Embolic Occlusion of Patent Foramen Ovale
A Syndrome Occurring in Pulmonary Embolism

By G. B. Elliott, M.B., and R. E. Beamish, M.D.

In the presence of a patent foramen ovale, pulmonary embolism may result in a right-to-left interatrial shunt which serves to alleviate the effects of the embolism. When the shunt is occluded by a subsequent embolus, sudden death occurs. The clinical and pathologic features of the syndrome are described.

RECENTLY two cases of pulmonary infarction were encountered which showed atypical clinical manifestations. At autopsy, in each instance a thrombus was found straddling a patent foramen ovale, suggesting a functional shunt during life. It is the purpose of this paper to describe the associated syndrome.

Although some 48 instances of a clot caught in a patent foramen ovale have been described since 1859, there has been no uniformity in terminology or clarity of thought regarding it. Many have been described as "paradoxic embolism" and a recent paper refers to it as "Lodging of an Embolus in a Patent Foramen Ovale." The term "paradoxic embolism" was originally devised by Zahn in 1885 to describe a condition in which emboli derived from the systemic venous system reached the systemic arterial system by passing through an abnormal communication between the chambers of the heart. It is apparent that this occurs when a small thrombus passes through a cardiac septal defect, but this is quite different from the event of a large thrombus being caught in the septal defect with consequent occlusion of its lumen. Thus embolic occlusion of a patent foramen ovale is to be distinguished from "paradoxic embolism" which may or may not accompany it.

Autopsy specimens showing a thrombus caught in the act of traversing an atrial septal defect have been of interest only as anatomic confirmation that paradoxic embolism does occur. Many of the patients were thought to have died of "further pulmonary embolism," and the surprise finding of a thrombus occluding a patent foramen ovale has been regarded as a coincidental occurrence. According to Vimtrup, the earliest account of such a condition is that of Wallman, who in 1859, wrote: "I have found a tough clot resembling thrombus in the auricular and ventricular cavities of three hearts, in which a firm cord of thrombus passed through a valvular patency of the foramen ovale from the left into the right auricle. In these three instances one supposes that at the end of life, when auricular pressure relationships may alter, a transfer of blood from one to another auricle might occur." Despite the implication that such an occlusion might have had serious hemodynamic consequences, it does not appear that these have been recognized although both Kyber and Johnson briefly alluded to the possibility.

CASE REPORTS

Case 1. C. M., a 66 year old male with a previously diagnosed duodenal ulcer, was admitted to the Winnipeg General Hospital on Nov. 4, 1950, with an acute gastrointestinal hemorrhage. He received several transfusions and improved; a few days later, however, bleeding recurred and it was decided that a gastrectomy should be performed. Because he had had an infected hematoma of the left leg following injury three years previously, his legs were carefully examined and no evidence of thrombophlebitis found. He was prepared for surgery and on November 14, a partial gastrectomy was carried out uneventfully. His condition postoperatively was good and he was up in a chair the day following operation. Progress continued unimpeptfully until the evening of November 19 (fifth postoperative day). At 10:40 p.m., while on a bed
pan, he developed sudden severe dyspnea without pain. He was seen to be pale and cyanosed; the heart was rapid (130 to 150) with a triple rhythm and blood pressure was 70/40. Neck veins were moderately distended. Clinical examination of the lungs and heart was negative. It was thought at first that he had either a concealed internal hemorrhage or a massive pulmonary embolism.

