The Heart in Old Age, and its Morbid Conditions.

Walter Dickson
Charco, in his "Lectures on Senior Disease," says, "the importance of a special study of the changes of old age would not be contested at the present day."

It is agreed, in fact, that if the pathology of childhood requires close consideration of a special kind, which, it is indispensable to be prac-

ically acquainted with; since pathology too has its difficulties, which can only be surmounted by long ex-
perience & a profound knowledge of its peculiar characters.

The changes which occur, with advancing life, in the heart & blood vessels are of great interest, partly, as developmental changes, & largely when they present the characters of disease, & this interest is accentuated in the senile by the dependence of the Nervous System, more especially on the higher centres, upon the efficiency of the Vascular. The bodily systems change & the tissues of the organs; functions are modified, or, for the most part gradually impaired; the heart, blood vessels, the blood obey the same laws in their structural changes & altered employment.
Still, life is compatible with health and a progress that is truly physiological, although more prone to disease, we do not regard disease as otherwise than an accident that affects the norm. The decline of vigour in man after the meridian of life has been passed is apt to incline the mind to regard the later period of life, as a progressive decay affecting every organ till life expires with the failure of some vital function. But this progressive decay or atrophy in age has some important exceptions. Bizzar taught that the heart in old people underwent an actual hypertrophy & many writers hold that the heart & kidneys evade the laws of a universal atrophy.

The same applies in a limited sense to some of the higher capacities of the individual. The intellectual creative powers decline after the decline of the animal reproductive power; but the judgement, depending upon the accumulation of a multitude of life-long impressions, & a just estimation & co-relation of these, implores in healthy old age until the encroachment of mental decay extinguishes it.
"Si donc à trente ans" says Bigot, "le cœur doit avoir le volume du poing au sujet, à cent ans il doit être plus volumineux, sous peine d'être dans une condition anormale. Le mieux est, dans les deux sexes, l'époque de la vie à laquelle le cœur offre le volume le plus considérable."

Coleridge states in his "Lectures on General Pathology" that the heart of very old persons ... rather increases in mass and volume. Denkle deduces the inference that was sure to be arrived at by some one "that only those reach advanced life who have been originally possessed of large and strong hearts."

Chacor, in the work already referred to, regards it as "a pathological state" following what is called the senile alteration of the arteries. At the same time "the network of capillary vessels" on its side becomes progressively thin, not only in the principal viscera, but also in the substance of the skin and mucous membranes. And again, "the involuntary muscular fibres do not escape from fatty degeneration; you will have a frequent opportunity of satisfying yourselves that the muscular walls of the"
"heart are almost always affected with it" in women who die at an advanced age.

With this alteration of the cardiac tissue are connected the phenomena of deficient cardiac contraction, which are so frequent—only observed in old people, even when they appear to enjoy good health.

By some it is stated that the enlargement mainly affects the left ventricle (Bigot) but there have not been wanting others who claimed that the right side was relatively more involved (Black, Lancet 1872).

Dr. George Balfour discussing in "The Senile Heart" the views expressed by Charcot & Bigot, takes exception to the view that the heart of age is a pathological state. He argues to the effect that it is a universal condition, therefore not pathological; the change in the senile blood vessels is sometimes only a loss of elasticity; obsolescent capillary areas cause intermittent flow from the arteries; therefore, raised arterial blood pressure, calling for increased left ventricle output—"the peeling of the myocardium at each"
"Pulsation with blood at a pressure considerably above the normal,—hence, other things being equal,—metabolism is more complete & nutrition more perfect. May so favourable are these conditions says Balfour "that weak hearts even hearts mechanically defective are able to profit by them, so that many hearts at seventy are stronger & better fitted for the discharge of their functions than they were at sixty."

Now, the discussion of whether to call the state of things pathological or not, must be largely an academic one, but what opportunity I have had of observing the hearts of old people, has not lead me to regard them as increasing their reserve of energy with advancing age. Practically we find nearly every heart of old persons we have an opportunity of handling after death, shows some evidence of fatty degeneration or connective tissue invasion or pigmentary deposit, not to mention arteriosclerotic changes & its sequelae, all which do not permit us to regard the aged heart as an organ whose functional power is on the increase. Its manifestation of energy in a
more susceptible & more frequent failure
is of course not necessarily indicative
of a healthier & more potential infor-
cardium. But, the recuperative power
in old age is great & I am sure that
this is, to say the least, as frequently
drawn in the heart as it is in some
forms of pulmonary embarrassment.
Still this recuperative power is not
absolute & it is important to remember
that the general reaction to a local
 lesion is less in old age than in the
adult or the child & is such as
often to mask the real state of things.
Perhaps this fact partially explains
why angina pectoris is an acute &
striking affection at fifty, rare as a
symptom at seventy; & we must
also remember that the hearts that
have shown signs of failure at sixty
have often improved in power & rhythm
at a later age because they have been
relieved of every such extra call
upon their reserve of energy as arose
in the daily business of the individual.

In discussing the pathology of
the heart of old age I will partly,
follow the usual classification of
heart diseases & attempt in the course
of the modifying influences of old age before alluding to the myocardial degenerations most intimately associated with this train of symptoms peculiar to the senile heart.

In this scheme I have addressed myself first to endocarditis & the valvular lesions & under the head of senile heart change more especially, hypertrophy, degeneration & dilatation of the heart, angina, & incidentally, the pulse modifications symptomatic thereof.

Acute Endocarditis is for the most part an affecting early life & inflammatory conditions of the endocardium in late life do not fall under this heading but I have often felt though I have not had an opportunity of verifying post-mortem that some cases of heart disturbance arising from influenza attacks in old people have been due to specific acute inflammation of the endocardium.

Clinically one cannot say whether this is a new thing or an old inflammatory process rekindled, but I have certainly seen a pericarditis
arise in this way in a robust old man, who, I had good reason to believe, never had a heart symptom before, or in his case I suspected a concurrent endocarditis at the time. Later on, when writing of the influence of Rheumatism on the Heart, I have cited some cases of the acute endocarditis which have occurred, in connection with the more chronic forms of that malady, frequent in old age. But, Acute Endocarditis or Pericarditis is sometimes a much more obscure process in life, because of the law of a less manifest general reaction to the local lesion. So doubts the in};

flammation is often overlooked, the temperature is misleading.

Although the temperature may be increased, on the other hand, it often falls. When the endocarditis, or pericarditis, arises in the course of another acute disease e.g. pneumonia, the onset may be marked by a drop (Charcot). The symptoms of local or reflex pain may be entirely absent. But, Chronic Endocarditis or Valvular Atherosclerosis is often evident in the heart after middle life, following, maybe
the acute process of some earlier state, or without any special history at all.
It is generally associated with the similar condition in the inner coat of arteries — arterio-sclerosis or athrombus. When occurring as an idiopathic affection it has been attributed to long continued high pressure within the aorta, the result of actual physical strain. Fayre regards more than half the cases as Rheumatic in origin. It, Pott, Bright's disease, alcohol, syphilitis, Chronic plumbism, with their consequent high arterial tension are all proved causes of the affection.

But, most important from our point of view is the fact that the condition seems to arise as a purely senile change, secondary to the athrombus in the arteries generally, or probably, in part determined by the higher arterial tension in old age.
The aorta values are most frequently affected, the endocardium of the left heart shows the condition very much more frequently than the right.
One finds the edges of the valves thickened & the valves perhaps adherent at their
extremities. The endocardium in the chambers of the heart may show this change, the chordae tendineae may be thick and tough. Here and there fatty or chalky degeneration may have taken place, much in the same way as in the inner coat of arteries.

In the aortic valves the process begins around the *corona arantii*, in the auriculo-ventricular valves, just within the valve margins.

The circatrificial contraction, producing great valvular insufficiency, may be established without much obvious valvular malformation. This is more frequently the case with the aortic orifice. Obstruction to the outflow does not so often accompany insufficiency as in the case of the auriculo-ventricular orifices. But in advanced cases the aortic lumen is diminished by the cicatrization.

Gelatinous mural endocarditis are often present as well as the changes in the papillary muscles - chordae tendineae.

The greatest degree of this chronic endocarditis I ever witnessed was in the congenitally malformed heart of a middle
aged woman, the particular of which I published (Edin. med. Journal 1871).
As the result of these changes stenosis occurs but incompetence is said to be the more frequent result, and the symptoms are generally those of the valvular affection.

