

The Intrapulmonary Arterial Pattern in Infants with Transposition of the Great Arteries Associated with Interventricular Septum Defect*

A Microangiographic and Histological Study

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Received February 13, 1968

*Das intrapulmonale, arterielle Gefäßmuster bei Kindern
mit Transposition der großen Arterien und Ventrikelseptumdefekt*

Eine mikroangiographische und histologische Untersuchung

Zusammenfassung. Bei 7 Obduktionsfällen mit Transposition der großen Arterien und Ventrikelseptumdefekt wurden mikroangiographische und histologische Untersuchungen (einschl. Serienschnittstudien) der Lunge durchgeführt. Bei 5 Fällen lagen auch andere kardiovaskuläre Mißbildungen vor: Pulmonalklappenstenose, Venenanomalien, Vorhofseptumdefekte und Coarctatio aortae. Das Alter der Fälle betrug 4 Tage bis 11 Monate. Abnorm gewundene intralobuläre Pulmonalarterienäste wurden bei 3 Fällen beobachtet. Pulmobronchialarterien kamen in normaler Anzahl vor und zeigten keine Abweichungen hinsichtlich Größe oder Verlauf. Eine geringere Anzahl arterieller, bronchopulmonaler Anastomosen (Lumen 50—350 μ) wurde bei 2 der untersuchten Fälle beobachtet. Das Lumen der Hauptäste der Bronchialarterien war normal. Bei 2 der älteren Fälle war die Zahl der Bronchopulmonalarterien mäßig erhöht. Die Bronchialarterien des Lungenparenchyms zeigten jedoch keine so starke Vergrößerung und reiche Verästelung wie bei gleichaltrigen Fällen mit isolierter Transposition der großen Arterien.

Summary. Microangiographic and histological studies, including serial sectioning, were carried out on the lungs of seven autopsy cases of transposition of the great arteries associated with interventricular septum defect. In five of the cases other cardiovascular abnormalities were also present — valvular pulmonary stenosis, pulmonary and systemic venous anomalies, atrial septal defect and coarctation of the aorta. The ages of the subjects varied from four days to 11 months. Tortuosity of the intralobular pulmonary arteries was observed in three subjects. The number, size and course of the pulmobronchial arteries were normal. A few arterial bronchopulmonary anastomoses (diameter range 50—350 μ) were demonstrated in two subjects. The diameter range of the main bronchial arteries in the aorta-injected specimens was within normal limits. The number of bronchopulmonary arteries was moderately increased in two of the older subjects. The systemic-artery supply of the pulmonary parenchyma, however, was not as prominent as in infants of the same age with isolated transposition of the great arteries.

This is the fourth part of a study on the pulmonary vasculature of the human fetus and infant. Previous papers have dealt with the intrapulmonary arterial pattern in the normal late fetal and neonatal age (ROBERTSON, 1967), in infancy

* This investigation has been supported by grants from the Swedish National Association against Heart and Chest Diseases, Karolinska Institutes "Reservationsanslag", "Carin Tryggers fond", and "Stiftelsen Therese och Johan Anderssons Minne".

and early childhood (ROBERTSON, 1967) and in neonatal cases of transposition of the great arteries without other cardiovascular malformations (ROBERTSON, 1968).

The increased bronchial-artery supply of the pulmonary parenchyma demonstrated in infants with transposition of the great arteries (ROBERTSON, 1968) offers an auxiliary pathway for poorly oxygenated blood to reach alveolar capillaries. The present investigation was undertaken in order to determine whether, in transposition of the great arteries, the extent of bronchial-artery supply of the pulmonary parenchyma is influenced by the presence of interventricular septum defect.

As in the abovementioned studies (ROBERTSON, 1967, 1968), attention will also be paid to the incidence of other types of aberrations from the basic arterial pattern of the lung, such as arterial bronchopulmonary anastomoses and pulmobronchial arteries.