It soon became clear that his shocked state was not due to hemorrhage, and he was treated for a pulmonary embolism with oxygen, blood, intravenous procaine and heparin. An emergency electrocardiogram was taken, but, apart from tachycardia, it was not remarkable. During the night his condition remained unchanged: he was pale; cyanosis persisted in spite of oxygen; he perspired profusely; his skin was cool; his heart rate remained rapid and his pulse small. Blood pressure after four bottles of blood, remained at 75/60, and he was almost anuric. Next morning (November 20) the electrocardiogram was repeated and showed no change from the evening before. (See fig. 1.) There was little change in this

![Fig. 1. Electrocardiogram of case 1. Apart from tachycardia, the tracing is within normal limits. (On vector analysis, record is consistent with, but not diagnostic of, pulmonary embolism.)](image1)

![Fig. 2. View of opened atria in case 1. The intracardiac embolus is caught in the act of traversing an atrial septal defect, and the end projecting into the left atrium is intact.](image2)
state throughout November 20 and November 21, and his sensorium remained clear. Considerable misgivings were felt about the working diagnosis of pulmonary embolism when he neither got worse nor better as time wore on. Late in the evening of November 21, he became more dyspneic and the blood pressure fell to 50/40. A continuous adrenaline drip was started, but his general condition deteriorated and he died at 1:40 a.m., November 22, 50 hours and 45 minutes after his collapse.

An autopsy eight hours later showed deep cyanosis of face and limbs. Fluid blood distended the right atrium and ventricle so that they formed three-fourths of the presenting surface of the heart. An embolus 10 cm. long, arching up from the opening of the inferior vena cava, was tightly impacted without adhesion in a 1.0 cm. diameter patency of the foramen ovale (fig. 2). The venous end was broken and laminated. In contrast, 3.0 cm. of firm unbroken embolus tapering to a rounded tip projected into the left atrium. A hemorrhagic infarct 3.0 cm. in diameter occupied part of the lower lobe in each lung. All pulmonary arteries except the main branch to the right middle lobe, one tertiary division to the right upper lobe, and two to the left upper lobe, were occluded by brittle, moderately adherent emboli. Considering that there are usually 10 tertiary subdivisions of the pulmonary artery, this represents about 50 per cent occlusion of the arterial tree. Lightly adherent antemortem thrombi up to 15.0 cm. in length were expressed from each femoral vein. Microsections of viscera confirmed the character of emboli and infarcts.

Comment. The prolonged unexplained survival of this patient in an unusual state, together with the remarkable embolic occlusion of a possibly large right-to-left shunt in the heart, suggested that the patent foramen ovale served as a palliative shunt and that its occlusion was the precipitating cause of death. This interpretation led to a review of clinical features of cases which had been reported from a pathologic viewpoint. During this investigation, a second case, complicated by a paradoxical air embolism was observed.

Case 2. Mrs. C. C., a 24 year old primipara, was delivered on June 16, 1951, and developed a persistent systemic postpartum infection after removal of retained placenta. She was admitted to the Winnipeg General Hospital on July 19 with acute diffuse peritonitis and paralytic ileus. Slow symptomatic improvement followed intubation, antibiotic therapy and intravenous feeding. On August 8, pulmonary embolism was diagnosed, following an attack of sudden, substernal, breath-catching pain with dullness in the right lower lobe posteriorly. Two days later clear fluid aspirated from a right pleural effusion yielded no growth or culture. By August 14 it was evident that the peritonitis had localized with pelvic and subphrenic abscess formation.

The right twelfth rib was resected at an elective operation for drainage of the latter on August 17. While separating adhesions manually over the dome of the liver, several hundred cubic centimeters of clear yellow fluid gushed forth, and the surgeon believed he had inadvertently entered the right pleural cavity. The patient immediately blanched, respiration ceased, and the radial pulse became feeble, then imperceptible. After hearing an initial precordial churning murmur, the anesthetist heard no heart sounds and blood pressure was unobtainable. A new epicardial incision showed apparent cardiac standstill; direct cardiac massage gradually restored beating after a three minute pause. Further exploration was abandoned. The pulse was again found to vanish when turning the patient on her right side during dressing, to reappear when supine. A portable radiograph showed a right pneumothorax. No intracardiac translucencies, as described by Taylor,7 were distinguishable on review. Her condition deteriorated two hours later and 500 cc. of air were aspirated and closed pleural suction drainage set up. Convulsive twitching of limbs was noticed in the next hour. Deep coma persisted with profuse perspiration, ashen cyanosis relieved by oxygen, labored respiration at 30 per minute, irregular tachycardia of 150 per minute, and a blood pressure of 160/100. The blood pressure fell abruptly 45 hours after operation to 100/80 and there was steady deterioration to death 40 minutes later.