This process which I have briefly described is a most important one in the pathology of the semi-lunar valves. The change is such an insidious one that it may be invading the cardiac tissues to a marked degree while no signal of its advance is shown.

The general symptoms of increasing age often the related condition in the coats of arteries gives the clue, but sometimes the first intimation to the physician is the gross heart change that is induced. The obstruction to the flow, or, reflex through defective valves, calls for such hypertrophy as the heart is capable of; or also the chambers dilate, sometimes suddenly during over-strengthen strain, or, gradually under prolonged difficulty.

It is often remarked of the general
practice of a physician, that while he has been trained by the study of classical types of disease, it is not always, in the daily discharge of his profession, that he meets with them. The extremes of youth and old age are among the most prominent factors that modify these classical portraits, and in discussing the valvular lesions I shall keep this point in view. Michael Stenonos, for example, is not an affection that would supply us with its best clinical picture amongst the aged, but I have judged it as well to discuss these lesions separately.

Aortic incompetence is however of the highest interest in our particular study.

The diagnosis of this condition has to do with the reflux of blood, arising from imperfect closure of the aortic orifice during diastole. Thus we usually have the evidence of a murmur of greater or less intensity, duration according to the circumstances, which murmur is produced by the flow of the fluid column from its containing...
channel, into a larger channel or chamber beyond. This backward current, when the valves no longer interpose, is caused by the elastic recoil of the blood vessels, or the suction exerted by the relaxing ventricle. This suction power or negative pressure is less in old age or dilated hearts, but whether it plays a large part in the production of the reflex is open to some doubt. It probably coincides with diastole but many thought it was attained at the end of systole.

Moore, — shortly before the systolic maximum.

The murmur produced by the de-gurgitation is not generally most audible over its place of origin, but is conducted in other directions. Its place of origin at the semilunar valves would be marked upon the chest wall at a point on the left side of the sternum, adjacent to the third left costal cartilage.

But this is not usually the point where it is loudest. Indeed, that point is a variable one.

It is usually to be heard in the aortic area (in the second right intercostal
space or over the second costal cartilage, close to the sternal junction. It may be traced upwards with diminishing intensity. It is often heard along the sternum, or, to the left of it, sometimes most loudly in the fourth or fifth interspaces to the left. Sometimes, it is audible at the apex. It is said to be sometimes most distinct in the third left interspace, over the pulmonary valve; almost limited to this position, it may act as an explanation of the line of conduction, that the pulmonary arterial interspaces between the aortic orifice and the chest wall.

In the adult heart the murmur is sometimes, under ordinary conditions, inaudible, though the pulse may be characteristic. Other signs point to imperfect closure of the valve. Its existence is a most reliable index of insufficiency. It is usually a soft, long murmur.

Professor Osler says, that is also in a large proportion of cases a systolic murmur heard at the aortic region, usually shorter, often rougher in quality, which may be propagated.
"upward into the neck. A common mistake is to regard this as indicating "stenosis, whereas, in the great majority "the usual instances of aortic insufficiency "there is no natural narrowing of the "murmur is produced by roughening of "the segments, or of the end of the "aorta.

Since I have directed my attention to this subject, I seem to have very rarely had any experience in aged "hearts of the second sound, without the "first. The first is generally of such "character as to impress an observer "the more forcibly.

A murmur at the apex, peculiarity "in time is sometimes heard, but this "may have no connection with mitral "disease. Dr. Austin Flint has ex-
plained this as being due to the "interference with the mitral flaps, by "the backward stream from the aortic "orifice, setting up vibrations that "are communicated to the ventricular "wall.

The pulse is often characteristic. "The waves are short, feeble. If the "patient is lying down or raises the "hand to show that there is not the gravity
of a supporting column of blood, we feel it may be the quick pulsation of sudden collapse. In pronounced cases we get a capillary pulse, an abnormal pulse. Sometimes we get a double wave, the pulse bifurcating, feeble on moderate pressure, another feature, — elevated by some to the position of a prime diagnostic point, — is the delay in the pulse.

Ordinarily, the velocity of the pulse wave is such that the time interval is hardly appreciable as between heart or carotids or radial. (carotid to radial 0.074 of a second, laenans) but in aortic incompetence, where the vessels are placid & empty, between the beats, we can appreciate the interval.

Important however, from the point of view of cerebro pathology is the fact, that vascular atheroma accelerates the transmission.

Irregularly, the pulse is not a special characteristic of the lesion but, in later stages, it occurs, it is often represented by a short supplement, —any beat followed by a longer interval, —the heart changing step as
it were, to recover its control.

Rosenstein, in an article in Steinem's Cyclopaedia says "The physioglogh-
tracing exhibits for aortic insuff-
ciency, perhaps the most characteristic-
curves of all heart diseases, but it-
is most certainly not specific . . . .

Besides the tracings are not at all-
constant. They vary with the degree-
of hypertrophy & the strength of the-
left ventricle, & further they vary-
according as the aortic insufficiency-
is complicated with much atheroma-
of the arteries, or produced by the-
endo-carditic process alone.

I give here two tracings, the first-
of an old lady with marked hyper-
 trophy - corary blood vessels, in whom-
I could detect no murmur until she-
had been in bed for a fortnight as the
recurs of a bronchial catarrh, when a harsh aortic, and later, a low tonal diastolic murmur developed in the aortic area.

The latter had a very distinct double aortic murmur, with a less degree of hypertrophy, & other atherosomatous vessels. It was one of those cases where the systolic murmur was very intense but the tracing negated stenosis. There was a more rapid pulse & less driving power than in the first case, & the sphygmograms perhaps illustrate this for Rosenkrantz's remarks.

The prognosis in this as in all heart affections is a most important one for patient & physician & we therefore have to look for special signs that indicate the gravity or otherwise, of the condition. It is a tenet of all heart pathology that the loudness of the murmur is no indication, per se, of the extent of the vascular mischief.

Broadbent ("Heart Disease" 1900) writing on the estimation of the lesion says "speaking generally, a long, loud murmur shows that a considerable
"degree of pressure is kept up in the aorta, which is a desirable thing in itself, is a proof that the heart is acting with vigour, or else that the leakage of the valves is not excessive; it is usually therefore among the favourable signs.

On the other hand, a weak short murmur indicating an opposite state of things may be a note of impending danger or death. But there are so many exceptions to the rule hinted at that it is not to be relied upon.

A favourable element is the existence of the second sound heard in the cardiac area, or over the carotids in the neck, as its existence will be positive evidence that the valves still discharge partly their function, but perhaps of greatest importance in this as in other heart conditions is our familiarity with the pulse of the individual, any variations that we may observe, induced by over dilatation of the left ventricle or the superinfection of mitral incompetency. While a markedly collapsing pulse points to a high degree of incompetence, a diminution of the pressure is not a reassuring sign, cardiac crises are accompanied by the enigmatical
of pulse that I have alluded to.

Heart pain & angina attacks, breathlessness & suffocating & some oedema are serious symptoms, but it is often one or other of these that first leads the patient to solicit our assistance. The following notes of a case illustrate some points of aortic incompetence.

Some months ago, I saw for the first time a lady aged seventy, who said she had always fairly good health till the exceedingly hot weather which had prevailed about a fortnight before.

She had then frequent attacks of great breathlessness & her sister told me she became pale & livid as the attack reached its worst. At such a time she had to sit leaning her breast against the back of a chair to support the stomach with her hand. She was of a very stout habit. Hot bogs or brandy seemed to afford her little relief, but the attack gradually passed off. A rather alarming attack of this nature occurred the night before I saw her. When I saw her I found her suffering from rather intense breathlessness & she admitted experiencing some degree of this for a
good time before she thought there was anything amiss. This breathlessness
assaulted her on any slight exertion, which she said she always avoided
on that account. She had had atten-
dance by a medical man for this &
defensive trouble, two years before.
Her countenance was slightly pallid
& her lips showed just a trace of
cyanosis; but for the rather hurried
respiration she was placed not
at all anxious about her condition.
I found her pulse 84, of low tension,
& although there were no intermissions
there was great variation in the volume
of the different pulse waves. On in-
specting the chest the motions of the
heart were very distinct, but I could
not feel any thrill on placing my
hand over the precordial area.
The aper beat was diffuse in the
fifth intercostal, almost on the
anterior axillary line & the heart
delusion was increased. There
was a loud prolonged systolic at the breast
& the second sound was almost inaudible
muffled by a soft diastolic breath.
Every few pulsations there occurred a
sharp forcible thump in which the
Character of the systolic murmur was lost. Both murmurs were very audible in the aortic area, were heard down the sternum but lost in a flapping systolic sound at, just outside the apex. There was great tympanitic distension of the stomach. After a week's absolute rest in bed, she improved greatly and her breathing became perfectly regular and she was able to lie down in bed without any propping of the shoulders. At this time she had occasional irregular intermissions of the pulse, the intermissions occupied by no pulse were those that the finger recognised or, there was just the suggestion of a faint beat. The intermission of the heart was equally obvious on auscultation and listening there with the finger on the pulse. I had illustrated the note of a remark of Dr. Stacey, apropos of Cardiac softenings - "no connection in time being traceable between it (the pulse) and the systole of the heart."

