Cardiovascular Effects of Breathing 95 Percent Oxygen In Children With Congenital Heart Disease

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The hemodynamic effects of breathing 95% oxygen were evaluated in 26 children with congenital heart disease. Aortic, pulmonary arterial, right atrial, and pulmonary arterial wedge pressure, aortic and pulmonary artery oxygen saturation, and blood gas, cardiac index, and heart rate were measured in room air and after each patient had breathed 95% oxygen for 10 (n = 26) and 20 (n = 5) minutes. Measurements were repeated with the patient again breathing room air for 10 (n = 11) and 20 (n = 6) minutes. After 10 minutes of 95% oxygen, arterial partial pressure of oxygen increased from 85 ± 13 to 420 ± 89 torr (p <0.001). Aortic mean pressure increased from 80 ± 10 to 83 ± 10 mm Hg (p <0.01), and systemic vascular resistance increased from 20 ± 7 to 26 ± 8 U (p <0.001). The cardiac index decreased by 21% from 3.96 ± 0.94 to 3.12 ± 0.74 liters/min/m^2 (p <0.001) and the stroke index decreased by 11% (p <0.001). A 23% de-crease in oxygen consumption (p <0.001) was observed, and oxygen transport decreased from 763 ± 179 to 600 ± 161 ml O$_2$/min/m^2 (p <0.001). Cardiac index, stroke index, and systemic vascular resistance did not return to normal until 20 minutes after cessation of oxygen breathing. To determine whether reflex bradycardia is responsible for these oxygen-induced hemodynamic changes, heart rate was kept constant by atrial pacing in a second group of 5 patients. In these children, significant decreases in cardiac index, stroke index, and oxygen consumption, and increases in systemic vascular resistance also occurred with 95% oxygen. Thus, in children with acyanotic congenital heart disease, hyperoxia increases aortic pressure and systemic vascular resistance and decreases cardiac index, stroke index, oxygen consumption, and oxygen transport.

The medical and surgical therapy of children with heart disease frequently includes administration of oxygen. The goal is to improve systemic oxygen delivery in patients with cyanosis, heart failure, or pulmonary edema. Previous studies have indicated, however, that oxygen itself may significantly alter cardiovascular hemodynamic function. In animals and in adults without heart disease, high inspired oxygen concentrations have been shown to increase systemic arterial pressure and systemic vascular resistance (SVR), and to decrease heart rate and cardiac index (CI).[1-6] Similar studies have not been performed in children or in patients with heart disease. The present study was therefore designed to evaluate the hemodynamic effects of oxygen administration in children with acyanotic congenital heart disease.

Methods

The study involved 31 children with congenital heart disease who were undergoing diagnostic cardiac catheterization. There were 18 males and 13 females ranging in age from 0.2 to 15.5 years (mean 5.5). Twenty patients had previously undergone surgical repair of the following congenital heart defects: ventricular septal defect (n = 10), endocardial cushion defect (n = 3), patent ductus arteriosus (n = 3), D-transposition (n = 2), tetralogy of Fallot (n = 1), and total anomalous pulmonary venous return (n = 1). The remaining patients, who had not been operated on, had aortic stenosis (n = 3), cardiomyopathy (n = 3), aortic regurgitation (n = 1), mitral regurgitation (n = 1), pulmonary stenosis (n = 1), L-transposition with pulmonary stenosis (n = 1), and primary pulmonary hypertension (n = 1). No patient had an intracardiac shunt documented at cardiac catheterization although several appeared to have a small intrapulmonary right-to-left shunt. Five patients (Table I: Patients 2, 12, 14, 20, 25) had congestive heart failure requiring treatment with digoxin and diuretics. Patients 2, 24, 25 and 26 had moderate to severe pulmonary artery hypertension (mean pulmonary pressure ≥ 0.5 mean aortic pressure). Patients 1, 2, 5 to 7, 26 and 31 had multiple episodes of pneumonia and evidence on chest roentgenography of chronic pulmonary disease.

