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Pulmonary Shunting during Anesthesia with Deliberate Hypotension

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Pulmonary shunting ($\dot{Q}_s/\dot{Q}_t$ with $FI_{O_2} = 1$) was measured in 18 anesthetized patients during deliberate hypotension. Hypotension was induced in 12 patients with sodium nitroprusside and light halothane anesthesia and in six others with deep halothane anesthesia and mechanical hyperventilation. Similar results were observed in the two groups. During the hypotensive period mean arterial pressure (MAP) was reduced to 49 ± 2 torr, a 37 per cent decrease from the control level after the onset of operation and a 40 per cent decrease compared with the recovery level during closure of the wound. $\dot{Q}_s/\dot{Q}_t$, however, remained unchanged throughout the study: 3.2 ± 0.9 per cent initially, 3.4 ± 0.8 per cent during hypotension, and 4.7 ± 0.5 per cent during recovery. It is concluded that pulmonary shunting need not develop during deliberate hypotension induced with either technique.
(Key words: Anesthetic techniques, hypotension, induced; Lung, shunting; Ventilation, shunting; Anesthetics, volatile, halothane.)

DELIBERATE HYPOTENSION is commonly used as an adjuvant to general anesthesia when brisk bleeding could jeopardize the safety of the patient during the surgical procedure. Although coronary, cerebral, renal and hepatic ischemia have all been reported to occur following hypotensive anesthesia,[1] the risk is not generally considered to be increased.[2] Hypotensive anesthesia, however, may alter pulmonary gas exchange in several ways. Eckenhoff *et al.* demonstrated increased respiratory deadspace, suggested that ventilation/perfusion inequalities increase, and recommended "careful control of respiration, with higher than normal tidal volumes and oxygen concentration during deliberate hypotension."[3] Others have since reported the association of induced hypotension with decreased arterial oxygen tension[4–6] and an increased $A\text{-}aD_{O_2}$ gradient.[4,6] This impaired pulmonary oxygenation during deliberate hypotension might be due to increased ventilation/perfusion inequalities, as suggested by Eckenhoff *et al.*, but increased intrapulmonary shunting ($\dot{Q}_s/\dot{Q}_t$ with $FI_{O_2} = 1$), another physiologic mechanism that can cause faulty oxygen exchange, must be considered.

This study was undertaken to determine whether deliberate hypotension alters the pulmonary shunt during anesthesia and operation. Two hypotensive techniques (sodium nitroprusside with light halothane anesthesia and deep halothane anesthesia with mechanical hyperventilation) were examined and compared.

Methods

Intrapulmonary shunting ($\dot{Q}_s/\dot{Q}_t$ with $FI_{O_2} = 1$) was measured in 18 patients undergoing neurosurgical or radical neck procedures with hypotensive anesthesia. The study was approved by the institutional review committee on human investigation, and informed consent was obtained from all patients. The average age was 32 years, with only one patient more than 40 years old. All had been in good general health and had been active until admission to the hospital. Some of the neurosurgical patients were sedated and kept on bed rest for several days prior to operation. Twelve smoked cigarettes, but none gave a history of or had physical findings indicative of pulmonary disease. Routine chest x-rays were reported as normal, but extensive preoperative pulmonary function testing was not attempted.

Although the dosage was variable, premedication consisted of intramuscular administration of atropine and either diazepam or secobarbital. A narcotic was also given to eight of the patients. Halothane and oxygen were the sole inhalational agents used for anesthesia following induction with thio-

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pental and endotracheal intubation facilitated by succinylcholine. Body position and table tilt were then established. Depth of anesthesia, degree of muscle relaxation, and Pa_{CO_2} differed among patients, as premedication, induction, and the course of anesthesia were managed by anesthesiologists apart from the study. Ventilation was assisted in three patients (radical neck surgery), and in the rest *d*-tubocurarine was given and respiration was controlled with an Air-Shields Ventimeter ventilator to provide a tidal volume of 10 ml/kg. Blood and other fluids were administered as needed.