So the same on the impression of a systole followed by no pulse, due very likely to the marked delay of the reflex in a state of low systolic pressure.
The aortic ruins was louder at the end of her sleep's rest and the breathing became quieter she was able to get almost again. There was no drop in the "pitter" but an appearance of sub-endocardic fulness. She became very fond of a nitroglycerin tablet occasionally. I heard that she died almost suddenly a few weeks after her return to her home in London.

From my experience of acute regurgitation I have come to regard the development of drop as a serious symptom in prognosis. It may be of a different type than the drop of mitral disease, a puffing of the backs of the hands or one leg perhaps, not great in degree nor of such rapid development, but, especially in aged people, less influenced by treatment. The precursor of total syncope.

The mind is sometimes affected in aortic incompetence and I have seen some instances of this. In my experience it has taken the form of a simple melancholia or, a degree of moroseness irritability. In a case of migransent dilatation.
following aortic incompetence in a woman of 67. I recollect just such a condition accompanied by delusions of grandeur. But it has been especially in my senile cases that I have observed it, and it has seemed to be more attributable to the early manifestation of senile dementia, hastened by the thickening of the cerebral blood supply than alteration of mind special to this disease.

Death may occur quite suddenly.

Aortic stenosis is one of the heart diseases of old age. It is the result of the valvular affections of the left side of the heart. It is characterized by a systolic murmur heard in the aortic area. This murmur is however not alone pathognomonic as I have already instanced its occurrence in incompetence, where there was no suspicion of narrowing of the valve.

The murmur coincides with the first sound and is variable in quality. It may be prolonged or short, and conducted up towards the sternoclavicular articulation or heard one, the carotids, or in the back. It may be conducted
down the sternum & heard faintly at the apex. Sometimes there is a perceptible thrill over the second, third intercostal spaces on the right.

The systolic aortic murmurs in youth are most often anechoic, & in old age, the accompaniment of incompetence with deformed cusps, or dilatation of the first part of the aorta. This deformity of the cusps is, however, the more frequent cause of stenosis, independent narrowing of the orifice being quite negligible as a cause.

As the murmur is only contributory evidence of the existence of stenosis we have to seek from the pulse other evidence before arriving at any opinion as to the existence of the condition.

The pulse wave is notably different from that of incompetence; it is long & slow, & the vessel is obviously fuller between the beats; the wave ascends slowly to its maximum, subsides in the same way in contrast distinction to the "collapsing" pulse.

In old age, when the condition exists without the symptomatic pulse, the modification usually arises from coexisting incompetence; 

then the
A typical pulse & the aortic breakup
gave the clue to its existence.
In the lesser degrees, if compensation
is sufficient, the pulse may still be of
fair volume.

The heart hypertrophies & there may be
slight dilatation but this is the
affection that presents us with the
best examples of concentric hypertrophy.

The prognosis in pure uncomplicated
Stenosis is less serious than where
the valve is incompetent, but when
failure occurs we get dilatation of the
cavity & a weakened myocardium,
which may be intensified by the process
at the valves & recesses of Valvular
implicating the coronary arteries &
obstructing their lumina at the source.

Then as a sequence we may get incom-
petence at the mitral valve from
stretching of the chordae tendineae, or
actual fibrosis of these valve segments
or the backward pressure giving rise
to all the general symptoms which
follow this affection when aggravated
by failing compensation.

There is not the risk of sudden death
that there is in incompetence, but when
there are signs of backward pressure
failing compensation it is grave, more so than in cases where the origin of the cardiac back pressure symptoms is at the mitral valve alone.

Cardiac pain is not so common as in incompetence of the aortic valves, but gasiness in old people from central anemia, in arthritis, is a symptom that might mislead us if attributing it to a purely cardiac cause. The pulse may be slower than normal.

Mitral incompetence affords us some of the most vivid pictures of heart disease, for it is a lesion that we may see, quite markedly in old age even when the trouble has existed some time, for the heart can often truth effectually for long periods with a mitral regurgitation, for the symptoms are often ameliorated by treatment.

More than in any other type of heart affection in the aged, does a case of this kind come repeatedly under our attention, for I have descriptively before my mind an old man, over 70 years, who periodically paid admission to the Union Hospital here.
breathless, with legs enormously swollen. A pulse hardly perceptible, his voice a hoarse whisper between his gasps for breath. After rest in bed for a time, it disappeared in a diabetic mixture these symptoms would pass away & he would return to his usual life till another heart failure brought him under our charge again. He died from acute intestinal obstruction when convalescent from a heart attack.

Of course the important diagnostic is the systolic thrill at the conducta outwards from the apex, heard sometimes between the spine & scapulae, in the back. Sometimes it is best heard in the 5th or 6th intercostal in the middle line.

Aortic & tricuspid murmurs may prove very confusing in aged hearts. The former loses in perhaps because of the different rates of Conduction.

The failure at quite an early stage may be full & regular, but irregular, is characteristic. Old people with this disease & advanced arteriosclerosis often exhibit extreme angina.

One may feel intermissions in the beats.
variations in the force of individual
pulse waves—sometimes a thump-
ing beat, a rare, g minor ones, or
no two alike.

The physiological influences at
work are of interest in bearing upon
irregularities in intermissions of
the heart. We know a good deal
of the retarding or accelerating of
perhaps tonie move influence on
the heart. We also know that it
pulscates regularly, automatically,
when quite isolated from its nerve
communications, & there is no reason
to think that the nervous system
has anything to do, strictly, with
disordered rhythm. Early in cases
of clinical incompetence, when com-
pensation is good, an intermission
in the pulse occurs, long before we
get anything like marked irregularity

Broadhead in his recent work on
"Heart Disease" says "When there
is incipient irregularity, careful
observation will almost always show
that the break in the rhythm occurs
at the moment when there is a change
in the intra thoracic pressure, at the
beginning or end of inspiration or
expiration. The influence of Respiration cannot prove to be the essential cause though it plays a part in effecting the phenomenon, most probably from mechanical reasons as Broadhurst suspects, than reflexly through the Pulmonary branches of the Vagus.

The regular discharge of muscular energy seems to be a property of the vascular substance itself as shown, very largely by the experiment with Curarum. It is only to be expected that whatever interference disturbs the protoplasmic metabolism will disturb its function.

Therefore it seems to me that Lucas's experiment with the excised heart in a Ray's tonometer presents some points of interest bearing upon this matter. He supplied a frog's heart, placed in this instrument, with pure serum. He observed groups of pulsations with a long diastolic pause between every two groups.

The pulsations were on a crescendo scale till the maximum was reached, then occurred the pause. The pause disappeared when defibrinated blood or serum with hemoglobin, or normal
salaric solution were used. They occurred when the blood was dark colored or ureaether was added. Therefore the nature of the circulating medium will give rise to recurring intermissions in the excised frogic heart.

As any rate, it seems to me that the rhythm of the heart is closely dependent upon the healthy contractility of the sarcoens elements themselves, unless under circumstances that show the disturbed rhythm to be manifestly nervous in origin.

Mitral incompetence is commonly met with in old age and may have been of long standing as the result of a valvulitis; in other cases it supervenes as a symptom of senile decay with arteriosclerotic vessels or a dilating heart. The efficiency of the mitral valve depends a good deal on the papillary muscles, when a fibrous or other degenerative process has invaded these, the action of the valve must be interfered with.