At autopsy three hours later, the cranial cavity was first explored with care, then the thoracic cavities opened under water, using the technics recommended to demonstrate air embolism. Bubbles up to 2 mm. diameter were enmeshed in postmortem clot in the sagittal sinus, with minute bubbles in the superficial cerebral vessels and emissary veins in all areas. The brain showed no gross changes. A right pneumothorax at atmospheric pressure was detected by needle exploration with manometric attachment. A few tiny bubbles were present in branches of the coronary artery over the anterior surface of both ventricles. Early fibrinous pericarditis was present. Some bloody froth occupied the ventral parts of the right ventricle and pulmonary artery with small amounts in the right atrium. A nonadherent antemortem thrombus, 5.3 cm. long and 0.3 cm. in diameter was held in a slit-like anterior valvular patency of the foramen ovale 0.5 cm. in diameter. Some 1.8 cm. projected into the left atrium, neither rounded end showing fragmentation (fig. 3).

The completely atelectatic right lung showed fibrinous pleuritis and two brownish friable infarcts, 3.0 and 1.8 cm. diameter in the lower lobe, the larger being septic. The latter lay posteromedially, an-
chored superiorly by a band-like adhesion. A tear beginning in this attachment extended for 3.5 cm. through the infarct and intact lung to end in mediastinal pleura. Blue ink injected into the inferior vena caval area terminated, after clamping off the heart, returned through the medial end of this tear, but the communicating vein was not identified. In the depth of the packed laparotomy wound was a 6.0 cm. split in muscle forming the posterior part of the right diaphragmatic dome. This appeared to be the site of accidental entry into the pleura. The remaining lesions were an unopened right subphrenic abscess, two small septic hepatic infarcts posterior to this abscess, fibrinous peritonitis, small septic infarcts in both kidneys, and bilateral tubovarian abscesses. Friable, free, septic, antemortem thrombi lay in the left common iliac, left internal hypogastric, and right femoral veins. A hemolytic, coagulase-positive staphylococcus was isolated from the septic sites, but no gas-producing organisms were found. Microscopic examination confirmed the character of the lesions.

**Comment.** The massive atelectasis of the right lung, together with infarction, constituted a major obstruction to the pulmonary circulation during life. In this instance, deep coma probably due to cerebral air embolism replaced the clear consciousness shown by the first case. The observed clinical sequence was otherwise similar, peripheral paradoxic emboli being an autopsy finding. In addition to being of interest in connection with the syndrome under consideration, this case has some significance in relation to paradoxic air embolism.

The physiologic basis of air embolism, both in venous and arterial forms, has been reviewed by Durant while Merkel has reviewed paradoxic air embolism, a very rare variety.

In this patient, the clinical collapse with a classic churning murmur over the right

![Fig. 3. View of intracardiac embolus in case 2, found straddling an anterior valvular patency of the foramen ovale. The larger portion lies in the right atrium and both ends are intact.](image_url)
assume that this shunt was the route of the arterial air emboli.

**Discussion**

Evidence for the anatomic and physiologic basis of paradoxical blood flow from right-to-left atrium has been presented in several reviews.6, 10 After birth the tension in the right atrium falls, and the higher pressure in the left atrium now becomes sufficient to establish competence of the valvelike flap over the foramen ovale. Lesions reversing this adult atrial pressure relationship re-establish the fetal shunt where patency persists. Foremost among such lesions are pulmonary embolism and infarction, where the following sequence develops: (1) pulmonary embolism occurs and the pressure in the right atrium rises; (2) when this exceeds the left atrial pressure, blood flows through the foramen ovale. Clearly a subsequent embolus arriving in the right atrium may then, if small, pass into the left atrium and arterial circulation, but if large, impaction results.