Indwelling radial artery catheters were inserted to monitor blood pressure and blood gases. A second catheter was threaded from an antecubital vein and advanced into the heart with its tip lying in the pulmonary artery in four patients and in the right ventricle in eight others. Catheter position in these 12 patients was determined by pressure tracings. In the other six patients catheter advancement ceased after the observation of an abrupt change in the P-wave configuration of the EKG V lead recorded from the catheter tip.[7] Arterial and mixed venous blood samples were drawn anaerobically into iced heparinized glass syringes and analyzed expeditiously for P_{O_2}, P_{CO_2}, and *p*H on an IL 313 blood-gas analyzer at 37 C. One *p*H and one CO_2 electrode sufficed for both arterial and venous blood, but two oxygen electrodes were used so that the high oxygen calibrating gas closely corresponded to the P_{O_2} of each sample. All determinations were done in duplicate. When a delay of more than 10 minutes occurred between sampling and blood-gas analysis, a correction factor was employed to compensate for oxygen decay in the arterial blood.[8] The time-corrected P_{O_2} was determined from the measured P_{O_2} + [(minutes elapsed − 10) × decay factor]. The decay factor was 0.17 torr/min for measured P_{O_2}'s of more than 570 torr, 0.16 between 569 and 540 torr, 0.15 between 539 and 500 torr, and 0.14 between 499 and 470 torr. Venous samples were not time-corrected. A second correction was applied to the time-corrected Pa_{O_2} value to compensate for the oxygen electrode blood-gas P_{O_2} difference, which was determined by tonometry and found to be 7 per cent deficient for P_{O_2}'s above 525 torr, and 6.5 per cent for P_{O_2}'s between 524 and 450 torr.[8] All blood-gas values were corrected to body temperature.[9] The shunt equation used is:

$$\dot{Q}s/\dot{Q}t = \frac{Cc'_{O_2} - Ca_{O_2}}{Cc'_{O_2} - C\bar{v}_{O_2}} \times 100$$

O_2 content was calculated: $C_{O_2} = (1.39 \times Hb \times \text{per cent saturation}) + (\alpha \times P_{O_2})$, where c', a, and $\bar{v}$ represent end-pulmonary capillary, arterial, and mixed venous values, respectively. End-pulmonary capillary P_{O_2} was assumed equal to alveolar P_{O_2}, which was calculated by subtracting Pa_{CO_2}, P_{H_2O}, and $P_{halothane}$ from the barometric pressure. Per cent O_2 saturation was derived from measured blood gases using the data of Severinghaus[9] and Roughton and Severinghaus.[10] A normal P_{50} was assumed for the O_2 dissociation curve. Solubility of O_2 in blood (α) was corrected for temperature and hemoglobin.[11] Cardiac output was measured by the dye-dilution method using cardiogreen dye and a Beckman cardiodensitometer. Cardiac index ($\dot{Q}_I$) was computed by dividing the cardiac output by the body surface area. Oxygen delivery to the tissues was calculated as the product of the cardiac index and the oxygen content of the arterial blood × 10. Whole-body oxygen consumption was not measured but was derived from: $\dot{V}_{O_2} = \dot{Q}_I \times (a\text{-}\bar{v}C_{O_2}) \times 10$. Statistical significance was determined by the Student's t test for paired data whenever possible; however, analysis of variance was substituted when the groups to be compared were not of equal size.

During the operation, after a period of at least 30 minutes of stable vital signs, control measurements were made. Hypotension was then induced utilizing one of two techniques. With the aid of a Holter infusion pump, patients in Group A (n = 12) received a 0.04 per cent solution of sodium nitroprusside. Ventilation and inspired halothane concentration were not altered during hypotension. Group B (n = 6) received no additional drug, but ventilation was increased by doubling both tidal volume and respiratory rate. Although the mean airway pressure increased, end-expiratory pressure returned to zero. The inspired halothane concentration was then