I imagine that alterations in the character of the murmur must sometimes be due to this cause, especially such as when little sign of valve insufficiency...
after death.

The prognosis in old age depends a good deal on our previous acquaintance with the case. I don't think there is any much account as in acute incompetence, but when the symptoms of dilatation show themselves as a source of distress it becomes a very grave affection in old people, especially, if appropriate treatment does not at once yield some improvement. Pulmonary complications, bronchitis, or emphysema, constitute, of course, an increased risk but some wonderful recoveries occur under these conditions.

Atrial stenosis is not a cardiac affection that occupies a large place in senile pathology for my own part I can say little or nothing of its aspects peculiar to old age.

It is among the most interesting of heart lesions as an earlier age is commoner in the female sex. More than once I have had cases where a hereditary tendency to dilatation of the mitral valve was shown, incompetence in one member, stenosis in another,
rheumatism in, at an early two
families, played a large contributory
fact than anemia, which has been
ascertained as a possible cause of its
more frequent occurrence in girls than
boys.

The heart is usually enlarged but the
left ventricle may show little hyper-
trophy or none. The narrowing of the
mitral orifice is a sequel of chronic
endocarditis just as in another case
its effects lead to pure incompetence.

Why are yet the incompetence in some
cases of stenosis in others has not been
conclusively shown but Dr. W. Stewart
(Brit. Medical Journal 1900) advances
a theory that coincides with clinical
experience. He points out that the
nature of organic incompetence is
persistent from the first, while the
early auscultatory signs of stenosis
are inconsistent only such as to
arouse one's suspicions, and these is
generally no complication by regurg-
itation. He thinks the determining
factor is to be sought less in the
position of occurrence of the lesion than
in the action of the valve. His conclu-
-1-0
the original contraction is imperfect, & stenosis; in those cases where that original or "high closure" is maintained — & "stenosis" he says "... would" be the disease or taking strong, normally acting hearts; incompetence the disease of yielding atomic myocardiia. This theory would largely account for its rarity as a pure uncomplicated lesion of old age.

The elements of diagnosis are founded upon these — a pulse usually at first of small volume & rather high tension, later irregular, some of the beats feeble or imperceptible at the wrist though the heart sounds are audible. While the apex may, or may not be much displaced, we may be able to find increased cardiac dulness along the 3. & 4. intercostal spaces & from dilatation of the right auricle, even beyond the right margin of the Sternum. We may feel a thrill just inside the apex & there may be pulsation of the epigastrium.

The distinctive auscultatory sign is a precordial murmur between the apex & the Sternum — a vibratory murmur ending in an abrupt short sound.
As a later stage the second sound of the heart becomes inaudible at the apex and we have to auscultate carefully to avoid confusing a presystolic bruit ending in an abrupt taffy-like sound with the murmur of mitral incompetence quickly followed by an accentuated second. In the latest or most embarrassed state of the heart the diastolic and presystolic murmur is lost, probably from exhaustion of the left auricle & the degeneration of the mitral incompetence. So far as symptoms go, the patient may at an early stage show no obvious signs of heart disease until the final appearance of heart failure & backward pressure.

I believe that a mitral systolic murmur which has an initial & final sound of different quality may simulate a presystolic bruit.

I might indicate the sound waves diagrammatically thus: --

To show the sound and distinctly it may be misleading but close attention to the time of the cardiac cycle is a good.

It is usually a serious cardiac affection & progressive.
An over distended reflex heart is not uncommon in the middle aged and elderly. Sometimes we can percuss increased dulness to the right of the sternum and assume ourselves of the Whiffing bruit of tricuspid regurgitation, one third of the distance from the left margin of the sternum on a line drawn to the middle line. But in truly Senile cases a murmur here is often very difficult to detect. For probably have an obvious mitral systolic murmur in the immediate neighborhood, but the jugular distension & pulsation are outstanding signs of its existence.

As much then for the defects of the heart valves which I have treated in cases as affording an opportunity of referring to some special characters & modifications in old age, also to assist in adjusting the focus in examining the general symptoms & signs of the various corids at that period of life, or it is a large field of disease in the Senile that is occupied by heart trouble presenting few or none of the signs of vascular affection, but in which the symptoms are clamant enough
although the signs presented by an un-
published or dilated myocardium are not
as striking, yet it is precisely this
failure of the heart muscle that is the
most notable incident in some cardiac
disease judged from the clinical stand-
point, & its early symptoms may
ever appear trivial, & examination by
the stethoscope may reveal nothing
approaching what one might in contra-
denation call the more gross anatomic
changes perceptible in valvular lesions.

For some years I had occasionally
attended a gentleman, aged a little
over 60, a very stout man & the sub-
pject of a ventral hernia. He was a
man who lived well but in consequence
of some edema & a temporary appear-
ance of albumen in the urine he
was induced to restrict himself
by limit his aliment to a small
dose of whisky taken in the evening.
This course reduced his weight con-
siderably & he enjoyed very fair
health for about a couple of years.
Then in common with other members
of his family he had an influenza
attack which he seemed to get over
pretty well as he adopted every precau-

About two months afterwards I saw him for breathlessness. He told me that on the previous evening he had to keep aside his pipe as it had made him, for the first time in his life, feel faint. He felt breathless when he moved about but otherwise was much as usual. He had presented no signs of anterior sclerosis but none any evidence of heart affection unless possibly a weak, apoplectic after sound, which one could not be certain about in a big man with large mammae and deep chest. Now for the first time in my experience of his case I noticed once in every twenty beats or so, an intermission. The heart sounds seemed to show no change except the recurring pause. He gave up tobacco altogether during the next month of his life as he experienced the unpleasant effect upon his heart but was able to attend to his business. One day he kept his bed from slight symptoms of cedema contrary to the directions of my partner who saw him in the morning. He ate a hearty lunch; shortly afterwards he was sick, realized impending death as he emitted...
a hurried testament with his solicitor
whom he had summoned, and died
shortly afterwards in the presence of his
partner with the symptoms of increas-
ing angina. Such a case as this
one illustrates the fact that there is
often no obvious sign of gross cardiac
lesion though the disease is in the more
ominous, since much as it resides
in the muscular fibres themselves, the
carrier's signs are often very ob-
scure and produce only trifling mod-
ifications of the heart sounds to the ear.
In quite the earliest type of the
Senile Heart change,—that is the
prodromata of degeneration succeeding the
physiological increase of the old heart,—
what we probably notice is a faint
impulse, or none; the rigid thorax in
old people always lessens the perceptible
impulse. On listening over the heart
the sounds may be faint, the first sound
perhaps a little fainter than normal,
the second more of a tap than usual
—yet such a heart may be quite
incapable of any strain, mental or
physical without manifesting forlorn
the signs of failure. Here it is that
an occasional intermission in life—.
posture in propoer s, sometimes only occurring now and again while we keep the forefingers on the pulse for a long time, but more important than when we find it in a younger person with complains of much anxiety about their "weak heart". Like many another thing of disease, this is not absolute, for some old people live for years with very irregular and intermittent hearts, but it is important as a point of evidence.

Balfour ("Senile Heart") says "very important information as to the condition of the myocardium & the state of the blood is thus to be obtained from the greater or less readiness with which the clarity is evoked," & we usually have frequent means of estimating this from such trivial causes as the taking of food, slight exercise, or a pipe of tobacco, up to the systemic disturbance of a bronchial attack. Soon the morbid effect upon the heart proceeding from degenerative changes in its substance & the greater or less arterial sclerosis of the blood vessels with the consequent higher tension & resistance to the outflow, is a degree of Dilation of the chambers which
is the source of a great many of the more acute symptoms in the cardiac failure. It is not always easy in these cases of dilatation to perceive an area of increased dulness.

Empyema of a thorax is unimportant as the steam of a castle make a difference.

Careful percussion may fail in many cases to satisfy us that there is an increased area, though the symptoms point that way; then again a degree of dilatation with great embarrassment of heart action may exist where the deep dulness is not slightly increased & the apex beat is on the same plane.

The pulse is generally irregular & may present variations of intermission or great differences in the pulse waves.

It is often a sudden short compressible beat. In youth it may be regular & slow, often it is unduly rapid though still regular.

