SECTION ON
CARDIOVASCULAR DISEASES

Proximal Interruption of a Pulmonary Arch
(Absence of One Pulmonary Artery):
Case Report and a New Embryologic Interpretation

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Congenital absence of the right or left pulmonary artery is a rare anomaly, with less than 50 cases in the medical literature. Although Frantzel1 is generally credited with having described the first case of this type, his case was atypical, since it involved an “aortic-pulmonic” defect and had a right pulmonary artery arising from the ascending aorta. This pulmonary artery was probably an “ectopic” pulmonary artery, resulting from abnormal aortic-pulmonic septum formation, and not an “absent pulmonary artery” in the usual sense. The first typical autopsied case to be reported was that by Döring.2 Subsequent cases have been diagnosed at autopsy3-6 and at operation.7, 8 Madoff, et al,8 in their review of the subject in 1952, were the first to report the successful clinical diagnosis of this condition, utilizing angiocardiography. Since then, numerous authors9-19 have reported cases similarly diagnosed. Cardiorespiratory data were included in some of these case reports.9, 11, 11

To the best of our knowledge, no published case has had proved pulmonary hypertension. The following case report is therefore unique in this regard.

Case Report

B. P. (U. H. 887346), a male infant, was born on February 11, 1955, following an uncomplicated, full term pregnancy. Delivery was spontaneous and normal. The birth weight was 4225 grams. There was no neonatal complication, although from birth he was noted to have deep but not labored respirations. At the age of seven weeks he had the first bout of “pneumonia,” characterized by low grade fever and rapid respirations. He was treated at home with intramuscular penicillin, with apparent recovery in a few days. During subsequent months the infant had two similar episodes and was again treated at home.

Between the ages of four and six months, he had two rather severe bouts of pneumonia, both of which required hospitalization. Cardiomegaly was first noted at the age of five months, together with circumoral cyanosis on exertion and crying. Because of the repeated bouts of pneumonia and enlarging heart size, he was referred to University Hospitals on August 10, 1955, for further evaluation. Growth and development up to this time had been normal.

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On physical examination the infant was noted to have slight tachypnea and mild circumoral cyanosis. His weight was 7800 grams and his length was 70 centimeters. A few moist rales were heard in both lungs. The heart was enlarged to percussion. A grade 1 systolic murmur was heard best in the fourth and fifth left intercostal spaces along the left sternal border and the pulmonic second sound was noted to be slightly accentuated. The liver was palpable just below the right costal margin. The electrocardiogram showed right axis deviation, right ventricular preponderance greater than normal for age, and abnormally peaked P waves. Roentgenographic studies showed a moderately enlarged heart with prominence of the pulmonary artery segment and the right ventricle. The pulmonary vascular markings in the left lung appeared increased but those in the right lung appeared decreased (Figure 1). An angiocardiogram showed absence of filling of the right pulmonary artery (Figure 2). He responded well to digitalization and was therefore discharged on September 11, 1955.

He was readmitted three weeks later because of dyspnea, circumoral cyanosis, and pulmonary congestion. He improved with antibiotic therapy. Because of the known occurrence of associated cardiac malformations in patients with absence of a pulmonary artery, cardiac catheterization was done on October 10, 1955. The findings suggested a patent ductus arteriosus (Table 1), so a retrograde aortogram was done the following day in an attempt to better identify such a defect. This study demonstrated multiple fine collateral vessels arising from the aorta and passing to the right chest, as well as a larger vessel arising from the left subclavian artery and crossing over to the right chest (Figure 3). No patent ductus arteriosus was demonstrated.

The patient was discharged from the hospital on October 24, 1955, but subsequently required repeated hospitalizations because of severe dyspnea and cyanosis. He failed to gain weight and his cardiac status gradually worsened. Auricular fibrillation and flutter developed. A repeat angiocardiogram was performed in the antero-posterior view, to check for a possible localized obstruction of the right pulmonary artery. Again, there appeared to be complete absence of the right pulmonary artery (Figure 4). Because his condition was deteriorating rapidly, a repeat cardiac catheterization was done.

