SECTION ON CARDIOVASCULAR DISEASES

Patent Ductus Arteriosus in the Adult
With Partial Reversal of Flow

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The clinical and physiological characteristics of the uncomplicated patent ductus arteriosus in the adult have been well-defined and understood for many years. A typical continuous murmur usually develops early in life; the electrocardiogram is usually normal or shows a left axis deviation; and heart fluoroscopy ordinarily shows only mild to moderate left ventricular enlargement, vigorous aortic pulsation, and increased pulmonary blood flow. In addition, cyanosis does not occur unless severe congestive cardiac failure develops in the terminal stages of the disease.1

In 1925, following a review of 16 atypical cases of patent ductus arteriosus in which right ventricular hypertrophy was present, it was pointed out that reversal of flow through the ductus arteriosus and the consequent development of lower extremity cyanosis and clubbing could and probably did occur in at least six of the patients studied.2 Recently the syndrome of patent ductus arteriosus with pulmonary hypertension and reversal of flow through the ductus arteriosus has been well-described and documented in scattered reports.3-9 The following cases are believed representative of that group.

Case Reports

Case 1: M. C., a 44 year old white woman entered the hospital for the first time on December 30, 1953. She gave a life-long history of dyspnea on effort and of becoming tired easily. As a child she had been "sickly" and never able to keep up with her playmates. Cyanosis on exertion was first noted in 1940 at the age of 30. Hemoptysis of approximately one-half cup of blood first occurred in 1947 and was subsequently followed by eight similar episodes over a period of four years. She had previously worked as a beauty shop operator but after 1946 was limited to manicure work because of weakness and dyspnea. There was no history of dyspnea at rest or of orthopnea. She noted hoarseness on several occasions.

She had been studied in hospitals for the first time in 1951 when she complained of severe dyspnea on exertion and could not walk over 20 feet without stopping to rest. On examination in 1951, she was noted to have cyanosis of the lips and fingers and marked cyanosis with definite clubbing of the toes. The blood pressure was right arm 108/92, left arm 112/90, right leg 140/94, and left leg 142/90 mm. Hg. The lungs were clear. The heart was generally enlarged. The pulmonary second sound was louder than the aortic second sound. A low-pitched diastolic murmur was heard in the fourth inter-

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TABLE I
CATHETERIZATION DATA—1953—CASE 1

<table>
<thead>
<tr>
<th>Site of Blood Sample</th>
<th>Pressure Mm. Hg.</th>
<th>O₂ Content Vol. Per Cent</th>
<th>O₂ Saturation Per Cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary artery</td>
<td>85/60</td>
<td>18.4</td>
<td>63</td>
</tr>
<tr>
<td>Right atrium</td>
<td></td>
<td>18.0</td>
<td>62</td>
</tr>
<tr>
<td>Femoral artery (left)</td>
<td>110/89</td>
<td>17.6</td>
<td>60</td>
</tr>
<tr>
<td>Brachial artery</td>
<td>117/88</td>
<td>27.6</td>
<td>94</td>
</tr>
</tbody>
</table>

The pulmonary artery and systemic artery pressures were not recorded simultaneously, which may account for the considerable difference between them.

costal space to the left of the sternum. There was no peripheral edema. The hemoglobin was 21.1 gm./100 ml., the venous pressure 7.0 cm. of Na. citrate solution. The arm to tongue circulation time (decholin) was 36 seconds while the vital capacity was 88 per cent of normal. An electrocardiogram showed right axis deviation and right ventricular strain. Cardiac fluoroscopy showed considerable enlargement of the right ventricle and less marked enlargement of the left ventricle. The findings at cardiac catheterization are recorded on Table I. She was discharged with a probable diagnosis of Eisenmenger's complex.

