cerned, but please do not talk about hypermetropia as old sight.

A few weeks ago a lady brought her son to me, aged about 8, because her medical man, finding that the boy was wearing + 1 D spheres for hypermetropia, told the mother that he had the sight of a man of 45, these being the glasses that a man of that age would ordinarily be wearing for reading. The mother was very distressed because, as you may know, the general public consider that deterioration of vision is necessarily an accompaniment of advancing years, which leads to the remark one so constantly hears from old people, if they do not come up to full standard, "Well—what can you expect of a person at my age?" This idea, by the way, keeps many people from consulting anyone about their sight when they ought to be having advice and treatment.

This lady, therefore, imagined that her son was thirty-two years nearer this inevitable deterioration of vision than he otherwise would be.

Perhaps I may be allowed to remind you that hypermetropia means a shorter eyeball than normal, and that this refractive error is corrected by convex glasses, and such glasses have to be worn by patients with hypermetropia if the amount is more than they can comfortably deal with by the continued exercise of the accommodation, or if through failure of accommodation the standard of vision falls below the normal.

Presbyopia, or failure of accommodation at 45 and over in normal sighted people, is also corrected by convex glasses, but the physiological cause for wearing this type in such cases is different to the anatomical cause in the hypermetropes. But the practitioner, in interpreting the cause for wearing glasses in the above case, had made the apparent error of regarding the wearing of the + 1 D lens in the same light in both cases, in spite of the fact that in the boy the glass was prescribed for distant vision, while in the man of 45 it would be ordered only for reading.

I may have omitted many of the apparent difficulties of general practitioners, which they themselves may regard as real difficulties; or on the other hand may not, being possibly under the impression that they are giving sound advice and treatment; but if I can be of any help by answering any questions you may wish to put to me, I will do my best in the short time left at our disposal.

ANGINA PECTORIS.

A POST-GRADUATE LECTURE DELIVERED AT QUEEN MARY'S
HOSPITAL FOR THE EAST END.

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LITTLE progress has been made in the study of this symptom during the past 150 years, since Heberden in 1768 published his original description, in which he found that the attack was relieved by alcohol and stimulants; that opium at night warded off attacks, and that at post-mortem there were no very gross changes, except early atheroma of the aorta.

One might venture to think that the cause of such little progress is the comparative infrequency of angina pectoris in hospital patients in this country, in comparison with the numerous cases of all types of angina pectoris met with in private practice. Most of the recent investigations have been done in America, where possibly the greater incidence among hospital patients would be due to the fact that these patients are derived from wider classes of society than in this country.

Though, as I have said, little progress has been made in our knowledge of the causal factors, we can certainly claim that recent work has given us a clearer classification of the types of angina pectoris, which materially helps us in both treatment and prognosis.

I would classify angina as follows:—

(a) Angina pectoris with cardiovascular lesions—

- (1) In old rheumatic carditis.
- (2) Syphilitic aortitis.
- (3) Arterio-sclerosis and hypertension.
- (4) Coronary occlusion (thrombotic, embolic).

(b) Angina pectoris without cardiovascular lesions—e.g., pseudo-angina.

Mild attacks of pain are experienced in cases suffering from rheumatic heart lesions, both in acute stages and in the later chronic stages, when myocardio failure has set in. These cases would appear to be due probably to dilatation of the heart muscle under strain, and are relieved by the usual remedies given in cases of cardiac failure, such as digitalis, venesection, &c. Severe pain is often experienced in patients who have syphilitic aortitis, resulting in some degree of dilatation, or even aneurism of the first part of the aorta. These subjects are usually under the age of 45.

Probably the largest group is that of cases of angina pectoris associated with arterio-sclerosis and hypertension, in which there is always some accompanying myocardial degeneration, in which changes of either a fatty or fibroid nature are present, or frequently both are found together. The blood-pressure usually, both systolic and diastolic, is raised. This is the type that readily responds to inhalations of amyl nitrite, and presents the classical features of an anginal attack, which is called by many true angina pectoris, death frequently occurring during an attack, and is so well known to you to need no further description here.

