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Cardiac shunt
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Ductus Arteriosus Shunts in the Respiratory Distress Syndrome

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Extract

The arterial blood gas tensions in the right radial artery and the lower abdominal aorta were compared in 21 infants suffering from respiratory distress syndrome. Simultaneous sampling was carried out using a radial artery catheter inserted by a cut-down technique and an umbilical arterial catheter inserted into the abdominal aorta. One hundred thirty-nine simultaneous blood samples were analysed for paO_2, pH, and pCO_2. In no case was there any significant difference in pH and pCO_2 values in samples of blood drawn from the two sites. Differences in paO_2 were observed but owing to inherent errors in measurement, only differences of greater than 10 mm Hg were regarded as significant. In 27 out of the 139 determinations, paO_2 from the right radial artery exceeded that from the abdominal aorta by more than 10 mm Hg. The babies studied were divided into the following groups: Group 1, 6 babies with no significant paO_2 difference between the two sampling sites. Group 2(a), 6 babies with significantly higher oxygen tensions in blood drawn from the radial artery but only at aortic paO_2 levels of more than 140 mm Hg. In this group there were no significant differences when the abdominal aorta paO_2 was less than 140 mm Hg. Group 2(b), 3 babies with slightly higher radial artery oxygen tensions only when they were being ventilated with a positive pressure respirator. Group 2(c), 6 babies with significantly higher paO_2 levels in the radial artery at arterial oxygen tensions of less than 100 mm Hg. In two of these babies, the right radial paO_2 (and therefore presumably the retinal artery paO_2) would have exceeded 160 mm Hg had the ambient oxygen concentration been raised sufficiently to produce an aorta paO_2 of 100 mm Hg and had the total shunt and the proportion passing through the ductus arteriosus remained constant. In two of these babies, however, increasing ambient oxygen concentration decreased the total shunt and the proportion of the shunt passing through the ductus. In the remaining four babies in Group 2(c), the severity of the disease was such that it was impossible to raise the aorta paO_2 to 100 mm Hg.

Speculation

From these data it seems that most of the R → L shunt in respiratory distress syndrome must pass through the foramen ovale or the lungs. Only rarely does more than 15% of the R → L shunt pass through the ductus arteriosus.

The final quantitative differentiation between shunting in the lungs and through the foramen ovale is most likely to be made by dye dilution studies from peripheral vein or right atrial injections.

Introduction

It has been established that the major cause of the hypoxemia in respiratory distress syndrome (RDS) is a net right to left shunt of cardiac output [26, 27]. There are three possible sites for the shunt: the foramen ovale, the ductus arteriosus, and the lungs, either by direct vascular bypass through the bronchial circulation or by pulmonary arterial blood coming into contact with unventilated alveoli. There is good qualitative evidence that bidirectional shunts occur in all these sites [12, 17, 25]. During the first 24–48 hours of life, the pulmonary artery pressure is close to systemic levels and falls steadily during this time [5]; nevertheless, R → L shunting through the ductus has been demonstrated in normal newborn babies at this age [4, 16, 19]. In the hypoxic neonate with RDS, the pulmonary artery pressure is likely to rise and closure of the ductus will be delayed [2, 13, 15, 23]; both these factors will increase the likelihood of a R → L shunt occurring through the ductus arteriosus. In very sick babies with RDS studied by cardiac catheterization, however, net R → L shunts through the ductus arteriosus were not demonstrated [22].

In recent years, measurement of arterial oxygen tension has been used as a guide to oxygen therapy in newborn infants with respiratory illnesses. The umbilical artery has provided the most convenient source of arterial blood. Umbilical arterial catheters are usually placed to lie in the abdominal aorta and will therefore be sampling post-ductal blood. One purpose of measuring paO_2 is to prevent dangerously high levels that might cause retrolental fibroplasia. If there is a net R → L shunt through the ductus arteriosus, blood reaching the retina will have a higher oxygen tension than that in the abdominal aorta, and dangerously high retinal levels could occur at normal aortic paO_2 values. STAHLMAN [25] and OLIVER *et al.* [18] have reported babies who had much higher paO_2 levels in left atrial blood than in peripheral arterial samples. Recently, CHU *et al.* [3] have reported the presence of R → L shunts at a ductus level in 14 babies by comparing temporal artery and abdominal aorta pO_2 values.