During the next two and one-half years, she worked 15 to 30 hours weekly until late in 1953 when she re-entered the hospital because of increasing fatigue and dyspnea. On examination she was found to be a malnourished, chronically ill 44 year old woman with severe dyspnea on effort. The blood pressure was 100/60 mm. Hg. The lungs were clear. The heart was generally enlarged with a diffuse maximal impulse over the fourth intercostal space to the left of the sternum. The pulmonary second sound was accentuated. A grade II blowing diastolic murmur was heard along the left sternal border radiating to the apex when the patient was in the left lateral decubitus position. A rough systolic murmur was heard intermittently at the base. There was no peripheral edema. The hemoglobin was 23.3 gm./100 ml. Electrocardiogram showed right ventricular preponderance and strain (Figure 2). Chest x-ray film (Figure 3)

FIGURE 1: Photograph of hands and feet of Case 1 showing distinct clubbing of the toes without similar changes in the fingers.
TABLE II
EFFECTS OF BREATHING 100 PER CENT O₂ ON FEMORAL ARTERY SATURATION—CASE 1

<table>
<thead>
<tr>
<th></th>
<th>1951</th>
<th>1953</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral saturation at rest</td>
<td>53</td>
<td>60</td>
</tr>
<tr>
<td>Femoral saturation after breathing 100 per cent O₂</td>
<td>74</td>
<td>69</td>
</tr>
</tbody>
</table>

In 1951, there was a considerable drop in the amount of desaturated pulmonary artery blood shunting into the aorta after breathing 100 per cent oxygen. Much less change was produced two years later under the same circumstances, suggesting that the pulmonary resistance was less altered by high oxygen tensions in the inspired air.

and cardiac fluoroscopy showed primarily right ventricular enlargement. The pulmonary arteries were enlarged but the peripheral pulmonary vessels were diminished in size. Arterial oxygen studies are recorded in Table II. A retrograde aortogram through the left brachial artery outlined the aorta with no evidence of coarctation. A slight outpocketing of the medial portion of the arch in the position where a ductus arteriosus is usually found was seen but there was no flow of dye from the aorta to the pulmonary artery. As a result of the studies it seemed likely that the patient had

FIGURE 2: Electrocardiogram from Case 1, interpreted as showing marked right ventricular hypertrophy and strain, normal sinus rhythm.
a patent ductus arteriosus with severe pulmonary hypertension and a shunt from pulmonary artery to the distal aorta, with no other apparent cardiac defects.

The seriousness of an operation was stressed for this patient. However, she desired to undergo any type of corrective surgery which might benefit her. While in the hospital she was a total invalid because of exertional dyspnea. The plan was to place a small diameter shunt between the pulmonary artery and the left atrium before cross clamping the ductus arteriosus. Then if the patient tolerated closure of the ductus

FIGURE 3: Posteroanterior and right-anterior oblique chest views of Case 1. Calcification in the ductus arteriosus is visible.

FIGURE 4: Photomicrographs of small pulmonary arteries from Case. Striking intimal proliferation is present.
arteriosus it was to be divided and ligated. It was thought that this technique would decrease right heart work by decompressing any intolerable right ventricular pressures developing after the shunt through the ductus arteriosus had been eliminated. Moreover, it was hoped that this leak would contribute a volume flow adequate to sustain sufficient blood volume in the systemic circulation, especially the coronary arteries during an interval when the right heart was incapable of pumping enough blood through the arteriosclerotic pulmonary circuit. Perhaps with time for readjustment a regression of the lung lesions would follow. Once her clinical status had improved enough, at a second operation the arterial shunt between the pulmonary artery and the left atrium could then be disconnected. It was appreciated that such an anastomosis would result in mild generalized cyanosis rather than the regional cyanosis already present.

At operation the ductus arteriosus was estimated to have a diameter of 20 to 25 mm. It was thickened and contained calcium. There were evident thrombi in all branches of the right pulmonary artery with wide spread calcific deposits in the arterial wall. The proposed anastomosis of the left atrium to a branch of the pulmonary artery could not be carried out because of thromboses throughout the smaller pulmonary arteries. A tentative clamping of the ductus arteriosus was attempted to note the heart's tolerance of the altered hemodynamics. The cross clamping unfortunately eventuated promptly in ventricular fibrillation. A normal rhythm could not be re-established despite recourse to a variety of resuscitative techniques.