In cases of massive pulmonary embolism, it has been supposed by Johnson6 that “a patent foramen ovale would be instrumental in prolonging life by providing a much needed shunt.” Kyber6 thought of it as “a sort of emergency outflow.” The behavior of case 1 indicates that this is so, and in addition that when this shunt is blocked, death is precipitated. The clinical course of this patient would appear to constitute a syndrome as follows:

1. A clinical sequence of collapse from pulmonary embolism, incomplete recovery, sudden deterioration and precipitate death.

2. The recovery phase in its most recognizable form is characterized by a state of prolonged survival with greyish pallor, moderate cyanosis unrelieved by oxygen (because of the shunt), profuse perspiration, persistently low blood pressure, a fast thready pulse, and only moderate venous distention. Peripheral paradoxical embolism may occur in this phase.

3. Absence of electrocardiographic changes diagnostic of acute cor pulmonale both in the initial collapse and recovery phases, presumably due to the decompressing action of the shunt. (See fig. 1.)

4. At death cyanosis and venous distention are conspicuous due to acute cor pulmonale supervening when the decompressing shunt is blocked.

Table 1 presents an analysis of the 29 cases of embolic occlusion of foramen ovale by thrombus occurring after 1926. The previous 18 reports are exclusively in the older European literature not generally available, and only 10 contain clinical detail. It is seen that there are 14 cases (cases 1–14) which show the clinical sequence of the syndrome described. However, five (cases 10–14) of these were complicated by cerebral embolic lesions, and rapid death is not spectacular in patients already comatose. Nevertheless, Young’s11 case (case 12) died suddenly, and Lindley12 noted that “death occurred suddenly while stuporous” in his case (case 13). In our case 2 (case 14), there was terminal worsening in the last 40 minutes, during deep coma. Vimtrup’s1 case (case 11) died in coma one and one-half hours after cerebral embolism, and Wittig’s13 case (case 10) shows a closely similar termination. In contrast, the 12 cases of coma due to presumptive paradoxical cerebral embolism, reported after 1930,10, 14–18 and not associated with foraminal occlusion, do not show this abrupt demise. Most survive for weeks after the cerebral episode, the shortest survival being eight hours. There is only one instance of sudden death and this was due to an obvious massive pulmonary embolism. Thus it is apparent that cerebral embolism, per se, is not a decisive cause of precipitate death; it is rather our belief that the sudden demise is due to the intracardiac embolism.

Of the remaining 15 cases the demise is undescribed in five (cases 15–19), three of whom had paradoxical cerebral embolism of comparatively short duration. Five cases (cases 20–24) showed sudden or precipitate death without apparent clinical pulmonary embolism. However, of these, Elliott’s19 case (case 20) showed a septic infarct in one lung, and Merkel’s6 case (case 22) showed old as well as recent pulmonary emboli, while recent
Table 1.—Cases of Embolic Occlusion of Patent Foramen Ovale Reported after 1926