Previous Investigations

Previous investigations on the pulmonary vasculature in transposition of the great arteries and in ventricular septal defect have mainly been concerned with changes in the pulmonary arterial bed. Intimal fibrous proliferation, medial hyperplasia in muscular pulmonary arteries, thrombotic and plexiform lesions have been described in infant cases of both transposition of the great arteries (NAEYE, 1963; WAGENVOORT et al., 1964; FERENCZ, 1966) and ventricular septal defect (ROSENBERG et al., 1960; WAGENVOORT, 1962; NAEYE, 1966). Increased bronchial-artery supply of the lungs has been reported in infants and older subjects with transposition of the great arteries (COCKLE, 1863; ABBOTT, 1937; CAMPBELL and SUZMAN, 1951; LAPP, 1951; ASTLEY and PARSON, 1952; CUDKOWICZ and ARMSTRONG, 1952; EDWARDS, 1960; CAMARRI and MARINI, 1965; FERENCZ, 1966), but these findings were not correlated to the presence of associated cardiac defects.

Apart from the work by ROBERTSON (1968), the microangiographic technique has not been applied to the study of the pulmonary vasculature in transposition of the great arteries.

Material and Methods

The material, which was collected consecutively, consisted of lungs from seven autopsy cases of transposition of the great arteries associated with ventricular septal defect. The type and size of this defect differed. In five cases there were additional associated cardiovascular abnormalities — pulmonary stenosis, atrial septal defect, pulmonary and systemic venous anomalies and coarctation of aorta. The ages of the subjects varied from four days to eleven months. Three cases had been subjected to cardiac surgery shortly before death. In two of these (A 88, A 123) an artificial atrial septal defect had been produced *ad modum* HANLON-BLALOCK (1950). In the third case (A 137), a widely patent ductus arteriosus had been ligated and the main pulmonary artery had been banded with reduction of its circumference from 60 to 40 mm. In one case (A 61), there were multiple visceral malformations including asplenia, symmetric liver and hyperlobation of both lungs. In no instance was there gross or histologic evidence of but recent non-inflammatory lesions of the pulmonary parenchyma — atelectasis, edema, hyaline membranes, aspiration, intraalveolar and interstitial hemorrhage. The birth weights, ages, surgical procedures and main autopsy findings in case series are given in Table 1.

The pulmonary or the bronchial arteries were injected with 7.5 per cent aqueous suspension of fine barium sulphate ("Micropaque", Damancy & Co.). The ductus arteriosus — when not anatomically closed — was ligated before the injection. The lungs were unexpanded and in atmospheric conditions during the injection procedure.

In three subjects (A 45, A 61, A 64) the injection was made into the pulmonary arteries of each lung. The injection was continuous and the pressure was recorded and kept around 100 mm Hg (80—120 mm Hg). In two other subjects (A 123, A 124) the injection was made into the thoracic aorta in order to fill the bronchial arteries of both lungs. The injection pressure in these subjects was also kept around 100 mm Hg (80—120 mm Hg). In the remaining two subjects (A 88, A 137), the injection was first made into the thoracic aorta (pressure 80—120 mm Hg). After the filling of the bronchial arteries had been confirmed in survey

Table 1. *Birth weights, ages, cardiac surgery, associated cardiac defects and other main*

Case no.	Birth weight (g)	Age		Cardiac surgery			Main autopsy findings	
		mth	d	type of	Age at		Associated cardiac defect	diameter of cardiac defect (mm)
					mth	d		
A 124	4280		4	—			VSD (single ventricle) Coarctatio aortae, preductal	3
A 45	2740		11	—			VSD, posterior Pulmonary stenosis, severe Fibroelastosis, both ventricles	8
A 137	4290		26	Banding of PA, Division of PDA	24		VSD, posterior	20
A 61	?	1	18	—			Ostium AV communis (cor biloculare). Totally anomalous pulmonary venous drainage to v. portae. Persistent left superior v. cava, emptying into left part of the atrial cavity	
A 123	3110	2	17	H-B	2	16	VSD, anterior	16
A 88	?	4	28	H-B	4	27	VSD, posterior ASD (ostium-secundum type) Moderate fibroelastosis, both ventricles	14 5
A 64	3700	11		—			VSD (single ventricle) ASD (ostium-secundum type)	20 12

? = no recording available. H-B = artificial ASD *ad modum* Hanlon-Blalock.

radiograms of the entire specimen, the left lung (A 88) or the right lung (A 137) was injected into the pulmonary artery (pressure 80–120 mm Hg). The injection time was at least 30 minutes; 60 minutes in the double-injected specimens.