TABLE 1. Hemodynamic and Blood-gas Data from Individual Patients before,

	MAP (torr)			HR (Beats/Min)			pH_a			Pa_{CO_2} (torr)			Pa_{O_2} (torr)		
	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery
Patient 1	77	50	88	74	81	68	7.50	7.49	7.49	31.6	30.6	30.2	608	622	612
Patient 2	74	37	78	80	100	70	7.48	7.49	7.45	28.0	26.1	28.3	509	548	574
Patient 3	85	52	84	75	90	72	7.39	7.38	7.43	40.6	35.0	32.4	636	621	644
Patient 4	71	34	76	74	82	66	7.63	7.57	7.56	22.5	25.1	25.3	636	642	630
Patient 5	63	42	67	52	73	70	7.53	7.51	7.51	29.0	29.8	29.2	659	658	665
Patient 6	76	40	83	61	82	67	7.56	7.51	7.55	28.3	29.6	27.1	633	628	587
Patient 7	78	41	70	68	65	58	7.64	7.64	7.68	19.2	19.3	17.2	669	673	645
Patient 8	61	47	75	90	87	74	7.53	7.54	7.53	29.7	26.8	27.5	624	603	611
Patient 9	68	52	80	60	75	70	7.47	7.52	7.31	28.3	24.3	47.1	623	582	604
Patient 10	—	50	73	—	83	74	—	7.41	7.47	—	36.5	30.8	—	517	587
Patient 11	90	50	100	85	106	80	7.46	7.52	7.47	31.3	22.3	27.5	595	597	617
Patient 12	76	43	78	80	92	80	7.59	7.58	7.57	21.5	21.4	25.3	678	665	646
MEAN	74	45	79	73	85	71	7.53	7.51	7.50	28.2	27.2	29.0	625	613	619
SEM	3	2	3	3	3	2	0.02	0.02	0.03	1.7	1.5	2.0	14	14	8
P	<0.001	<0.001		<0.02	<0.01		NS	NS		NS	NS		NS	NS	

slowly increased until the desired level of hypotension was achieved. The mean arterial blood pressure (MAP) rarely decreased by more than 50 per cent with either technique; when the surgical field became sufficiently dry before that level was attained, further reduction was not attempted. During hypotension, heart sounds, lead II of the EKG, arterial and right heart pressure waves, and urinary output were meticulously monitored. Measurements during hypotension were made after the lower arterial pressure had remained stable for 30–60 minutes. Hypotension was terminated prior to the completion of the operation. During closure of the wound and at least 30 minutes following the resumption of normal arterial pressure, recovery measurements were made. We attempted to have the anesthetic conditions and the patient's arterial blood pressure in this recovery period match those in the control period.

Results

Data from individual patients are presented in tables 1 and 2, and mean data for all 18 patients are illustrated in figure 1. During

TABLE 2. Hemodynamic and Blood-gas Data from Individual Patients before, during and after Hypo-

	MAP (torr)			HR (Beats/Min)			pH_a			Pa_{CO_2} (torr)			Pa_{O_2} (torr)		
	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery
Patient 13	73	56	83	60	55	55	7.52	7.76	7.51	29.4	12.9	27.9	565	537	594
Patient 14	75	67	97	75	85	65	7.64	7.74	7.58	17.4	10.6	20.6	618	590	604
Patient 15	93	46	80	110	95	80	7.45	7.62	7.44	31.2	16.0	28.8	576	571	603
Patient 16	78	57	—	90	110	—	7.60	7.59	—	20.1	18.9	—	619	663	—
Patient 17	75	58	93	95	86	90	7.48	7.72	7.65	29.3	16.0	19.6	596	553	612
Patient 18	90	53	82	80	80	70	7.37	7.80	7.61	24.7	14.3	22.6	562	585	592
MEAN	81	56	87	85	85	72	7.55	7.71	7.56	25.4	14.8	23.9	589	583	601
SEM	4	3	3	7	7	6	0.03	0.03	0.04	2.3	1.2	1.9	10	18	4
P	<0.001	<0.001		NS	NS		<0.01	<0.02		<0.01	<0.01		NS	NS	