![Figure 1](image1.png)  ![Figure 2](image2.png)

*Figure 1:* Roentgenogram of the chest (antero-posterior view), demonstrating cardiomegaly, prominent main pulmonary artery segment, and decreased pulmonary vasculature on the right side.—*Figure 2:* Angiocardiogram (right posterior oblique view), demonstrating absence of the right pulmonary artery.
on March 9, 1956, in the hope of revealing a correctable intracardiac defect. This study showed a further marked increase in pulmonary artery pressure but no left to right shunt (Table I).

He was admitted to the hospital for the last time on April 4, 1956, with marked cardiomegaly and severe congestive failure. He died two days later.

**Autopsy findings:** There was complete symphysis pleurae on the right side. The heart was markedly enlarged, particularly the right ventricle. The valves and endocardium were normal. The ventricular septum was intact and the foramen ovale was closed. The great vessels arose in the usual manner but the main pulmonary artery gave rise only to an enlarged left pulmonary artery (Figure 5). The coronary vessels and pulmonary veins were normal. A ductus arteriosus, measuring 1.5 millimeters in diameter, connected with the left pulmonary artery but not patent. An obliterated vessel, measuring 1.5 millimeters in diameter (probably a right ductus arteriosus) arose from the proximal portion of the innominate artery and passed to the hilum of the right lung. At the hilum this vessel joined a widely patent vessel 4 millimeters in diameter which then branched to supply the various lobes of the lung. The branch of the left subclavian artery demonstrated by aortography was not identified, and apparently supplied the surface of the lung rather than the hilum. The left lung was firm and had lost its crepitance. On cut section, this lung exuded large amounts of bloody froth. The right lung was smaller than the left and had several areas of congestion and atelectasis. Sections of both lungs were examined microscopically by Dr. Donald F. Ferguson of the Veterans Administration Hospital, Minneapolis, Minnesota. A Verhoeff-Van Gieson elastic stain was used. The left lung showed severe
medial hypertrophy of the small arteries but no intimal proliferation was noted in any vessels less than 150 microns in diameter. The veins and capillaries appeared normal (Figure 6). Sections from the right lung were strikingly different. The pulmonary arteries in this lung had very thick and dense adventitia, and seemed rather small in comparison to the adjacent bronchi. The smaller arterioles, so prominent in the left lung, were hardly distinguishable. There was no evidence of medial or intimal lesions in the pulmonary arterial vessels in the right lung (Figure 7). The veins and capillaries were normal.

Discussion

Embryologic Features

Among published cases of absent left or right pulmonary artery there is a preponderance of the former. The former tends to be associated with an intracardiac defect, particularly tetralogy of Fallot, while absence of the right pulmonary artery generally occurs as an isolated finding.\textsuperscript{18, 20} There have been at least five cases of absent right pulmonary artery described with an associated patent ductus arteriosus,\textsuperscript{1, 2, 4, 11, 19} and in two of these there was also a coarctation of the aorta.

McKim and Wiglesworth\textsuperscript{12} reviewed the embryologic aspects of this defect in considerable detail, and emphasized that the aortic arch is usually on the side opposite to the absent pulmonary artery. Thus, absent left

\textbf{FIGURE 3} 
\textbf{FIGURE 4}

\textit{Figure 3:} Retrograde aortogram (antero-posterior view), demonstrating bronchial arteries and an anomalous branch from the left subclavian artery passing to the right chest.—\textit{Figure 4:} Angiocardiogram (antero-posterior view), demonstrating absence of the right pulmonary artery.
pulmonary artery often occurs in tetralogy of Fallot having a right aortic arch. These authors described an anomalous vessel running from the innominate artery to the left lung hilum in three cases with right aortic arches and interpreted this vessel as an obliterated left ductus arteriosus. They considered that the heart end (the ventral portion of the sixth arch) of the left pulmonary artery had disappeared, while the ductus arteriosus (the dorsal portion of the sixth arch) had remained in continuity with the distal portion of the left pulmonary artery, first as a functioning vessel and later as a fibrotic non-patent structure. McKim and Wiglesworth also suggested that the aortic bud (dorsal portion of the sixth arch) may have grown directly into the pulmonary plexus without there having been a pulmonary artery per se, but considered this a less likely possibility in view of the normal anatomical development of the hilar and intrapulmonary arteries in these cases.

