With the exception that the kidney here presents a more clearly lobulated appearance, and that there is no arterial abnormality, the case is almost identical with the preceding. The reduplication occurs only on the left side, there are two pelves, the upper being the smaller, the superior ureter crosses in front of the inferior, and its separate orifice is also along the edge of the Trigone, in front, and to the inner side of the main orifice, between that and the urethra.

It is a curious fact that in nearly all the recorded cases of this peculiarity it has occurred in the *left* side. The two cases just mentioned are on the left side; Tangl's[1] celebrated case, and Gangolphe's[2] likewise occurred on this side. Baum[3] has lately published a case in which it occurred on the right side. There may be no special significance to be attached to this *left*-sided tendency, but still it appears to obtain.

Dr. SHEPHERD had met with a great many cases of abnormal blood supply of the kidney, and partial double ureter, but the only other case that he had seen of complete reduplicat on was the one taken by Dr. Adami from the museum to compare with the case reported.

*Mitral and Tricuspid Stenosis.*—Dr. FINLEY exhibited a heart in which both mitral and tricuspid stenosis was well marked. The orifice of the mitral valve admitted the tip of the little finger, that of the tricuspid the first finger. The changes in the left ventricle were not marked; if anything, its cavity was somewhat smaller and its walls thin; the right ventricle, while its walls were slightly thickened and its cavity dilated, did not present that extreme degree of enlargement commonly found in mitral stenosis; the right auricle was the largest of all the cardiac cavities. The lungs presented numerous reddish patches, which on microscopical examination proved to be hæmorrhagic infarcts.

The following is the history of the case:—The patient, a female, æt. 33, was admitted to the Montreal General Hospital in November, 1893, for pain in the side and cough. She had suffered from repeated attacks of sore throat, sometimes going on to suppuration, but had never had rheumatism or chorea. Dyspnœa on exertion had been present for three months before her admission. She had never had hæmoptysis. The family history presented no feature of importance, and there were no rheumatic tendencies. The present illness began four days previous to admission, with a slight chill, cough and pain in the right side.

*Physical Examination.*—Moderate emaciation, slight cyanosis of lips and cheeks, with stellate venules on face. Temperature subnormal. Cardiac impulse forcible and somewhat heaving over lower sternal region. Apex in fifth space ¼ inch inside nipple. Marked presystolic thrill at the apex. Cardiac dullness normal. A harsh, rumbling presystolic murmur is heard, but to inner side of the apex, and localized over a space two inches in diameter. The first sound is abrupt, greatly accentuated and snapping in character. A soft systolic murmur is heard between the lower sternal area and the nipple. The pulmonary second sound is enormously accentuated and reduplicated. Below the angle of the scapula on the right side, dullness, feeble breathing, with diminished vocal resonance and fremitus. A small quantity of clear serum was drawn off a few days later with a hypodermic syringe. The first sound at the tricuspid area is feeble. The pulse 102, small, regular and of low tension. The other organs are normal, and the urine reddish yellow in color, s.g. 1025, no albumen or casts. Ordered digitalis m.x. ter in die.

Nov. 14.—Fluid in pleura reaches fourth rib in front. Temperature 99 to 100 in the morning and about 100 at night, became normal at this date. Digitalis dropped on account of vomiting.

Feby. 15.—The presystolic murmur and thrill disappeared, and ten days later pulse became extremely weak, paroxysmal attacks of intense dyspnœa and cyanosis came on, death resulting apparently from cardiac failure. The urine averaged 20 to 30 ozs. daily whilst under observation. There was at no time any œdema of the extremities or serous sacs.

The physical signs left no doubt that the mitral valve was narrowed, but there was, during life, no evidence made out indicating disease of the tricuspid. On looking back, however, on the case, he was inclined to think that the systolic murmur heard in the lower sternal area was possibly a tricuspid sound.

It was impossible to find the onset of the disease—there was no history of rheumatism; but judging from the condition of the cardiac orifices, it must have been of a good many years' standing, and the case furnishes another instance of the extreme degree to which cardiac disease may advance and yet compensation is maintained. A point of interest in connection with the first sound of the heart in mitral stenosis is the cause of its peculiar snapping character. It is, perhaps, difficult to give any satisfactory explanation. The point has been much debated, and many authors think that the thickened condition of the valve, in itself, would preclude the possibility of its emitting such a sound. Recently a paper has been published by Fenwick and Overend in *Am. Jour. Med. Sc.*, 1893, stating that the peculiarity of the first sound of the heart occurring in mitral stenosis is really due to the closure of

1 Virchow's *Archiv.* 118 (1889) p. 414.
2 Loc. cit.
3 Archiv. of Gynœkol. 42, p. 339 (1892).