TABLE I Hemodynamic Effects of 95% Oxygen in 26 Children With Congenital Heart Disease: Baseline Data in 21% Oxygen and Data in 95% Oxygen for 10 Minutes

Patient	Diagnosis	Age (yr)/BSA	21% Oxygen (Baseline)								95% Oxygen (10 Minutes)							
			HR	CI	SI	AoP	SVR	VO_2	PaO_2	$PaCO_2$	HR	CI	SI	AoP	SVR	VO_2	PaO_2	$PaCO_2$
1	PDA ligation	6.5/0.70	79	4.57	58	72	14	125	70	41	72	3.14	44	78	23	86	482	43
2	Primary pulmonary hypertension	6.2/0.89	124	1.91	15	91	47	159	55	35	125	1.79	14	98	51	126	382	38
3	VSD repair	6.8/0.78	107	5.00	47	75	14	186	97	37	113	4.48	40	80	17	129	460	40
4	PDA ligation	10.5/1.10	68	3.09	45	82	23	126	98	40	58	2.36	41	82	30	94	442	42
5	VSD repair	3.2/0.48	120	4.79	40	83	16	173	82	51	105	4.48	43	84	18	...	399	50
6	VSD repair	2.1/0.43	120	4.27	36	62	13	132	85	44	112	3.67	33	70	17	108	466	44
7	ECD repair	2.0/0.51	110	4.15	38	77	18	139	83	44	100	3.17	32	73	22	...	368	45
8	VSD repair	6.2/0.71	105	5.40	51	90	16	196	92	39	85	3.66	43	100	26	162	488	30
9	VSD repair	1.0/0.36	80	3.61	45	70	18	188	84	37	70	2.77	40	75	25	149	342	43
10	AS	8.5/0.90	78	4.11	53	88	20	119	86	43	67	3.22	48	90	26	116	448	41
11	ECD repair	3.1/0.58	96	4.17	43	80	18	134	84	39	74	3.44	46	80	22	109	348	44
12	MR	11.8/1.00	86	3.30	38	80	22	117	99	43	79	2.46	31	80	30	96	450	37
13	AS	6.1/0.72	104	3.82	37	103	26	100	88	36	75	2.94	39	101	32	93	510	36
14	Cardiomyopathy	2.1/0.46	96	4.39	46	70	15	145	100	36	94	3.58	38	72	19	123	480	39
15	TAPVR repair	12.2/1.08	67	3.64	54	75	19	123	77	39	64	3.24	51	80	23	129	448	35
16	D-TGA, Mustard	1.6/0.47	115	3.19	28	78	23	118	73	36	108	2.72	25	85	29	98	260	49
17	ECD repair	5.0/0.57	107	3.75	35	72	17	147	83	46	102	3.30	32	80	22	134	392	38
18	AS	15.5/1.90	85	4.53	53	90	18	175	93	40	96	4.10	43	95	22	92	459	33
19	PS	1.4/0.40	116	3.92	34	90	22	113	81	39	96	3.35	35	90	25	80	495	34
20	AR	0.2/0.21	114	2.76	24	70	24	70	100	40	115	2.52	22	72	26	85	486	35
21	VSD repair	2.0/0.53	85	4.39	52	70	15	143	100	37	77	3.21	41	77	22	96	240	42
22	L-TGA, PS	12.0/1.25	135	3.91	29	85	21	123	80	37	140	3.21	23	88	26	87	489	37
23	Cardiomyopathy	0.3/0.20	141	6.25	44	103	16	242	80	40	126	4.16	33	103	24	147	510	40
24	VSD repair	3.5/0.81	88	2.96	34	70	21	108	82	39	73	2.44	33	68	25	78	515	39
25	Cardiomyopathy	3.0/0.46	143	2.39	17	85	31	182	56	40	126	1.54	12	77	43	99	401	36
26	PDA ligation	7.8/0.63	111	4.60	41	69	14	171	63	53	101	3.96	39	78	18	76	169	62
	Mean		103	3.96	39.8	80	20.1	144	84	40	94.3	3.12	35.3	82.9	26	112	420	40
	±SD		±21	±0.94	±11.1	±10	±6.9	±37	±13	±4	±22.4	±0.74	±9.7	±9.9	±7.7	±23	±89	±6
	Significance*										†	†	†	†	†	†	†	...

* Compared with baseline value in 21% oxygen.
† $p < 0.001$.
‡ $p < 0.01$.