At autopsy two areas of infarction were present in the upper lobe of the right lung. The right and left pulmonary artery branches were dilated and showed a marked degree of atherosclerosis. A laminated thrombus was present at the bifurcation of the right pulmonary artery and extended into the main lobar branches with complete occlusion of the opening to the upper lobe. The ductus arteriosus entered the aorta 2 cm. beyond the left subclavian ostium. The aorta in this area showed moderate atherosclerosis and was calcified. The left pulmonary artery was markedly atherosclerotic with calcification and sclerosis of the intimal layer. Marked plaque formation and ulceration of the intima were present in the left pulmonary artery where the ductus arteriosus entered. The right ventricle was hypertrophied and dilated, its walls measuring 1.2 cm. in thickness. The left ventricle measured 1.4 cm. in thickness. The valves were all normal. No other cardiac defect was present.

Microscopic examination of the lungs showed areas of atelectasis and infarction. The smaller pulmonary arteries showed striking changes, mainly of thickening due to intimal fibrosis with fragmentation and duplication of the elastic fibers (Figures 4 and 5). Vascular lesions in the form of small cavernous channels supported by connective

![Image](https://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21313/)

FIGURE 6: Chest views of Case 2. Enlargement of both ventricles and the pulmonary artery segment is present.
tissue containing endothelial cells were present. Sections of the right pulmonary artery showed severe atherosclerotic changes with calcification as well as a laminated thrombus.

**Case 2:** This patient was a 21 year old woman who gave a history of life-long inability to keep up with other people of her age. Her symptoms were primarily those of shortness of breath on effort. In addition, she had noted paroxysmal nocturnal dyspnea and ankle edema. She had never been cyanotic. Because of her cardiac symptoms she had been forced to discontinue school at an early age. There had been many episodes of respiratory tract infection and bronchitis. There was no antecedent history of rheumatic fever.

The physical examination at the time of her first hospital admission was negative except for the heart and for a palpable thyroid gland adenoma, which with her hyperactivity and a fine tremor led to the clinical diagnosis of thyrotoxicosis. The heart was not enlarged on physical examination. There was a grade III systolic murmur heard best along the left sternal border and at the apex. In addition, a grade I diastolic blowing murmur could be heard along the left sternal border. There was no cyanosis of either the mucus membranes, the finger nails, or the toe nails. The blood pressure was 118/74 mm. Hg.

The pertinent laboratory findings on the first hospital admission were as follows:

The urine was within normal limits. The hemoglobin was 15.3 gms. and the white blood count was 6,600 with 49 per cent neutrophils, 42 per cent lymphocytes, 5 per cent monocytes, 3 per cent eosinophils, and 1 per cent basophil. The radioactive iodine uptake was 53 per cent of the administered dose at the end of 24 hours. An electrocardiogram showed atrial fibrillation at a slow rate. The frontal plane axis was about +80°. There were marked ST segment and T wave changes in leads II, III, AVF, and V6 and V5 suggestive of digitalis effect or possibly a left ventricular strain pattern. No evidence of right ventricular hypertrophy could be seen. Cardiac fluoroscopy showed marked enlargement of the heart which was thought to involve both the left and right ventricles. Mild left atrial enlargement was also observed in the left anterior oblique view. The pulmonary artery segment and the central and peripheral pulmonary arterial branches were considerably enlarged. The aorta was thought to be small (Figure 6). The findings were consistent with a left to right shunt and because of the slight but definite left atrial enlargement it was thought that either a ventricular septal defect or a patent ductus arteriosus would be the more likely possibility.

Cardiac catheterization was carried out and the significant findings are indicated in Table III. Despite severe pulmonary hypertension a considerable left to right shunt at the level of the pulmonary artery was found and was thought to be consistent with either patent ductus arteriosus or an aortic pulmonic window.