during and after Hypotension Induced with Sodium Nitroprusside, Group A

$P\bar{v}_{O_2}$ (torr)			$\dot{Q}_s/\dot{Q}_t$ (Per Cent)			$\dot{Q}_t$ (l min m^2)			$a\text{-}\bar{v}C_{O_2}$ (ml 100 ml)			$\dot{V}_{O_2}$ (ml min m^2)			O_2 Delivery (ml min m^2)		
Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery
44	45	38	3.0	2.1	2.3	1.43	1.99	1.39	4.69	4.48	5.95	67.0	89.2	82.7	276	367	259
44	56	47	11.4	13.4	8.5	1.76	2.44	2.02	4.09	2.68	3.61	71.9	65.4	73.0	307	377	314
63	49	47	2.1	3.7	2.2	—	—	—	3.18	3.57	4.01	—	—	—	—	—	—
48	58	46	3.9	4.1	3.9	1.64	3.25	2.11	3.71	3.03	4.15	60.9	98.6	87.5	340	651	422
42	59	42	1.4	2.2	0.9	1.75	3.29	1.47	4.65	2.93	4.50	81.3	96.3	66.1	328	547	245
50	77	46	2.7	4.8	6.0	—	—	—	3.91	2.44	3.97	—	—	—	—	—	—
45	39	26	2.5	1.9	2.9	1.50	1.59	1.43	3.36	3.98	5.81	50.5	63.2	83.0	257	269	231
73	66	56	5.5	7.9	5.8	2.79	3.02	2.00	2.44	2.59	3.21	68.1	78.2	64.3	499	539	362
43	30	70	2.3	2.8	4.3	—	—	—	5.97	9.38	3.11	—	—	—	—	—	—
—	37	45	—	6.6	6.0	—	2.14	1.86	—	6.70	4.43	—	—	—	—	—	—
84	28	34	9.9	3.3	3.0	—	—	—	2.14	7.47	6.29	—	—	—	—	—	—
41	54	39	1.0	2.4	2.8	1.80	2.63	1.81	4.48	2.99	4.55	80.6	78.7	82.4	325	438	292
52	50	45	4.2	4.6	4.1	1.81	2.54	1.76	3.87	4.35	4.47	68.6	81.4	77.0	333	455	304
4	4	3	1.0	1.0	0.6	0.17	0.22	0.10	0.3	0.7	0.3	4.1	5.3	3.5	30	49	26
NS		NS	NS		NS	<0.02		<0.01	NS		NS	NS		NS	<0.05		<0.01

deliberate hypotension the mean arterial pressure decreased from 76.6 ± 2.2 torr to 48.6 ± 2.0 torr ($P < 0.001$), and with recovery it returned to 81.5 ± 2.2 torr. The shunt fraction, however, remained unaffected, as $\dot{Q}_s/\dot{Q}_t$ values were 5.2 ± 0.9 per cent, 5.4 ± 0.8 per cent, and 4.7 ± 0.5 per cent, respectively ($P > 0.1$).

Sodium Nitroprusside, Group A, Table 1

The hypotension achieved in Group A was more profound than that in Group B, but this was not intentional. The hypotension induced by sodium nitroprusside was accompanied by significant increases in cardiac index and heart rate, but stroke volume did not change. Respiration was constant throughout the study. Mild respiratory alkalosis was present, but no change in acid–base balance developed with induced hypotension. Pa_{O_2} was usually well over 600 torr and was not altered by hypotension. The shunt fraction remained between 4 and 5 per cent, and was thus unaffected by sodium nitroprusside. Oxygen delivery to the tissues increased during hypotension, but the apparent in-

tension Induced with Deep Halothane Anesthesia and Mechanical Hyperventilation, Group B

$P\bar{v}_{O_2}$ (torr)			$\dot{Q}_s/\dot{Q}_t$ (Per Cent)			$\dot{Q}_t$ (l min m^2)			$a\text{-}\bar{v}C_{O_2}$ (ml 100 ml)			$\dot{V}_{O_2}$ (ml min m^2)			O_2 Delivery (ml min m^2)		
Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery	Control	Hypotension	Recovery
36	26	41	6.5	7.3	6.5	1.29	1.26	1.61	5.11	6.14	4.21	65.9	77.4	67.8	221	223	266
29	30	31	3.1	6.5	4.1	—	—	—	7.42	4.66	6.65	—	—	—	—	—	—
61	49	59	10.5	10.3	7.7	2.61	1.90	1.99	2.74	2.88	3.23	71.5	54.8	64.3	457	320	405
37	36	—	4.7	1.1	—	1.94	1.88	—	4.43	4.74	—	—	—	—	—	—	—
55	33	41	7.4	8.2	5.3	2.67	1.10	1.57	3.07	4.66	4.14	82.0	51.3	65.0	451	190	281
52	37	43	10.8	8.0	7.3	1.27	1.30	1.37	2.86	3.62	3.74	36.3	47.1	51.2	208	220	238
45	35	43	7.2	6.9	6.2	1.96	1.49	1.64	4.27	4.45	4.39	63.9	57.7	62.1	334	238	298
5	3	5	1.3	1.3	0.7	0.30	0.17	0.13	0.7	0.5	0.6	9.8	6.8	3.7	69	28	37
<0.05		NS	NS		NS	NS		NS	NS		NS	NS		NS	NS		<0.05

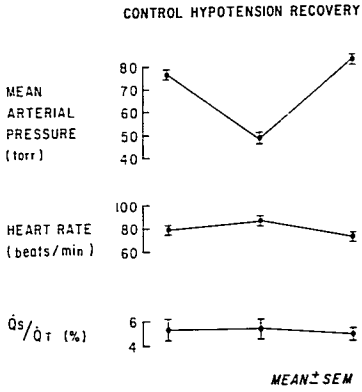

FIG. 1. Effects of deliberate hypotension on mean arterial pressure, heart rate, and pulmonary shunt.

crease in oxygen consumption was not statistically significant.