Thus, when using abdominal aorta paO_2 as a guide to ambient oxygen concentration, there is a risk of being misled, in the presence of substantial R → L shunts through the ductus arteriosus, and of subjecting babies to dangerously high retinal blood oxygen tensions.

In order to study this problem, we supplemented our routine management of babies with respiratory distress [20] by inserting catheters in the right radial artery. Radial artery catheterization for sampling preductal blood was chosen for several reasons. First, direct arterial puncture is painful and makes the baby cry, which is known to raise the pulmonary artery pressure and therefore the R → L shunt. Second, we have had no success with the technique of cannulation of the temporal artery for serial samples using a scalp vein needle. Third, capillary samples may be particularly unreliable for measurement of paO_2 [14]. We chose the right radial artery because the left subclavian artery occasionally arises beyond the ductus arteriosus and also because turbulence in the aorta from blood coming out of the ductus may cause some deoxygenated blood to enter a normally situated left subclavian artery. Our technique allowed the blood to be sampled frequently without disturbing the sick newborn baby.

This report presents the results of blood gas estimations on samples obtained simultaneously from the right radial and umbilical arteries in 21 infants with RDS.

Material and Methods

The babies were studied during a two-year period. Eleven were born in Hammersmith Hospital. Ten were born in outside units and transferred to this center for management. They were all diagnosed clinically as having RDS by exhibiting two or three of the following signs at four hours of age: a respiratory rate of 60 or more, expiratory grunting audible without a stethoscope, and chest retraction. X-rays were taken and were compatible with the clinical diagnosis. Apart from diagnosis of RDS, no selection of cases was carried out other than that imposed by the availability of research staff and technical facilities at the time of admission and diagnosis. The babies were nursed in Oxygenaire Series III incubators at high humidity. The incubator temperature was regulated to maintain the baby's rectal temperature between 35 and 37°. Occasionally, readings were outside that range, usually immediately after admission, when slightly lower values were recorded. The lowest recorded rectal temperature was 34.4° and the highest, 37.8°. In the latter half of the study, babies were also covered by a radiant heat shield as described by HEY and MOUNT [11]. An early feeding regime was used [24] and infants were given intravenous therapy only for hypoglycemia (true glucose less than 20 mg/100 ml) or if they were not tolerating oral feeding. Antibiotics were not given routinely but if suspicion of infection existed, patients were treated with penicillin, kanamycin and polymixin.

Umbilical artery catheters were inserted as soon as possible after admission to the Neonatal Ward. FG 5 polyvinyl feeding tubes were inserted to a distance calculated to place the tip of the catheter just below the diaphragm [9]. To sample preductal blood, a FG 2 polyvinyl intravenous catheter of external diameter

0.63 mm was inserted into the right radial artery at the wrist by a cut-down technique. The procedure is particularly easy in very small premature infants who have little subcutaneous fat. The deep fascia was divided and the artery cleaned. The superficial palmar branch may be large and torn at this stage. Gentle pressure over the artery, however, usually staunches the flow and the procedure is not jeopardized. The artery was tied distally with catgut and a small nick made in its wall. Hemorrhage can usually be easily controlled by traction on the distal tie. If this fails, a small bulldog clamp can be applied proximally with good control of bleeding. The catheter was then inserted and not pushed farther than the division of the brachial artery into its ulnar and radial branches. It was held in place with catgut and maintained patent with heparinized saline (10 units/ml). Constant perfusion was not found necessary. The skin was closed with interrupted atraumatic 5–0 black silk sutures. To remove the catheter, it was simply pulled out and pressure applied to the artery for 5 to 10 minutes. The radial artery pulse usually disappeared afterwards but the ulnar pulse became more marked. At no stage before or after the procedure did we observe any signs that circulation to the hand had been compromised. Hemorrhage was never a problem and the infants had no sequelae apart from an absent radial pulse.