AoP = mean aortic pressure (mm Hg); AR = aortic regurgitation; AS = aortic stenosis; BSA = body surface area (m²); CI = cardiac index (liters/min/m²); ECD = endocardial cushion defect; HR = heart rate (beats/min); MR = mitral regurgitation; PaO_2 = arterial partial pressure of oxygen (mm Hg); PDA = patent ductus arteriosus; PS = pulmonary stenosis; SD = standard deviation; SI = stroke index (ml/m²); SVR = systemic vascular resistance (Wood units); TAPVR = total anomalous venous return; TGS = transposition of the great arteries; VO_2 = oxygen consumption (ml/min/m²); VSD = ventricular septal defect.

TABLE II   Hemodynamic Effects of Breathing 95% Oxygen for 10 Versus 20 Minutes

| | 21% Oxygen | | | | | | | 95% Oxygen | | | | | | | | | | | | | |
| | | | | | | | | 10 Minutes | | | | | | | 20 Minutes | | | | | | |
Patient	HR	CI	SI	AoP	SVR	VO_2	PaO_2	HR	CI	SI	AoP	SVR	VO_2	PaO_2	HR	CI	SI	AoP	SVR	Vo_2	PaO_2
10	78	4.11	53	88	20	119	86	67	3.22	48	90	26	116	448	67	3.42	51	90	25	125	488
11	96	4.17	43	80	18	134	84	74	3.44	46	80	22	109	348	74	3.40	46	78	22	94	478
12	86	3.30	38	80	22	117	99	79	2.46	31	80	30	96	450	78	2.40	31	80	31	85	472
16	115	3.19	28	78	23	118	73	108	2.72	25	85	29	98	260	109	2.87	26	85	28	87	263
20	114	2.76	24	70	24	70	100	115	2.52	22	72	26	85	486	114	2.49	22	72	28	78	492
Mean	98	3.50	37	79	22	112	88	89	2.85	35	81	27	101	398	88	2.92	35	81.0	26	94	439
±SD	±16	±0.54	±12	±6	3	±24	±11	±22	±0.39	±12	±6	±3	±11	±93	±21	±0.43	±13	±7	±4	±18	±98
Signifi- cance*	...	...	...	...	...	...		...	...	...	...	...	...	...	NS	NS	NS	NS	NS	NS	†

* Compared with value in 95% oxygen for 10 minutes.
† $p < 0.05$.
NS = not significant at $p < 0.05$. Other abbreviations as in Table I.

Before catheterization, each patient was premedicated with morphine sulfate (0.15 mg/kg) and diphenhydramine (1.0 mg/kg). After right and left heart catheterization had documented the underlying lesion, and before angiography, the study was performed. Baseline hemodynamic variables were measured with the patient breathing room air. Heart rate was determined from the electrocardiogram and averaged over a 10-second period. Pressures were measured in the aorta, right atrium, pulmonary artery, and pulmonary artery wedge position. Aortic and pulmonary artery blood was sampled for blood gas (IL 813 analyzer) and oxygen saturation (reflection oximetry) measurements. All blood gas samples were collected in cold heparinized syringes, placed in ice, and analyzed within 5 minutes. Hemoglobin concentration was measured in an IL 182 co-oximeter, and oxygen-carrying capacity was calculated by multiplying the hemoglobin concentration by 1.36. CI was measured by thermodilution technique (IL 701 thermodilution computer) and taken as the average of 3 determinations. In 5 patients, CI was also measured by dye dilution technique. Oxygen consumption (VO_2) was measured using a continuous flow-through system in room air,[7] and was also calculated from thermodilution CI and oxygen content difference according to the Fick principle in both room air and 95% oxygen: VO_2 = thermal dilution cardiac output (10 × [oxygen capacity × aortic saturation + 0.003 × aortic PO_2]) − (oxygen capacity × pulmonary artery saturation + 0.003 × pulmonary artery PO_2), where PO_2 = partial pressure of oxygen. Core temperature was measured with thermodilution thermistor situated in the main pulmonary artery.