DEEP HALOTHANE ANESTHESIA AND HYPERVENTILATION, GROUP B, TABLE 2

The 30 per cent reduction in MAP achieved with this technique was sufficient to produce the desired surgical conditions. No change in heart rate occurred. Severe respiratory alkalosis was induced together with hypotension, but this excessive hyperventilation, which used to be advocated for craniotomies in our institution, has now been renounced and abandoned. No base deficit or base excess developed. In all three study periods the patients in Group B had Pa_{O_2} and $P\bar{v}_{O_2}$ values that were lower than those in Group A, so the corresponding shunts were higher; however, the hypotensive phase was not accompanied by any change in either $\dot{Q}s/\dot{Q}t$ or Pa_{O_2}. Cardiac index, stroke volume, oxygen delivery, and oxygen consumption all decreased during hypotension induced by deep halothane anesthesia and hyperventilation, but statistical significance was not achieved.

COMPARISON OF HYPOTENSIVE TECHNIQUES

As indicated above, there were differences in the states of hypotension induced by the two techniques. However, the shunt was unaffected by the degree of hypotension, the level of ventilation, or the change in cardiac index, as $\dot{Q}s/\dot{Q}t$ correlated poorly with blood pressure ($r = 0.04$), Pa_{O_2} ($r = 0.06$), and $\dot{Q}t$ ($r = 0.04$). During the control and recovery periods, when the anesthetic conditions of the two groups matched one another, cardiac index was less than 2.0 l/min/m². Hypotension induced by deep halothane anesthesia and hyperventilation resulted in an even greater depression of cardiac index, whereas sodium nitroprusside-induced hypotension was accompanied by an increased cardiac index, to 2.5 l/min/m², so the two groups differed significantly during the hypotensive period ($P < 0.01$). The change in cardiac index had no effect on arterial oxygenation, as $\dot{Q}t$ correlated poorly with Pa_{O_2} ($r = 0.1$). However, the change in cardiac index correlated well with tissue oxygen delivery ($r = 0.97$, $P < 0.001$), which increased during the administration of sodium nitroprusside and decreased with deep halothane anesthesia and hyperventilation. Oxygen consumption appeared to be similarly affected, but no significant change in $\dot{V}_{O_2}$ was induced by either hypotensive technique. Only when oxygen consumption in the hypotensive phase of Group A was compared with that in the hypotensive phase of Group B was a significant difference substantiated ($P < 0.02$).

In each study period $\dot{Q}s/\dot{Q}t$ was larger in Group B than in Group A, but small sample size kept this difference from being significant. Pa_{O_2} and $P\bar{v}_{O_2}$ were likewise higher in Group A, and again this difference was insignificant except for the comparison of $P\bar{v}_{O_2}$'s in the hypotensive phase ($P < 0.05$). The pulmonary shunt averaged 5 per cent during the study and was unaffected by hypotension induced by either technique.

Discussion

Impairment of pulmonary oxygen exchange during anesthesia is usually attributed to increased pulmonary shunts ($\dot{Q}s/\dot{Q}t$ with $FI_{O_2} = 1$), to increased ventilation/perfusion inequalities, or to both factors. Decreased arterial oxygen tensions and increased alveolar–arterial oxygen gradients have been observed during anesthesia with deliberate hypotension,[4–6] and ventilation/perfusion ab-

normalities have been suggested as the responsible physiologic mechanism.[3-6] However, increased pulmonary shunting has not been ruled out, and in this study we found that $\dot{Q}_s/\dot{Q}_t$ remained unchanged during deliberate hypotension induced with two different techniques. We did not apply positive end-expiratory pressure or other ventilatory maneuvers especially designed to retard alveolar collapse. The level of hypotension achieved was sufficient to produce the desired operating conditions, and matched in degree that reported to decrease Pa_{O_2}.[1-6] One hundred per cent oxygen was employed because it permitted an isolated examination of a single component of the alveolar–arterial oxygen gradient, *i.e.*, the shunt fraction. During ventilation with 100 per cent oxygen any appreciable decrease in Pa_{O_2} cannot be caused by ventilation/perfusion abnormalities or diffusion limitations, and therefore must be attributable to an increase in the shunt fraction.