Once catheters were *in situ*, the babies were managed in our conventional way [8, 20]. Sufficient oxygen was given to maintain paO_2 at satisfactory levels and acidosis was corrected by intravenous injections of THAM or sodium bicarbonate. Eight of the babies deteriorated sufficiently to need artificial ventilation [7]; four of them survived.

Blood samples were withdrawn simultaneously from both catheters, the dead spaces of which were filled with heparin (1000 units/ml), into new disposable plastic syringes. The baby's temperature was noted and the oxygen concentration in the incubator measured with a Beckman D2 oxygen analyzer. The blood gases were measured immediately after blood was withdrawn. Levels of paO_2, pH, and pCO_2 were measured at 37° on an IL blood gas apparatus, which was calibrated with gases of known oxygen and CO_2 concentrations and buffers of known pH. In calculating pO_2, corrections were applied for the difference between the measured pO_2 of a gas mixture and the pO_2 of blood tonometered with it, a value 3 % lower than that of true pO_2 [20]. Temperature corrections for pCO_2 and for pO_2 less than 200 mm Hg were made according to BRADLEY *et al.* [1]; for pO_2 more than 200 mm Hg, the factors of HEDLEY-WHITE and LAVER [10] were used. The pH was corrected to body temperature of the subject by the method of ROSENTHAL [21]. The R → L shunt was calculated by the method of STRANG and MCLEISH [26], assuming an A/V oxygen content difference of 4 ml/100 ml of blood. It is recognized that, at low environmental oxygen concentrations, there are errors in the calculation of the shunt due to ventilation-perfusion imbalances. These errors would tend to increase the calculated size of the shunt and are recognized in the discussion of the results.

Results

In the 21 babies studied, between one and seventeen simultaneous blood samples were taken to estimate paO_2, pH, and pCO_2. In no case was there any significant difference between pH and pCO_2 values obtained from the two sites. Many values for paO_2 differed in the radial and umbilical arterial samples, and in all cases the radial value was higher than the umbilical one. There is an error of ± 5 mm Hg in using the pO_2 electrode [6]. We had confirmed this figure in trials in which we had measured the pO_2 of tonometered blood; therefore we decided to regard as not significant all pO_2 differences of 10 mm Hg or less. On this basis, it was possible to divide the babies into two groups.

Group 1. Those babies with no significant paO_2 differences (i.e., no pair of blood samples had paO_2 values differing by more than 10 mm Hg). There were six such babies (table I).

Group 2. Those babies who sometimes showed paired samples with a radial paO_2 value more than 10 mm Hg higher than that of the aortic paO_2 value. They were divided into three groups.

(a) Six babies who showed occasional radial paO_2 more than 10 mm Hg higher than values for aortic paO_2, but only when aortic paO_2 values were more than 140 mm Hg (often obtained during a nitrogen washout), never when aortic paO_2 values were below this (table II).

(b) Three babies who showed occasional radial paO_2 values of more than 10 mm Hg higher than aortic paO_2 values while on intermittent positive pressure ventilation. These differences were again at paO_2 values higher than we usually attempt to maintain our babies (table III).

(c) Six babies who showed occasional radial paO_2 values of more than 10 mm Hg higher than aortic paO_2 at aortic paO_2 values of less than 100 mm Hg (table IV).

