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Comparison of Direct Blood Pressure Measurements at the Radial and Dorsalis Pedis Arteries during Sodium Nitroprusside- and Isoflurane-induced Hypotension

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Deliberate systemic hypotension can be induced by vasodilators (nitroprusside and nitroglycerin),[1] ganglionic blockers (trimethaphan),[2] and potent volatile anesthetics (halothane and isoflurane).[3] A potential hazard of deliberate hypotension is cerebral ischemia.[4] Other organs susceptible to ischemic damage include the heart, gastrointestinal tract, and liver.[5] Hence arterial blood pressure is important to monitor during deliberate hypotension.

The most frequently used site for direct arterial blood pressure measurement is the radial artery (RA). Other sites, such as the dorsalis pedis artery (DPA), have been used.[6] Although differing pressures in RA and DPA may exist,[7] the influence of deliberate hypotension and drugs used to induce hypotension on this difference have not been clarified. We compared arterial blood pressure obtained from RA and DPA during deliberate hypotension

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induced with either sodium nitroprusside (SNP) or high concentrations of isoflurane in patients undergoing clipping of an intracranial aneurysm.

METHODS

Twenty ASA Class III patients (28–69 yr) scheduled for elective clipping of a cerebral aneurysm were randomly allocated to receive either intravenous (n = 10) or isoflurane (n = 10) anesthesia. Their age, weight, and sex distribution were similar (table 1). Their medication included epsilon-aminocaproic acid 1 $g \cdot h^{-1}$ iv and phenobarbital 30–60 mg every 8 h by mouth. Patients with known intolerance to SNP were excluded. None of the patients suffered from peripheral vascular or cardiopulmonary disease. The study was approved by the Hospital Ethics Committee. All patients gave written informed consent to the study. Anesthesia was induced with fentanyl 4 $\mu g \cdot kg^{-1}$, droperidol 0.15 $mg \cdot kg^{-1}$, and thiopental 3–5 $mg \cdot kg^{-1}$, all given iv, followed by lidocaine 1.5 $mg \cdot kg^{-1}$, given to block the response to laryngoscopy and endotracheal intubation, which was facilitated with pancuronium 0.1–0.15 $mg \cdot kg^{-1}$ iv. Ventilation was controlled using a circle system of 67% nitrous oxide in oxygen. Pa_{CO_2} was maintained at 34 ± 2 mmHg. Additional pancuronium was administered to provide paralysis during the surgical procedure.

Patients allocated for intravenous anesthesia received incremental doses of fentanyl 1.5 $\mu g \cdot kg^{-1}$ throughout surgery. Thiopental (50–100 mg) was used if hemodynamic hyperactivity or signs of inadequate anesthesia could not be controlled by the earlier mentioned narcotic supplementation. In the other ten patients, anesthesia was maintained with supplemental 0.75–1.5% inspired concentration of isoflurane.

In all patients, 20-gauge Teflon® cannulae were inserted into the RA and DPA for direct pressure monitoring and blood sampling. To secure equal dynamic responses, both cannulae were connected by two identical arterial pressure tubings to the same pressure transducer[8] calibrated and placed at the level of the external auditory meatus. The pressure transducer was attached to three preamplifiers that continuously displayed systolic (SYST), diastolic (DIAST) and electronically obtained mean arterial pressure (MAP). Analysis of arterial blood gases was done at regular intervals.

In patients who received intravenous anesthesia, hypotension was achieved by SNP 50 mg in 500 ml of 5% glucose, given iv as a continuous infusion. The infusion rate was adjusted to achieve a radial MAP of 50–60 mmHg during the dissection and clipping of the aneurysmal sac.

In patients who received isoflurane anesthesia, the inspired isoflurane concentration was gradually increased until the same level of hypotension was achieved.

TABLE 1. Patient Characteristics

	Intravenous Anesthesia (n = 10)	Isoflurane Anesthesia (n = 10)
Age (yr)	40.6 ± 12.6	39.1 ± 10.4
Weight (kg)	62.1 ± 7.6	65.3 ± 6.8
Sex (F/M)	7/3	7/3

Data expressed as mean ± SD where applicable.

SYST, DIAST, and MAP were obtained from the two arteries in rapid succession (fig. 1) before, during, and 20 min after hypotension. A minimum of 5 min of stable arterial pressure was allowed before the measurements were taken.

The differences between RA and DPA values were analyzed using Student's *t* test. Probability values less than 0.5 were regarded as significant. Results are expressed as the mean ± SEM.

