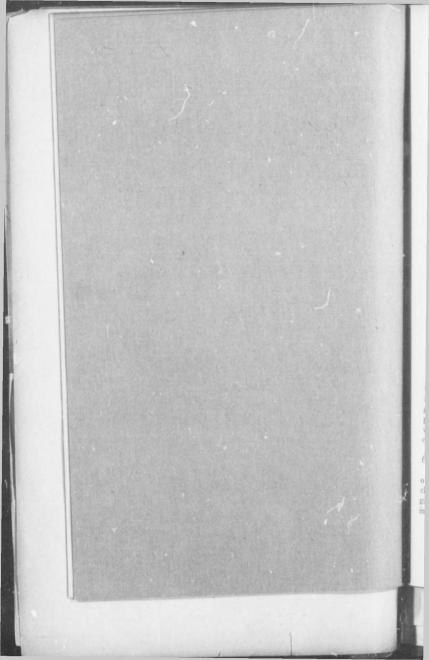


# Two Cases of Widely Patent Foramen Ovale

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## TWO CASES OF WIDELY PATENT FORAMEN OVALE.

### By MAUDE E. ABBOTT, M.D., McGill University, Montreal.

A slitlike or valvular patency of the foramen ovale, sometimes large enough to admit a lead pencil, is recognized by all those with autopsy experience as a very common phenomenon, occurring according to combined statistics in some 30 per cent of all cases. In most of these the opening does not gape, but is closed by the pressure of the overlapping edges of the valvula foraminis ovalis in the left auricle and the annulus ovalis in the right. Such cases cannot be regarded as an anomaly, both because of their frequency, and because, under ordinary conditions, they present no disturbances in the circulation. A widely gaping foramen, in which an opening remains permanently between the free borders of the annulus ovalis and the valvula foraminis ovalis, which cannot be approximated, permitting the free passage of blood from one auricle to the other, without the interposition of any valvular fold, is not usual in cases uncomplicated with other anomalies and must be ranked among cardiac defects, as presenting a permanent interauricular communication.

Like other defects of the interauricular septum the majority of these cases are characterized by an entire absence of cyanosis except as a terminal phenomenon, (cyanose tardize of the French writers) and by the presence of the characteristic physical signs in the form of precordial bulging and a rough presystolic or systolic murmur and thrill with maximum intensity in the neighbourhood of the 3rd left interspace. Marked pallor and a general gracility of form is not uncommon, for the reason that such wide patency is frequently associated with hypoplasia of the aorta, whether as a primary or secondary condition is not altogether clear.

This combination presents a definite symptom-complex which frequently enables a correct clinical diagnosis to be made.

The two cases presented here of uncomplicated patency are of interest, first because they presented during life this definite clinical picture to be ascribed to a patent foramen of the widely gaping type, (which enabled the diagnosis to be made antemortem in the first case) and secondly, because they form an interesting comparative study of the effects upon the hearts of such cases of an associated mitral stenosis, which was present in the second of these cases, but not in the first, and which led to the most extensive degree of back pressure in the right auricle, evidenced by extreme dilatation of its wall, aneurismal dilatation of the coronary sinus and its tributaries, and an indirect communication of the latter with the cavity of the left auricle through an opening supplied with a subsidiary valve. This second case was further remarkable in that there was associated with it an anomalous fenestration of the annulus ovalis and of the Thebesian valves, probably due to a faulty involution of the embryonic valvula venosa sinistra, to which also persistent patency of the foramen ovale has been ascribed by several authorities. Both cases showed also a coarctation of the aorta.

My thanks are due to Dr. C. F. Martin, in whose public service these patients were, for the privilege of observing these two cases during life, and for permission to report them here.

#### CASE I.

Large Patent Foramen Ovale with Cribriform Fossa Ovalis, Hypoplasia of the Aorta, and Stenosis at the Isthmus. Simple Hypertrophy of the Left Ventricle. Hypertrophy and Dilutation of both Auricles. Hypoplasia of external genitals. Diagnosis made before death on the ground of pallor, absence of cyanosis, and presence of characteristic physical signs of Auricular Septal Defect, and of Hypoplasia of the Aorta.