This latter group, 2(c), must be regarded as the most significant since the differences occurred within the range of paO_2 at which we normally maintain babies. The records of these six babies were carefully scrutinized to try to identify some common feature that might separate them from the other 15. None was found. Their weights and gestational ages covered a large span from 1260–2930 g and from 31 weeks to term

Table I. Group 1. Babies with no recorded differences between radial and aortic paO_2

Baby No.	Birth weight	Gestational age	Age	F_IO_2	paO_2, mm Hg		% R → L shunt calculated:		% shunt through ductus	Comment
	g	weeks	h		Aortic	Radial	Aortic	Radial		
1	1320	$28^6/_7$	3	0.60	60	63	35	34	+1	died with
			3 ¼	1.00	121	117	32	32	—	IVH, pneu-
			4	0.85	105	99	29	29	—	mothorax
			8 ¾	0.86	56	57	42	41	+1	and hyaline
			13 ¼	0.80	37	37	58	58	—	membrane
			16 ¾	0.93	60	58	40	41	—1	disease
2	1160	$31^4/_7$	52	1.00	15	16	>75	>75	—	died with
			53 ¼	1.00	30	29	68	69	—1	IVH and hyaline membrane disease
3	1840	34	17 ½	0.88	78	81	35	34	+1	alive and
			19 ¾	0.88	65	58	39	42	—3	well
			21 ½	0.96	106	100	33	33	—	
			30 ¾	0.92	91	101	33	32	+1	
			40 ¾	0.72	50	50	44	44	—	
			44 ¾	0.70	36	36	58	58	—	
			56 ¼	0.94	60	63	41	40	+1	
			65 ¾	0.94	86	76	35	37	—2	
			69	0.96	87	96	35	33	+2	
4	1220	29	5 ½	0.38	135	135	10	10	—	alive and
			6	0.20	64	61	18	20	—2	well
			7	1.00	326	318	21	21	—	
			14	0.34	75	80	21	19	+2	
			14 ¾	0.20	73	75	12	11	+1	
			15 ¼	0.20	49	45	32	36	—4	
5	1600	30	19 ½	0.92	48	48	48	48	—	died with
			24 ¾	0.92	50	51	47	47	—	IVH and hyaline membrane disease
6	2790	37	5	0.50	71	71	28	28	—	alive and
			5 ¾	1.00	117	115	32	32	—	well
			9 ½	0.68	79	76	29	30	—1	
			10	0.74	59	61	38	37	+1	

F_IO_2 = Inspired oxygen 1.00(I) = 100 % oxygen by intermittent positive pressure ventilation
IVH = Intraventricular hemorrhage.

Table II. Group 2(a). Babies with radial paO_2 > 10 mm Hg higher than aortic paO_2 at high paO_2 levels

Baby No.	Birth weight	Gestational age	Age	F_IO_2	paO_2, mm Hg		% R → L shunt calculated:		% shunt through ductus	Ductus shunt / Total[1] shunt	Comment
	g	weeks	h		Aortic	Radial	Aortic	Radial		%	
10	1760	31 ½	5 ½	0.72	161	176*	21	20	+1	4.8	alive and well
			7 ¼	0.48	65	63	30	29	+1		
			11 ¼	0.56	83	83	26	26			
			15 ¾	0.56	128	132	19	18	+1		
			21 ½	0.42	97	101	17	16	+1		
			38 ½	0.40	80	84	21	19	+2		
			44 ½	0.40	76	71	23	24	—1		
11	1400	29	120	0.95	143	188*	29	26	+3	10.4	alive and well
12	1800	37	3 ½	0.94	283	343*	21	17	+4	19.1	alive and well
			4 ¼	0.52	183	179	12	13	—1		
			4 ¾	0.34	118	113	10	11	—1		
13	1300	32	3 ¼	0.30	99	96	10	10			alive and
			3 ¾	1.00	298	327*	22	20	+2	9.1	well
			16 ¾	0.28	97	98	10	10			
			21 ¼	0.25	78	76	14	16	—2		
			21 ¾	1.00	326	369*	21	19	+2	9.1	
14	1500	32	4 ½	0.74	170	190*	21	19	+2	9.55	alive and well
			6	0.56	120	116	20	20			
			19	0.30	92	90	16	16			
			22	0.40	96	97	17	17			
			22 ¼	1.00	220	230	26	26			
15	2260	37	9 ½	0.48	61	59	31	32	—1		alive and
			9 ¾	1.00	156	182*	30	28	+2	6.6	well
			10 ½	0.58	61	61	34	34			
			26	0.90	72	71	37	38	—1		
			32 ½	0.94	66	63	40	41	—1		
			37 ½	0.90	49	55	47	43.5	+3.5		
			47	0.92	47	49	48	47	+1		
			52 ½	0.90	49	49	47	47			
			56 ½	0.92	49	50	47	47			
			61 ½	0.90	50	53	47	45	+2		
			71 ½	0.97	70	67	38	39	—1		
			75 ½	0.97	63	68	40	39	+1		
			127 ½	0.64	76	75	30	30			
			128	1.00	209	224	26	25	+1	3.8	
			146 ½	0.38	53	58	35	29	+6	17.2	
			147	1.00	177	182	29	29			

