Fig 2 shows a typical MCA flow waveform obtained by transcranial Doppler ultrasonography at period 3.

The IABP efficacy in group 2 was monitored at the femoral artery. The pulse pressure, dP/dt, –dP/dt, and inflation times were 24 ± 8 mm Hg, 645 ± 64 mm Hg/s, –454 ± 49 mm Hg/s, and 211 ± 33 ms (mean ± SD), respectively, during the three 20-minute periods of CPB.

Discussion

One recent study reported that normothermic CPB has a more adverse effect on intelligence than hypothermic CPB.[18] Croughwell and colleagues[8]

Table II. *Variable parameters in pulsatile or nonpulsatile groups during perioperative period*

		Measurement time						
Parameter	*Mode*	*1*	*2*	*3*	*4*	*5*	*6*	*7*
CI ($L \cdot min^{-1} \cdot m^{-2}$)	Pulsatile	2.7 ± 0.8	2.6 ± 1.0	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.1	3.0 ± 0.6	3.0 ± 0.9
	Nonpulsatile	2.8 ± 0.7	2.6 ± 0.8	2.5 ± 0.1	2.5 ± 0.1	2.5 ± 0.1	3.2 ± 0.6	3.1 ± 0.5
MAP (mm Hg)	Pulsatile	90 ± 21	104 ± 15	64 ± 14[a]	61 ± 12[b]	66 ± 14[c]	83 ± 10	85 ± 14
	Nonpulsatile	85 ± 10	102 ± 15	69 ± 11	59 ± 8	63 ± 10	79 ± 9	81 ± 11
IJP (mm Hg)	Pulsatile	10 ± 2	11 ± 2	7 ± 2	7 ± 3	6 ± 3	9 ± 3	10 ± 3
	Nonpulsatile	10 ± 3	10 ± 3	7 ± 3	7 ± 3	6 ± 3	9 ± 3	10 ± 3
$Paco_2$ (mm Hg)	Pulsatile	35 ± 2	37 ± 2	40 ± 5	36 ± 4	38 ± 3	36 ± 5	40 ± 3
	Nonpulsatile	38 ± 4	36 ± 4	37 ± 3	36 ± 3	38 ± 3	40 ± 4	37 ± 5
Hb ($g \cdot dL^{-1}$)	Pulsatile	108 ± 1.3	10.5 ± 1.6	7.0 ± 1.2[d]	6.9 ± 1.0[e]	7.2 ± 0.9[f]	7.9 ± 0.9[g]	8.9 ± 0.6[h]
	Nonpulsatile	11.5 ± 1.2	11.0 ± 1.1	7.3 ± 1.1	7.6 ± 1.3	7.5 ± 1.7	8.0 ± 1.4	8.7 ± 0.9
TT (°C)	Pulsatile	35.7 ± 0.7	35.5 ± 0.7	34.9 ± 1.3	35.3 ± 1.2	35.5 ± 1.2	35.8 ± 1.1	35.7 ± 1.1
	Nonpulsatile	35.2 ± 0.8	35.5 ± 0.8	35.0 ± 0.8	35.3 ± 0.9	35.4 ± 0.8	35.8 ± 0.8	35.8 ± 0.9

Values are means ± SD. *CI,* Cardiac index; *MAP,* mean arterial pressure; *IJP,* internal jugular pressure; *Hb,* hemoglobin concentration; *TT,* tympanic temperature. Measurement times: *1,* After the induction of anesthesia and before the start of the operation; *2,* after sternotomy; *3,* 20 minutes after the onset of CPB; *4,* 40 minutes after the onset of CPB; *5,* 60 minutes after the onset of CPB; *6,* 30 minutes after the end of CPB; and *7,* at the end of the operation. *P* values compared to measurement time 1 are as follows: *a,* .036; *b,* .021; *c,* .041; *d,* .008; *e,* .007; *f,* .014; *g,* .029; *h,* .041.

reported that a reduction in $Sjvo_2$ was closely associated with postoperative neurologic disorders. Thus we attempted to determine whether or not pulsatile perfusion, a method used by Mutch and associates[9] to ameliorate the reduction in $Sjvo_2$ during the rewarming period, could prevent the reduction in $Sjvo_2$ during normothermic CPB. However, in the present study, no differences in $Sjvo_2$ and rSo_2 were observed between pulsatile and nonpulsatile perfusions under conditions of normothermia.

$Sjvo_2$ has been thought to be an index of flow/metabolism coupling.[6] The present study suggested that pulsatile perfusion generated by IABP does not improve the state of global cerebral oxygenation. Few studies concerning the effects of pulsatile perfusion on $Sjvo_2$ during normothermic CPB have been reported in human beings.[9,10] Our findings contrast with the results obtained by Mutch and associates,[9] who found significantly lower $Sjvo_2$s during rewarming after hypothermic CPB when apulsatile flow was used than when pulsatile flow was employed. Given the strong effects of hypothermia on $Sjvo_2$ during CPB found by Cook and coworkers,[7] it is likely that the discrepancy between the results of this study and that of Mutch are due to the fact that this study used normothermic CPB and Mutch employed hypothermic CPB. Many studies have reported that the most important factor related to cerebral blood flow is the mean MCA flow velocity.[14] In our study, no differences between the 2 groups in mean MCA flow velocity were observed. In addition, Cheung and associates[16] reported that IABP did not produce an increase in mean MCA flow velocity. This is consistent with our result. MCA flow velocity measurements do not correlate well with cerebral blood flow measurements during CPB.[19,20] However, the fact that mean MCA flow velocities were the same between the pulsatile and nonpulsatile groups during CPB offers reasonable support for the contention that mean cerebral blood flow was approximately equivalent between the groups. Furthermore, the cerebral perfusion pressure, as in our study, was greater than 50 mm Hg throughout CPB in both groups. Sadahiro, Haneda, and Mohri[21] demonstrated that cerebral autoregulation should be intact when cerebral perfusion pressure is maintained at more than 50 mm Hg. This also indicated that cerebral blood flow in this study was approximately equivalent between the 2 groups. Furthermore, in our study CPB was in a normothermic condition, and thus the decrease in $Sjvo_2$ value at hypothermia was likely not induced solely by the high blood-brain temperature gradient, as suggested by Hindman and colleagues.[4] In normothermic conditions, we believe that $Sjvo_2$ is really an index of flow-metabolism coupling in the brain. Above all, we believe that pulsatile flow generated by IABP, which when estimated by MCA flow velocity waveforms was shown to have an effective and physiologic pulsatility, did not produce any beneficial effect on global cerebral circulation.