After acquisition of baseline data, each patient was administered 95% oxygen. This was the highest inspired oxygen concentration that could be reliably administered by a hood system. Inspired oxygen concentration (F_IO_2) was determined using an Applied Electrochemistry oxygen analyzer. Measurements of heart rate, pressures, blood gases and saturations, CI, and VO_2 were made as just described after oxygen breathing for 10 minutes (n = 26) and 20 minutes (n = 5). Hemodynamic measurements were then repeated with the patient once again breathing room air for 10 minutes (n = 11) and 20 minutes (n = 6).

To evaluate the hemodynamic significance of reflex bradycardia during oxygen breathing, heart rate was kept constant by atrial pacing (rate 5 beats/min above the spontaneous sinus rate) in 5 patients. Hemodynamic measurements were made in room air and after breathing 95% oxygen for 10 minutes as described previously.

Data were evaluated for significance using the two-tailed t test for paired observations. All values are expessed as mean ± standard deviation.

TABLE III   Duration of Oxygen-Induced Hemodynamic Changes

| | 21% Oxygen (Baseline) | | | | | | 95% Oxygen (10 Minutes) | | | | | |
Patient	HR	CI	SI	AoP	SVR	VO_2	HR	CI	SI	AoP	SVR	VO_2
10	78	4.11	53	88	20	119	67	3.22	48	90	26	116
12	86	3.30	38	80	22	117	79	2.46	31	80	30	96
14	96	4.39	46	70	15	145	94	3.58	38	72	19	123
18	85	4.53	53	90	19	175	96	4.10	43	95	22	92
22	135	3.91	29	85	21	123	140	3.21	23	88	26	85
25	143	2.39	17	85	31	182	126	1.54	12	77	43	99
2	124	1.91	15	91	47	159	125	1.79	14	98	51	126
5	120	4.79	30	83	16	173	105	4.48	43	84	18	...
16	115	3.19	28	78	23	118	108	2.72	25	85	29	98
17	107	3.75	35	72	17	147	102	3.30	31	80	22	134
19	116	3.92	34	90	22	113	96	3.35	35	90	25	80
21	85	4.39	52	70	15	143	77	3.15	41	77	22	96
Mean	107	3.71	37	82	22	143	101	3.07	32.0	85	28	104
±SD	±21	±0.83	±13	±8	±9	±25.1	21	±0.81	±11.4	±8	±10	±18
Signifi- cance*												

* Compared with baseline value in 21% O_2.
Abbreviations as in Table I.

Results

After 10 minutes of breathing 95% oxygen, mean arterial PO_2 increased from 86 ± 13 to 420 ± 89 torr (p <0.001) and arterial oxygen content increased from 17.0 ± 1.9 to 18.7 ± 2.0 ml/100 ml (p <0.001). There was a slight increase in these values after 20 minutes of oxygen breathing. The increase in mean arterial PO_2 to only (420 ± 89 torr) after 10 minutes of breathing 95% oxygen suggests that some degree of intrapulmonary right-to-left shunting was present in our patients. Arterial pH, PCO_2, and temperature did not change during the study.

Significant hemodynamic effects were noted after 10 minutes of oxygen breathing (Table I). Heart rate decreased from 103 ± 21 to 94 ± 22 beats/min (p <0.001). Mean systemic arterial pressure increased from 80 ± 11 to 83 ± 10 mm Hg (p <0.01), and mean pulmonary arterial pressure decreased from 24 ± 12 to 22 ± 11 mm Hg (p <0.01). Mean right atrial pressure increased from 5 ± 2 to 6 ± 2 mm Hg (p <0.01), while the small observed increase in pulmonary arterial wedge pressure from 10 ± 5 to 11 ± 5 mm Hg (p = 0.15) did not reach statistical significance.

CI measured by thermodilution technique was diminished in every patient after 10 minutes in 95% oxygen. Mean CI decreased by 21% from 3.96 ± 0.94 to 3.12 ± 0.74 liters/min/m^2 (p <0.001). Stroke index decreased by 11% from 39.9 ± 11.1 to 35.5 ± 9.7 ml/m^2 (p <0.001). In 5 patients, CI determined by dye dilution did not differ significantly from simultaneous thermodilution measurements.

Oxygen breathing resulted in a significant elevation of SVR from 20.1 ± 6.9 to 25.5 ± 7.7 U (p <0.001). Pulmonary vascular resistance did not change significantly.