See table I for abbreviations.

* Significant differences between aortic and radial paO_2.

[1] Calculated when the % of the cardiac output passing through the ductus was > 5 % and when the radial paO_2 was > 10 mm Hg higher than the aortic paO_2.

Table III. Group 2(b). Babies with radial paO_2 > 10 mm Hg higher than aortic paO_2 when being artificially ventilated

Baby No.	Birth weight	Gestational age	Age	F_IO_2	paO_2, mm Hg		% R → L shunt calculated:		% shunt through ductus	Ductus shunt / Total[1] shunt %	Comment
	g	weeks	h		Aortic	Radial	Aortic	Radial			
7	1380	33	11	1.00	16	18	> 75	> 75			alive and
			11 ¼	1.00 I	108	105	33	33			well
			22 ¼	1.00	88	88	34	34			
			22 ¾	1.00	38	36	59	60	—1		
			27 ½	1.00 I	59	54	43	46	—3		
			31 ½	1.00	90	90	35	35			
			36 ½	1.00	61	60	42	42			
			50 ½	1.00	106	117*	33	32	+1	3.2	
			102 ½	0.62 I	127	141*	21	20	+1	5	
			125 ½	0.94	71	70	38	38			
			127 ½	0.94	33	32	65	66	—1		
			133	1.00	38	44	59	53	+6	10.3	
			133 ¾	1.00 I	79	79	28	28			
			143 ½	1.00	34	36	62	61	+1		
			144	0.60 I	98	98	34	34			
			150 ½	1.00 I	126	137*	32	31	+1	3.1	
			155 ½	1.00	78	79	38	37	+1		
8	2000	32	8	0.86	50	50	48	48			died with
			18 ½	1.00 I	37	45	60	52	+8	13.3	IVH,
			22 ½	1.00	47	57	50	44	+6	12	pneumo-
			26 ¾	1.00	63	81*	41	37	+4	9.75	thorax, and
			27	1.00	33	36	65	61	+4		hyaline
			27 ¼	1.00 I	101	126*	33	30	+3	9.1	membrane
			32 ¼	1.00	90	89	35	35			disease
			41 ½	1.00	107	106	33	33			
			47 ½	1.00	131	128	31	31			
9	1640	32	11 ¼	1.00	6	5	> 76	> 76			died with
			11 ½	1.00 I	17	18	76	76			IVH, hya-
			13 ¾	1.00	33	36	65	60	+5	7.7	line mem-
			15	1.00	34	36	63	60	+3		brane
			16	1.00	25	24	73	74	—1		disease,
			18	1.00 I	26	28	72	69	+3		hypogly-
			27 ½	1.00	71	69	40	40			caemia,
			30 ½	1.00	54	68*	46	40	+6	13.1	and supe-
											rior mes-
											enteric
											artery
											thrombosis

See tables I and II for abbreviations.

* Significant differences between aortic and radial paO_2.

[1] Calculated when the % of the cardiac output passing through the ductus was >5 % and when the radial paO_2 was >10 mm Hg higher than the aortic paO_2.

