

CARDIAC AND GENITO-URINARY ANOMALIES IN THE SAME SUBJECT

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F. S., male, 38 years of age, was admitted into the Great Northern Central Hospital on March 6, 1919. The previous history obtainable was of "heart-trouble" for three years and "pain in the head" for four weeks. Two nights previous to admission, the headache was severe and on awaking from sleep, ptosis of the left upper eyelid was observed. There was also a history of a severe cold with cough and some haemoptosis in 1916.

On March 7, the mental condition is noted as having been "strange" and the patient is stated to have become cyanosed at times. On the 8th he is described as "deranged," restless and complaining of headache. The heart examined at this date is stated to have shown a loud, "flapping" first sound and a systolic bruit not conducted outwards to the left. The pulse rate is stated at the same time to have been "slow," regular and feeble. There was complete oculo-motor paralysis of the left eye, but no motor or sensory disturbance was notable elsewhere. There was some stiffness of the left leg and an exaggerated knee-jerk in the same limb. The urine was free from albumin and sugar.

On March 10th signs of intracranial pressure were marked and the visiting physician examined him on the afternoon of that day. The patient was found to be delirious and incapable of answering any questions. The oculo-motor signs described were well-marked. On examining the heart, one was struck by the peculiarity of the auscultatory signs. The apical systolic bruit was not audible in the left paravertebral groove and so marked was the accentuation of the first sound, that it could be described by no other term than as a *loud smack*. The condition of the patient precluded more detailed examination and he died the same day.

On *post mortem* examination, the deceased was found to have tuberculous meningitis, and the lungs showed several small cavities at the pulmonary apices and scattered tuberculous foci in the lower lobes. The heart and genito-urinary organs showed abnormalities to be described more fully. The following description of the heart is given by Dr Alexander Blackhall-Morison:

The *right ventricle* is dilated and hypertrophied, the thickest portion of the wall having a diameter of 5 cm. The chamber measures 9 × 7 cm. The columnae carnae are decidedly hypertrophied and especially so is the mass running downwards and outwards from the base of the posterior cusp of the

pulmonary arterial valve (Pl. XVI, fig. 1). These cusps are themselves normal. The transverse measurement of the pulmonary artery is 6 cm. The endocardium below the pulmonary valves is opaque. The tricuspid orifice is dilated, easily admitting three fingers and measuring 5 cm. transversely and the same antero-posteriorly. The internal or septal cusp is fleshy, sessile and of no valvular value. The *anterior cusp* measures 4×5 cm. and is attached in its normal position. The *posterior cusp* at its right boundary coalesces with the right limit of the anterior cusp, but is attached abnormally low in the ventricle and its left portion is divided into two pieces. A large, tough, umbrella-like flap is attached to the ventricle close to its apex and a second smaller portion is, like the internal cusp, fleshy, sessile and of no valvular value. The *right auricle* measures 8×6 cm. and is hypertrophied. This chamber is also dilated. The *foramen ovale* and venous entrances are normal and the *coronary sinus* is provided with a well-developed Thebesian valve.

The *left auricle* is normal, as usual opaque in lining and smooth in surface.

The *left ventricle* measures 7×6 cm. and its wall at its thickest 2 cm. The mitral and aortic cusps are normal. The *mitral* orifice measures $7\frac{1}{2}$ cm. transversely and the *aortic* orifice 5 cm. There are two coronary arteries and the base of the aorta is slightly atheromatous.

The anatomical point of chief interest in the cardiac conditions described, as bearing upon clinical diagnoses, has reference to the abnormal tricuspid segments. The very strikingly exaggerated loudness or accentuation of the first sound of the heart was manifestly due to the impact of blood in systole upon the redundant tricuspid segments. The effect reminded one of the sudden slap of a loose sail rendered taut by a gust of wind. The great audibility of the sign may also in a less degree have been due to the superficial position of the ventricle affected, for, dextral signs caused by organic valvular disease are for this anatomical reason more pronounced than those arising in the deeper left ventricle. An indirect anatomico-physiological interest likewise attaches to this case as elucidating the chief cause of the accentuated first sound in mitral stenoses. Of this a variety of explanations has been offered, but this case strongly supports the view that that diagnostic sign is chiefly attributable to the impact of ventricular blood in systole on the more or less fixed aortic segment of the mitral valve. Dr Ernest Henry Shaw gives the following description of the genito-urinary anomalies in the case:

Congenital absence of one kidney with abnormal development of ureter of same side (fig. 2).

The specimen consists of the bladder with the vesiculae seminales and portions of the vasa deferentia, and the malformed ureter of the left side. The bladder is normal in size and its wall is natural in thickness. Internally a rounded prominence is seen to the left of the trigone about $\frac{3}{4}$ in. in diameter. It is formed by a thin layer of tissue which is easily depressed by the finger into a large sac in and behind the bladder wall. No ureteral opening is visible on the left side. A ridge of firm muscular tissue runs down from the promi-