Emanuel and Pattinson\(^\text{2}\) called attention to the presence of developmental defects of the bulbus cordis (as in tetralogy of Fallot) in cases

![Diagram of heart and pulmonary vessels](image_url)

**Figure 5:** Drawing of autopsy specimen, showing connection of right ductus arteriosus (obliterated lumen) to right pulmonary artery.
where a left pulmonary artery is absent, and attributed the latter to faulty absorption of the left sixth arch. They stated that if the conventional view of symmetrical development of the two pulmonary arteries is accepted, the foregoing association could not be explained. They pointed out that this still did not explain the association of absent right pulmonary artery with normal intracardiac anatomy, though they suggested that in such cases perhaps the proximal portion of the right sixth arch became involved in the normal absorption of the left sixth arch.

To us, the observations and interpretations of both the foregoing groups, as well as those of the embryologists, Bremer and Congdon, can best be brought together as follows. The aortic (fourth) arches begin symmetrically, but unilateral dominance develops rapidly. Similarly, the sixth arches begin symmetrically, but deviate at about the time that the arterial trunk is becoming divided (Figure 8a). The dorsal or ductal portion (B) of the pulmonary arch on the side opposite to the dominant pulmonary arch (carrying the greater blood flow) normally undergoes degeneration. This allows a straightening out of the pulmonary artery on this side, and also combines with the action of the increasing blood current to permit the pulmonary trunk and other pulmonary arch to align. The result in the normal individual is that the main pulmonary artery is derived from the pulmonary trunk and some of the proximal portion (A) of the left pulmonary arch; the left pulmonary artery consists of
the primitive left pulmonary artery; and the right pulmonary artery consists of the primitive right pulmonary artery plus a portion (D) of the right pulmonary arch (Figure 8c). Thus there is definite asymmetrical

FIGURE 8: a. Schematic diagram of pulmonary arch system at early stage of normal development. The terminology of Emanuel and Pattinson has been followed for ease of comparison. Proximal (ventral) portion of pulmonary arch designated as “A” on left and “D” on right, and distal (dorsal or ductal) portion designated as “C” on left and “B” on right. Left and right primitive pulmonary arteries designated as “LPA” and “RPA.” Pulmonary trunk indicated by “TP,” and left and right descending aortae, by “LDA” and “RDA.” b. Comparable diagram in case where the right pulmonary arch becomes dominant. c. Standard adult pattern of pulmonary vessels, with embryologic derivatives indicated. “MPA” indicates main pulmonary artery. d. Comparable adult pattern where right pulmonary arch was dominant. e. Adult pattern in case of absent right pulmonary arch. The left ductus arteriosus (shown here as being patent) indicated by “C,” the right ductus arteriosus (shown here as being obliterated) indicated by “B,” and the underlying arch defect (interruption) indicated by “D.” f. Adult pattern in case of absent left pulmonary arch. The right ductus arteriosus (shown here as being patent) indicated by “B,” the left ductus arteriosus (shown here as being obliterated) indicated by “C,” and the underlying arch defect (interruption) indicated by “A.”
development of the definitive pulmonary arteries. The direction of asymmetry is probably dependent primarily on the characteristics of the aortic sac and anterior arches, the fourth in particular (which determines the location of the definitive aortic arch). Blood flow is certainly involved in this deviation in symmetry, but it is difficult to distinguish cause from effect in this regard. It is well to remember that the normal interruption of the right pulmonary arch (ductal portion or B) takes place prior to the normal interruption of the right fourth arch.

One can theorize that if the dorsal portion (B) of the pulmonary arch on the side opposite the definitive aortic arch persists (perhaps due to blood flow factors), there will be interference with the development described above, and the ventral portion (D) of this pulmonary arch may then be obliterated. This will result in an "absent pulmonary artery" on this side, or rather in "proximal interruption of the pulmonary arch" (Figure 8e). The primitive pulmonary artery will remain intact, but will connect with the dorsal (B) portion (which will usually be subsequently recognized only as a fibrotic structure best identified as a ductus arteriosus, but which may persist as a patent vessel) rather than with the ventral portion (D) of the pulmonary arch (which would normally be incorporated into the definitive pulmonary artery). The absent pulmonary artery will always occur on the side opposite the larger ductal (dorsal) arch segment. Since the normal heart usually has a left aortic arch and a left ductus arteriosus, the absent pulmonary artery will usually be on the right side. In abnormal hearts with right aortic arches, the larger ductal segment would generally be expected to be on the right side (Figure 8b), and an absent pulmonary artery would then be on the left side (Figure 8f). In cases of absent left pulmonary artery with defects of the bulbis cordis, but with left aortic arches, one must assume that the dominant pulmonary arch was on the right. Likewise, for those very rare cases of normal heart with left aortic arch and absent left pulmonary artery, one must assume that the dominant pulmonary arch was on the right. The development of large bronchial vessels from the aorta to the affected lung in these cases can be viewed as a secondary process.