Table IV. Group 2(c). Babies with radial paO_2 > 10 mm Hg higher than aortic paO_2 at normal aortic paO_2 values

Baby No.	Birth weight	Gestational age	Age	F_IO_2	paO_2, mm Hg		% R → L shunt calculated:		% shunt through ductus	Ductus shunt / Total[1] shunt %	Comment
	g	weeks	h		Aortic	Radial	Aortic	Radial			
16	2930	40	12	0.95	30	34	67	62	+5	7.5	died at 194 h
			12 ¾	0.95	41	53*	55	46	+9	16.3	with airways
			17	0.94	35	41	61	54	+7	11.5	obstruction
			23	0.94	42	47	54	55	—1		from re-
			26	0.94	29	32	68	66	+2		tained secre-
			28	0.96	30	35	68	61	+7	10.3	tions on ventilator, IVH, ASD present
17	2060	33 1/7	4	0.84	65	77*	38	34	+4	10.5	alive and
			7	0.82	41	48	54	47	+7	13	well
			10 ¾	0.93	51	64*	47	40	+7	14.9	subse-
			11 ½	0.94	89	96	34	33	+1		quently
			21 ¾	0.96	64	63	41	41			had to be
			22 ¼	0.94	54	53	45	46	—1		ventilated
			27 ¼	0.90	83	78	34	35	—1		for 2 days
18	2100	35	9	0.32	56	70*	30	22	+8	26.6	alive and
			9 ¼	1.00	250	278*	25	23	+2	8	well
			13 ¼	0.52	136	145	16	15	+1		
			16 ¾	0.52	73	72	27	28	—1		
			20 ¾	0.56	82	78	26	27	—1		
			21 ¼	1.00	231	226	25	25			
19	1260	31	2 ¼	0.46	57	93*	33	20	+13	39.3	alive and
			2 ¾	1.00	195	290*	27	22	+5	18.5	well
			3 ¾	0.54	74	120*	28	19	+9	32.2	
			13 ¼	0.36	61	59	28	29	—1		
			14 ¾	1.00	173	262*	29	24	+5	17.3	
			25 ½	0.44	111	109	16	16			
20	1800	33 4/7	36 ¾	0.94	78	73	36	38	—2		alive and
			45 ½	0.94	76	97*	37	32	+5	13.5	well had Ap-
			46	0.94	82	114*	35	31	+4	11.4	gar of 0 at 1
			56 ½	0.96	131	125	30	31	—1		min and arti-
			64 ¾	0.94	57	59	43	42	+1		ficially ventilat-
			68 ½	0.94	67	65	39	40	—1		ed for 1st 20 h of
			83	0.94	52	56	46	44	+2		life subsequent-
			89	0.94	64	64	40	40			ly thrived
21	1730	35 6/7	26 ¾	0.60	127	122	21	21			alive and well
			27 ¼	1.00	238	238	26	26			subsequent-
			29	0.64	133	132	21	21			ly had to be
			40 ½	0.88	64	108*	39	30	+9	23.1	artificially
			41	0.82	48	53	47	44	+3	6.3	ventilated
			43	0.86	147	161*	27	25	+2	7.4	for 60 h

See tables I and II for abbreviations.

* Significant differences between aortic and radial paO_2.

[1] Calculated when the % of the cardiac output passing through the ductus was >5 % and when the radial paO_2 was >10 mm Hg higher than the aortic paO_2.

ASD = Atrial septal defect.

Table V

Baby No.	Time of observed readings in hours	F_IO_2	Measured paO_2, mm Hg		% R → L shunt		Column I	Column II	Column III
			Aortic	Radial	Aortic	Radial	Max. radial paO_2 in 100 % O_2 if aortic paO_2 could not be increased to 100 mm Hg in 100 % oxygen at % shunt shown	Radial paO_2 when aortic paO_2 = 100 mm Hg with shunt as shown	Aortic paO_2 if radial paO_2 restricted to 160 mm Hg with shunts as shown
16	12 ¾	0.95	41	53	55	46	53	—	—
17	4	0.84	65	77	38	34	94	—	—
	10 ¾	0.84	51	64	47	40	67	—	—
18	9	0.32	56	70	30	22	—	220	80
	9 ¼	1.00	250	278	25	23	—	120	135
19	2 ¼	0.46	57	93	33	20	—	320	60
	2 ¾	1.00	195	290	27	22	—	160	100
	3 ¾	0.54	74	120	28	19	—	210	80
	14 ¾	1.00	173	262	29	24	—	160	100
20	36 ¾	0.94	76	97	37	32	120	—	—
	45 ½	0.94	82	114	35	31	130	—	—
21	40 ½	0.88	64	108	39	30	140	—	—
	43 ½	0.86	147	161	27	25	—	120	135