VO_2 was calculated from thermodilution cardiac output according to the Fick principle. In room air, this calculated VO_2 did not differ significantly from VO_2 measured by a continuous flow-through system (150 ±

26 versus 156 ± 24 ml/min/m^2, respectively). After 10 minutes of breathing 95% oxygen, VO_2 decreased by 24% (p <0.001) (Table I). Oxygen transport, the product of arterial oxygen content and cardiac output, decreased as well, from 762.9 ± 179.4 to 600.2 ± 161.3 ml O_2/min/m^2 (p <0.001).

After 20 minutes of oxygen breathing, no further hemodynamic changes were noted. The data in Table II indicate that hemodynamic measurements made after 20 minutes did not differ significantly from those made after 10 minutes of breathing 95% oxygen. Thus, oxygen seems to exert its major cardiovascular effects within 10 minutes. Ten minutes after cessation of oxygen breathing, however, all values had not returned to baseline (Table III). CI, stroke volume, oxygen transport, and VO_2 remained significantly depressed and SVR remained elevated (p <0.01). These and all other hemodynamic parameters returned to normal after 20 minutes after oxygen breathing.

The effects of breathing 95% oxygen on the 5 patients with congestive heart failure are presented in the Figure 1. These data are highlighted since the hemodynamic effects of oxygen in such patients may be of particular clinical relevance. When compared with the 21 patients without failure, oxygen decreased CI and stroke volume and increased SVR in the 5 children with congestive failure. CI decreased from 2.95 ± 0.95 to 2.38 ± 0.79 liters/min/m^2 (p <0.05), stroke index decreased from 28.1 ± 13.4 to 23.6 ± 11.1 ml/m^2 (p <0.05), and SVR increased from 27.7 ± 11.8 to 34.0 ± 13.1 U (p <0.05).

To evaluate the role played by reflex bradycardia during oxygen breathing, heart rate was held constant by atrial pacing in 5 patients. Hemodynamic values measured before oxygen and after breathing 95% oxygen for 10 minutes are presented in Table IV. CI and stroke index both decreased by 12% (p <0.05). VO_2 also decreased from 146.2 ± 42.2 to 97.0 ± 12.0 ml/min/m^2 (p <0.05). SVR increased in oxygen from 22.8 ± 5.1 to 26.4 ± 5.5 U (p <0.05).

TABLE III (Continued)

| | Return to 21% Oxygen | | | | | | | | | | | |
| | 10 Minutes | | | | | | 20 Minutes | | | | | |
Patient	HR	CI	SI	AoP	SVR	VO₂	HR	CI	SI	AoP	SVR	VO₂
10	79	3.88	49	100	...	118	...	...	...	...	...	...
12	92	2.80	30	80	26	110	...	...	...	...	...	...
14	96	3.91	41	70	17	130	...	...	...	...	...	...
18	94	4.26	45	90	18	148	...	...	...	...	...	...
22	142	4.24	30	100	22	133	...	...	...	...	...	...
25	137	1.80	13	72	...	134	...	...	...	...	...	...
2	...	...	...	...	...	...	130	1.93	15	90	45	164
5	116	3.60	31	66	16	118	120	4.79	40	66	12	157
16	112	2.76	25	78	26	102	115	3.19	28	78	23	124
17	107	3.51	33	72	18	145	107	3.85	36	72	16	159
19	105	3.38	32	90	26	108	110	3.75	34	90	23	120
21	84	3.64	43	66	17	124	85	4.33	51	68	14	148
Mean	106	3.43	33.8	80	21	124	111.2	3.64	34	77	22	145
±SD	±20	±0.69	±10.2	±13	±4.2	±15	±15.2	±0.84	±12.1	±11	±11.8	±19
Significance*	NS	†	‡	NS	†	‡	NS	NS	NS	NS	NS	NS