Our theory appears to explain all cases. It differs greatly from that of Emanuel and Pattinson in that these workers implicated faulty absorption of the segment on the side of the ductus (dominant ductal side), whereas we implicate degeneration of the corresponding segment on the opposite side. It differs from the interpretation of McKim and Wiglesworth in that the latter workers did not refer to possible reversal of the dominant pulmonary arch in their cases of absent left pulmonary artery, and did not stress the asymmetrical development of the pulmonary arteries. Since we ascribe great significance to the location of the dominant pulmonary arch (and its ductal segment), the next question which arises has to do with the factors determining arch dominance. We have already assumed that the development features of the fourth arches will influence those of the sixth arches. In addition, one can assume that faulty differentiation of the aortic sac and arterial trunk (common trunk) will
affect the location of the dominant pulmonary arch through its effect on the fourth arches, but may also be of more direct influence at times (as in tetralogy of Fallot with left aortic arch but with absent left pulmonary artery, and therefore a dominant right pulmonary arch by our theory). Actually, the bulbus cordis defects may well be a result of faulty aortic sac and arterial trunk differentiation, and the association of bulbus cordis defects with absent pulmonary artery probably is not one of cause and effect.

The association noted between absent right pulmonary artery and abnormalities of the great vessels does not conflict with this theory, but is just as one might expect if arch differentiation is abnormal. The infrequent occurrence of bilateral ductus arteriosus in cases of otherwise normal hearts and pulmonary arteries is certainly in line with our theory, yet its rare occurrence in no way negates it. Moreover, it fits those cases of truncus arteriosus having absence of one pulmonary artery. If the left pulmonary artery is absent in such cases, the right pulmonary artery arises from the right posterior aspect of the truncus; if the right pulmonary artery is absent, the left pulmonary artery arises from the left posterior aspect. Collett and Edwards23 list one case (Dickson and Fraser24) which would appear to be an exception, inasmuch as the right pulmonary artery was listed as arising from the left side of the truncus; however, examination of the original paper shows the description and drawing to be indeterminate and confusing on this point. Another case (Shapiro25) of truncus is pertinent to the earlier discussion; this case had a left pulmonary artery arising from the left side of the trunk and a right innominate artery giving off a branch to the right lung; this latter vessel was not referred to as a ductus arteriosus, but it is certainly very suggestive of such a structure in view of the embryologic features already described.

Diligent search for residual ductal structures in specimens of truncus arteriosus lacking both pulmonary arteries (given a separate classification by most workers) may demonstrate such structures. This would of course immediately identify them as close relatives of cases with pulmonary arteries, since the presence of a ductus arteriosus indicates the earlier presence of a pulmonary arch. It is much easier to accept faulty differentiation of the pulmonary arches, with subsequent degeneration of various structures, than to visualize complete failure of the sixth arches to form. Failure of clear-cut dominance of one or the other pulmonary arch may result in two pulmonary arteries each with a functioning ductus arteriosus or in bilateral interruption of the proximal portions of the pulmonary arches. Although this suggestion is included largely for speculation, the observation that unilateral absence of a pulmonary artery is apparently not due to an absence of a pulmonary arch should make one hesitant in invoking such an explanation for absence of both pulmonary arteries. There may well be similar or closely related factors involved in the development of all defects of the aortic and pulmonary arches, truncus, and bulbus cordis.

Since in all cases of absent pulmonary artery there appears to be
simply a break in pulmonary arch continuity, it would appear more appropriate to term this defect as "proximal interruption of the pulmonary arch." The primitive pulmonary arteries *per se* remain intact, whereas the proximal arch component of the definitive pulmonary artery is deficient. Normally there is "distal interruption" of the pulmonary arch, so one must be careful to refer to these cases as "proximal interruption." Interruption of the pulmonary arches has much in common with interruption of the aortic arches, since in the latter too there is normal as well as abnormal interruption. Likewise in each there may be failure of interruption, giving bilateral ductus arteriosus in the former and double aortic arch in the latter.