After injection, the lungs were fixed in 10 per cent neutral formalin for 4–7 days. Frontal slices of the lungs, 2–3 mm thick, were then radiographed and representative specimens were taken from all lobes of the lungs including the hilus and especially from areas with evidence of transmission of contrast from one arterial system to the other.

After embedding in a mixture of "Histowax" (Matheson, Coleman & Bell) and yellow beeswax the selected specimens were cut into 1,500 μ thick blocks, which were stereomicro-radiographed by a method previously described (LJUNGQVIST, 1963). An average of 9 pairs of microangiograms measuring about 3 \times 5 cm were produced from each pair of lungs. Areas of particular interest, found in the microangiograms, were cut out from the blocks and reembedded in paraffin for histologic examination. The sections, 6–7 μ thick, were stained with Verhoeff's or with Weigert's elastic tissue stain and counterstained with van Gieson's stain. Serial sections were cut from all areas with evidence of arterial bronchopulmonary anastomoses or pulmobronchial arteries in the microangiograms, and from other areas with unusual appearances. An average of 5 blocks were serially sectioned from each pair of lungs.

autopsy findings in case series (7 infants with transposition of the great arteries)

Heart weight (g)	Ductus arteriosus	Foramen ovale	Lung: gross and histologic findings	Other findings
33	patent	patent	Atelectasis; Congestion; Focal interstitial hemorrhage	Multiple erosions in the stomach mucosa
19	patent	?	Atelectasis, right middle lobe	—
35	surgically divided	patent	Slight aspiration; Septal edema	Mild post-op. fibrinous pleuritis Hemorrhagic gastroenteritis
27	patent	—	Hyperlobation of both lungs (4 + 5)	Hernia diaphr. (stomach) Asplenia Symmetric, enlarged liver (195 g) Mesenterium commune
?	closed	(H-B)	Prominent intraalveolar and interstitial hemorrhage, right lung	Post-op. fibrinous pericarditis and pleuritis. Multiple erosions in the stomach mucosa
55	closed	(ASD)	Congestion; Focal intraalveolar hemorrhage	—
95	closed	(ASD)	Intraalveolar edema Thin hyaline membranes	—

Results

Pulmonary Arterial System

Basic Pattern. In microangiograms from pulmonary-artery injected specimens (five subjects), increased tortuosity of intralobular pulmonary arteries was demonstrated in three subjects, and particularly in one of these (A 137, age 26 days) (Fig. 1). In two subjects the basic pattern of the pulmonary arterial system did not differ significantly from the normal (Table 2). In the two remaining subjects, in which the injection had been made into the bronchial arteries only, the limited transmission of contrast to the pulmonary arterial system did not allow a microangiographic analysis of the pattern of the latter (indicated by “?” in Table 1). Funnel-shaped portions of the intralobular pulmonary arteries were not demonstrated in any of the subjects. Obliterative features, in the form of intimal cushions of endothelial and smooth muscle cells, were not encountered.



Fig. 1. Tortuosity of intralobular pulmonary arteries in transposition of the great arteries associated with interventricular septum defect. Age 26 days (A 137). Pulmonary-artery injected lung specimen. Microangiogram $\times 10$

In one case (A 61) a few pleural branches of the pulmonary artery (diameter $35\text{--}50\ \mu$) were demonstrated at the costal and mediastinal aspects of the lung, $2\text{--}4.5\text{ cm}$ from the hilus.

Pulmobronchial Arteries. A few pulmobronchial arteries (i.e. intrapulmonary bronchial arteries originating from branches of the pulmonary artery) were demonstrated by serial sectioning in two subjects. These arteries send concurrent as well as recurrent branches in the bronchial wall (Fig. 2). Their diameter at the point of origin ranges from 75 to $200\ \mu$ (Table 3). In their first portion the pulmobronchial arteries usually display the mural elastic pattern of a pulmonary artery. After they join the bronchial wall, however, their wall structure cannot be distinguished from that of ordinary bronchial arteries. They do not have the structure of "Sperr-

Table 2. *Basic pattern of the pulmonary arterial system in 7 infants with transposition of the great arteries and ventricular septal defect*

Case no.	Age		Injection of	Basic pattern of the pulmonary arterial system	
	mth	d		Normal	Tortuosity of intralobular pulmonary arteries
A 124		4	BA	?	?
A 45		11	PA	—	moderate
A 137		26	BA, PA	—	prominent
A 61	1	18	PA	+	—
A 123	2	17	BA	?	?
A 88	4	28	BA, PA	+	—
A 64	11		PA	—	moderate

BA = Bronchial arteries; PA = Pulmonary arteries.