Recent animal studies have demonstrated that pulsatility does not produce any further beneficial effects on regional cerebral circulation.[5,22] Hindman and coworkers[5] reportedly found no differences between cerebral blood flow and metabolism during pulsatile CPB, nor during nonpulsatile CPB in normothermic animals. Lodge and colleagues[22] also demonstrated that pulsatile perfusion did not improve regional cere-

Table III. *Transcranial Doppler data for the 2 groups*

	Group 1	*Group 2*	*P value*
Mean MCA velocity (cm/s)	20 ± 5	24 ± 5	.44
Peak MCA velocity (cm/s)	—	47 ± 7	
Pulsatile index	—	1.8 ± 0.5	

Values are mean ± SD.

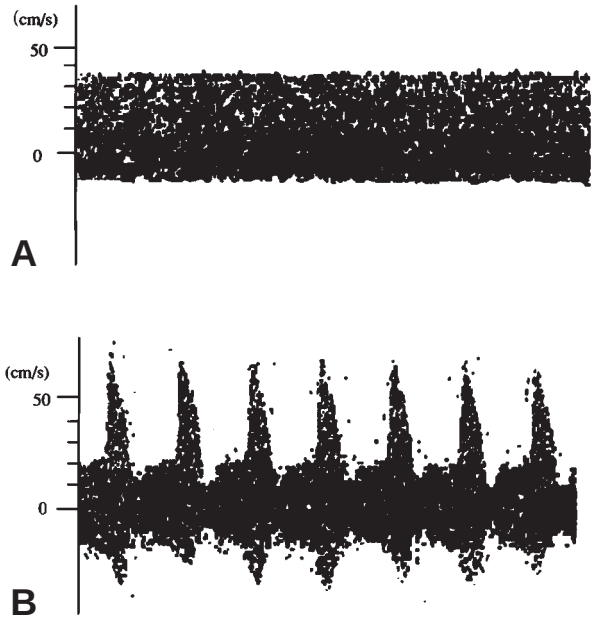

Fig 2. A typical MCA flow waveform obtained by transcranial Doppler ultrasonography at period 3. **A,** Group 1. **B,** Group 2.

bral blood flow in the infant animal model. In contrast, several reports have reportedly found benefits of pulsatile perfusion. In some of these reports, the quality of pulsatility was below acceptable levels.[2,3] The fact that no effects of pulsatility on rSo_2, as measured by NIRS, were observed in the present study indicated that the state of rSo_2 is not preserved even when pulsatility generated by IABP during normothermic CPB is applied in human beings. To date, there have been no reports describing the effect of pulsatility on rSo_2 during CPB under conditions of normothermia in human beings. Sapire and associates[23] reported that the state of rSo_2, as measured by NIRS, decreased during hypothermic nonpulsatile CPB in human beings. Nollert and coworkers[24] reported the same phenomenon during hypothermic nonpulsatile CPB. In contrast, several animal studies reported advantages associated with pulsatility for maintaining the state of rSo_2.[2,3] Matsumoto, Wolferth, and Perlman,[2] in an animal model study, reported that pulsatile perfusion was superior to nonpulsatile perfusion with regard to cerebral capillary collapse, intravascular sludging, and vasodilation. This discrepancy might be partly attributable to the difference in anesthetic method or anesthetic dosage. Newman and associates[25] suggested that propofol may reduce the embolic load to the brain and thus have a cerebral protective effect. In contrast, Souter, Andrews, and Alston[26] reported that propofol could not ameliorate the reduction in $SjvO_2$ value during the rewarming period. The difference in anesthetic dosage likely had a major effect on cerebral circulation.

Study limitations

No significant differences in the cardiac index with or without the use of IABP were observed. IABP produces an approximate 20% increase in cardiac output. However, a number of factors modulate this effect, including the size of the balloon, how proximally the balloon is situated in the aorta, compliance of the aorta, aortic pressure, left ventricular function, and cardiac rate and rhythm.[27] Patients with IABP in this study had good left ventricular function. Thus in the present study we concluded that IABP does not produce any further beneficial effects on cardiac index.

Although NIRS has been used as a noninvasive, real-time, on-line monitor for determining the state of rSo_2 in animals and human beings,[11] a technical limitation exists, as noted by Pollard and Prough.[28] Furthermore, other types of NIRS machines that are capable of assessing the state of mitochondrial redox in the brain are needed to estimate the degree of microcirculation.[11,17,23]

The most recent reports from the Duke group suggested that a reduction in $SjvO_2$ has only a minor independent effect on neuropsychologic outcome.[29] However, a reduction in $SjvO_2$ in patients with a preexisting neurologic disorder may have great influence on neurologic outcome.[30]

In conclusion, pulsatility generated through the use of IABP did not produce any beneficial effect on $SjvO_2$ and rSo_2 at normothermia.

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