RESULTS

With SNP-induced hypotension, the SYST, DIAST, and MAP were higher at the DPA than at the RA (fig. 2). This difference persisted throughout normotension, during hypotension, and on return to normotension; it was statistically significant during hypotension for SYST and MAP: RA SYST 84.4 ± 3.5 mmHg, DPA SYST 97.3 ± 4.0 mmHg ($P < 0.05$); RA MAP 57.7 ± 1.8 mmHg; DPA MAP 64.2 ± 2.2 mmHg ($P < 0.05$); and post hypotension for SYST: RA SYST 118.2 ± 4.6 mmHg, DPA SYST 134.1 ± 4.8 mmHg ($P < 0.05$). The mean SNP dose was 2.1 ± 0.4 (SD) $\mu g \cdot kg^{-1} \cdot min^{-1}$, and the mean duration of hypotension was 45 ± 6 (SD) min.

With isoflurane-induced hypotension, the SYST, DIAST, and MAP were higher at the RA than at the DPA. This difference lasted throughout the study and was statistically significant before hypotension for MAP: RA MAP 84.2 ± 2.1 mmHg, DPA MAP 77.3 ± 2.3 mmHg ($P < 0.05$); and during hypotension for MAP: RA MAP 58.7 ± 1.2 mmHg, DPA MAP 54.3 ± 1.2 mmHg ($P < 0.05$) (fig. 2). The mean duration of hypotension

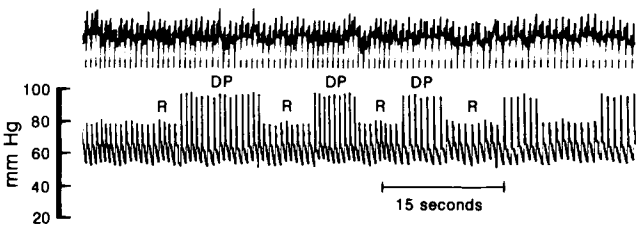

FIG. 1. Tracing demonstrating alternate pressures recordings from the radial (R) and dorsalis pedis (DP) arteries during nitroprusside-induced hypotension.

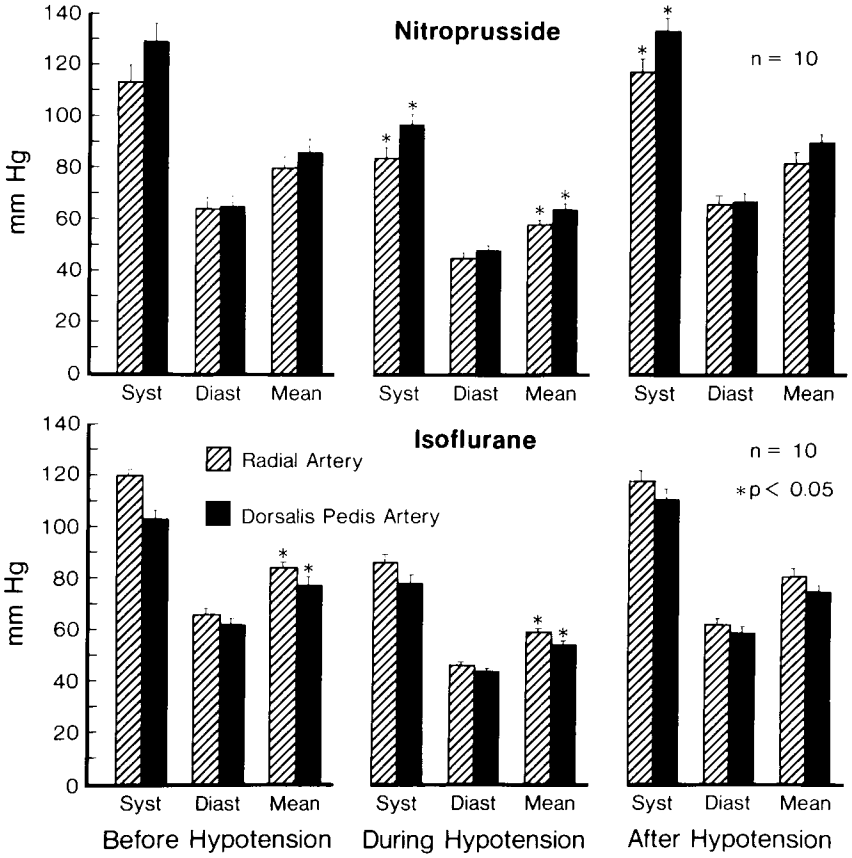

FIG. 2. Changes in systolic, diastolic, and mean arterial pressures at radial and dorsalis pedis arteries, before, during, and after nitroprusside- and isoflurane-induced hypotension. Values are mean ± SEM. * Significant difference between RA and DPA values ($P < 0.05$).

was 41 ± 9 (SD) min, which was obtained with an inspired isoflurane concentration of 2.0–2.5%.