On inspecting the precordia we may see a large area of pulsation, perhaps with the apex beat below the normal, but I think more often there is nothing to be seen & the apex position is better recognized on auscultation.
usually in the fifth interspace more or less outside its proper area and not so sharply delimited; but after the degree of palpitation as well as the character of the sound is better arrived at, listening with the solid stethoscope over the 3rd or 4th interspaces & approximating inward.

Else it may be quite possible to feel the apex beat with the hand & also to discriminate in some cases whether what we feel is the apex of the left ventricle or the surface of the over-lapping right, with a degree of epigastric pulsation. The Right ventricle is very much "to the fore" in dilatation of the heart.

As regards palpation in these cases Walshe in his Practical Practice says, "the apex beat though visible & though the movement be nowhere else visible for frequently cannot be distinguished by the finger from the impulse of the ventricle generally— an evidence of extreme motor feebleness " as often exhibits a quasi undulatory character.

When I chanced upon this remark by this author I was much impressed by its coincidence with certain types in
In my own experience, I have sometimes been deceived in the detection of the quasiduplex sound by the impression it seemed diminished rather than intensified when the hand was placed on the pericardial area. He says the shock is feeble in proportion to the purity of the delation.

Apart from the question of conduction the quality of the sound is more indefinite - we in delination than in vascular lessons generally. Sometimes the first sound is loud and rather abrupt; the second is distinct, sometimes in later stages feeble but distinct.

Most frequently we get a feeble or rather muffled first with an accentuated second, or very often at a stage when the condition is quite established there is an alternation in the pause in time between the first and second sounds or an alternation of the long delitative pause. In the former case, the second sound may abruptly succeed a long pause, or the interval between the first and second may appear lengthened. In the latter case, the sounds may be equidistant like the ticking of a clockpiece, or in some like the embryo cardiac sounds with the two sounds
Alike in character, or the second the
more distinct of the two, apt to be
mistaken for the first. This is often
the case with senile hearts beating
over 90 or 100 to the minute.
Then in many of these cases of di-
lation, when the condition is fully
established, we get a systolic bruit
of mitral incompetence audible be-
tween the 2nd and 3rd ribs to the left of the
sternum. So the murmur we so
often hear in the young and anemic
the left auricular appendage, but recog-
nized in connection with the senile
heart to be of grave import. It may
be a temporary murmur at first, but
with increasing dilatation becomes per-
mmanent & audible all over the heart;
while I have found it extend to the
auricular area when the sound at the
apex would have given no suspicion
of its existence, though there were at
the time, general symptoms of heart-
break pressure of the circulation.
Again in some other cases, it is
first detected at the apex, conducts
outwards, & I presume that the po-
thesis of the murmurs is influenced greatly
by the greater or less degree of overlapping
of the right ventricle. This leads in a dilating senile heart, it seldom to do with any marked change in the valve segments, although marked change in these or the chordae tendineae or annuli papillaris, help to precipitate the establishment. It is more intimately connected with a failure of the structural contraction which does not permit of the free ascent of the valves being laid to one another during the force of systole.

Expecting dilatation, Schottlen, in his monograph, 'Cyclographia' has the following. But how we must remember that even in normal conditions the heart sounds are so variable in intensity, that if we have not been familiar with them before in a given individual, we cannot come to any accurate conclusions as to a possible change in sound. The second sound in the aorta is very often intensified for the reason that aorta walls have partially lost their elasticity.

Hardly to be distinguished clinically from senile dilatation on its being entirely regular in 

Fatty heart giving rise as it
often does to precisely the same symp-
thonies although post mortem the
organ may show more fatty degener-
ation than dilatation. It is appro-
priate to this paper to notice it, es-
pecially as it is largely an affecting
middle and old age, and weighing the
evidence, it may sometimes be possible
to attribute the evil to the degenerative
process rather than to dilatation of the
cardiacs, although perhaps, our pro-
gnoses and suggestions for treatment
may not be much influenced by the
refinement of diagnosis in a given case.
The apex beat is further diffuse in
pure cases the area of cardiac delayed
is not increased. The first sound
is usually shorter and weaker, and a
more noticeable interval between the
first and second; the second sound
relatively the more delayed. The first
sound is perhaps heard better to the
right than the left, the right is the
second sound over the second left
than right costal cartilage.

But the weakness or muffling of
the first sound is important.
Just as in dilatation we may get an
atomic central sound. Its appearance
is likely the signal of commencing dilatation.

There is no doubt that there exists a class of cases where alarming symptoms and death may occur from the faulty infiltration without there having been any previous symptoms to draw attention to the heart. In short, the condition may exist while an individual is in the active discharge of duties, intellectual or physical.

But on the other hand we may encounter every kind and variety of symptom, of greater or less degree, to which a heart struggling to maintain its output gives rise.

The pulse may be regular, 70 to 80 beats a minute, in an artery somewhat rigid from sclerosis, but the pulse may be irregular or slow. There is reason to connect some of the true cases of Bradycardia with a faulty degeneration.
I took it from an old lady, between 70 and 80 years of age,头脑清晰 but as slow and deliberate as her pulse.

She had slowly developed a dark red-rimmed, marked auricular ecchymosis, her countenance was dusky - a fact pointing red-tinted blue throughout. Her heart seemed slightly hypertrophied in every dimension but I could not satisfy myself of the existence of any tumour. Her pulse was 36.

When this tracing was taken I have never found it faster than 38 or 39. She has had this slow pulse for a good many years but she says "the liver is her trouble" not her heart, & two smart attacks of congestive jaundice justify her opinion.

An alteration in the ordinary pulse is often coincident with commencing delirium - then we may get the "gallop rhythm" & a shortening of the diastolic pause.

Shortness of breath on the slightest exertion or none, is often an early symptom of degeneration in the heart attacks also occur from this enfeeblement of the heart muscle. It is often brought about by advanced coronary
Sclerosis. There may be occasional syncopal attacks or nausea & the patient may awake with symptoms of cardiac asthma. I shall have a word or two to write about this when treating of Angina.

There may be slight edema of the limbs but dyspnoe is not marked as a rule & this edema is generally of late appearance, though not very progressive, serious enough.

While fatty myocardium affects old people of the most spare & attenuated frame, it often occurs in old people of a full habit, who tend to increasing obesity with the advance of years. It is said to be associated with a similar condition in the diaphragm in some, & death occasionally follows the overdistension of a full stomach, as in the case I narrated, or this is not so hastened by a weak & yielding diaphragm.

Sometimes coldness of the extremities & general lethargy characterize the condition, but some of the symptoms which connect themselves more especially with the Nervous System are of great importance, & it is well to describe
them here although they are not ex-
clusively due to faulty hearts but rather
to the degenerated senile heart.
They are of great interest as they
generally simulate lesions of the nervous
system & so may be mistaken for them.
They constitute the Adams-Stokes
Syndrome or Malattia dell'Adamo of
Huchard & there is a good description
of the matter in Lectures on "Anemia
Victoris & Allied States" by Adler 1897.

Syncope, Apoplectic, Epileptic etc.
are states of cardiac origin have been de-
scribed. The Syncope may be to
the extent of unconsciousness, or not,
the face of the body pales & cold & the
attack generally of some duration.
The pulse is weak & may be unperceptible
& these attacks sometimes closely precede
a fatal issue. But on the other hand
they may not. In our workhouse
hospitals there is a peculiar woman
who has lost her sight from repeated
attacks of glaucoma & some three
years ago she had double lobar pne-
monia which one anticipated would
carry her off. However she recovered,
but for a long time she had extremely
frightful action of the heart without any
obvious gross change but was liable to these syncopeal attacks, so much so, that care had to be exercised even in setting her up in bed. Then I have seen it repeatedly in an active old lady of 77 whose vessels were atrophied, but whose heart, excepting rather feeble high pitched sounds seemed normal. The first of her attacks I witnessed was while examining her wrist which she had hurt by tripping over an obstacle & again some months afterwards when I was removing cerumen from her ear. On each occasion I noticed the change impending, she became abstracted & had to lie down on a sofa; the surface of the body was cold & the face rather drawn & although not unconscious we were as she said “far away”. She was able to swallow a little brandy but lay on the couch for the better part of an hour, moaning from time to time. Recently, when attending her for asthma, & when she was in bed I saw symptoms of the syncope, but more evanescent. Sometimes now she has a pulse intermission. During her attacks the pulse became weaker & more compressible but
kept regular at its usual rate, generally 80 beats. The heart sounds were des.

tant.

