In operating upon tumours in the bladder, I have frequently noticed a peculiar condition of the bladder wall, and which seems worthy of mention. After working in the bladder for half or three-quarters of an hour the mucous membrane becomes thick and rather oedematous in appearance, and the bladder muscle contracts and thickens, so that the exposure of the interior of the viscus becomes more difficult. These changes are probably due to the prolonged exposure to air, but they are followed by no ill-effects.

Having carefully freed the bladder from blood-clot, and removed gauze pads or swabs from the post-trigonal area, the question of closing or draining the bladder will arise. After a trial of suturing the cystotomy wound in these cases, and after one or two failures from serious hæmorrhage, I have abandoned the method. If the bladder is sutured and the urine, by any chance, such as the blocking of the catheter by a clot or the slipping of the tied-in catheter, accumulates and distends it, hæmorrhage is encouraged, and should hæmorrhage take place a retained catheter, however large, is not a sufficient means of drainage. I now drain the bladder in these cases from the first by means of a large rubber tube, three-quarters of an inch in diameter. A stitch or two are usually required to close the bladder wound around this tube. In addition I place a small rubber tube, which dips into the prevesical space, at the lower angle of the wound. This ensures the removal of any urine which may escape into this space. The abdominal wound is carefully repaired by uniting the cut portions of recti and approximating the recti muscles by means of catgut sutures.

In the after-treatment I use a very simple method of removing the urine which is neither a syphon nor a suction method, but merely drains the overflow. One end of a yard of medium-sized soft rubber Paul's colotomy tubing is drawn over the prominent end of the rubber drainage-tube and tied round with a piece of silk to retain it. This is brought through the dressings and passing over the edge of the bed dips into a jar. To ensure that the surfaces do not adhere and block the flow of urine two drachms of olive oil are poured through the tube before adjusting it. The tube is changed at each daily dressing, when the bladder is irrigated with oxycyanide of mercury (1 in 10,000). On the fourth day the drainage-tubes are both removed and the urine may be allowed to soak into large pads of cellulose wadding, or an Irving apparatus may be applied. In my experience the wound heals more quickly without any apparatus.

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ON CHRONIC BRONCHITIS AND EMPHYSEMA AS THE RESULT OF ACUTE PNEUMONIA.

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It is not surprising that so severe a disease as pneumonia should be followed by serious nutritive changes in the lungs. Of the local changes in the consolidated parts it is not necessary to speak. I wish to draw attention to less recognised results which affect the lungs generally. The two affections I refer to are bronchitis, on the one hand, and emphysema on the other.

The signs of bronchitis associated with pneumonia in its acute stage have a different significance according as they have preceded the attack of pneumonia or developed in the course of it. Where the pneumonia is subsequent—i.e., occurs in the course of bronchitis—the association of the two affections is awkward but not necessarily of very grave significance. Where, on the other hand, the physical signs of general bronchitis develop in the course of the pneumonia they indicate not a mere bronchitis or complication of the pneumonia but pulmonary failure, a very serious condition and almost necessarily fatal.

In a severe case of pneumonia the pathological changes are not limited to the parts which become consolidated but are widespread and affect the whole of both lungs. This is recognised pathologically, but clinically is usually disregarded unless during the acute stage the signs of congestion develop

in the rest of the lungs. After the pneumonia is past these general nutritive changes leave behind a delicacy of the lungs which may last for months and show itself in a tendency to bronchitis on slight provocation. If this were recognised and dealt with appropriately the patient might be spared much distress and saved the risk of becoming a chronic bronchitic.

Besides bronchitis, there is also emphysema to be considered. Vicarious or complementary emphysema is, of course, common enough during the acute stage of pneumonia, but this is only transient and rapidly passes away. True or ordinary emphysema may be observed, and not rarely if it be looked for, to develop after pneumonia in a patient who before the attack presented no signs of it, so that I cannot doubt that the nutritive disturbances produced during the acute attack may, and not infrequently do, initiate those atrophic changes which end in the wasting and atrophy of the alveolar walls characteristic of ordinary emphysema.

Much might be done to obviate these risks if it were known and remembered how profoundly the general nutrition of the lungs is affected in any severe case of pneumonia, and that the delicacy thus engendered may lead to recurrent bronchitis and to permanent emphysema.

It should therefore be the rule of practice to take great care of a convalescent from pneumonia for at least 12 months. This rule is often disregarded and with disastrous results, for the reason that the resolution of the local lesion is so rapid and complete that the possibility of any widespread changes such as those to which I have referred is not recognised or suspected.