Sodium Nitroprusside

Parson and Sullivan observed that in anesthetized dogs breathing oxygen, shunting remained constant during hypotension induced with sodium nitroprusside.‡ This report confirms their findings in man. $\dot{Q}_s/\dot{Q}_t$ remained approximately 4 per cent before, during, and after hypotension induced with sodium nitroprusside (Group A, table 1).

Deep Halothane Anesthesia

The pulmonary shunt in the presence of high concentrations of halothane has been examined previously. In dogs, Stone and Sullivan found that hypotension induced by halothane caused no significant change in $\dot{Q}_s/\dot{Q}_t$, which remained about 3 per cent.[12] Prys-Roberts *et al.*[13] measured shunting in man under hypotensive conditions similar to those employed here in Group B (table 2), although the respiratory alkalosis was not as profound. They, too, reported that $\dot{Q}_s/\dot{Q}_t$ did not increase with hypotension; however, in both human studies the shunts measured about 7 per cent throughout. This level of shunting is abnormally high[14] and probably reflects the advanced ages of Prys-Roberts' subjects and the preoperative sedation and bed rest imposed on our younger patients with cerebral aneurysms. Others have administered increasing concentrations of halothane vaporized solely in oxygen to healthy young volunteers.[15-17] With 2 per cent halothane, hypotension was significant, but no decrease in Pa_{O_2} occurred. Mixed venous blood P_{O_2} was not reported; thus, we cannot be assured that $\dot{Q}_s/\dot{Q}_t$ remained unchanged.

To our knowledge no one has measured the shunt fraction in anesthetized man during deliberate hypotension induced with ganglionic blocking drugs. Marshall[18] reported that $\dot{Q}_s/\dot{Q}_t$ increased from 6 to 30 per cent in dogs given trimethaphan, and implicated the bronchoconstrictive action of histamine as the cause. Similar results were observed by Skene and Sullivan,§ who found that the shunt fraction in anesthetized dogs increased from 3 to 8 per cent during trimethaphan administration and then decreased toward normal when the drug was discontinued. In any case these are the only two reports of increased shunting during deliberate hypotension, and this evidence may not be applicable to man as trimethaphan alters canine pulmonary circulation.[18] In fact, it is probably not germane to generalize about the pulmonary effects of all hypotensive agents on the basis of data obtained from a single drug or even a single class of drugs.

In summary, we found no increase in pulmonary shunting ($\dot{Q}_s/\dot{Q}_t$ with $FI_{O_2} = 1$) during deliberate hypotension induced with either sodium nitroprusside or deep general anesthesia. What is more, we are not aware of reports of such changes, and so feel justified in concluding that the shunt fraction does not increase during hypotension induced by sodium nitroprusside or high concentrations of halothane. However, there are reports of impaired pulmonary oxygenation during deliberate hypotension with $FI_{O_2} < 1$.[1-6] These occurred during both sodium nitroprusside[5,6] and trimethaphan[4] infusions, but not during hypotension induced with halothane and nitrous oxide alone.[16,19] An increase in ventilation/perfusion inequalities has been

‡ Parson NL, Sullivan SF: Effect of sodium nitroprusside hypotension on pulmonary blood gas distribution (personal communication).

§ Skene DS, Sullivan SF: Hypoxemia during anesthesia and induced hypotension (personal communication).

proposed as a physiologic explanation for the observed Pa_{O_2} decrease during deliberate hypotension,[3-6] and indeed this explanation may be correct, for in this study we were unable to demonstrate that hypotensive anesthesia caused any change in the shunt fraction. However, in order to state that ventilation/perfusion inequalities do increase during deliberate hypotension, these measurements must be made.