From my experience I should say that this form of syncope was hardly
as sudden or complete as the syncope
we are familiar with in younger people,
but more enduring.

The skin may be dry instead of clammy;
there may be frequent excitations, or after
the worst of the attack is over, a good
deal of panting and an occasional deep
sighing respiration.

Then besides these syncope attacks
others of an apoplectic form or epileptic
kind may occur & these have generally
been described by writers in association
with fatty heart. Oster says "Don’t
forget that slowness of heart’s action is
the special feature" — not apparent
slowness from imperceptible beats at the
wrist. The patients are usually advanced
in years, & thus often an extreme
grade of arteriosclerosis, the arteries
feeling as Stokes remarked, both full &
hard.... Huckabee regards the slow
pulse as a result of changes in the vague
centres, due to decrease of arteries in the
medulla, he calls this form of action arterios
"the cardiac bell." It is worth re-

membering that, physiologically, there

is a centre in the medulla which

can excite general a phase of contract-

ions. Hence, or sudden anemia

can stimulate this centre.

At first, there is nothing to distinguish

the unconsciousness of cardiac origin

from the central. The apoplectic

fit may be sudden or profound & the

breathing noisy & temporary hemiplegia

has been described. But the attack

is not of long duration as a rule & we

may form an opinion as to the cause

from the invariably weak, almost

irreducible heart sounds & pulse pulse.

The epileptiform variety, with muscular

spasms & unconsciousness is I think, 

not an uncommon accompaniment of

the degenerated cardiac heart but is

perhaps more often seen by the nurse

than the physician. I imagine

that these central manifestations

are sometimes present at night &

I give here tracings of the pulse of

an old man with pulse first sound

at the apex, & distant tapping second

in the carotid area. I will describe

very altercations. We hear frequently,
had attacks of an epileptic character, or rather attacks that have been described to me as such, of brief duration. When I have been called to them he has usually been so far recovered that he was just vaguely conscious of his surroundings. General convulsions are not as common as in the epilepsy of youth, but there is always the possibility in infancy cases that it is a manifestation of early epilepsy.

The patient is affected, and the patient may be apt to fall, because giddiness is also present. Such a fall ends the life of many a poor person. There is often depression and irritability.

These attacks are more frequent: the nature of the fit is such with a brief period of unconsciousness and obliteration of recent memory.

Of the epileptiform variety, the pseudo-epilepsy of Stokes—Adam—first published a case in Vol. 70 of the Dublin...
Hospital Reports, It was a man of 68 years who had not less than twenty attacks in seven years, followed by much failure of memory. The pulse was very slow; temperature during the attacks. There was never any paralysis & he died after one of the seizures. The heart was very fatty.

This tracing I took a few days after an attack. It was that of a very old man—over 80, considerately, who had a hot bath on admission to the workhouse, & soon after getting back into bed passed into a state of unconsciousness. He breathed hardly & his extremities, especially his head & face were blue from cerebral congestion. He had a weak, irregular pulse of about 40, & I could not hear the heart sounds at all. There were a few rales about the lungs. By the following day he was better. It showed no signs of paralysis; in a few days he was clothed out of bed & his body...
from attacks since a big frame.

"Of the nervous symptoms" says Stokes

"the most important are the attacks of" apoplexy, or pseudo-apoplexy to which these patients are so liable. This affection differs from ordinary congenital apoplexy in these particulars, namely, the frequent repetition of the seizures, the variety of consequent paralyses, the fact that there is no only danger from an antiphlogistic treatment, but benefit, both remedial and preventive, from the use of stimulants.

In the other variety, the epileptiform variety, the mind is affected sometimes slowly but progressively. In the former they may be careless in matters of personal cleanliness, Traying all day & setting up walking about at night, in the latter irritable & prone to dislikes.

In the second the writing is affected. As a typical example we find the commencement of a sentence written clearly, well, the concluding words drift into an illegible scrawl with not a letter in it.

Most writers refer to the "phenomenon of Chaque" as it was called when the
causing the alteration of respiration that may occur from the fatty heart, although disclaiming it as a peculiar diagnostic or frequent accompaniment of the condition. I have certainly seen instances, not infrequently, in the last stages of degenerated senile hearts, but the rhythmic variation portrayed by the classic description of Stokes has been best realised in my own experience in connection with central apoplexy. A few weeks ago I saw it most notably in a man of 60 who died after a seizure with right hemiplegia. Older says "you will find it more frequently associated with chronic myocarditis than with any other form of heart disease".

I observed a peculiar modification recently in the case of a man between 70-80, who was dying from the failure of an hypertrophied heart. He had a distinct ticking second sound in the aortic area, but the heart sounds were elsewhere inaudible & his lungs were emphysematous. He had a full flapping pulse of about 130 beats a minute. For a fortnight before death he had much dyspnoea & during the
last two days it was of the description; he would breathe almost half-gently and regularly for the space of a minute, then, there would follow
15 to 20 quick forcible respirations, then he would resume his former comparatively placid period. He was perfectly conscious till within an hour of death, but the forced respiration was as regular intervals as less vigorous as he neared his end. It was not a gradual increase to a maximum then a decline in his case, but short sharp spells of dyspnoea.

Speaking of the Cheyne Stokes Variety, Etheridge in his Essay on "The Antagonism of Therapeutic Agents" 1878 says, "This is a most interesting phenomenon to observe, it is closely allied in its nature to the fall of the respiration in the rabbit produced by a toxic dose ofaconite, & its restoration when again under the stimulus of an antitonic dose of belladonna or strychnine. The respiratory centre becomes temporarily embarrassed; the respirator moves in fits, the increasing insufficiency of the blood awakens up the respiratory centre, the blood is again somewhat aerated."
then the action of the center fails until
the viscosity of the blood is such that
respiratory efforts are once more provoked.

Most frequently in the failure of the
generated hearts we get rapid shallow
respirations, sometimes with a deep
gaping one every now and again, so
we doubt that the act assists the di-

stended chambers of the heart, or in cases as I have mentioned it
seemed to be more adjacent to the
circulation, than to increase colic affection.

This would seem to be especially
in those cases where the respiratory
acts are controlled by the will and
patient is quite conscious, or the
subject, more cerebral anemia,
then anoxemia.

I remember seeing the Cheyne
stokes repeatedly in an old man
in the latter stages of a failing chronic
hypertrophied heart or acute incompatibility
with dyspnea of the lungs to a degree
of mental torpor with mild delusions.
The symptoms was exhibited a few
weeks or more before his death and
seemed to coincide with the development
of the dyspnea & the impaired circulation.

There is one diathesis which is
much associated with the train of symptoms exhibited by the enfeebled heart, with hypertrophy, dilatation of the chambers, that is, the Gouty.

It is especially in middle life, or at a later period that joint is seen to participate as a factor in the symptoms arising from the state of the heart and blood-vessels. Looking back over my ten years of practice, I should say that the recognition of the joint disease was more important in the treatment of a case than the recognition of treatment of a morbid state of heart in itself. So detect the basis towards the condition, constitutional or acquired is all important here.

Lathrop has a remark that "Observation has traced back, with fearful fidelity, a long line of fornicable fatal diseases to their path - the pathological parentage of the heart. But that which is constructed of preceding or coincident events is hitherto less perfect; yet observation has been able to assign to some diseases of the heart a cause, or, quid erat? a still continued alliance with, diseases of other organs."
or of the constitution at large.

This is peculiarly applicable to gout in some of its less obvious manifestations, for we might be led to attribute these to a morbid condition of heart which are more truly secondary to the general condition.

Chances in the work that I have alluded to, come up in the third subdivision of his seventh lecture much that is important in the gouty heart. The influence of gout, the rays of redness, as in rheumatism, towards endocarditis, pericarditis or valvular affections. These lesions in the heart depend on alcoholism or Bright's disease. Fat, degeneration is the great tendency. At the commencement it is but slight in degree. Retrocession of gout to the heart is not frequent. In the second stage it is definitely present. The symptoms are those arising from that condition. The rational symptoms are misleading. Here are paradoxical attacks—palpitations, dyspnea, a tendency to syncope; we may notice central symptoms taking the form of apoplexy although there has been no stroke.
cranial hemorrhage; sharp pain are fell in the precordial region, radiating along the arm, thus stimulating angina pectoris, which itself is often considered as an affection of heart or origin. Sudden death is frequent, rather from fatty heart than rheumatic gout. The atherosclerotic vessels may give rise to central hemorrhage.