†p <0.01.
‡p <0.05.
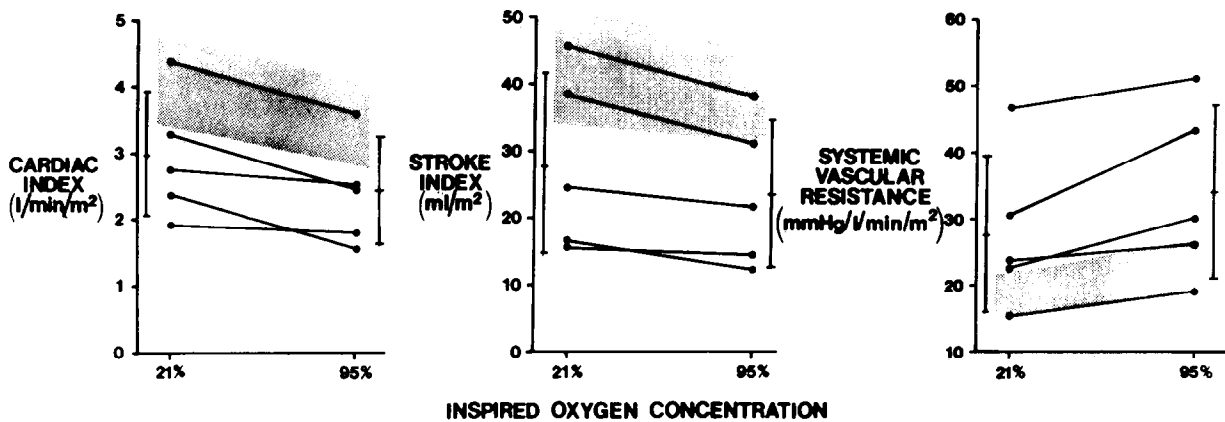
FIGURE 1. Cardiovascular effects of oxygen.

Discussion

This study documents significant hemodynamic changes induced by the breathing of 95% oxygen in children with congenital heart disease. Oxygen breathing increased SVR and arterial pressure, and decreased heart rate, CI, and stroke volume. A marked decrease in VO_2 was observed as well. These hemodynamic changes occurred in all patients regardless of cardiac diagnosis or the presence of intrapulmonary right-to-left shunting.

Several studies have documented an increase in SVR induced by hyperoxia.[1-6,8] Eggers et al[1] noted a 22% increase in 10 healthy adults breathing 100% oxygen for 20 minutes. Torbati et al,[5] studying rats exposed to high oxygen tension, concluded that the elevated SVR was due primarily to vasoconstriction in large muscle groups. This is consistent with observations, using venous occlusion plethysmography, that forearm blood flow is reduced by 18.9% in adults breathing oxygen at 2 atm pressure.[8] Hyperoxia has been shown to affect SVR by increasing precapillary sphincter tone.[9] The mechanism by which oxygen increases SVR remains unclear, however. A direct constrictor effect of oxygen on arteriolar smooth muscle and chemoreceptor-mediated reflex vasoconstriction have been postulated.[20] The observa-

tion that significant hemodynamic effects persist for 20 minutes (40 minutes in Egger et al's study[1]) despite normalization of arterial oxygen content suggests that a circulating vasoconstrictor substance may also be involved in mediating the cardiovascular response to hyperoxia.

A decrease in heart rate during oxygen breathing has also been reported by a number of investigators.[1-3,5,11,12] Daly and Bondurant[2] demonstrated an inverse linear relationship between heart rate and inspired oxygen concentration in 15 normal adults. After administration of atropine, however, heart rate did not decrease in response to high inspired oxygen concentrations. Thus, it appears that hyperoxia-induced bradycardia may be mediated by the baroreflex in response to an increase in arterial pressure.

CI decreases in response to hyperoxia.[1-6,13] Our data indicate that this is due to the combined effects of a decrease in both heart rate and stroke volume. CI decreased by 21% after 10 minutes of breathing 95% oxygen. In the 5 patients whose heart rates were kept constant by atrial pacing, CI decreased by 12%, entirely the consequence of a diminished stroke volume. This 12% decrease in stroke volume was almost identical in magnitude to that observed in the 26 nonpaced patients.

