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Pulsed Doppler Ascending Aortic, Carotid, Brachial, and Femoral Artery Blood Flows during Caudal Anesthesia in Infants

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Hemodynamic effects of caudal bupivacaine anesthesia were studied in eight infants 6.5 ± 0.5 months old (mean ± SD), weighing 7.4 ± 4.4 kg anesthetized with halothane 0.2% end-tidal and 60% nitrous oxide. Heart rate and systolic, diastolic, and mean arterial pressure remained unchanged. Cardiac index and stroke index assessed by pulsed Doppler and total vascular resistances were not altered by the caudal block. However, after caudal anesthesia, the authors observed a significant decrease ($P < 0.05$) in brachial blood flow assessed by pulsed Doppler (from 49.0 ± 28.3 to 31.7 ± 24.6 $ml^{-1} \cdot min$) and a significant increase ($P < 0.05$) in brachial vascular resistance (from 2.9 ± 1.7 to 5.5 ± 1.0 $mmHg \cdot min \cdot ml^{-1}$). Blood flow and vascular resistance in both the femoral and carotid arteries did not change. This study suggests that, in supine position, caudal anesthesia in infants induces a blood pooling in the denervated lower extremities and a reflex vasoconstriction in innervated areas which maintains cardiac output. We conclude that volume loading is not necessary in normovolemic infants after caudal anesthesia with cutaneous analgesia below T5. (Key words: Anesthesia: pediatric. Anesthetic techniques: caudal; epidural. Hemodynamic: cardiac output; peripheral blood flows. Pulsed Doppler.)

CAUDAL ANESTHESIA can be used in infants and children for lower abdominal and genitourinary surgery.[1] Although arterial blood pressure has been shown to be well maintained with this technique in infants,[2,3] little is known about the changes in cardiac output and peripheral blood flow that occur.

Recent progress in Doppler technique permits the non-invasive determination of peripheral blood flow and cardiac output in neonates and children, as well as in adults.[4–8] The aim of this study was to evaluate diameter, mean cross sectional blood flow velocity, and blood flow in the ascending aorta (as cardiac output),[7–12] carotid, femoral, and brachial arteries before and after bupivacaine caudal anesthesia in normal infants.

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Materials and Methods

PATIENTS

Eight infants 6.5 ± 0.5 months old (mean ± SD), weighing 7.4 ± 4.4 Kg, and 69 ± 17 cm in length were studied. They were free of cardiac, renal, or hepatic disease, and were scheduled for herniorraphy or circumcision. The study was approved by Human Investigation Committee, and parental consent was obtained prior the study.

All infants had fasted for 6 h before anesthesia (range 5.5–6.5 h and received no premedication. Heart rate was continuously recorded from the ECG, and arterial pressure was measured by automated blood pressure cuff every 5 min. Anesthesia was induced, using a partial rebreathing system, by inhalation of 60% nitrous oxide in oxygen plus halothane sufficient to achieve an end-tidal concentration of 1%. After induction, end-tidal concentration of halothane measured with a Datex Normac® anesthesic agent monitor,[13] was decreased to 0.2%. During the procedure, end-tidal CO_2 partial pressure was monitored using a Datex® capnograph. An intravenous catheter was inserted in a brachial vein and 5% dextrose in Ringer's lactate solution was infused at the rate of 4 $ml \cdot kg^{-1} \cdot h^{-1}$. After 10 min at a steady-state concentration of 0.2% end-tidal concentration of halothane in 60% nitrous oxide which defined the control state, blood flow was measured in the aorta, carotid, femoral, and brachial artery in the arm without the blood pressure cuff. Then, the concentration of halothane was increased to 1% end-tidal concentration. The infants were positioned on their side, and a 22-gauge needle was inserted into the caudal epidural space, and 0.5% bupivacaine (3.25 $mg \cdot kg^{-1}$) without epinephrine was injected. The halothane end-tidal concentration was again lowered to 0.2%, and, 30 min later, a second set of measurements was obtained before surgery. At the end of surgery (35 ± 5 min after the second set of measurements), when awakening occurred, the upper level of cutaneous analgesia was assessed by pinprick in each infant.