During breathing of oxygen Pa_{O_2} is primarily determined by the magnitude of the shunt. But if the shunt fraction remains fixed during oxygen breathing and deliberate hypotensive anesthesia, then arterial oxygenation can be noticeably altered by secondary determinants. Thus, if cardiac output changed during hypotensive anesthesia, Pa_{O_2} should be directly altered in a manner described by the Fick equation. However, even when the shunt fraction remains fixed, its magnitude controls the degree to which cardiac output alters Pa_{O_2}. When $\dot{Q}_s/\dot{Q}_t$ is small, as it was in this study, the effect on Pa_{O_2} is minimized, and it is not surprising that we were unable to detect any variation in Pa_{O_2} with either type of induced hypotension. However, had a large shunt been present during the hypotension induced by deep halothane anesthesia, then the effect of the decreased cardiac output would have been magnified, and the Pa_{O_2} would have been substantially reduced. On the other hand, the decreased Pa_{O_2} caused by that same hypothetical large fixed shunt would have been partially reversed by the increased cardiac output induced by sodium nitroprusside. In any case, it is important to realize that when cardiac output varies, the shunt fraction may be unaffected, as was the case here, but Pa_{O_2} is always influenced to some extent.

Oxygen Delivery and Oxygen Consumption

Tissue oxygen delivery depends upon both the quantity of oxygen in the arterial blood and the flow of that blood to the tissues. We did not observe a change in the arterial oxygen content during either hypotensive technique, but the delivery of oxygenated blood was reduced by deep halothane anesthesia and markedly enhanced by sodium nitroprusside. Thus, hypotension produced a change in tissue oxygen delivery that was directly and proportionately related to the cardiac index.

Deep halothane anesthesia decreases whole-body oxygen consumption,[12-15] and hyperventilation during light anesthesia increases it, though not to as great an extent.[20] Prys-Roberts *et al.* previously induced hypotension by combining 2 per cent halothane with hyperventilation and found a small reduction in $\dot{V}_{O_2}$,[13] which our data seem to confirm. In both studies of this form of hypotension, cardiac index was depressed more than oxygen consumption, reflecting increased capillary oxygen extraction.

The hypotension induced by sodium nitroprusside was accompanied in our patients by a 40 per cent increase in $\dot{Q}_t$, but not by any significant change in $P\bar{v}_{O_2}$, $a\text{-}\bar{v}C_{O_2}$ difference or $\dot{V}_{O_2}$. Others have reported that sodium nitroprusside increased cardiac index about 20 per cent during anesthesia,[21-23]‡ but whole-body oxygen consumption and oxygen contents of arterial and mixed venous blood remained unchanged.[21-23]‡ We cannot, however, explain how $\dot{Q}_t$ can increase without a commensurate change in either $\dot{V}_{O_2}$ or $a\text{-}\bar{v}C_{O_2}$ difference, or both.

Whole-body oxygen consumption and tissue oxygen delivery have been derived or calculated in this study, and as such are extremely dependent upon the accurate determination of their components. Caution is therefore warranted, as interpretation may be no more than speculative. However, it does appear that hypotension induced by sodium nitroprusside is accompanied by an increase in oxygen delivery to the tissues without any significant change in whole-body oxygen consumption, and that this favorable relationship does not hold for hypotension induced by deep halothane anesthesia and hyperventilation.

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Monitoring

MODIFIED ALLEN TEST One hundred twenty-eight adults (mean age 40.6 years) of both sexes underwent the standard Allen test. The test consisted of simultaneous compression of ulnar and radial arteries at the wrist while the subject opened and closed his hand several times until the hand was exsanguinated. The subject was then asked to open his hand fully while the radial of the ulnar artery was released. Complete capillary blush of the hand within 6 seconds indicated functional continuity of palmer arch. Ninety-three subjects (73 per cent) showed defects in reperfusion of the hand. A modified test was then performed on the same subjects in a similar manner except that subjects were carefully instructed not to hyperextend the fingers or the wrist upon release of the clenched fist. All but one person had normal capillary refill after release of either radial or ulnar arteries. A portable Doppler ultrasonic velocity detector with a transmision frequency of 7.9 MHz revealed normal radial and ulnar signals and normal augmentation of velocities in these vessels when the opposite artery was compressed in all but the same person mentioned above, who had no radial artery, showing complete correlation with the modified Allen test. *(Kamienski RW, Barnes RW: Critique of the Allen test for continuity of palmer arch assessed by Doppler ultrasound. Surg Gynecol Obstet 142: 861–864, 1976.)*