DISCUSSION

Our study demonstrated that isoflurane reverses the difference in pressures between the RA and DPA. Also, during hypotension the MAP measured at the RA differed significantly from that measured at the DPA. With SNP, the MAP at the DPA was significantly higher than at the RA and with isoflurane, the reverse was true. This would indicate that during SNP hypotension, the MAP might be excessively decreased when the arterial pressure is measured at the DPA, resulting in ischemic complications. Conversely, isoflurane-induced hypotension might yield low MAP readings at the DPA when the actual arterial pressure is higher than the acceptable lowest limit. This could be detrimental in cases where the lowest possible MAP is needed, as in a difficult aneurysm clipping.

The reason the MAP behaves differently during controlled hypotension with different hypotensive drugs remains to be elucidated. Johnstone and Greenhow[9] suggested that the DPA and the RA could be used interchangeably for invasive blood pressure monitoring. They described differences between the RA and the DPA pressure curves, consisting of a larger pulse pressure with a higher systolic peak and a loss of the incisure when moving distally in the arterial tree. They attributed these changes to the increased impedance in the more peripheral arterial vasculature and to the reflections of previous waves at bifurcations. They also found that the systolic DPA pressure was 5–20 mmHg higher than the reference RA pressure.

Spoerel *et al.*[10] reported that by taking simultaneous measurements in humans, the systolic pressure and the pulse pressure were higher at the DPA than at the RA. In four patients, during normotension and controlled hypotension with trimethaphan, they found that the MAP was slightly higher at the RA than at the DPA but the differences were not significant. In view of a low incidence of complications in their series, they concluded that the DPA was an appropriate site to monitor arterial blood pressure when collateral flow was adequate. Youngberg and Miller[6] tried to evaluate the validity of DPA blood pressure measurements by comparing them with the blood pressure measured simultaneously with a cuff on the arm and concluded that the DPA was a good measuring site because measurements correlated well with the arm cuff

pressures. They also mentioned a low incidence of complications, consisting mainly of thrombosis of the cannulated artery, which they reported to be 6.7%.

Our results lead to different conclusions. The dissimilar behavior of the MAP observed in response to the two hypotensive agents probably results from the dissimilar effect of these two drugs on the vascular resistance of the arterial tree downstream of the measuring point. Miletich and Ivankovich[11] noted that while SNP has no effect on the myocardial muscle and does not change the cardiac output of the healthy heart, it reduces significantly the portion of the cardiac output delivered to the skin (0.08% to 0.05%, $P < 0.05$), but maintains the blood flow to skeletal muscles. In contrast, Stevens *et al.*[12] found the skeletal muscle blood flow to increase by 199% at MAC 1 of isoflurane, and 227% at MAC 2; skin blood flow increased, up to 700%. Cardiac output was not appreciably changed.

The smaller vessels downstream from the RA and the DPA mainly supply skeletal muscles and skin. SNP may decrease blood flow distal to the measuring sites by increasing the distal impedance as a result of an increase in sympathetic response, as proposed by Miletich and Ivankovich.[11] This would preserve the systolic and pulse pressure amplification that is seen during normotension. Our results seem to confirm this hypothesis. Conversely, isoflurane diminishes the impedance distal to the measuring sites by decreasing the resistive component. This would then lower the pressure for a given flow and cancel the amplification effect previously described. Our results using isoflurane during normotension and hypotension agree with this explanation. Moreover, as the vascular bed distal to the DPA is larger than that distal to the RA, isoflurane would result in a larger drop of impedance at the DPA than at the RA and, hence, in a larger drop of the MAP at the DPA. Stern *et al.*[13] demonstrated a reversal of the usual relationship between aortic and RA pressure in patients immediately following cardiopulmonary bypass. They attributed their observation, in part, to vascular changes in the arm. Indeed, both systemic vascular resistance and forearm resistance were found to be low, implying vasodilation. Our results in patients who received isoflurane anesthesia may be explained on the same basis.

In conclusion, our results suggest that the DPA might not be an acceptable pressure measuring site during controlled hypotension, because it yields values that are at a variance from those recorded at the reference RA site, either higher or lower, depending on the mode of action of the hypotensive drug used. These differences could become clinically important in some critical situations. Moreover, Husum *et al.*[14] found in their series a complication rate as high as 25% when all the cannulations of the DPA were routinely checked for possible thrombosis. This, allied with the finding of Spoerel *et al.*[10] that 16% of the population has inadequate collateral circulation in the foot, would actually make the DPA an undesirable site for cannulation.

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