In view of the co-existing thyrotoxicosis it was thought advisable to control this portion of her illness first. Initial attempts using propylthiouracil were not effective and it was necessary eventually to administer relatively large doses of radioactive iodine. By February of 1956 she was somewhat hypothyroid, the radioactive iodine uptake being 6.6 per cent at 24 hours.

She was readmitted to hospital in February of 1956 for an attempt at corrective surgery. There had been little change in her symptomatology except that she had noted cyanosis with exercise on several different occasions. Her symptoms of nervousness were well controlled and her other symptoms directly referable to the cardiovascular system were unchanged despite the fact that she was then hypothyroid.

### Table III

<table>
<thead>
<tr>
<th>Chamber</th>
<th>Pressure Mm. Hg.</th>
<th>O₂ Content Vol. Per Cent</th>
<th>O₂ Saturation Per Cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA</td>
<td>128/64</td>
<td>17.54</td>
<td>83</td>
</tr>
<tr>
<td>RV</td>
<td>100/7</td>
<td>13.21</td>
<td>63</td>
</tr>
<tr>
<td>RA</td>
<td>6</td>
<td>12.98</td>
<td>62</td>
</tr>
<tr>
<td>SVC</td>
<td>12.79</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td>IVC</td>
<td>13.01</td>
<td>62</td>
<td></td>
</tr>
<tr>
<td>FA</td>
<td>19.66</td>
<td>92</td>
<td></td>
</tr>
</tbody>
</table>

![Image](http://journal.publications.chestnet.org/pdaccess.ashx?url=/data/journals/chest/21313/ on 06/26/2017)
The physical examination at the time of her last admission showed the blood pressure to be 100/78 mm. Hg. The cardiac murmurs were unchanged. It was thought that distinct but mild cyanosis of the nail beds was present.

There were no significant changes in the laboratory studies other than in the arterial saturation. A femoral artery blood sample collected at rest showed the oxygen saturation to be only 88 per cent. In view of this finding simultaneous samples were taken from the right brachial artery and the left femoral artery in which the arterial saturations were 80 per cent and 88 per cent respectively. Since the brachial artery saturation was lower than that of the femoral artery, it was thought that the diagnosis of aortic pulmonic window was more likely than that of patent ductus arteriosus with reversal of flow.

At the time of operation a diagnosis of patent ductus arteriosus was confirmed. A lung biopsy showed considerable medial hypertrophy and intimal proliferation in the small branches of the pulmonary artery. These findings were especially prominent in the arteries measuring under 100 micra in diameter where the arterial lumen was found to be nearly obliterated in several instances. This was thought to be grade III pulmonary arterial and arteriolar sclerosis. A corrective operation was not attempted at the time of exploratory thoracotomy.

The two cases described both presented the common complaint of progressively severe exertional dyspnea. Case 1 presented the more typical physical finding of regional cyanosis and clubbing limited to the lower extremities. Variations of this sign have been reported since there may be retrograde flow of pulmonary artery blood into the left subclavian artery or even into the root of the aorta causing cyanosis of the upper portions of the body in general. This latter and more atypical physical finding was present in Case 2 at the time of her exploratory operation. The retrograde flow of desaturated pulmonary artery blood into the aortic root had been well demonstrated in one other instance. The first case showed the usual findings of polycythemia, right ventricular hypertrophy on fluoroscopy, and a right ventricular strain pattern electrocardiographically with large pulmonary artery segments. In her case, a well calcified ductus arteriosus could also be seen and demonstrated on the x-ray films. Case 2 had apparently not been in a state of reversal long enough to result in the development of polycythemia. However, a regurgitant murmur of the Graham-Steell type was heard although the pulmonary systolic murmur was much the more prominent of the two.

That pulmonary hypertension may be progressive and that reversal of flow may occur relatively late in life, supporting the idea that the hypertension may be acquired rather than congenital, is demonstrated by the clinical course of Case 2.