HEMODYNAMIC MEASUREMENTS

Pulsed Doppler System. We used two zero crosser Doppler (Alvar®) apparati with different ultrasound

frequencies: 4 MHz for ascending aortic blood flow measurement because of the depth of the aortic orifice[14] and the high blood flow velocities, and 8 MHz for superficial vessels. However, the system design was exactly the same. Briefly, the system used has, in addition to the pulsed emission, an adjustable range gated time system which selects the time delay from emission (depth) and the duration of the reception (sample volume size). These times are converted echographically into depth and width of Doppler sample volume. A pedal incorporated within the apparatus enabled the investigator to automatically vary the depth and the width of the sample volume step by step.

Aortic Blood Flow Measurements.[7-14] The measurement of aortic blood flow as an index of cardiac output requires: 1) the cross sectional area of the aorta (CSA), 2) the mean frequency of the Doppler shift integrated over time to give mean blood velocity in the ascending aorta (BVa) during one cardiac cycle, and 3) the heart rate. Cardiac output was then calculated as:

$$CO = CSA \times BVa \times HR \text{ in } ml \cdot min^{-1}$$

The 4 MHz pulsed Doppler used has a repetition rate of 10 kHz. The transducer was applied to the suprasternal notch and directed inferiorly toward the proximal aorta. The sample volume was moved to cover selected distance from the transducer. We used a divergent probe 1 cm in diameter with a beam width of 3 cm at a distance of 5 cm from the transducer.[10] The sample volume was the smallest compatible with an acceptable signal-to-noise ratio. The depth of the width varied between 30 and 45 μsec; the sample volume was then located at a distance of 2.25–3.25 cm from the transducer. Ascending aortic diameter was recorded and measured for each infant using an M-mode Echocardiograph (Echovar 3.5 MHz, Alvar®) coupled with the Doppler device. The echo transducer was placed along the left sternal border at the third or fourth intercostal space and directed toward the base of the heart. Tilting the transducer superiorly and medially permits the beam to go through the root of the aorta. Hard copy of M-mode echographic tracing was obtained when evidence of aortic valve motion appeared.

The diameter (D) was measured from leading edge to leading edge of the anterior and posterior wall, respectively, at mid-systole. Then CSA was computed as follows:

$$CSA = \pi D^2/4 \cdot \text{in } cm^2$$

Mean Doppler frequency shift (ΔF) was obtained by a zero crossing system. Then the frequency shift of reflected ultrasonic signals was converted into velocity *via:*

$$V = \frac{\Delta F \times C}{2Fe \times \cos \theta}$$

Where V is the red cell velocity in $cm \cdot sec^{-1}$, ΔF is the Doppler shift frequency in KHz, cos θ is the incidence angle between the ultrasonic beam and the flow direction, Fe is the ultrasound emitted frequency, and C is the speed of ultrasound in tissues: 1550 $m \cdot sec^{-1}$. V was recorded on a Gould® ES 1000 recorder using 100 $mm \cdot sec^{-1}$ paper speed. Then mean blood flow velocity during one cardiac cycle was calculated manually on a digitizing tablet linked to a microcomputer (Apple® IIe). V value represented the average of ten cardiac cycles.

Peripheral Blood Flow Measurements.[4-6] Brachial, femoral, and carotid blood flows were obtained with an 8 MHz pulsed Doppler. This apparatus enables the arterial diameter and blood velocity to be measured using two fundamental characteristics: a bidimensional recording the Doppler of signals which considerably minimizes the error due to incidence angle and a range gated time system of reception. The probe contains two transducers, with an angle of 120° between them. With this double transducer probe, when Doppler signals recorded by each transducer are equal in absolute value, the incidence angle with the vessel axis is 60°.[4,5] Using the pedal, the sample volume size was adapted to the smallest convenient for an adequate signal-to-noise ratio (0.035 mm). Then, using another pedal, the depth of the sample volume was modified with incremental or decremental steps of 0.4 mm, to determine the arterial diameter. Finally, the velocity over the vessel cross-section was obtained by adjusting the time delay to the proximal arterial wall and the gate width to the diameter. Mean cross-sectional blood flow velocity (Vm) was continuously calculated by electronic integration during ten cardiac cycles of the phasic velocity curve. Mean arterial blood flow was then calculated as the product of mean cross-sectional blood velocity (Vm) and cross-sectional area (CSA) calculated from diameter (D) according to the equation:

$$\text{Flow} = \pi D^2/4 \times Vm \times 60 \text{ in } ml \cdot min^{-1}$$

Measurements. During each set of measurements, the following data were measured or calculated: heart rate by EKG, arterial blood pressure by automated blood pressure cuff (Dinamap®), aortic diameter (cm), aortic peak flow velocity ($cm \cdot sec^{-1}$), left ventricular ejection time (seconds), cardiac index (ascending aortic blood flow) ($ml \cdot min^{-1} \cdot m^{-2}$), systemic vascular resistance as the direct ratio between mean arterial blood pressure and cardiac output ($mmHg \cdot min \cdot ml^{-1}$) neglecting central venous pressure; brachial, femoral, and carotid diameters (mm), blood flows ($ml \cdot min^{-1}$), and vascular resistances ($mmHg \cdot min \cdot ml^{-1}$). In order to evaluate blood volume partition, we calculated the following ratios: brachial/femoral, and brachial or femoral blood flow/cardiac output.

TABLE 1. Systolic Diastolic, Mean Arterial Pressure, and Heart Rate (Mean values ± SD) Before and After Caudal Anesthesia in Eight Infants

	Before Caudal Anesthesia	After Caudal Anesthesia
Systolic arterial pressure (mmHg)	80.6 ± 6.5	78.9 ± 6.6
Diastolic arterial pressure (mmHg)	40.2 ± 5.3	41.5 ± 6.3
Mean arterial pressure (mmHg)	54 ± 6.5	56.5 ± 6.0
Heart rate (beat · min^{-1})	120 ± 20	119 ± 18

No significant difference between before and after caudal anesthesia.

TABLE 2. Aortic Peak Flow Velocity, Left Ventricular Ejection Time, Cardiac Index, Stroke Index, and Systemic Vascular Resistance (Mean Values ± SD) Before and After Caudal Anesthesia in Eight Infants

	Before Caudal Anesthesia	After Caudal Anesthesia
Peak flow velocity (cm · sec^{-1})	53.5 ± 10.1	55.7 ± 10.7
Left ventricular ejection time (seconds)	0.278 ± 0.047	0.268 ± 0.049
Cardiac index (ml · min^{-1} · m^{-2})	4505 ± 920	4164 ± 1204
Stroke index (ml · m^{-2} · $beat^{-1}$)	35.7 ± 7.2	35.0 ± 8.3
Systemic vascular resistances (mmHg · ml^{-1} · min)	13.8 ± 2.7	14.6 ± 4.9

No significant difference between before and after caudal anesthesia.

STATISTICAL ANALYSIS

Data were analysed by non parametric Wilcoxon paired test ($P < 0.05$). All values were expressed as mean ± SD.

Results

Neither cardiovascular nor neurologic side effects were observed during the study. The cephalad extent of cutaneous analgesia was T10 ± 3 (range T6–L1). The end-tidal CO_2 partial pressure did not change significantly (37 ± 2 mmHg and 38 ± 2 mmHg, respectively, before and after caudal anesthesia).

Systemic Hemodynamics. The aortic diameter was 1.4 ± .2 cm. Caudal anesthesia did not modify systolic, diastolic, mean arterial pressure, or heart rate (table 1). Aortic peak flow velocity, left ventricular ejection time (table 2, fig. 1), cardiac index, stroke index, and systemic vascular resistance were not modified by caudal anesthesia (table 2).

Peripheral Hemodynamics. Brachial, femoral, and carotid diameters were, respectively, 2.9 ± 0.6 mm, 3.3 ± 1.0 mm, and 3.5 ± 0.8 mm. Both femoral and carotid blood flows and respective vascular resistances were not altered by caudal anesthesia (table 3, fig. 1). However, brachial blood flow decreased, and brachial vascular resistance increased significantly after the onset of caudal block (table 3). The brachial/femoral blood flow ratio decreased significantly after caudal block ($P < 0.05$).

The brachial blood flow/cardiac output ratio decreased significantly (3.2% ± 1.6 to 2.2% ± 1.5; $P < 0.05$), and the femoral blood flow/cardiac output ratio did not change.