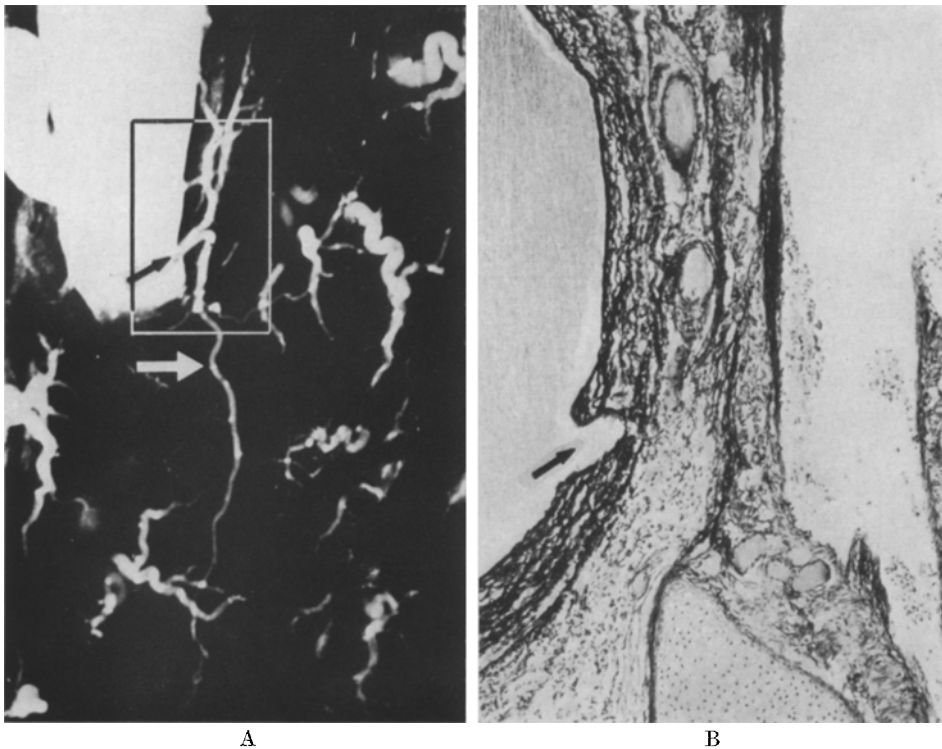


Fig. 2 A and B. Pulmobronchial artery of normal type, diameter 75μ , with recurrent (A, *white arrow*) and concurrent branches in the bronchial wall. The point of origin is indicated by corresponding *black arrows* in A and B. Transposition of the great arteries associated with interventricular septum defect. Age 26 days (A 137). Pulmonary-artery injected lung specimen. A: Microangiogram $\times 13$. B: Serial histologic section from framed area in A. Elastin-van Gieson $\times 66$

Table 3. *Diameter and site of 5 pulmobronchial arteries demonstrated in pulmonary-artery injected lung specimens from two neonatal autopsy subjects with transposition of the great arteries and ventricular septal defect*

Case no.	Age (d)	Diameter (μ) of pulmobronchial artery	Site Lung lobe
A 45	11	125	RL
		125	RM
A 137	26	200	RU
		75	RL
		200	RL

arteries", i.e. their lumen is not narrowed by intimal bundles of smooth muscle cells. One of the pulmobronchial arteries formed an anastomosis of side-to-side type (diameter 75μ) with an adjacent bronchial artery. Otherwise, the pulmobronchial arteries did not communicate on the precapillary level with the neighboring true bronchial arteries.