This description indicates tertiary, the changes induced in the heart, or to be looked for when the atherosclerosis has been long established. But preceding all, we have the essential conditions leading up to the degenerative change, the lithiasis, the raised vascular tension, arterio capillary fibrosis, the uric acid kidney change, the hypertrophy of the heart, more especially of its left chamber. And though death as the ultimate issue may be attributable to the heart, it is usually only responsible as the last ordeal to yield to the advancing forces of disease.

The increased arterial tension, with strong muscular and forcible heart sounds are early characteristics indicate the commencing hypertrophy. Thus we can generally soon demonstrate by an en-
larged area of dulness. Very often in
these cases, a free indulgence in alcohol
participates as a factor in the early heart
change. Before the booming aortic
sound at the apex, is evident, we
may get exaggeration of the first sound
at, or in the immediate neighbourhood
of the aortic area, especially, if the
patient is suffering from one of his
"liver" attacks. I have heard it here,
strong & sudden as the blow of a foot,
I could little wonder at the change
that must in turn be induced in the
heart & aorta.

Although it is not immediately per-
tinent to the subject of this paper, I
have seen heart trouble in the second
or third generations of a family
with an ancestral history of gout.

Here is the pulse tracing of an old
Gentleman, aged 63, who is marked
gouty. His heart is slightly hypertro-
phied but there is no evidence of
valvular disease. His urine is pres-
ently abundant & creamy, or nearly
loaded with protein alkalies. He is a
short, stout, powerfully complexioned man,
with a longitudinally ridged finger nail.

He had a calculus removed by crushing
SIX YEARS AGO. AUTUS, EYES AND WINE ARE TALKED OF LONGER FOR AN EAR WERE THE DIABOS OF THE PAST AEGYPTIAN KING. THE HEAL INJUNCTIONS GENERALLY MEANS A BRIEF EXHILARATED PERIOD, GREAT VIVACITY, THEIR ABULIC FAVOURITE BILLIARDS, TO BE FOLLOWED ON THE MORROW BY THE REMAINS OF "LUNA-LAGO", ACUTE DEJAVU TROUBLES AND VIOLENT HEADACHE. A DAUGHTER, WHO, ABOUT TWENTY, WITHOUT ANY PRECEDING ILLNESS SEEMS TO HAVE DEVELOPED HEART TROUBLE, VIO, SUFFERED, THE SUBJECT OF A MITRAL LESION IN CAPABLE OF MUCH EXERCIION, WITHOUT DYSPEPSIA, HER CONDITION WAS LATELY PROCEEDED BY THE TELL LADY HANEN CLARK.

Then I have attended a grandchild, a girl, who became ill with a fluctuating pulse temperature, finally, acquired evidence of the presence of endocarditis. The mitral valves became incompetent, the developed aneurysms, which I have stopped, removing on each occasion, a week, being full of fluid. The agitation in full doses seemed to lend just the stimulus required to attain the enormous hyperkinesia of the heart, re-
Quired to permit of the proper main-
tenance of her circulation, she has
suffered since with very intractable
prorosis. I also attended a
little boy, a brother of the above, aged
5, who, while in the convalescent
period of scarlet fever, developed an
acute infective endocarditis, which
ended fatally within three days of
the first appearance of fever. Perhaps
Rheumatism played an unsuspected
part in the heart troubles of the heart,
but the drug in the grandfather is
and outset. At any rate, the occur-
rence in this family is of some
interest, as there does not appear
to be much evidence of hereditary
preroposition to heart affections in
gout, unless as a mere or less serile
change, dependent upon the other
conditions peculiar to this discussion.

Does the Rheumatic Disease, as
we see it in old age, play a part
in decline in the circulatory sys-
tem? The connection is so close, in
the acute forms of Rheumatism, that
the relation cannot be dismissed
without a word. But Rheumatism
is a term of such wide application,
amongst the public, and many medical men, that it is as well, to indicate clearly, the nature of what one is talking about, because even in standard works, there is some confusion in reference to articlar affections of this class.

Of course, Rheumatic fever stands out prominently as a most frequent cause of valvular affections, endocardial, pericardial inflammations, the effects of which are often times obscure enough in the simple heart.

But what of chronic Rheumatism? the so-called Rheumatoid, or Osteo-Arthritis?

"The use of the adjective Rheumatic" says Paget in his "Principles and Practice" "ought to be limited to the strict sense of the term; but in practice, it is often employed loosely for various affections of the muscles of other parts, of which the only common character is, that they are caused by cold. Nevertheless, it is much better to speak of Myalgia than of muscular Rheumatism; for the common usage of the term implies a connection..."
"of which there is no proof, between"

"rheumatic affections and rheumatism"

Mr. Busson describes "Chronic Rheumatism"

- extremely rare - as a sequel of
  acute rheumatism & mentions that
valvular lesions, due to sclerotic changes
are not uncommon. - Chances, although
admitting that "the more tendency the"
disease has to assume a chronic form,
the rarer it is to see such lesions

"develop during its course," - yet

...goes on to narrate evidence of their
occurrence in what he terms "chronic
or nodular rheumatism."

He points out that rheumatic endo-
carditis is very often latent during
life although leaving traces recogniz-
able after death. - This is a fact which
most practitioners can well appreciate
- i.e., as one encounters so many
late valvular affections, where there
is absolutely no history of acute
rheumatic affection. He instances
a case of Kombergi (Klinische Erfah-
rung, Berlin 1846): an analogous
case of Todd's in 1843; two of Travers's
out of his own, two of aortic lesions
in non-declarative rheumatism described by
Dean - Dr. Hecker.
There, he says, have generally occurred amongst those who formerly had an attack of acute rheumatism, "but I have collected," he goes on to say, "a considerable number of cases in which endocarditis has developed in chronic rheumatism without the disease having ever assumed an acute form."

One was a case of Dr. Ball's, a woman of 60, with evidence of chronic rheumatism only, who died from pericarditis, endocarditis affecting the aortic valves, and these conditions appeared to be of recent origin. Also a woman of 84 died in the edematous stage of cancer of liver, stomach, and intestines, "the lesions of dry arthritis," in the shoulders, elbows, knees. The heart was large and flabby. "On the aortic valves there were manifest traces of former endocarditis.

Also he says, "pericarditis probably occurs frequently in chronic rheumatism." He sums up thus, "endocarditis, pericarditis undoubtedly occur in some cases of chronic articular rheumatism; these affections present the same characters as in acute rheumatism. They appear especially when..."
there is an exacerbation of the disease, when there is some approach to the acute state. But these affections are generally less grave in character when they appear in the course of chronic rheumatism. This modus rheumatism he describes as distinct from what he terms "partial chronic rheumatism" or Heberden's nodosities, which he terms "two important forms of this disease" i.e. chronic rheumatism. He says further these may often be accompanied by vesicular lesions (asthma, heart disease); he is referring here to what we understand as osteo-arthritis.

I believe that Dr. Spender of Bath, has called attention to rapid action of the heart at an early stage in osteo-arthritis but I have not observed any special liability for the heart to be attacked. I noticed recently some heart disturbance in a patient afflicted with osteo-arthritis, which may or may not have had some connection with the general condition. It was the case of an old lady of 74, whom I had regularly attended for three years, who had gradually developed the affection...
In both hands till they became greatly distorted, the elbows & shoulders being also affected in lesser degree, & the neck in such a way that there was a forward inclination of the head & projection of the chin. Her pulse was always about 72 or 74, quite regular, & the second wave rather firm from atheroma. The heart was noticeable for only a weak first sound towards the apex with no perceptible ejection. Sometimes in November of last year the pulse became more frequent, as fast as 96, with sharp tapping heart sounds, & this condition lasted about a week, & then one day she had a sudden sharp pain over the heart. When I saw her I found her suffering from a most disordered state of heart rhythm.

The pulse beats were utterly irregular in time & force & the strongest beats abrupt rather than powerful. The second sound of the heart was distant to the first sound, then audible, muffled, but not attended by any appreciable bruit. I quite anticipated from her aspect that she would have died but the pain subsided by the second
day after her attack and the pulse had resumed a regular beat of 76. She was unaffected mentally and there was no dyspnoea.