Clinical History: L. L., female Russian Jewess, aged 26. Admitted to R. V. H., March 16th, 1014, complaining of trouble in vision, frequent headaches, sweiling of the legs and face, shortness of breath on exertion, morning vomiting, irregular menstruation, palpitation and pain over the heart. These symptoms had persisted one year. She was unmarried, and had been in Canada for two years, gave no history of any previous illness, scarlet fever or rheumatism. Menstruation had commenced at thirteen and was regular every five to six weeks; amenorrhoea had been present nine months. The family history was negative.

On admission showed no signs of cardiac embarrassment, no dyspneea, cyanosis nor clubbing. The pulse was 114, regular and small, and the blood pressure rather high, 170.

*Examination of the heart and great vessels.* Inspection showed some precordial bulging, and marked throbbing involving most of the front of the chest, the neck veins very much congest-

ed. but no positive venous pulse, epigastric pulsation, and the apex beat in the fifth left interspace in the anterior axillary line, the veins over the front of the chest slightly dilated. On palpation a marked systolic thrill was felt at the base of the left sternal line and to the left of this down nearly to the fourth left rib or third left interspace. There was no diastolic shock, but tracheal ungging was present. The cardiac dulness began above at the second rib and extended laterally from 4 cm. to the right and 11.5 cm. to the left of the median line at the fourth rib and 13.5 cm. to the left at the fifth rib. On auscultation a loud harsh systolic murmur having its maximum intensity at the third left cartilage 4.5 to 5 cm. from the mid-sternum, was heard all over the front of the chest and in the left back. At the apex there was a suggestion of a presystolic murmur and a systolic blow was audible at the pulmonary cartilage.

 $Urine\ {\rm showed}\ {\rm a}\ {\rm sp.g.}$  of 1010, alkaline reaction, a few hyaline and granular casts.

On April 15th, 1914, a pericardial friction rub developed, also ptosis of right upper lid and definite external squint. Examination of the blood haemoglobin 45 percent, red blood corpuscles, 3,590,000. Died suddenly on April 16th, 1914.

Autopsy showed the conditions enumerated above, also an acute fibrinous pericarditis and pleurisy, cloudy swelling of the liver, fibrotic cardiac spleen, atheroma of the aorta, anasarca, chronic interstitial nephritis, both kidneys very small, weighing together 55 gms. (? congenital bypoplasia). The brain was not examined.

Examination of the heart showed a moderately enlarged organ covered by an extensive exudate of recent fibrin. Both auricles were dilated to about double their capacity and their musculature especially that of the right was considerably increased. The auricular septum presented a gaping hole admitting the thumb which was bounded above and anteriorly by the thick annulus ovalis, and below and behind by the thin crescentic margin of the valvula foraminis ovalis, which was considerably enlarged and bulged into the right auricle and presented numerous fenestrations, as well as a hole admitting a bone knitting needle at its upper border. The Eustachian and Thebesian valves were normal as well as the auricular walls elsewhere and the tricuspid and mitral orifices. The right ventricle was about normal size being the only chamber of the heart unaltered. The pulmonary orifice was moderately dilated, measuring 6 cm. as also the pulmonary artery which was thin walled and otherwise normal. The oblit-

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erated ductus was plainly seen as a small ligament about .25 cm. long and extending from the base of a depression at the bifurcation of the aorta just beyond the subclavian where its insertion was marked by deep puckering and by a patch of a theroma. The left ventricle was markedly hypertrophied, its wall measuring 18 cm, in thickness, but was only slightly dilated, a simply hypertrophy existing. The aortic orifice was very narrow measuring 4.5 cm, in circumference. The aorta itself widened slightly at its origin, but was abruptly narrowed at the point of insertion to the left subclavian artery to a circumference of 3 cm, widening shortly below this to 4 cm. Several patches of atheroma were scattered over the arch and thoracic aorta.

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#### CASE II.