I think it is unnecessarily complicating matters to describe milder attacks of classical angina pectoris as angina minor. In most cases of true angina of any duration, there are variations in the severity of attacks, and, indeed, some cases of true angina first start with these mild attacks, which later develop into a severe form, and it is simply a question of our ability to recognize the significance

of early symptoms at such a time when we have the best opportunity to prescribe treatment and alter the patient's regime, with a view to preventing, if possible, further cardiovascular and myocardial degenerative changes, and so obviate further attacks.

Following recent investigations into post-mortem changes, we now come to recognize in greater detail another type of angina, due to coronary occlusion, either partial or complete, usually called coronary thrombosis, as this is the condition most frequently found at post-mortem in such cases, although embolism is occasionally found. Briefly the left coronary artery more usually than the right, is the site of the thrombus, and the descending branch of the left coronary artery is probably the commonest site. There is a resultant infarction of the heart wall, with some over-lying pericarditis, the heart generally is dilated, and the pericardial sac may contain blood-stained fluid.

The clinical picture in this type of case differs widely from that found in the classical anginal attack. The onset may be sudden, usually while the patient is at rest. The pain is longer continued, lasting some hours: there is great restlessness rather than rigidity and fixation: the patient presents an ashen-grey colour, and breaks out into a cold clammy sweat. The blood-pressure, both systolic and diastolic, tends to become lowered, the systolic more markedly so than the diastolic, resulting in the lowering of the pulse-pressure—the pulse-rate may rise, reaching 100 or more. Premature beats and other irregularities of rhythm may be found. On percussion, the cardiac dullness may be somewhat rapidly increased in the course of a few hours, showing rapid dilatation. On auscultation, pre-existing murmurs may become softer and even disappear. The character of the heart sounds alters, becoming less clearly defined and softer, and a pericardial friction rub may be heard. Changes in the electrocardiogram may show alterations in conductivity, with some degree of block. Later there may be some dyspnoea

and Cheyne-Stokes respiration may be present. Râles may also be heard at the bases. Sometimes emphysema may be a marked feature, and so mask any cardiac dilatation. Death may be sudden, either in the early stage, or later on. Some cases recover and may never have another attack for years, until possibly over-confidence has led them to take liberties with their regime. A few cases go on to progressive cardiac failure.

A TABLE SHOWING THE MAIN POINTS IN THE DIFFERENTIAL DIAGNOSIS BETWEEN "TRUE" ANGINA PECTORIS AND CORONARY THROMBOSIS.

Exerting cause	Emotion Exertion Dyspepsia	None Onset while at rest
Pain	Duration short, may be radiating	Duration long, usually localized
Appearance ...	Pallor and fixation	Ashen grey, sweating, very restless
Pulse	Regular and unaltered	Increasing pulse- rate and rhythm may be changed
B. P.	Usually high	Lowering of pulse- pressure
Pericardium ...	<i>Nil</i>	May be friction
Treatment ...	Amyl nitrite c. relief Morphia good	Amyl nitrite no use ? Morphia in large dose

Lastly I come to pseudo-angina. I dislike this term, as it implies that angina is a disease. And at the outset I would venture to warn you of the dangers of making such a diagnosis. It is often thought that the differential diagnosis between true and pseudo-angina should be easily and confidently made; in the young and in women possibly this is true, but in men past the third decade of life, where some mild cardiovascular changes are present, it may be extremely difficult to arrive confidently at a diagnosis of pseudo-angina. Pseudo-angina is usually found in women frequently without any obvious cardiovascular disease. Exertion plays no part in the case, but emotion and indigestion most decidedly do.

Toxic factors are frequently present, namely, excess of tobacco, alcohol, tea, &c., and intestinal stasis. Many such patients are labelled "Neurasthenic," probably most unjustly, owing to our failure to find the chief causal factor.

I now propose to discuss the methods of treatment in true angina, false angina, and coronary thrombosis, in the following three stages :—

- (1) During an attack.
- (2) Immediately after an attack.
- (3) To prevent further attacks.

