Disease of the valves may be the result either of acute endocarditis, usually rheumatic, or of chronic inflammatory or degenerative change.

The kind and degree of deformity may vary greatly, and we have to endeavor to see our way to the prognosis under these diverse conditions. In some cases, the disease will prove fatal in a few months or years; in others, it may exercise little influence on the health or life. In two instances mentioned, mitral regurgitation had been ascertained to exist twenty years since in patients still living and well; and in another case, repeatedly examined within the last three years, the patient had been condemned to life-long inactivity after acute rheumatism, thirty-five years ago, but was still, at the age of sixty-four or sixty-five, doing strenuous work, a mitral murmur having been known to be present all this time. It is very common to meet with mitral regurgitation at and after the age of seventy, but its duration cannot be ascertained. This form of disease is very frequent among the out-patients of hospitals, and sends into the wards numerous cases of dropsy which recover. It is, in effect, not a deadly kind of valvular affection, and is probably less serious than aortic stenosis, which is placed lowest in the scale of danger. It is not simply that, in a large proportion of cases, the valvular change is comparatively slight; even considerable disease may be long survived, if not progressive.

The first inquiry will be as to the signs and symptoms by means of which the amount of regurgitation may be estimated.

In some cases the character of the murmur affords much information. When it is not conducted much beyond the apex, and is not heard in the back, especially when the first sound of the left ventricle is not lost, but is still audible at the apex, and particularly when the murmur does not begin with the first sound, but follows it at a brief interval, *i.e.*, post-systolic, the leakage is usually inconsiderable. Care must, of course, be taken not to take the soft short murmur of a gaping orifice and a weak heart as an indication of slight mischief, and not to mistake the short sharp first sound of mitral stenosis for a normal left ventricular first sound. Mitral systolic murmurs are often musical, and a musical note would seem to imply a narrow chink and small regurgitant stream; but observation alone must determine the significance of any particular kind of murmur.

For further evidence, the effects of regurgitation through the mitral orifice must be followed. The first of these will be distension, and then dilatation of the left auricle; but, from the deep-lying situation of this chamber, an early stage of enlargement is not easily made out. But the obstruction to the entry of blood into the auricle from the pulmonary veins will give rise to increased pressure in the pulmonary artery, audible evidence of which will be accentuation of the pulmonary second sound. It is not easy, however, to define the degree of intensification of this sound, which must be accepted as proof of obstruction at the left side of the heart; especially as obstruction may arise in the pulmonary capillaries. From the increase of pressure in the pulmonary artery, another effect follows, hypertrophy and dilatation of the right ventricle, manifested by displacement of the apex to the left, and undue impulse of the right ventricle. But the effects do not end here; the pressure maintained in the left auricle must be such as to afford resistance to the return of blood from the ventricle; and must, therefore, in extreme cases, almost equal the pressure in the aorta. Such pressure must, during the ventricular diastole, drive the blood violently into this cavity, and act as a dilating force, and dilatation will involve hypertrophy. The situation, then, of the apex-beat will be the resultant of hypertrophy and dilatation of two ventricles, and the volume of the heart, as a whole, will be greatly increased.

These changes, the result of mitral incompetence, becomes for us its measure. A systolic mitral murmur, without any of the signs enumerated, and without symptoms, is not attended by much regurgitation, and is not a source of present danger; unless it marks the beginning of progressive mischief, it may be disregarded. As the indications of pressure in the pulmonary artery and of changes in the heart increase, we infer increase in the amount of reflux, and look for diminished stability of the compensation, and expect less power of regaining a working equilibrium if it is once overthrown. These statements are of course subject to the qualifications enumerated in the first lecture.

At the risk of being tedious, one more qualification must be added. It has been pointed out how the right ventricle comes to the aid of the left by maintaining a degree of pressure in the left auricle which resists the reflux through a gaping mitral orifice. The resistance must, when the barrier of the valve is withdrawn, be greater than the pressure in the arterial system. Everything depends, therefore, upon the resistance in the capillaries and the arterioles, and this varies greatly in different conditions. If this be considerable, the arterial tension is high, the backward pressure on the auricle and pulmonary veins is great, and the demand upon the right heart is heavy—all circumstances tending to the production of dilatation and hypertrophy. If, on the other hand, the arterial tension be low, the conditions are reversed. In the case mentioned of mitral regurgitation known to exist for thirty-five years, the pulse was extremely soft and short; and as there were at one time disquieting symptoms—a sense of constriction of the chest on slight exertion, and a liability to slight fainting attacks—it was feared that the heart was failing. The family pulse, however, is of extremely low tension, and to this the patient owes, in part, at least, his immunity.

The pulse of mitral regurgitation has still to be considered. Its characterisic is irregularity both in rhythm and force, which in advanced cases is