Large Patent Foramen Ovale with Calcified lower Border, Fenestrated Annulus Ovalis and Anomalous Septum in right Auricle, Anomalous Pocket on Wall of Left Auricle, Aneurismal Dilatation<sup>1</sup> of Coronary Sinus and of Right Auricle, Dilatation of the Pulmonary Artery, Slight Hypoplasia and Coaretation of the Aorta.

Clinical History. Mrs. B., aged 38. Admitted to R. V. H. in August, 1912, in an advanced state of failing compensation, marked ascites, anasarca and orthopnoca. Menstruation set in at the age of 21 and disappeared at 28. Had one child who died early. Acute rheumatism at the ge of 14, and since then had symptoms of myocarditis with exacerbation in 100 and 1903, asthma and chronic bronchitis; in 1094 right hemplegia with aphasia and a slight degree of recovery. Failing compensation with severe dyspnoca in 1906, right sided pneumonia in 1908, dysent in 1909, pneumonia and jaundice in 1911.

Condition on admission. A pale, emaciated woman, with marked ascites and generalized oedema, dyspnoea and orthopnoea and precordial pain, the right arm and leg paretic, of good intelligence, nervous system normal, the liver much enlarged, a trace of albumin in the urine but no casts.

Examination of the heart showed great irregularity (auricular fibrillation), marked venous pulsation (positive venous pulse) in the neck, very diffuse impulse, increased pear-shaped cardiac dulness, a presystolic murmur at the mitral area with accentuated second sound. Examination by Dr. Martin on two occasions showed a faint thrill and presystolic murmur localized towards the middle of the precordium in the fourth left interspace. The symptoms of failing compensation increased gradually, bronchitis with marked oedema set in and *terminal cyanosis*. Died November 13th, 1912.

Autopsy showed the condition of the heart detailed above, also fibroid myocarditis, general anasarea, hydrothorax, ascites, emphysema, osteo-myelitis, passive congestion of organs, anaemia.

Examination of the heart showed an enormously large organ, the increase in size being due to alterations in both auricles and in the right ventricle. The auricular septum presented a huge gaping foramen ovale, incapable of closure, 2 cm. long by 1.5 cm, wide. The valvula foraminis ovalis was greatly thinned and increased in size measuring 6 cm. in diameter, bulging into the left auricle and presenting a crescentic free border, in part calcified, which formed the lower border of the permanently patent foramen. This was bounded above on the side of the right auricle by the flattened and much fenestrated annulus ovalis, a direct continuation of which extends to the wall of the right auricle where it was inserted just above the Eustachian valve, cutting off a shallow triangular sulcus, into the depths of which several channels open from the surrounding musculature. The Eustachian valve is large, but is not fenestrated. It is separated from the base of the tricuspid valve by a large bulging area caused by localized dilatation and hypertrophy of the musculature so marked as to form practically an accessory chamber in the depths of which lay the fenestrated Thebesian valve, guarding the orifice of the greatly dilated coronary sinus, which presented a varicose aneurism in its course. chamber was evidently a compensatory development to accommodate some unusual condition of the circulation. vestigation of its floor showed several openings admitting a large probe, one of which passed for a long distance beneath the endocardium of the left auricle. Examination of the left auricle showed its endocardium greatly thickened and that covering its anterior wall the seat of a curious formation in the form of a narrow thick walled pocket 2 cm. deep, with a crescentic upper free border attached by slender cords to the wall of the auricle. The depths of this pocket lay in close contiguity to the muscular channel running from the accessory chamber in the right auricle, and suggested that a communication existed here between the two auricles though none was definitely made out.

The right auricle was enormously dilated and very thin walled, the tricuspid orifice was dilated, and the right ventricle was greatly hypertrophied and dilated, especially in its conus: the pul-

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 $-6\,-\!\!\!\!$  monary orifice and artery were greatly dilated measuring 9 cm. in circumference.

The left auricle was dilated and its wall also distinctly hypertrophied, the mitral valve was markedly thickened and contracted, an extreme button-hole stenosis existing. The left ventricle was practically unchanged. The aorta was smaller than the pulmonary artery, measuring 7 cm. at its orifice, and only 4 cm. at the orifice of the left subclavian (slight coarctation). A short ligamentum arteriosum connected the aorta at this point with the pulmonary artery.