Bronchial Arterial System

Basic Pattern. Microangiograms and histologic sections from the aorta-injected specimens (four subjects), revealed an essentially normal basic pattern of the bronchial arterial system. The bronchial arteries largely follow the course of the bronchi, supplying the bronchial structures and lymph nodes. Pleural branches of the bronchial arteries occur at the mediastinal aspect of the lung and at the interlobar fissures.

In two of the pulmonary-artery injected specimens, there was transmission of contrast to the bronchial arterial system, rendering at least part of the latter visible in the microangiograms. In these two cases, too, the basic pattern of the bronchial arterial system appeared normal.

Measured in the microangiograms, the inner diameter of the main bronchial arteries ranged from 175 to 475 μ (Table 4). In no instance did the bronchial arteries have the mural struture of "Sperr-arteries".

Table 4. *Diameter of the main bronchial arteries in the hilus of the lung in 7 infants with transposition of the great arteries and ventricular septal defect*

Case no.	Age		Injection of	Diameter of main bronchial arteries (μ)
	mth	d		
A 124		4	BA	400
A 45		11	PA	300
A 137		26	BA, PA	475
A 61	1	18	PA	175
A 123	2	17	BA	475
A 88	4	28	BA, PA	425
A 64	11		PA	^a

^a No filling of the bronchial arteries after injection of the pulmonary arteries.

BA = Bronchial arteries; PA = Pulmonary arteries.

Bronchopulmonary Arteries. Bronchopulmonary arteries (i.e. branches from the bronchial arteries which enter the pulmonary parenchyma proper to ramify as capillaries of alveolar walls) were demonstrated in all aorta-injected specimens and in one of the pulmonary-artery injected specimens. Their number is small and within normal limits in three subjects and moderately increased in two subjects (Fig. 3). Their diameter ranges up to 125 μ (Table 5). The bronchopulmonary arteries are generally derived from intrapulmonary bronchial arteries in the medullary (circumhilar) zone of the lung but a few small bronchopulmonary arteries (diameter range up to 50 μ) originate from pleural branches of the bronchial arteries.

The mural structure of the bronchopulmonary arteries, after they enter the pulmonary parenchyma cannot be distinguished from that of ordinary pulmonary arteries of corresponding size.

In some areas supplied by bronchopulmonary arteries there was transmission of contrast from the bronchopulmonary artery to adjacent branches of the pulmonary artery (or *vice versa*) via a common alveolar capillary network.



Fig. 3. Bronchial-artery injected lung specimen with moderately increased bronchial-artery supply of the pulmonary parenchyma in the "medullary" zone of the lung. Transposition of the great arteries associated with interventricular septum defect. Age 2 months 17 days (A 123).
Microangiogram $\times 2$

Table 5. *Incidence and size of bronchopulmonary arteries in 7 infants with transposition of the great arteries and ventricular septal defect*

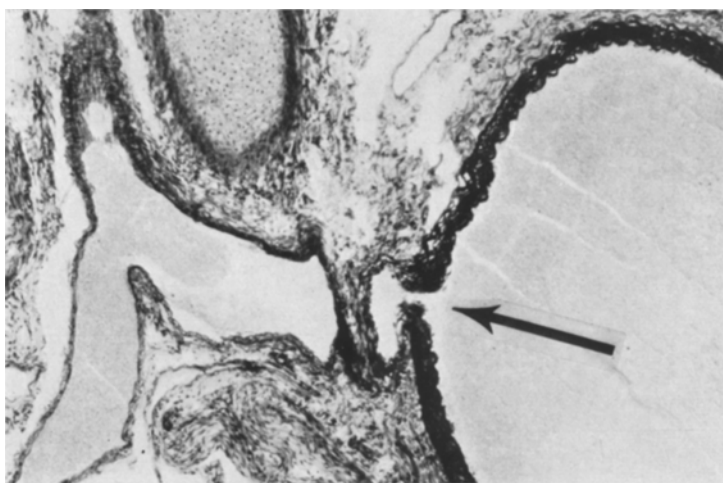
Case no	Age		Injection of	Incidence of bronchopulmonary arteries	Maximal diameter of bronchopulmonary arteries (μ)
	moth	d			
A 124		4	BA	normal	75
A 45		11	PA	normal	100
A 137		26	BA, PA	normal	120
A 61	1	18	PA	^a	—
A 123	2	17	BA	moderately increased	90
A 88	4	28	BA, PA	moderately increased	125
A 64	11		PA	^a	—

^a No bronchopulmonary arteries demonstrated (PA-injected specimens).