The functioning of the ductus arteriosus before birth probably accounts for the failure of large bronchial arteries to develop on the involved side. Also, it probably allows a near-normal development of the lung on the affected side. The decreased size of the lung on the affected side, as described in the majority of published cases, may well be due to differential postnatal growth resulting from differences in blood supply.

The autopsy findings in our cases followed exactly the pattern described by McKim and Wiglesworth, and like two of their cases, involved bilateral ductus arteriosus. Our case, like that of Emanuel and Pattinson, showed highly vascular adhesions between the affected lung and the chest wall. (We assume that the branch from the left subclavian artery demonstrated by aortography supplied blood to this area.)

**Clinical and Physiological Features**

If present as an isolated defect, congenital absence of one branch of the pulmonary artery has been usually described as essentially asymptomatic. However, there may be a slight decrease in exercise tolerance and pneumonitis is said to be fairly common in the involved lung. Hemoptysis occasionally occurs, and in at least one case rupture of an arteriosclerotic bronchial vessel was established as the cause of death.

Absence of one of the pulmonary arteries can often be diagnosed on the ordinary chest roentgenogram. The involved hemithorax is smaller and the heart and mediastinum are shifted to that side. The involved lung is very radiolucent. As mentioned earlier, angiocardiography provides an exact diagnosis. Cardiac catheterization has been performed in only a few of the reported cases, and in these the resting pulmonary artery pressures were normal, with only a slight increase on exercise. In the present case, aortography was useful in demonstrating the collateral blood supply to the affected chest, and in retrospect, indicated that the ductus arteriosus on each side was not patent.

Studies of pulmonary function in published cases showed either normal or slight reduction in vital capacity, residual volume, and total lung volume. Bronchospirometric studies using room air demonstrated normal ventilation in the involved lung but non-participation in oxygen uptake. It has been shown experimentally that as much as one third of the output of the left ventricle may go to the bronchial arteries of a lung in which the pulmonary artery has been ligated.
suggested that the patient with a congenitally absent pulmonary artery may be benefited by the removal of the lung on the affected side, thereby decreasing the load on the left ventricle.

The pulmonary pathology found in our patient was unusual. The medial hypertrophy in the small arteries of the left lung was severe, the average cross-section area of the lumen being only about 12% of the total average cross-section area of the artery. This degree of narrowing was probably sufficient to account for the high calculated total pulmonary resistance obtained at each cardiac catheterization. The left lung of this patient accepted the total right ventricular output, and pulmonary flow in this lung could therefore be assumed to be twice normal. These pulmonary changes differed somewhat from those seen in ventricular septal defect with "secondary" pulmonary hypertension. In the latter there is usually intimal proliferation of the small pulmonary arteries in addition to some degree of medial hypertrophy when the resistances are in the range calculated for this patient. The absence of intimal proliferation suggests a different pathogenesis between the pulmonary resistance in our case and the type seen in ventricular septal defect ("secondary"). The thickness of the media compared to the total vessel size is greater in this patient's left lung than in normal newborns. The total pulmonary resistance of 2585 dynes sec. cm.\(^{-5}\) obtained at the first cardiac catheterization is comparable to normal newborn resistances. However, the finding of an increased resistance of 6200 dynes sec. cm.\(^{-5}\) at 13 months of age indicates that this is a progressive phenomenon and therefore represents more than a mere persistence of normal medial hypertrophy of the newborn.

Even though the patient was over one year of age at the time of death, the finding of a closed foramen ovale suggests an interesting possibility. If the foramen ovale closed prematurely, from days to weeks prior to birth, it would be expected that the left atrium and mitral valve would not be as large as normal because of diminished flow into this chamber through this normal fetal passage. Thus, a relative mitral stenosis would be created. This, however, might be minimal enough so that it would not be easily measured or observed by routine measures (in our specimen, the mitral valve was slightly smaller than normal, but was not stenotic). Similar pulmonary pathology has been noticed in mitral stenosis patients. A combination of twice the normal pulmonary flow plus some degree of mitral obstruction might more readily account for the pulmonary pathology observed in this patient.