It is generally agreed that the division of a ductus arteriosus when there is a significant (net) right to left shunt unless the patient is supported by some other procedure designed to maintain flow into the left ventricle is likely to prove fatal. One case in which the two circulations seemed to be “in-balance”—virtually equal pressures in aorta and pulmonary artery with bidirectional shunt and not truly continuous reversal of flow—has been operated on with survival of the patient. In this instance the ductus arteriosus was not completely divided and there have been no follow-up studies to determine if the communication has been completely closed. The theoretical possibility exists that merely to divide such a “balanced” shunt, particularly in an adult, might do little to cure the existing pulmonary hypertension.
The effects on the pulmonary artery pressure of breathing 100 per cent oxygen have been well-studied and the chief result has been an inverse relationship between the oxygen tension of the inspired air and the pulmonary artery pressure. In the presence of patent ductus arteriosus with reversal of blood flow the result should then be an increase in the saturation of femoral artery blood. It has been well-demonstrated that the opposite occurs when the O₂ content of the inspired air is reduced to a level of 10 to 14 per cent, the amount of the reduction in saturation ranging between 2.2 and 9.8 vol. per cent. Whether or not these alterations occur in a given case would seem to depend on the degree of sclerosis of the small pulmonary arterioles and arterioles. In Case 1 the femoral artery saturation rose from 53 to 74 per cent in 1951 after breathing 100 per cent O₂ while a repeat similar study in 1954 showed a rise from only 60 to 69 per cent, suggesting the possibility that the pulmonary arteriosclerosis had progressed during the interval to the point that relatively little reduction in the pulmonary vascular resistance could accrue from the breathing of 100 per cent O₂ (Table II).

The basic reason why the adult with patent ductus arteriosus develops pulmonary hypertension and eventually reversal of flow is not entirely clear although the same problem exists in reference to other left to right shunts. Recent animal studies may give a clue.

From the relationship:

\[
\text{Pressure} = \text{Flow} \times \text{Resistance}
\]

it is apparent that increases in pulmonary artery flow can result in increased pulmonary artery pressure provided the pulmonary vascular resistance remains constant or does not decrease excessively. That this does occur has been amply demonstrated by experience with the closure of atrial septal defects wherein distinct elevations of pressure in the pulmonary artery prior to operation are no longer present following closure of the defect and reduction of the pulmonary artery flow to normal. The highest pulmonary artery pressure we have observed to date with a simple atrial defect was 72/34 mm. Hg.—moderately severe pulmonary hypertension apparently based on increased flow alone but not enough to cause reversal of the shunt and cyanosis. Following closure of the defect, the right ventricular pressure was found to be 28/4 mm. Hg.—a normal value. There are scattered reports of moderate pulmonary hypertension in the presence of a patent ductus arteriosus reverting towards normal following surgery—from 100 mm. Hg. to 40 mm. Hg. in one case—apparently because the elevation in pressure originally depended almost entirely on increased pulmonary blood flow. The same reduction occurs in certain instances following correction of ventricular septal defects.

A second aspect of the problem has to do with increased pulmonary vascular resistance resulting from increased pulmonary flow. In two series of experiments, pulmonary hypertension has been consistently produced in dogs by the anastomosis of a systemic artery and a branch of the pulmonary artery. In each series marked changes in the pulmonary arteriolar structure occurred, including the appearance of medial muscular
hypertrophy and intimal fibrosis. Serial lung biopsies demonstrated the
development of arteriolar medial muscular hypertrophy within a two-week
period following the anastomosis, followed in turn by intimal fibrosis and
and increase in the adventitial collagenous material over a two to three-
month interval. No changes in alveoli, capillaries, or veins were noted.\textsuperscript{12}
The striking similarity of the changes to those noticed in humans with
increased pulmonary vascular resistance has already been emphasized.\textsuperscript{11}
In some instances the smaller vessels are completely obliterated. That the
size of the lumen in the pulmonary arteriole is the more important factor
in determining pulmonary vascular resistance than is the increased viscosity
of blood relative to polycythemia or other similar factors has also been
demonstrated.\textsuperscript{13}