<table>
<thead>
<tr>
<th>Author</th>
<th>Pulmonary Embolism or Infarction Clinical: Autopsy</th>
<th>Recovery Phase Duration</th>
<th>Peripheral Arterial Embolism</th>
<th>Length and Mode of Terminal Collapse</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Barnard(^{13})</td>
<td>+</td>
<td>½ day</td>
<td>+</td>
<td>Sudden death while talking.</td>
</tr>
<tr>
<td>2. French(^{21})</td>
<td>+</td>
<td>23 days</td>
<td>−</td>
<td>15 minutes of acute clinical symptoms.</td>
</tr>
<tr>
<td>3. Taylor(^{28})</td>
<td>+</td>
<td>21 days</td>
<td>+</td>
<td>“Shortly after” acute exacerbation.</td>
</tr>
<tr>
<td>4. Koritschner(^{27})</td>
<td>+</td>
<td>3 hours</td>
<td>−</td>
<td>Sudden death.</td>
</tr>
<tr>
<td>5. Ingham(^{16})</td>
<td>III</td>
<td>4 days</td>
<td>+</td>
<td>“Suddenly became worse and died.”</td>
</tr>
<tr>
<td>6.</td>
<td>IV</td>
<td>2 days</td>
<td>−</td>
<td>Sudden exacerbation and death.</td>
</tr>
<tr>
<td>7.</td>
<td>V</td>
<td>½ day</td>
<td>−</td>
<td>Sudden death.</td>
</tr>
<tr>
<td>8. Vimtrup(^{3})</td>
<td>I</td>
<td>14 days</td>
<td>−</td>
<td>15 minutes of acute symptoms.</td>
</tr>
<tr>
<td>9. Present series</td>
<td>I</td>
<td>2 days</td>
<td>−</td>
<td>2½ hours—died in acute exacerbation.</td>
</tr>
<tr>
<td>10. Wittig(^{13})</td>
<td>+</td>
<td>14 hours</td>
<td>+ (Brain)</td>
<td>Unstated—died 2 hours after cerebral infarction.</td>
</tr>
<tr>
<td>11. Vimtrup(^{3})</td>
<td>II</td>
<td>1½ hours</td>
<td>+ (Brain(^{*}))</td>
<td>Died after brief coma.</td>
</tr>
<tr>
<td>12. Young(^{11})</td>
<td>+</td>
<td>7 days</td>
<td>+ (Brain(^{*}))</td>
<td>Sudden death.</td>
</tr>
<tr>
<td>13. Lindley(^{12})</td>
<td>+</td>
<td>12 days</td>
<td>+ (Brain(^{*}))</td>
<td>“Sudden death while stuporous.”</td>
</tr>
<tr>
<td>14. Present series</td>
<td>II</td>
<td>2 days</td>
<td>+ (Brain(^{*})) Air</td>
<td>40 minutes—died in coma.</td>
</tr>
<tr>
<td>15. Jones(^{29})</td>
<td>+</td>
<td>12 days</td>
<td>+ (Brain)</td>
<td>Died on 11th day of coma.</td>
</tr>
<tr>
<td>16. Ingham(^{16})</td>
<td>I</td>
<td>10 days</td>
<td>+</td>
<td>Unstated.</td>
</tr>
<tr>
<td>17.</td>
<td>II</td>
<td>7 days</td>
<td>+ (Brain)</td>
<td>Died on 4th day of hemiplegia.</td>
</tr>
<tr>
<td>18. Robinson(^{1})</td>
<td>+</td>
<td>3 days</td>
<td>−</td>
<td>Unstated.</td>
</tr>
<tr>
<td>19. Johnson(^{6})</td>
<td>I</td>
<td>2 days</td>
<td>+ (Brain)</td>
<td>Unstated—died 1 day after cerebral infarction.</td>
</tr>
<tr>
<td>20. Elliott, T. R.(^{19})</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>Sudden death.</td>
</tr>
<tr>
<td>21. Wilson(^{29})</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>5 minutes—sudden death.</td>
</tr>
<tr>
<td>22. Merkel(^{9})</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>Sudden death.</td>
</tr>
<tr>
<td>23. Hirchhooeck(^{28})</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>35 minutes of acute symptoms.</td>
</tr>
<tr>
<td>24. Ingham(^{16})</td>
<td>VI</td>
<td>Not stated</td>
<td>Not stated (^{?}) (Heart)</td>
<td>Sudden death and cyanosis.</td>
</tr>
<tr>
<td>25. Wolff &amp; White(^{21})</td>
<td>XI</td>
<td>Not stated</td>
<td>Not stated (^{?}) (Heart)</td>
<td>Sudden death.</td>
</tr>
<tr>
<td>26. Geipel(^{22})</td>
<td>I</td>
<td>Not stated</td>
<td>Not stated (^{?}) (Heart)</td>
<td>“Died a few days after myomectomy.”</td>
</tr>
<tr>
<td>27.</td>
<td>II</td>
<td>Not stated</td>
<td>−</td>
<td>Not stated.</td>
</tr>
<tr>
<td>28.</td>
<td>III</td>
<td>Not stated</td>
<td>−</td>
<td>“Death from pulmonary embolism.”</td>
</tr>
<tr>
<td>29.</td>
<td>IV</td>
<td>Not stated</td>
<td>−</td>
<td>“Death from pulmonary embolism.”</td>
</tr>
</tbody>
</table>