Dammann and Ferencz\(^{27}\) described the lung findings in McKim and Wiglesworth's case of "Eisenmenger complex" with absent left pulmonary artery.\(^{15}\) The small pulmonary vessels were thick walled in the right lung (lumen-wall ratio of 2.6) and thin walled in the left (ratio of 5.8). In this case, the right lung was said to be under the stress of a common ejective force. No physiological data were obtained, and therefore it is difficult to make comparisons with our case. However, because of the presence of a ventricular septal defect in their patient, we assume that there may have been some degree of "secondary" pulmonary hypertension.
Surgical Aspects

In our case, because of the presence and progression of pulmonary hypertension, it was considered inadvisable to remove the lung on the involved side. In retrospect, inasmuch as this case appeared to have normal hilar and intrapulmonary vessels on the involved side, it would have been possible to do surgical correction or alleviation by making an anastomosis between the hilar portion of the right pulmonary artery and the main pulmonary artery by means of an arterial graft. This same conclusion holds true both for cases of isolated absent pulmonary artery and for cases associated with tetralogy of Fallot. In the latter instance it will be important to be prepared at the time of corrective surgery to bridge the interrupted portion of the artery with a graft. Actually, one of our recent surgical cases was a four year old boy having a tetralogy of Fallot defect with left aortic arch and absent left pulmonary artery. At surgery, the ventricular septal defect was closed and the infundibular pulmonary stenosis corrected. The patient died and on routine autopsy no left pulmonary artery was identified. In view of the findings of McKim and Wiglesworth\(^1\), later examination was done, and this showed a blind pouch, 4 millimeters in diameter, corresponding to the hilar end of the pulmonary artery system; it gave rise to the normal system of pulmonary artery branches, and was connected proximally to a small occluded vessel which was undoubtedly a fibroscled ductus arteriosus.

Whether the above contemplated surgery would have helped in our case, or whether it is ever indicated in the usual case of isolated absent pulmonary artery remains problematical. It seems likely that the pulmonary stump or pouch at the hilus is about as large at birth as it will ever become, and thus surgery if it is to be done at all, perhaps should be done in infancy. Maier\(^4\) suggested that in one of his cases of absent right pulmonary artery with an anomalous vessel from the aorta supplying the lung, the proximal end of this anomalous vessel could have been transferred to the side of the main pulmonary artery. From the autopsy findings, this case would seem to be different from the usual case of absent pulmonary artery, resembling the "ectopic" pulmonary artery of Frantzel's case already described.

SUMMARY

Congenital absence of the right pulmonary artery is described in a male infant with pulmonary hypertension. Gross and microscopic pathological findings are presented, as well as cardiac catheterization data and angiocardiographic findings. A theory is presented which appears to explain all variations of absent pulmonary artery. It is suggested that a more accurate term for this defect would be "proximal interruption of pulmonary arch." There is reason to believe that this condition is surgically correctable by means of an arterial graft.

RESUMEN

La ausencia congénita de la arteria pulmonar izquierda se describe en el caso de un niño con hipertensión pulmonar. Se presentan los hallazgos
macro y microscópicos así como los datos de angiocardiografía y cateterización cardiaca.

Se muestra una teoría que parece explicar todas las variaciones de la arteria pulmonar ausente. Se sugiere que se use un término más exacto para este defecto, el que sería “interrupción proximal de un arco pulmonar.” Esta es una razón para creer que este defecto puede corregirse por injerto arterial.

RESUME

L'auteur décrit l'absence congénitale d'artère pulmonare droite, chez un bébé du sexe masculin atteint d'hypertension pulmonaire. Il présente les constatations anatomo-pathologiques macro- et microscopiques, ainsi que les résultats du cathétérisme cardiaque et les constatations angiographiques. L'auteur expose une théorie qui semble pouvoir expliquer l'absence d'artère pulmonaire quel qu'en soit le degré. Il suggère qu'un terme plus précis soit adopté pour désigner cette altération, et qui pourrait être “interruption proximale d’un arc pulmonaire.” Il y a des raisons de croire que cet état peut être corrigé chirurgicalement au moyen d’une greffe artérielle.

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REFERENCES