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AORTIC REGURGITATION.¹

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DILATATION and hypertrophy of the heart are carried to a greater extent in aortic regurgitation than in any valvular disease. Heaving pulsation of the lower chest is very evident until the heart is failing seriously. Often there is systolic retraction of the intercostal spaces near the sternum, due to atmospheric pressure and not to adherent pericardium. Also systolic retraction of the lower ribs at the back on the left, and even the right, may be caused by the hypertrophied heart pulling on the diaphragm, which is attached to these ribs. This used to be considered a sign of adherent pericardium, and is so if the heart is not much hypertrophied, but a large heart can pull on the diaphragm sufficiently to cause it without the pericardium being adherent.

At the apex the first sound is prolonged and not as loud as normal. A systolic murmur is often present, which may be conducted from the aortic orifice, or may be that of mitral regurgitation. If the murmur, traced from the aortic cartilage across the chest to the apex, becomes progressively less loud, one may conclude that it is conducted, but if it becomes less loud about half-way and increases towards the apex, it is mitral. Also when it is mitral, the first sound is diminished and may be absent at the apex, and the murmur is conducted outside the apex beat. A second sound is heard and usually a soft diastolic murmur.

Not infrequently a presystolic thrill can be felt and a loud presystolic murmur heard at the apex. Mitral stenosis would be incompatible with life, if there was much aortic regurgitation, blood would never get through a stenosed mitral orifice against the pressure in the left ventricle, and where the pulse is large and collapsing no mitral stenosis is found post mortem. The probable explanation is that the regurgitant stream from the aorta strikes against the anterior flap of the mitral valve and causes vibrations in it, or it may be that the valve is prevented by the aortic regurgitant stream from falling back, and so lies across the mitral orifice to a certain extent. This is sometimes called Flint's murmur, having been first described by Austin Flint in 1862. When there are aortic stenosis and regurgitation, in which the stenosis

¹ Portion of a paper read before the Brighton and Sussex Medico-Chirurgical Society.

predominates, mitral stenosis is not infrequent, but even then a presystolic murmur is not always heard. I remember one case where post mortem there were mitral and tricuspid stenosis as well as aortic stenosis, with some regurgitation, but no presystolic murmur was heard during the two months she was in hospital, but the first sound at the apex was short and loud.

Thayer, at the Johns Hopkins Hospital in 1901, found in 74 cases of aortic regurgitation followed by post-mortem examination 45 had a presystolic murmur during life, but in only 12 was there any mitral stenosis, and in these the pulse was noted as not large, and in most of them there was a short loud snapping first sound at the apex. But of the 29 with no presystolic murmur 4 had mitral stenosis. He illustrates the difficulty of diagnosis by a percentage table of physical signs, and concludes that the size of the pulse is the most reliable guide.

	Pure mitral stenosis, 17 cases.	Mitral stenosis and aortic regurgitation, 14 cases.	Aortic regurgitation and Flint's murmur, 33 cases.
	Per cent.	Per cent.	Per cent.
Small pulse	82.3	28.5	0
Presystolic thrill ...	64.7	50.0	42.0
Tapping systolic shock	76.4	50.0	15.1
Snapping first sound	64.7	78.5	30.3

The systolic blood pressure, as measured by the sphygmomanometer, is very high in aortic regurgitation, but the diastolic is low. The difference between the systolic and diastolic pressures is a fair measure of the amount of regurgitation, the greater the difference the greater the regurgitation.

The pulse is sometimes bisferiens; a distinct double beat may be felt when moderate pressure is put on the artery. In a tracing there is a double top to the wave, the second being higher than the first. This is often only present on the left side, while the right pulse is normal, and it is, I believe, invariably more marked on the left side. Some aortic stenosis or high peripheral resistance has been present in all the fully described cases. Roy and Adami, in their investigations on the hearts of dogs in 1890, found that a rise of peripheral resistance made the intra-ventricular pressure curve anacrotic, as if the contraction were reinforced by a renewed effort. It is probable, therefore, that in cases of aortic regurgitation with stenosis supervening the intra-ventricular pressure curve would be anacrotic, and, if the valves are much destroyed, the pressure curve in the aorta would correspond closely to the intra-ventricular, and an anacrotic or bisferiens pulse might be conveyed to the arteries. The explanation of the bisferiens pulse being more marked in the left carotid and radial is that the natural direction of the main blood stream is towards the left carotid (a fact often quoted as the cause of the greater frequency of cerebral embolism on the left side), and so the pressure curve of the aorta is more readily conveyed.