Discussion

METHODOLOGICAL DISCUSSION

Ascending Aortic Blood Flow. Both continuous and pulsed Doppler ultrasound have been used to measure blood flow velocity in the thoracic aorta. A major limitation of the continuous wave Doppler instruments for measurement of blood velocity in the great vessels is their lack of spatial resolution. Pulsed Doppler instru-

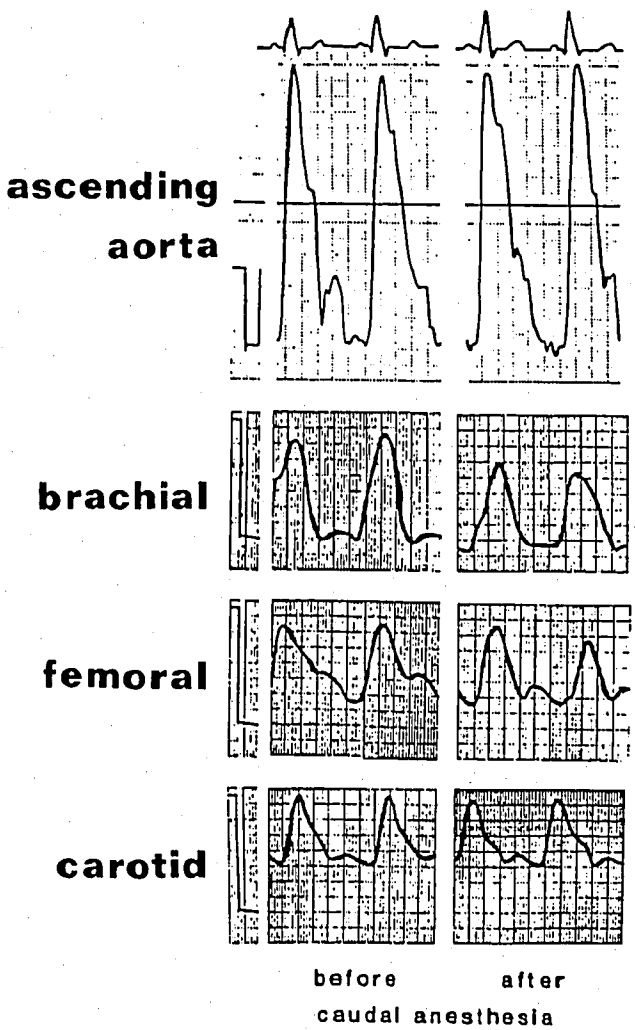

FIG. 1. Phasic ascending aortic, brachial, femoral, and carotid blood flows before and after caudal anesthesia in a 5-month-old infant.

TABLE 3. Brachial, Femoral, and Carotid Blood Flows and Vascular Resistances (Mean ± SD) Before and After Caudal Anesthesia in Eight Infants

	Before Caudal Anesthesia	After Caudal Anesthesia
Brachial artery		
Blood flow ($ml \cdot min^{-1}$)	49.0 ± 28.3	31.7 ± 24.6*
Vascular resistances ($mmHg \cdot ml^{-1} \cdot min$)	2.9 ± 1.7	5.5 ± 1.0*
Femoral artery		
Blood flow ($ml \cdot min^{-1}$)	37.4 ± 16.5	33.3 ± 17.6
Vascular resistances ($mmHg \cdot ml^{-1} \cdot min$)	1.6 ± 0.9	1.9 ± 0.7
Carotid artery		
Blood flow ($ml \cdot min^{-1}$)	153.8 ± 125.4	141.9 ± 108.2
Vascular resistances ($mmHg \cdot ml^{-1} \cdot min$)	0.56 ± 0.29	0.49 ± 0.42

* $P < 0.05$ statistical difference between before and after caudal anesthesia.

ments use range gating to define a small sample volume at a known distance from the probe which excludes extraneous signals from walls and adjacent blood vessels.[15] This improves the accuracy of the measurement of mean blood velocity. The probe used in this study has a beam-width of 2 cm diameter at a distance of 3 cm from transducer, which provides an illumination of the cross-sectional area of the aortic root. Consequently, the recorded Doppler signal contains information from the entire vessel. Three factors may distort the ascending aortic blood flow measurement:[14] aortic diameter determination, Doppler shift analysis, and precise knowledge of incidence angle.

It is generally accepted that the most probable source of error in flow measurement by Doppler is diameter measurement. We used M-mode echography because of its simplicity and convenience in the operating room, although it is less accurate than 2D echo. 2D echo permits assessment of the cross-section of the vessel. However, we used the results of serial velocity measurements with the same aortic diameter, so that this problem was minimized. Additionally, the inability to be certain that the sample volume exactly matched the location where the diameter had been measured might induce an error. However, we used the same sample volume size and depth adjustments in the two stages of the protocol.