BA = Bronchial arteries. PA = Pulmonary arteries.



A



B

Fig. 4 A and B. Arterial bronchopulmonary anastomosis (diameter 125μ) of side-to-side (*H*) type in pulmonary-artery injected lung specimen from infant with transposition of the great arteries, interventricular septum defect and pulmonary stenosis. Age 11 days (A 45). There is a gradual transition of the mural structure of the transverse vessel from pulmonary-artery type (*right*) to bronchial-artery type (*left*). Corresponding *arrows* in A and B. A: Microangiogram $\times 10$. B: Serial histologic section from framed area in A. Elastin-van Gieson $\times 66$

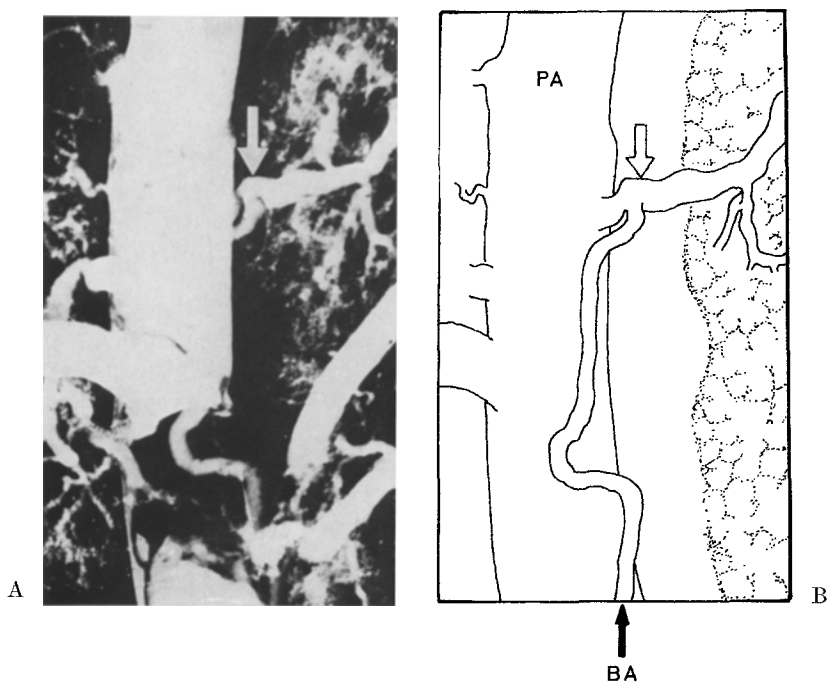


Fig. 5 A and B. Arterial bronchopulmonary anastomosis of end-to-side type (diameter $50\ \mu$) in pulmonary-artery injected lung specimen from infant with transposition of the great arteries, interventricular septum defect and pulmonary stenosis. Age 11 days (A 45). The anastomosis is indicated by corresponding arrows in A and B. A: Microangiogram $\times 26$. B: Diagram of A, based on serial sections. PA = pulmonary artery, BA = bronchial artery

Arterial Bronchopulmonary Anastomoses

A few arterial bronchopulmonary anastomoses (of side-to-side, end-to-side and end-to-end type) were demonstrated by serial sectioning in two subjects (Figs. 4 and 5). The diameter of the anastomoses ranges from 50 to $350\ \mu$ (Table 6). In the anastomoses there is a gradual change in the wall structure of the contributing vessels from bronchial-artery to pulmonary-artery type of mural elastic pattern. The anastomoses do not have the structure of "Sperr-arteries".