To estimate the amount of regurgitation the pulse is the most important factor. A large artery and very collapsing pulse, when the wrist is held above the level of the shoulder, indicate much regurgitation, and *vice versa*. But if the pulse is irregular proper compensation has not been attained, and there is more regurgitation than the pulse shows. In the last stages there may also not be sufficient force in the heart to produce a collapsing pulse at all. In atheromatous regurgitation the artery is large and tortuous from long-standing high tension, but there is not the same collapse of the pulse; sphygmographic tracings show a more sustained wave, and the amount of regurgitation is never very great.

The degree of dilatation and hypertrophy of the left ventricle is a help in estimating the extent of the regurgitation, but, if prolonged rest has not been insisted upon after the onset or after illnesses attended with fever, the dilatation will be in excess of what was necessary for compensation.

If the heart is much hypertrophied but the pulse is not correspondingly collapsing, either aortic stenosis predominates or the hypertrophy is the result of old high tension or kidney disease. If due to aortic stenosis there will be no second sound heard in the carotids, the pulse wave will be long and slow and the artery soft walled; but if due to old high tension

the second sound in the carotids will be loud and the artery thick walled and probably tortuous.

Prognosis.

"When, with a diastolic murmur, the aortic second sound is still audible in the neck, the pulse exhibits the collapsing character only in a moderate degree and the dilatation and hypertrophy of the heart are inconsiderable, the patient may be expected to enjoy life and do hard work for many years, provided the lesion is due to acute endocarditis," and not to syphilis or atheroma. In practically all such favourable cases the onset is before the age of 20.

Atheromatous cases arise so late in life that very little extra hypertrophy can be expected, and, unless specially guarded against, the high peripheral resistance, which was the main cause of the regurgitation, continues to act, and the atheroma progresses; the prognosis is therefore bad though the amount of regurgitation is not great.

When aortic stenosis supervenes in a case with considerable regurgitation, it improves the prognosis by lessening the amount of regurgitation. Dangerous symptoms will often disappear and the patient's life be much prolonged. When a valve ruptures, the strain put suddenly on the heart is too great, dilatation of the left ventricle is extreme, and hypertrophy never properly compensates. If there is dropsy the first thing to determine is whether it is due to mitral disease mainly, or to dilatation of the mitral orifice by the extent of the aortic regurgitation. If due to mitral disease the pulse will be irregular and not very large nor collapsing, and the second sound will be heard in the neck; the prognosis will be then rather better than when due to giving way of the mitral valve through the amount of aortic regurgitation.

Treatment.

Far the most important time for treatment is at the onset in rheumatic cases. Children with growing pains or a pain in any joint should always have their hearts examined; the onset of endocarditis is so insidious. During the acute stage salicin or aspirin given in full doses appears to be less depressing to the heart than salicylates. Small repeated blisters to the first, second, or third spaces on the left side, as recommended by Caton, are distinctly useful, as also is potassium iodide after the temperature has fallen. When the aortic valve is affected, rest in bed is essential, not only until the temperature comes down to normal, but for at least six weeks after, and then a very gradual return to being up all day, and no exercise or work should be allowed for six months or more. If so treated the murmur and all signs of the regurgitation will in rare instances completely disappear; I have seen this happen in a case recently. But when the regurgitation persists, as it usually does, compensation is attained with as little dilatation as possible, and so more reserve is left for future contingencies.

In syphilitic cases potassium iodide tends to prevent the increase of the lesion, and also does good by lowering the vascular tension. Atheromatous cases need tonics combined with measures to reduce the peripheral resistance.

In all cases tonics such as strychnine, arsenic, formates, ammonia, and bark are most useful, and belladonna if there is palpitation. The bowels should be kept regular, but no violent purgatives must be given because of the danger of fatal syncope. Flatulent distension of the stomach should be watched for and treated, since it may bring on angina pectoris earlier than need be. Whenever there are symptoms of cardiac distress, rest in bed for two weeks to a month should be ordered, and the getting up after this must be gradual. The patient ought also to be warned of the extreme danger of any sudden exertion. Hypodermic morphia is the best hypnotic if the nights are bad.

If venous congestion or dropsy come on, digitalis combined with vascular dilators and diuretics is useful until these symptoms have been got rid of. Then, if the mitral and tricuspid breakdown was due to the amount of the aortic regurgitation, the digitalis must be at once stopped, since it is a dangerous drug to use in aortic regurgitation. Bleeding is never safe, but a few leeches may sometimes help the digitalis when there is venous congestion. For angina pectoris the vascular dilators, spiritus aetheris nitrosi, trinitrin, or erythrol tetrinitrate are needed regularly, and capsules of amyl nitrite should be carried in case of an attack coming on. Arsenic or phosphorus and tonics improve the condition of the heart muscle and lessen the tendency to attacks.

Brighton.