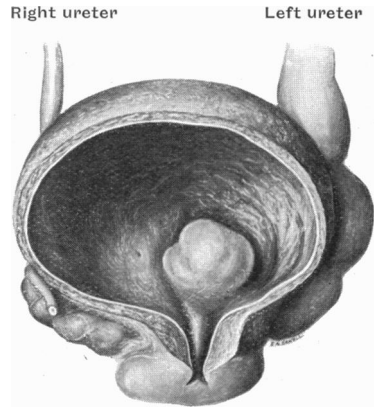
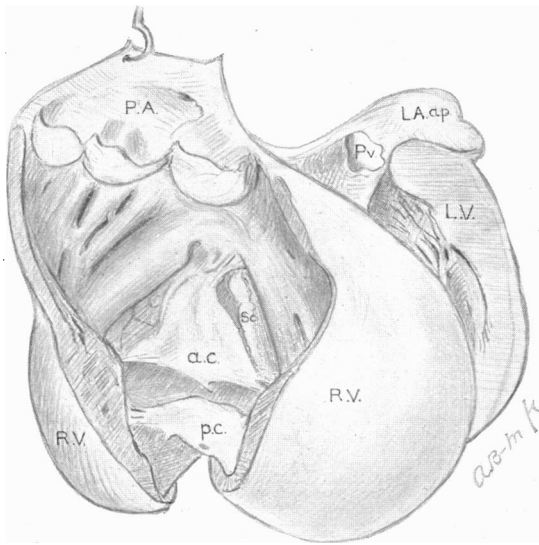


Fig. 2.

Fig. 1. R.V. right ventricle. P.A. pulmonary artery. S.c. septal cusp of tricuspid valve; a.c. anterior cusp and p.c. posterior cusp of the same. L.V. left ventricle. P.v. pulmonary vein. L.A.ap. left auricular appendix.

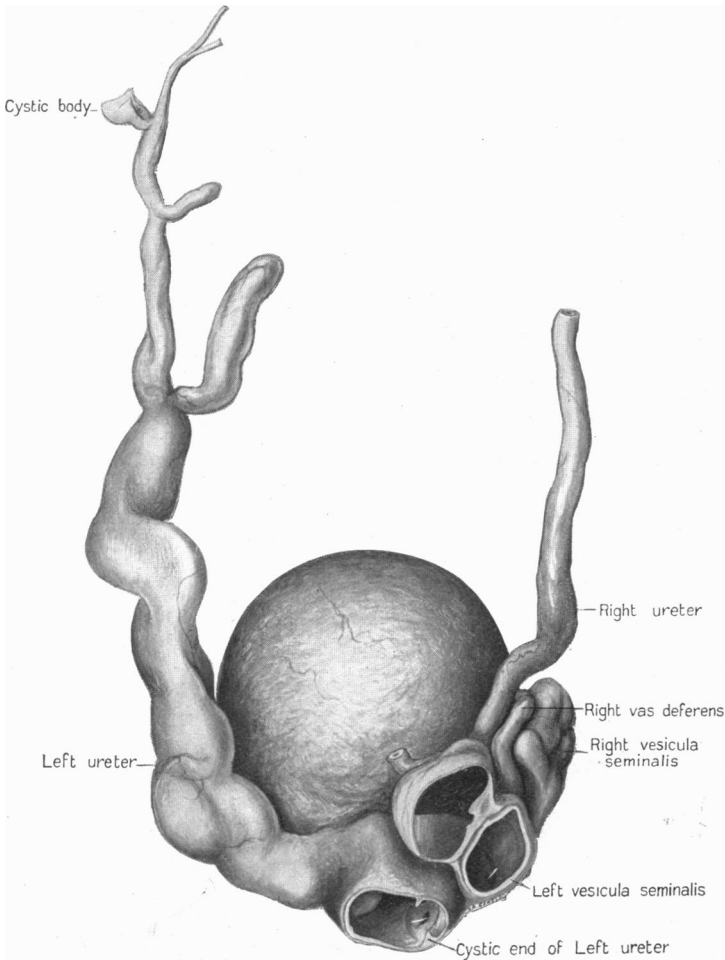


Fig. 3.

nence and becomes continuous with the verumontanum. The orifice of the right ureter is seen in its usual position and is natural in size.

On the posterior aspect a large sacculated cavity is seen in the left wall of the bladder and from this the ureter emerges. The left seminal vesicle is large and dilated to form a bilobed cyst, at the bottom of the cyst a rod is passed through a small hole into the large cyst at the lower end of the ureter. The left vas is much dilated below and then suddenly contracts to a narrow hollow rod as it enters the base of the prostate.

The left ureter begins below as a large thin walled sacculated tube which runs upwards in a convoluted manner and gradually becomes narrower. Towards the upper end it gives off a narrow branch about two inches long and further up a second branch about one inch long. Both branches are hollow and end blindly. The ureter ends above in a small cystic mass and from the upper part of this two strands of tissue taper away.

The vesiculae seminales and vas deferens on the right side are natural. The ureter is a little enlarged.

The left ureter was filled with turbid yellowish fluid which contained many cells of various shape and size, granular material, and a large number of spermatozoa. The latter were mostly small and ill-formed, but many were quite normal in size and shape. On injecting formalin solution into the dilated ureter it first passed into the left seminal vesicle which became distended, it then issued from the cut end of the vas deferens. A channel of communication between the ureter and vas was thus clearly proved. The hole between the ureter and seminal vesicle suggests that the channel is linked up by this organ. There is no communication between the left ureter and the bladder cavity.

The small cystic mass at the upper end of the ureter may represent a rudimentary kidney. Microscopically the cysts and some small tubes are lined with columnar epithelium.

The right kidney weighed 15 oz. and appeared to be normal.

The specimen appears to be valuable from a developmental point of view. It supports the theory of the origin of the ureter being formed by an offshoot from the Wolffian duct (vas deferens). The lateral branches of the upper part suggest the formation of the calices found in the normal kidney. The dilatation of the ureter is due to pressure by the accumulation of seminal fluid, and the tube may be said to form a huge seminal vesicle. The formalin solution did not escape from the ejaculatory duct and it is not possible to tell from the present state of the dissection whether this tube is present or if it is patent.