The zero-crossing detector for Doppler shift determination is an old technique which has some limitation in accuracy due to Doppler frequency spectrum, electrical noise, signal from moving structure, and complex flowing conditions.[15] Nevertheless, we obtained a quasi-linear increase in frequency-shift during early systole, reaching peak flow velocity and falling back to zero with little signal during diastole. We think that there is a relatively small dispersion of velocities during deceleration of the blood in infants, since flowing conditions are laminar. Because of the incidence angle close to 0, aortic vessel wall movements cannot generate Doppler effect. Finally, the electrical noise problem is partially overcome by the use of the set-reset system and high-frequency and low-frequency filters.[15]

An error in estimating incidence angle will affect the measurement of aortic blood velocity. However, from the suprasternal window, we can assume that the ultrasonic beam is essentially parallel to the direction of the flow, and that θ is, therefore, close to 0. Even if θ is assumed to be 0 but it is in fact 15°, $\cos \theta$ equals 0.965 and flow is underestimated by about 3.5%. Using the same external method in adults, we found an improvement of the regression equation between thermodilution and pulsed Doppler cardiac output determination, when $\cos \theta$ was corrected from 1 to .95 ($\theta = 18°$).[10] We conclude that, in the present study, the error due to the incidence angle can be neglected.

The 4 MHz pulsed Doppler apparatus allowed deep measurements (5 cm). The recurrence frequency of 10 KHz permitted velocity measurements up to 95 cm/sec corresponding to 5 KHz. The resolution for velocity measurement equals 0.1 KHz (1.9 cm/sec). The sum of the theoretical errors are taken into account in the standard deviation of cardiac output values which were little changed after caudal anesthesia.

Peripheral Blood Flows. Carotid, brachial and femoral blood flows were measured with an 8 MHz pulsed Doppler as previously described.[4–6,16–18] The resolution of this apparatus is 0.1 KHz (1.9 cm/sec), and the recurrence frequency is 32.4 KHz. Because of the influence of diameter measurements on flow calculations, it might be useful to mention previous validations.

With the same equipment and the same double transducer probe, Safar *et al.* demonstrated the accuracy of pulsed Doppler diameter measurements, compared to actual diameter of calibrated tubes, using an *in vitro* evaluation model.[4] The intercept of the linear regression was 0.35 mm, which meant that the Doppler overestimated the diameter by 0.35 mm. The smallest value of diameter measured in our study was 2.3 mm, so that the error can reach 15%. Even if these values were false, the impact on our study would be negligible, since we used a same arterial diameter value for each pair of measurements. Consequently, the observed flow variations were only due to cross-sectional blood flow velocity changes.

The error due to the incidence angle variation could be large for theta value used ($\theta = 60°$). The double transducer probe largely minimizes this error.[4,5] We took the greatest care at this step of measurement to ensure that the probe was in a proper position.

Caudal Anesthesia Effects. These results confirm pre-

vious studies concerning arterial blood pressure and heart rate stability in children during conduction anesthesia.[19,20] The absence of significant variation in ascending aortic blood flow suggests that cardiac output is maintained. The maintenance of aortic peak flow velocity, which is well correlated with left ventricular contractility,[21–23] demonstrates that left ventricular ejection was not influenced by caudal anesthesia.

However, musculo-cutaneous flow (brachial flow) in the innervated area decreased similar to that in previous studies in adults.[17] This reflex adaptation probably participates in cardiac output maintenance.[24–26] Moreover, this reflex adaptation in the unblocked area did not alter common carotid blood flow. This flow, which is related to mean cerebral blood flow,[4] is regulated by mechanisms other than modification of blood flow distribution. Even if external carotid artery flow was decreased, this was too slight to modify mean carotid blood flow.

Surprisingly, femoral blood flow did not increase in spite of sympathetic blockade. This could be explained by the fact that resolution of the apparatus is not sufficient to accurately detect the small diameter increase for this artery. Alternatively, the sympathetic nervous system in infants may be less well-developed than the adult, or lower-extremity blood volume may be smaller than that of the adult,[27] thus limiting the extent to which blood flow can increase.

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