TABLE IV   Hemodynamic Effects of Breathing 95 % Oxygen in 5 Children During Atrial Pacing

Patient	Diagnosis	Age (yr)	21% Oxygen (Baseline)								95% Oxygen (10 Minutes)							
			HR	CI	SI	AoP	SVR	VO_2	PaO_2	$PaCO_2$	HR	CI	SI	AoP	SVR	VO_2	PaO_2	P_aCO_2
27	Mustard repair of D-TGA	2.0	85	3.24	38	68	20	132	94	39	85	2.80	33	80	27	90	480	37
28	VSD repair	12.0	105	3.48	33	97	26	97	98	38	105	3.32	32	108	29	85	390	38
29	TOF repair	5.0	125	4.71	38	97	20	213	96	33	125	3.91	31	93	23	103	420	35
30	VSD repair	10.0	95	3.50	37	115	30	140	88	38	95	2.93	31	110	33	91	391	39
31	VSD repair	1.4	115	3.48	30	69	18	149	68	42	115	3.30	29	72	19	115	248	44
Mean	. . .		105	3.68	35	89.2	23	146	89	38	105	3.25	31	93	26	97	386	39
±SD			±16	±0.59	±3	±20.3	±5	±42	±12	±3	±16	±0.43	±2	±17	±6	±12	±85	±3
Significance*	. . .		. . .	. . . . . .			. . .	. . .	. . .	. . .	NS	†	†	NS	†	†	†	. . .

* Compared with baseline value in 21% O_2.
† $p < 0.05$.
NS = not significant at $p < 0.05$.
Abbreviations as in Table I.

Thus, the decrease in CI during oxygen breathing is due to a decrease in stroke volume as well as heart rate. In contrast, Daly and Bondurant[2] failed to demonstrate a statistically significant decrease in stroke volume in 6 patients during oxygen breathing when reflex bradycardia was abolished by atropine administration. Their data show, however, that stroke volume did decrease in 4 subjects and was unchanged in 2.

The oxygen-induced decrease in stroke volume may be due to multiple factors. First, it may reflect an increased SVR and left ventricular afterload. This factor may be of particular significance in children with heart disease. Second, the decrease in stroke volume may relate to myocardial depression by hyperoxia. Several studies have documented diminished ventricular function in animals exposed to high oxygen concentrations.[10–16] Kioschos et al,[15] for example, found a 14% decrease in the first derivative of left ventricular pressure in dogs during hyperbaric oxygenation at 3.6 atm for 15 minutes. Finally, hyperoxia has been shown to decrease coronary artery blood flow,[14,17] which may further compromise myocardial function.

The hemodynamic response to hyperoxia may be of particular clinical relevance in children with congestive heart failure and already compromised ventricular performance. Figure 1 illustrates some of the hemodynamic changes induced by breathing 95% oxygen for 10 minutes in 5 children with heart failure. In room air, these children already had a mean CI and stroke index well below, and a mean SVR well above the group as a whole. Nevertheless, oxygen administration caused a further 19% decrease in CI and 16% decrease in stroke index. SVR resistance increased by 23% from 27.7 to 34 units. These potentially adverse hemodynamic effects were noted in each child after 10 minutes of oxygen administration.

In the present study, VO_2 decreased by 25% after 10 minutes in 95% oxygen. A similar marked decrease in VO_2 has been reported during hyperbaric oxygenation.[13] The mechanisms involved are unknown but one can speculate that peripheral vasoconstriction and decreased cardiac output may cause underperfusion of metabolizing tissues. In fact, the present data have shown that oxygen transport, the volume of oxygen delivered to the tissues each minute, is depressed by hyperoxia. Oxygen in high concentration may also directly inhibit oxidative metabolism. In rabbits exposed to oxygen at 3 atm a marked lactic acidosis develops,

supporting the concept that aerobic metabolism is inhibited by hyperoxia.[18]

The present study has a number of potential clinical implications. First, at cardiac catheterization it is common to assess the hemodynamic response to oxygen breathing of patients with intracardiac shunts or pulmonary vascular obstructive disease. Our findings suggest that pulmonary blood flow may be overestimated and pulmonary resistance underestimated if one assumes that oxygen consumption in oxygen is the same as that measured in room air. Second, the medical and surgical therapy of children with acyanotic heart disease often includes administration of oxygen in the range of inspired oxygen concentration of 30 to 50%. Thus, further investigations are warranted to determine if similar adverse hemodynamic changes occur when these therapeutic concentrations of oxygen are given.

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