Table 6. Diameter and site of 6 arterial bronchopulmonary anastomoses demonstrated in pulmonary-artery (PA) or bronchial-artery (BA) injected lung specimens from two infants with transposition of the great arteries and ventricular septal defect

Case no.	Age		Injection of	Type of anastomosis, diameter (μ)			Site Lung lobe
	moth	d		SS	ES	EE	
A 45		11	PA	75	—	—	RM
				125	—	—	RL
				—	50	—	LU
				—	—	150	LL
				—	—	350	RL
A 88	4	28	BA, PA	200			LL

Comment

The results of the present investigation should be considered in relation to those of the similar study on neonatal infants with transposition of the great arteries without additional cardiac defects (ROBERTSON, 1968). The slight difference in age distribution between these two series is consistent with increased survival time in the presence of associated cardiac septal defects (KATO, 1930; HANLON and BLALOCK, 1948).

The intrapulmonary arterial pattern in isolated transposition, even in the neonatal period, has prominent abnormalities affecting both the pulmonary and bronchial arterial systems. These abnormalities include "funnel-shaped" portions of the intralobular pulmonary arteries ("Type I"-pattern), prominent tortuosity of intralobular pulmonary arteries ("Type II"-pattern), enlargement of the main bronchial arteries and a considerable increase in the number of bronchopulmonary arteries, particularly in the "medullary" zone of the lung (ROBERTSON, 1968). In the present study, the only observed difference from the normal microangiographic pattern of the pulmonary arterial system is the tortuosity of intralobular arteries, which was demonstrated in three cases and which corresponds to the abovementioned "Type II"-pattern. Tortuosity of the pulmonary arteries has been previously recognized in post-mortem angiograms from cases of pulmonary hypertension and is most probably not related to any specific type of cardiac defect (SCHOENMACKER and VIETEN, 1954; DOYLE et al., 1957).

In neonatal specimens of isolated transposition of the great arteries, the pulmobronchial arteries display abnormal features, including deviation from the bronchial wall in their terminal course (ROBERTSON, 1968). This is contrary to the findings in the present study, in which the size, number and course of the pulmobronchial arteries do not differ significantly from the normal.

The diameter range of the main bronchial arteries in the aorta-injected specimens of the present series is within normal limits, so is the diameter range of the bronchopulmonary arteries. The moderately increased number of bronchopulmonary arteries in two of the older subjects is similar to the pattern in isolated transposition, although not as prominent.

The presence of a common capillary network between bronchopulmonary arteries and adjacent branches of "ordinary" pulmonary arteries has also been recognized in isolated transposition, as well as in the normal infant lung (ROBERTSON, 1967, 1968).

With the exception of one H-anastomosis of unusual appearance, between a bronchial artery and a pulmobronchial artery, the few arterial bronchopulmonary anastomoses demonstrated in the present series were essentially normal in type. In one subject (A 45, age 11 days) some of the anastomoses were, however, wider than in the normal neonatal lung (WAGENVOORT, 1966, 1967; ROBERTSON, 1967). The slightly higher pressure used in the injection of the pulmonary arterial system in the present study, as compared to otherwise similar studies on the normal infant lung (ROBERTSON, 1967), may have contributed to this difference. Another possibly contributing factor in case A 45 is the presence of pulmonary stenosis, which is said to enhance the development of arterial bronchopulmonary anastomoses (LAPP, 1951; TOBIN, 1952; HARRIS and HEATH, 1962; LIEBOW, 1962; TURNER-WARWICK, 1963; CAMARRI and MARINI, 1965).

The present series of infants with transposition of the great arteries is not uniform with respect to the type of associated cardiac defect. The type and size of the associated ventricular septal defects varied up to the state of *cor biloculare*. In five cases there were additional cardiovascular malformations — valvular pulmonary stenosis, pulmonary and systemic venous anomalies, atrial septal defect and coarctation of the aorta. To what extent these various individual malformations might have influenced the microangiographic picture of the lungs has not been analysed in this study. It is obvious, however, that the intrapulmonary arterial pattern in the present series is much closer to the normal than is the neonatal pattern of isolated transposition (ROBERTSON, 1968). It seems, then, that the presence of associated cardiac defects increasing the “effective blood flow” (FERENCZ, 1966) through the lungs interferes with the development of an increased systemic-arterial supply of the pulmonary parenchyma, which is a prominent and consistent feature in neonatal cases of isolated transposition of the great arteries (ROBERTSON, 1968).

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