respectively. Four babies had severe respiratory distress and eventually had to be artificially ventilated (the discrepant paO_2 values were found while the infants were breathing spontaneously), but only one died. The other two babies were mildly affected. We have details of the state at birth in only three of the six babies (Nos. 17, 19 and 20), those born at Hammersmith Hospital. Baby No. 20 scored 0 at 1 minute (Apgar), whereas the other two were in good condition. None was overtly infected at the time of sampling. No baby had any cardiac murmur at the time of sampling and none of the five survivors had any clinical evidence of congenital heart disease, although Baby No. 16 was found to have a small ASD at postmortem examination. It should be noted that the recordings of radial paO_2 more than 10 mm Hg higher than aortic paO_2 were not persistent throughout the course of the baby's illness. In four of the six babies who showed high paO_2 differences, the relevant samples were taken during the first 24 hours of life. Subsequent samples showed no significant differences between radial and aortic oxygen tension. The other two babies had high paO_2 differences during the second day of life, when previous samples in Baby No. 21 and subsequent samples in Baby No. 20 showed no differences.

Discussion

In previous years it had been our aim to maintain the aortic paO_2 at approximately 100 mm Hg and always less than 160 mm Hg to prevent retrolental fibroplasia [20]. It would appear from the present study that the right radial and hence the carotid and retinal paO_2 can be significantly greater than the lower abdominal aorta paO_2 sampled via an umbilical arterial catheter.

Assuming a retinal paO_2 of 160 mm Hg as the maximum safe value [20], what is the highest safe abdominal aortic paO_2? It is clear from our data that the safe level of aortic paO_2 cannot be deduced accurately; however, a reasonable approximation can be derived by calculating the R → L shunts by the method of STRANG and MCLEISH [26].

The shunt calculation on the aortic paO_2 value will give the total R → L shunt through the foramen ovale, ductus arteriosus, and the lungs; the shunt calculation on radial paO_2 blood will give the R → L shunt through the foramen ovale and lungs only. The differences between these values will be the percentage of the cardiac output passing R → L through the ductus arteriosus.

Tables I, II, and III show that in the babies of Groups 1, 2(a) and 2(b), less than 5 % of the cardiac output passed from right to left through the ductus arteriosus—the only exception being a total of six observations on Babies Nos. 7, 8, 9 and 15, in whom the paO_2 was very low in 100 % oxygen and in whom there was no problem of oxygen toxicity to the retina. It should be noted that at low paO_2 levels, small differences in observed values, lying within the limits of experimental error, will cause large differences in the calculated shunt. Furthermore, the proportion of the total right to left shunt passing through the ductus exceeded 10 % only on these occasions and on one other occasion, in Baby No. 12, in whom the total shunt was very small.

In contrast, the babies in Group 2(c) sometimes showed R → L shunts through the ductus arteriosus of more than 5 % of the cardiac output at paO_2 levels within the normal range (table IV). The proportion of the total right to left shunt passing through the ductus often exceeded 15 %.

The shunt calculations on observed values of radial and aortic paO_2 having been made, the radial paO_2 was calculated for an aortic paO_2 of 100 mm Hg (table V, column II) and aortic paO_2 was calculated for a radial paO_2 of 160 mm Hg (table V, column III). It is assumed that the proportion of cardiac output shunted through the ductus arteriosus will remain constant at varying levels of paO_2. This assumption is no doubt incorrect but will err on the side of safety, since there is much evidence to suggest that ductus flows are decreased by raising paO_2 [1, 18].