\(^{*}\) Clinical evidence.

emboli were present in the remainder of the group. In addition, Wolff and White (case 25) make a very short note\(^{21}\) in which the condition of the lungs is not specifically mentioned and “embolism of the descending branch of the left coronary artery” is also described. Finally, in Geipel’s\(^{22}\) four cases (cases 26–29) there is insufficient clinical information on which to base any opinion. In summary, out of the 29 cases, 14 fall within the general clinical pattern described, five conform so far as their clinical detail goes, five showed sudden death alone, and five lack adequate history for appraisal. No striking exceptions occur.
Because these cases have nearly always complicated pulmonary embolism, death has been ascribed to "further pulmonary embolism" without direct proof. From the foregoing discussion, it would seem obvious that death was, in fact, due to the intracardiac clot, as has been tentatively suggested. That such a clot is indeed a terminating event is supported by several additional observations. Barnard's reported circumstances make it likely that the thrombus passed into the foramen ovale "as the heart was making its last beats," and French noted "it is evident there was no time for the embolus to be broken and thrown into fragments into the arterial circulation." In Young's case, the aorta also "contained an embolus which extended from the heart itself throughout the aortic arch." There are 21 detailed descriptions of the impacted embolus of the foramen ovale since 1925. The thrombi average 9.7 cm. in length, ranging up to 27.5 cm. but in no case is there evidence of fracture of the end in the left atrium. This feature is present even in those cases associated with peripheral arterial emboli. It is unlikely that brittle antemortem thrombi of such length could endure long in the atria without fracture. Of those dependent through heart valves, only a few pressure grooves are mentioned. It is noteworthy that 51.7 per cent (15 of 29 cases) showed no paradoxic emboli outside the heart itself. Thus, it seems clear that the heart stops within a few beats of the foraminal occlusion.

It is advantageous at this point to compare relevant features of uncomplicated pulmonary embolism. Using graded pulmonary arterial occlusions in cats, Haggart and Walker found no corresponding graded severity of symptoms. Although pulmonary arterial pressure rose, cardiac output was well maintained up to 60 per cent occlusion. Beyond this, a dramatic fall in cardiac output took place, with lowered systemic blood pressure and arterial oxygen saturation. In a clinical study, Thompson and Evans showed that depletion of the pulmonary circulation by more than a third was necessary to reverse atrial pressures, and that a sudden depletion of 50 per cent or more resulted in death, perhaps sudden, but within 30 minutes. In case 1 (case 9) although at least 50 per cent of the pulmonary arterial tree was occluded by adherent thrombi, death did not occur for over 50 hours. In table 1, the period of apparent recovery or palliation from pulmonary embolism ranges from one hour to four days. Evoy points out that in 246 fatal cases of pulmonary embolism, 92 per cent were dead within 24 hours, and 69 per cent were dead within one hour. Thus it is apparent that precarious survival for long periods is not a feature of uncomplicated pulmonary embolism. The essential difference between this and pulmonary embolism with patent foramen ovale is the decompression provided by the interatrial shunt. That the shunt does function to decompress is supported by electrocardiographic evidence available in four cases (cases 8, 9, 11, 13). In pulmonary embolism, the characteristic electrocardiographic picture is dependent on the production of an acute cor pulmonale. In the four cases associated with a patent foramen ovale, these changes have not been observed presumably because the right atrium was decompressed into the left until the terminal foraminal occlusion.