It was further observed that the total amount of increase in pulmonary
blood flow was not the sole factor in initiating the changes subsequently
observed in pulmonary arteriolar structure and in pulmonary artery
pressure. The type of anastomosis formed—whether end-to-end or side-to-
side—was contributory in some way, the end-to-end anastomosis resulting in
the greater amount of change. It has been suggested that the pulse wave
form is of some significance and that the pulsatile thrust of blood into the
pulmonary circuit is responsible in part for the changes observed.\textsuperscript{12} Whether
or not this is an important factor in the human cannot as yet be deter-
dined. The usual patent ductus arteriosus and aortic-pulmonic window
function as side-to-side anastomoses in which the shunt is not obligatory.
In the adult, for the former, the development of severe degrees of pulmo-
nary hypertension is uncommon. A large ventricular septal defect ap-
proaching functionally a single ventricle probably more closely simulates
the situation in the experimental animal and here marked pulmonary
hypertension is necessary for survival, provided that pulmonary stenosis
is not present. Simple atrial septal defects seem not to be ordinarily ac-
companied by severe pulmonary hypertension whereas atrioventricularis
communis defects seem commonly to have marked elevations in pulmonary
artery pressure. These are impressions not as yet statistically proved.
They would suggest, however, that the presence of a large, pulsatile
thrust through an obligatory shunt may be related to the development
of the severe forms of pulmonary hypertension. The ordinary Blalock
anastomosis does not duplicate this experimental situation. There are rare
instances in which end-to-end anastomoses have been made in the adult
between a systemic artery and the right or left pulmonary artery but none
of these have had post-operative studies that would shed any light on this
particular problem.

From the evidence at hand it would seem that a reasonable working
hypothesis can be formulated to explain the course of events in the adult
who develops pulmonary hypertension in the presence of a left-to-right
shunt. It is known that increased pulmonary flow may in itself produce
pulmonary hypertension and it is suggested that this in turn leads to an-
tomical changes within the small pulmonary arteries resulting in an in-
creased pulmonary vascular resistance and with it a further aggrava

\textsuperscript{11} E. D. Dollinger, personal communication.
\textsuperscript{12} For a full discussion of end-to-end anastomoses, see J. M. Blalock,
\textit{ibid.}, p. 172.
\textsuperscript{13} For a further discussion of end-to-end anastomoses, see J. M. Blalock,
\textit{ibid.}, p. 172.
of the pulmonary hypertension. The presence of a pulsatile thrust of blood into the pulmonary circuit may also be of great importance.

One interesting question concerns whether or not these changes observed in the pulmonary arteriole in the severe types of pulmonary hypertension are reversible. It is not known if such regression occurs in man. The experimental preparation does show a regression of the anatomical changes in the pulmonary arteriole following correction of the systemic artery-pulmonary artery anastomosis. These changes occur very slowly over a prolonged period of time and in no animal thus far have the pulmonary arterioles returned entirely to normal. The possibility of such regression occurring in man seems good enough to warrant further attempts at repairing surgically the patent ductus arteriosus in which reversal of flow has occurred.

The mechanism of death in the first case described, and in some others, has been ventricular fibrillation. It seems reasonable to assume that with obliteration of the ductus arteriosus and because of markedly increased pulmonary vascular resistance, the right ventricle is unable to maintain enough flow through the lungs and into the coronary circulation to perfuse adequately the myocardium. Under such circumstances ventricular fibrillation may easily occur. This constitutes a major problem that must be solved before the repair of this anomaly will become possible.

SUMMARY

1. Two examples of patent ductus arteriosus with pulmonary hypertension, reversal of flow, and cyanosis occurring in adults are described.
2. In Case 1 changes in the response to breathing 100 per cent O₂, as determined by studies of femoral oxygen saturations, suggest that the pulmonary vascular resistance became "fixed" as the disease progressed.
3. Clinical and experimental observations demonstrated that increased pulmonary blood flow results in pulmonary hypertension that may in turn be followed by pulmonary arteriolar, medial, and intimal thickening and increasing pulmonary vascular resistance. This is especially true if the flow is of the pulsatile type.
4. The pulmonary arteriolar changes in the experimental animal are but partially reversed during reasonable intervals of observation once the pulmonary pressure has been restored to approximately normal values.
5. For surgery to be successful in cases with marked reversal of flow some mechanism for maintaining adequate coronary flow and preventing ventricular fibrillation will be required.