It will be noted that in Babies Nos. 16, 17, 20 and 21 (first reading), it was not possible to raise the aortic paO_2 to 100 mm Hg, the level at which we wished to maintain the babies (table V, column I). In Babies Nos. 18 and 19, however, high radial paO_2 values, greater than the 'danger' level of 160 mm Hg, would theoretically have been possible in the presence of an aortic paO_2 of 100 mm Hg (table V, column II); conversely, a radial paO_2 of 160 mm Hg could have been achieved at aortic paO_2 values of 80, 60, and 80 mm Hg (table V, column III); however, both Babies Nos. 18 and 19 were in low oxygen concentrations at the time these samples were taken. At low ambient oxygen concentrations, and hence at low alveolar paO_2 tensions, there are considerably greater errors in the shunt calculations (due to ventilation-perfusion imbalances) than when the subject is breathing 80 % oxygen or more. Hence the calculated shunts in Babies Nos. 18 and 19 while breathing air with comparatively low oxygen concentrations are probably falsely high. Both babies were exposed to 100 % oxygen immediately after the readings, which showed large shunt differences in low environmental oxygen concentrations; in both cases, the anticipated reduction in total shunt took place. The proportion of the shunt passing R → L through the ductus arteriosus also decreased, indicating a constriction of the ductus arteriosus at the high arterial oxygen tension. The values recorded in 100 %

oxygen suggested that the retinal paO_2 would be unlikely to exceed the 'danger' level of 160 mm Hg in an ambient oxygen concentration necessary to raise the aortic paO_2 to 100 mm Hg, if the shunt remained the same.

It has been pointed out that the values for radial paO_2 of more than 10 mm Hg higher than aortic paO_2 did not persist throughout the course of the illness. When present, these usually occurred during the first 24 hours of life. This finding agrees with the greater incidence of bidirectional shunting and higher pulmonary artery pressure found at this time. Presumably, comparatively minor changes in pulmonary artery pressure or systemic blood pressure can alter the hemodynamics at the level of the ductus arteriosus and cause large R → L shunts of varying size to occur there. The babies in our series were not crying at the time of sampling and this is not the explanation of the variable shunts.

In babies in Group 2(a), the radial paO_2 values of more than 10 mm Hg higher than aortic paO_2 values that were recorded at high arterial oxygen tensions in high environmental oxygen concentrations are merely an indication that at these paO_2 values, very small differences in the R → L shunt will give big paO_2 differences (table II).

Babies who are artificially ventilated by positive pressure ventilation have an elevated pulmonary artery pressure. In Group 2(b), three of the babies on IPPR showed small but significant paO_2 differences between radial artery and aorta (table III). The considerable fluctuations in oxygen tension and acid base status found in these children may well have played a part in the vascular instability predisposing to R → L ductus shunts. Babies in Groups 2(a) and 2(b) are therefore of less clinical significance than those in Group 2(c).

The major clinical implication of these findings is that the measurement of aortic paO_2 values via an umbilical arterial catheter may not give a true indication of the retinal arterial pO_2. From our figures, however, it would seem very unlikely that retinal artery pO_2 would exceed 160 mm Hg if the umbilical arterial pO_2 never exceeded 100 mm Hg. The inspired oxygen concentrations required to reach these higher arterial pO_2 values will lower the pulmonary artery pressure and cause constriction of the ductus arteriosus, thereby decreasing the shunt through it. It seems reasonable, therefore, to try to maintain the umbilical arterial pO_2 at about 75 mm Hg, the normal value in the premature newborn during the first 24 hours of life, and not let it exceed 100 mm Hg. If this rule is adhered to, only in a very exceptional case, such as that reported by CHU *et al.* [3], would the retinal artery pO_2 reach levels likely to cause retrolental fibroplasia.

Summary

The technique of radial artery catheterization is described. Fifteen of 21 babies with respiratory distress syndrome had paO_2 values sometimes differing by more than 10 mm Hg when simultaneous pre- and post-ductal samples were analyzed. In only six was the difference of any clinical significance. The maximum safe abdominal aorta (and hence umbilical) paO_2 is 100 mm Hg.

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