The influence of other factors beside relative atrial pressure may be considered. "Pencil patency" (0.7 cm. diameter) of the foramen ovale persists in 1 per cent of adults of the average age at which confirmed paradoxic embolism occurs (51.5 years). However, reports of embolic occlusion of the foramen ovale are excessively rare. It seems to us that this infrequency depends on the relative sizes of the shunt and of the embolus rather than on the "element of chance." The average diameter of the foramen ovale in cases reported after 1925 is 0.68 cm. and in 80 per cent the source of the emboli was in veins of pelvis or leg where the caliber of thrombi is likely to approximate that of the foramen.

Johnson believed that "the magnitude of the stream through the foramen ovale might well be as great as through the pulmonary artery." That it may be even greater is suggested by the situations of the impacted foraminal emboli reported since 1925. In only two instances did one pass through the tricuspid valve (cases 27, 28), while in four instances it passed through the mitral valve.
(cases 1, 2, 16, 21), and in three others long emboli remained coiled up in the right atrium (cases 4, 20). In other words, the main flow was probably through the shunt.

We know of no case of comparable embolic occlusion of a patent interventricular septal defect. For reversal of interventricular flow to occur, a degree of pulmonary obstruction sufficient to cause immediate death would seem necessary. Thus, although the association of cerebral abscess with patent interventricular septum has been recorded at intervals since 1814, 31, 32 confirmation of its relationship as an embolic syndrome is lacking. While embolic occlusion of a patent foramen ovale may be rare, the lifesaving effect of the interatrial shunt may be commoner. It is conceivable that some patients who recover from massive pulmonary embolism do so because of this unrecognized mechanism.

**SUMMARY**

1. Two instances of a thrombus having been caught passing through a patent foramen ovale are described.

2. This is defined as “embolic occlusion of patent foramen ovale,” reserving the term paradoxical embolism to describe peripheral arterial embolic occlusions by venous thrombi.

3. It would appear that death in such cases is precipitated by acute cor pulmonale as a consequence of sudden obstruction of a decompression shunt between right and left atria and not to further pulmonary embolism as has been supposed.

4. The concomitant clinical syndrome is described. Of the 24 cases reported since 1925 on which there is sufficient data available, all conform to this pattern. The apparent exceptions are discussed.

5. The full clinical sequence consists of pulmonary embolism followed by a variable period of improvement (the “palliative shunt” phase), and finally sudden death with conspicuous cyanosis, when the shunt is occluded. In its most recognizable form, the shunt phase is characterized by pallor, cyanosis unrelied by oxygen, hypotension, profuse perspiration, moderate venous distention, and absence of electrocardiographic changes seen in acute cor pulmonale. During this phase, peripheral paradoxical embolism may occur.

6. In one of our cases, the foraminal occlusion was preceded by paradoxical air embolism. This is apparently the first instance of proven paradoxical air embolism, where the presence of a right-to-left interatrial shunt during life has been confirmed.

**ACKNOWLEDGMENT**

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**SUMARIO ESPAÑOL**

En la presencia de un foramen ovale patente, embolismo pulmonar puede producir un “shunt” inter-auricular de derecha a izquierda que alivia los efectos del embolismo. Cuando el “shunt” es ocluído por un embolo subsiguiente, muerte súbita ocurre. Los aspectos clínicos y patológicos del síndrome se describen.

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Embolism

Embolic Occlusion of Patent Foramen Ovale: A Syndrome Occurring in Pulmonary Embolism

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