RESUMEN

1. Se describen dos casos de conducto arterioso persistente con hipertensión pulmonar, inversión de la corriente y cianosis en adultos.
2. En el caso 1, los cambios en la respuesta a respirar 100 por ciento, como se pudo determinar por los estudios de la saturación de oxígeno
femoral, sugieren que la resistencia pulmonar vascular se volvió "fija" al progresar la enfermedad.

3. Las observaciones clínicas y experimentales demostraron que el aumento del flujo sanguíneo pulmonar da por resultado la hipertensión pulmonar que a su vez puede ser seguida por engrosamiento de la íntima arterial, así como engrosa miento de la media, y un aumento de la resistencia pulmonar. Esto es especialmente cierto si el flujo es de tipo pulsátil.

4. Los cambios pulmonares arteriolas en el animal de experimentación, sólo son parcialmente invertidos durante intervalos razonables de la observación una vez la presión pulmonar se ha restablecido aproximadamente a los valores normales.

5. Para que la cirugía tenga buen resultado en casos con marcada inversión del flujo se requerirá algún mecanismo para mantener el flujo coronario adecuado y para prevenir la fibrilación ventricular.

**RESUME**


2. Les altérations du cas I apparaissant lors de l'épreuve de la respiration à 100%, déterminée par l'étude de la saturation oxygénée de l'artère fémorale, donnent à penser que la résistance des vaisseaux pulmonaires s'était "fixée" alors que l'affection continuait sa progression.

3. Des observations cliniques et expérimentales ont démontré que l'augmentation de la circulation sanguine pulmonaire dépend de l'hypertension pulmonaire qui à son tour peut être suivie par un épaississement de la média y de l'intima des artérioles pulmonaires entrainant un accroissement de la résistance vasculaire pulmonaire. Ceci est particulièrement vrai si le débit est du type pulsatile.

4. Les altérations artériolaires pulmonaires chez les animaux d'expérience ne sont que partiellement reversibles pendant des intervalles raisonnables d'observation une fois que la pression pulmonaire est revenue à son taux approximativement normal.

5. Pour mener à bien les interventions dans les cas avec trouble marqué du débit, on doit arriver à maintenir un débit coronaire convenable, et prévenir la fibrilación ventriculaire.

**ZUSAMMENFASSUNG**

1. Es werden zwei Beispiele beschrieben von offenen ductus arteriosus mit pulmonalem Hochdruck, umgekehrter Strömungsrichtung und cyanose bei Erwachsenen.

2. Der Wechsel von 100% in der Antwort von Fall I bei der Atmung, wie aus Untersuchungen bei der Prüfung von femoralen Sauerstoffsättigungen hervorging, lässt vermuten, dass der pulmonale Gefässwiderstand zu einem "fixierten" wurde in dem Masse, wie sich die Krankheit weiter entwickelte.
3. Durch klinische und experimentelle Beobachtungen wurde nachgewiesen, dass eine erhöhte pulmonale Durchströmung eine pulmonale Hypertonie zur Folge hat, die ihrerseits gefolgt sein kann von einer pulmonalen arteriellen Media-Intema-Verdickung und erhöhtem pulmonalem Gefäßwiderstand. Dies ist besonders dann der Fall, wenn die Durchströmung vom pulsierenden Typ ist.

4. Die Veränderungen an den pulmonalen Arteriolen im Tierversuch lassen sich jedoch wenigstens teilweise wieder aufheben innerhalb übersehbarer Beobachtungszeiträume, wenn der pulmonale Druck erst einmal wieder auf annähernd normale Werte eingestellt ist.


REFERENCES


