Patency of the Ductus Arteriosus After Birth:  
A New Theory*

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The cause of patent ductus arteriosus has not yet been established. Spitzer1 in his ontogenetic and phylogenetic explanation considers patent ductus arteriosus as a simple malformation due to an arrest of development. He was not aware that patency of the ductus can be accompanied by severe pulmonary hypertension (so-called reversed ductus)2, 3, 4, 5, 6, 7 even though in most cases the pressures of the lesser circulation are normal or slightly elevated.8, 9, 10 While his interpretation could be applied to the presence of isolated, uncomplicated ductus, there is no stage of normal development which corresponds to the conditions found in ductus with pulmonary hypertension.

Kennedy11 in 1942 stated that “uncomplicated persistent patency of the ductus should not be regarded as malformation, but should be attributed to defective oxygenation of the blood during birth or shortly after.” Several subsequent publications12, 13, 14, 15 demonstrated fetal distress at birth and shortly after birth in a great number of the cases of patent ductus which were investigated. In statistical studies performed by Alzamora and co-workers,15 the occurrence of patent ductus in children born above 9,000 feet is strikingly high. Experimental studies also give16, 12 good evidence that asphyxia at birth may delay the closure of the ductus. Undoubtedly, as Record and McKeown12 state: “these observations ... do not dispose of the objection that closure might be expected to follow relief of embarrassment unless the possibility of closure is limited to a short period after birth.” It has been shown that the ductus may be obliterated many months after birth, even in the presence of anoxia, like in cases of tetralogy of Fallot,17 but this observation may not have direct bearing on the normal mechanism of closure. Spontaneous closure of the ductus has been described by several observers many months, and even years, after birth.18, 19, 20

While Record and McKeown state12 that, in their cases, asphyxia at birth is related to the occurrence of patency of the ductus, they do not try to explain the sequence of events. It is not probable that patency of the ductus is the direct cause of cyanosis or asphyxia, as found right after birth, because patency, at least in the first few minutes of life, is physiological: blood flow is directed from left to right and there is no reason to suppose that, under otherwise normal conditions, this may cause embarrassment.

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One could speculate that circulatory embarrassment might be due to failure of the left ventricle. This chamber is suddenly called to carry the entire blood volume through the greater circulation when, right after birth, the placental blood stream is abruptly cut off and pulmonary respiration begins.

One fetal condition might leave the left heart unprepared for its task at the moment of birth: premature closure of the foramen ovale. Patency of the foramen ovale in fetal life and some time after birth is necessary for the proper development of the left side of the heart during fetal life and for a proper balance of pressures after birth, until the left ventricle is adjusted to its task.\textsuperscript{13} The valve of the foramen ovale acts as an escape valve, and the blood flows from right to left in the fetal heart.

Brenner\textsuperscript{21} and Lehman\textsuperscript{22} pointed out that, if the foramen ovale closes prematurely, the following consequences may occur: overloading of the pulmonary circuit; increase of pressure in and increase of resistance of the pulmonary vessels; hypertrophy and dilatation of the right ventricle; atrophy of the left atrium; and decrease in size of the left ventricular chamber. During fetal life, with circulation maintained by the right ventricle—via ductus—oxygenation through the placenta is always adequate. After birth, the following sequence of events may occur: temporary or prolonged cyanosis caused by inadequate function of the left ventricle; slow adjustment of both ventricles to their new tasks; decrease of pressure in the pulmonary circuit; increase of pressure in the greater circulation. Abnormal conditions soon after birth may prevent closure of the ductus. They are anoxemia, high pressure in the pulmonary circulation, and hypertrophy of the walls of the ductus.

Patten\textsuperscript{13} stressed the fact that congenital malformations may be caused by either acceleration or retardation of development. He also pointed out that premature closure of the foramen ovale can be explained by a too rapid growth of either the overlapping valve or of the secondary membrane. If we accept that patency of the ductus can be the consequence of an isolated malformation, e.g. the premature closure of the foramen ovale, the different clinical forms could be explained with the different fetal age at which this closure occurred.

There is as yet no direct experimental evidence demonstrating that premature closure or abnormal narrowing of the interatrial opening will cause patency of the ductus. However, autopsies of infants, who died a few hours after birth, revealed the existence of a thick-walled ductus and a definitely closed foramen ovale.\textsuperscript{23, 21*} Recognition of premature closure of the foramen ovale at a necropsy effected later that the neonatal period is obviously a difficult task. Therefore, this theory will be purely speculative until experimental studies prove its truth.

\textsuperscript{*}In a recently published article, Kreutzer et al\textsuperscript{23} described the existence of closed foramen ovale in 3 cases of infants with a patent ductus arteriosus who died with left heart failure.
SUMMARY

The cause of patent ductus with different clinical manifestations and pathological findings—elevated or normal pulmonary pressures—can not be explained by Spitzer's theory of an arrest of development. Premature closure of the foramen ovale is the only isolated congenital malformation which could cause varying degrees of right ventricular hypertrophy, persistence of the fetal characteristics of the pulmonary vascular bed, and occasionally early left ventricular failure.

Two observations support this theory: (a) autopsy findings in infants only a few hours old, revealing a patent ductus with hypertrophied walls and a closed foramen ovale; (b) the knowledge that, except in complex congenital malformations, simultaneous patency of the ductus and of the foramen ovale is practically never found.

RESUMEN

La causa de persistencia del ductus arteriosus con diferentes manifestaciones clínicas: presiones pulmonares elevadas o normales, no puede ser explicada por la teoría de Spitzer de una detención del desarrollo.

El cierre prematuro del foramen oval, es la única malformación congénita que podría causar variados grados de hipertrofia ventricular, persistencia de las características fetales del lecho vascular pulmonar y ocasionalmente insuficiencia ventricular izquierda temprana.

Dos observaciones soportan esta teoría: (a) hallazgos de autopsy con sólo pocas horas de nacidos revelando un ductus abierto con paredes hipertrofiadas y un foramen oval clausurado. (b) el conocimiento de que, con excepción de las malformaciones congéntitas complejas, prácticamente no encuentran jamas simultáneamente persistencia del ductus y del foramen oval.

RESUME

La cause de la persistence du canal artériel avec ses manifestations cliniques variées et ses conséquences pathologiques (pressions pulmonaires élevées ou normales) ne peut s'expliquer par la théorie d'un arrêt du développement qu'invoque Spitzer. La fermeture prématurée du trou de Botal est la malformation congénitale qui pourrait à elle seule entraîner une hypertrophie ventriculaire droite, avec ses degrés variés, la persistance des caractéristiques fœtales du lit vasculaire pulmonaire, et éventuellement une insuffisance ventriculaire gauche précoce.

Deux observations vont à l'appui de cette théorie:

1) Des constatations d'autopsie chez les nouveau-nés, âgés de quelques heures seulement, où l'on a pu constater la présence du canal artériel avec des parois hypertrophiées et le trou de Botal fermé.

2) La notion qu'à l'exception de malformation congénitale complexe, on ne trouve jamais pratiquement associée la persistence du canal artériel et du trou de Botal.
REFERENCES