(1) *During an attack* of true angina, the immediate benefit of crushing and inhaling a min. 5 capsule of amyl nitrite is well known to you; likewise inhalation of chloroform may be given; or the hypodermic administration of $\frac{1}{4}$ to $\frac{1}{3}$ of morphia: relief is rapid, and the patient, though badly shaken, feels very much better.

In coronary thrombosis, amyl nitrite is of very doubtful benefit; many state that it is absolutely valueless, but I would not care to be so sweeping. Morphia is required in rather larger doses, and then is of benefit.

In false angina, amyl nitrite is of no avail, and although morphia relieves at once, it is a very dangerous remedy, and should be withheld till all other measures have been tried, such as bromides with large doses of carminatives and diffusible stimulants.

(2) *Treatment following an Attack.*—Rest in bed. Diet should be dry, light and non-flatulent. Any tendency to dyspepsia should be corrected, and the bowels kept open.

Drugs : (i) Nitrites and potassium iodide is usually given in all cases with hypertension.

(ii) Anti-syphilitic remedies are given in the appropriate cases.

(iii) Bromides with strong carminatives are usefully employed in quietening the patient and relieving any tendency to gastric over-distention with flatulence, and some such preparation as Sedobrol at night to promote sleep.

(iv) Theobromine and theobromine sodium

salicylate have been tried, and by some are thought to be helpful. But this drug was tried many years ago, and fell into disuse from the very disappointing results of its administration.

(3) *Later Treatment to Prevent further Attacks.*—As the common factor in all types of angina is exhaustion of the myocardium, we should direct our attention to helping this overtired and strained muscle as much as possible.

This should be done by relieving it of any further strains, e.g., avoid excesses of alcohol and tobacco, &c.; avoid all emotions, over-worry and physical strain; avoid extreme cold and chills, avoid dyspepsia, and reduce the weight when obese.

The patient who has had an attack of true angina or coronary thrombosis should be encouraged and persuaded into ordering his life along a lower plane of physical exertion, and avoid all mental worries and overwork.

It will probably be necessary to put it to the patient that he should retire from business, but it is equally important to see that the void is filled by some suitable hobby. Motoring is best avoided, and most certainly he should never be allowed to drive, both for his own sake and for the safety of others. Later, gentle games without excitement may be permitted, such as clock golf and putting. But I have encountered an accident in even this simple effort—it was not, however, the game, but the side bets that caused the attack in this case.

Several operative measures have been devised, such as section of the posterior roots of nerves, Cs to D1, and paravertebral alcohol block. The various authors of such treatments claim that they have had successful results, but it would appear that they have quite lost sight of the fact that they merely remove the pain, and hence any warning signs are lost.

Diathermy has more recently come to the front, and by many it is thought to be of great benefit. Perhaps it would be more

correct to say that some cases are symptomatically improved while others are quite unrelieved; but at present there is nothing to point out to us which case will derive benefit and which will not.

Balneological treatment as carried out at Bad Nauheim and other Spas both at home and abroad can be extremely efficacious. That this treatment is not more efficacious is due to the casual method of administration of the baths by the bathroom attendant, and insufficient supervision by the physician. But let me assure you that the "Nauheim" baths can be given to the patient in his own home with excellent results, and on the whole it is preferable that he should receive this treatment in his own home, or in a nursing home, than that he should have to undergo first a long and arduous journey, then have his daily excursions to the baths, undress and dress again, and return to his hotel!

SURGICAL RESURRECTIONS—II.

(Continued from p. 86.)

THE treatment adopted in the case first described influenced me in the conduct of the second case, in which death from severe hæmorrhage was narrowly averted. The details of the second case are as follows.

A major in the Artillery, between 30 and 40 years of age, was treated for some time as a case of typhoid fever on the grounds that he had continued pyrexia for which no other cause could be assigned. He was seen by a surgical friend of mine who thought that the fever might be due to an abscess in the liver. The patient had lived in the tropics, and the possibility of infection with the amœba of dysentery could not be excluded. An X-ray photograph rather supported the view that the liver was the site of