To sum up, one's experience of this Diaphoretic influence I would say that there was no special liability to heart affection in Osteo-arthritis but distinctly more than a fortuitous connection between these, especially endocardial, and Rheumatism in its more chronic forms. Acute or sub-acute rheumatism in the adult or adult-child may lead to an exacerbation of heart trouble, the signs of which are only found at a more or less remote period from the term of the attack, but the Rheumatism of the aged, or endocarditis, are less shocking, but nevertheless will be found in conjunction, when we remember that an acute endocardial inflammation is still possible in the Senile.

Arthritis deformans is an affection of which every physician carries a vivid impression, more, I should deem from the excellent portraits than from his own personal experience.
I have never myself, attended a case with that combination of symptoms — the appalling pain I still more striking sense of impending dissolution, the fear to breathe — pain that radiates to arm or head, although I have a lively recollection of a relative who died in the night from a third attack of the distressing malady.

In much of the literature relating to angina, associated conditions bulk very largely and many cases that are cited leave one in doubt as to whether they ought to be classed as anginapectoris at all. But although the true angina as described by Heberden is interesting to the physician because so striking and comparatively rare, the associated states that present some of the cardinal symptoms are interesting and important because they are more frequent occurrence.

"Where do you find it most?" asks Dr. Arnold of his physician during the interval of the fatal attack. "In large towns I think." "Why?" "Perhaps from anxiety and squalor competition among the higher and intemperance among the lower classes."
The answer must my own experience to
far as it goes, for although we rarely encounter many varieties of
heart pain and anxiety, I have not noted any pure cases of the affection.
My work for the last six years has
been among farmers, a clergyman
or two, some people of leisure, the
usual inhabitants of a little country
town in Berkshire, and the poor,
and I have met with no case during
that time.

The condition would be easier to
explain if it were of commoner occur-
cence. One would think that the
degenerated dilatable heart with ath-
romatous vessels, so often the condition
in aged persons, would frequently be
subject to angina. The pathological
changes which have been assigned as
causes so often exist, coronary
occlusions, dilated & atheromatous aorta,
high peripheral tension in atheromatous
arteries, aortic valve disease, fatty
degeneration, dilatation of the chambers.
And yet such hearts may fail
under stress without having ever exhib-
ted anything to be described as
anginapectoris.
But again there are many that partially exhibit symptoms and of this class these senile hearts occupy a considerable share. Such a case I mentioned when writing of acute incontinence, and there are many with heart pain and distress and interference of some kind with the respiratory act, but it is really not given in the aged restricted to a brief period of acute chest pain.

I have seen quoted somewhere an epigrammatic saying of Romberg's that "Pain is the prayer of a nerve for pure blood" and perhaps in all cases of angina that pain is a call for pure blood or for more. I suggested before that intermittent action of the heart may be dependent upon the blood condition and in chlorosis we get acute pain apart altogether from neurotic influences and cured when the blood is enriched.

Then if a vessel is plugged by embolism or thrombosis we have severe pain. I remember an old lady being suddenly attacked with the most terrible pain in the left leg which was accounted for a day or two later by the appearance of
gamprene, in the feet, and, even short
of embolism or total obstruction of
the coronary one can discern how
angina pectoris might arise from the
sudden deficiency in an already
defective blood supply. Certainly, an
impure blood can cause it. J. F.
Balfour cites a case (emphysema)
where retrograde flow gave rise to
unmistakable attacks and he says
further, that "an impure blood"
"supply is a factor common, not only"
to the conditions just referred to (i.e., the
"one or fatty myocardium) but also to
almost every condition of heart with
which angina has ever been found
associated.

To sum this state of things, it only
requires something to clinch the attack,
and short of extraordinary exertion,
the heart has its remorseless automatic
call to obey, seventy or more times
a minute, and while pain may be
allayed by cessation of movement or
function elsewhere, there is no gibe
here. A minute's pause, were such
a thing possible might evade the
fatal syncope in the right, just as
the voluntary arrest of all exertion,
even inspiration for a time, helps the heart to vanquish an attack.

As regards the pathology of angina, there are certain conditions demonstrated and occurring in the ageing organism, which play a large part in its production and severity.

Edward Jenner, about 1776, described atherosclerosis of the coronary arteries in two cases and in a letter to Aberden about John Hunter he says

"The importance of the coronaries, and how much the heart must suffer from their not being able duly to perform their functions (we cannot be surprised at the painful spasms), is a subject I need not enlarge upon, therefore I shall just remark that it is possible that all the symptoms may arise from this one circumstance."

The recognition of this fact was a great step in advance and coronary atherosclerosis with degenerating the heart muscle exists in the great majority of the cases.

These morbid conditions are accepted but much speculation has been indulged in tracing the clinical features to the pathological conditions.
The theory of intermittent Claudication, a condition of failure of the tendons with tingling or cramp pains following plugging of the main artery while a diminished collateral circulation was maintained, has suggested the analogy to angina. Boley described the condition in the horse in 1856.

Charles J. Goeckerman, among others, have discussed this and its relation to angina. Sometimes an attack seems to be produced by such a cause but we cannot deduce much more than the ascertained physiological facts that anemia impairs function, that a slowly blood supply through narrowed vessels very soon disturbs the action of the various substances, although the transitional heart fibers are more resistant in this respect.

Coronary sclerosis gives rise to diminished cardiac activity, altered rhythm and frequency, and breathlessness, sometimes unconsciousness, congestions and pulmonary edema, and it is often in aged people an exacerbation of these symptoms that goes by the name of Angina Pectoris.

W. J. Porter in the Journal of Experimental
Medicine, 1896, epitomises the knowledge of the effects of closure of the coronary and branches. The frequency of stoppage of the heart is in proportion to the size of vessel blocked; ligation of the arterial septa does not arrest, if the large circumflex, arrests in 61/2 per cent.

As regards the blood pressure with the heart, the ligation of a single branch causes a diastolic rise of blood pressure and this is not met by an increase of the pressure in the coronary. Indeed it falls, and, as there is little difference in blood pressure normally between the auricular contents and the outlets of the coronary veins "it is plain" says Forrester "that the entire coronary circulation can in fact, be interrupted by the ligation of one coronary artery".

Plugging of a coronary may induce contractions of a fibrillar type.

In our aged patients at some age or less throughout the vessels is probably productive of many of the anginalid heart symptoms, about middle life an aortitis may induce more narrowing of caliber at the
coronary orifices.

we may have only one coronary as in Arnold's case and others describe an extraordinary heart in a man of thirty-six who died suddenly. The left coronary was almost obliterated with only a few point channel and the right converted into a "fibroid cord."

A common condition he says is "thrombosis with infarction and anemic necrosis in the cases of some duration; and another sequence of slowly developing coronary artery disease."

"case" is "fibroid myocarditis at the apex with weakening of the wall and gradual formation of aneurysm of the heart."

The radiation of the pain is tersely described by D. James Ross (Lancet, 1871).

The local irritation "was conducted to the portion of the spinal cord from which the vesica derived its excentric nerve, and thence spread in the gray matter of the posterior horns, whence by the law of eccentric projection it was referred to the termination of the somatic nerves derived from the segment of the cord—the 2nd and 3rd dorsal in the case of the heart. This"
"explained the pain shooting between the shoulders and down the inner side of the arm (2nd dorsal) to the elbow and ulnar border of the forearm and hand and ulnar fingers (1st dorsal).

The sort of case I have been myself I may illustrate by the following cases which I would present as anyone peculiar but rather as the variations of the affection, more or less frequent in one's experience of the cardiac heart.

First, an old man of 70 who had to turn a windlass for a couple of hours a day, complained to me of a sudden pain below his left breast which made him stop his work at once, and it always attacked him when at work. He was a healthy looking old fellow but with markedly atheromatous arteries. There was no cardiac trace, but rapping heart sounds, and every now and then the second sound closed sharply on the first.

2nd. Another old man who lived a hard life, cold keeping, (he was aged 75) always complained of a winter cough and suffered from attacks of "rheumatism" in the left
arm. This pain was occasional and followed a bout of coughing and was accompanied by severe pain in the left hypochondrium and between the shoulders. The paroxysms of pain and "wind" as he called it sported many a night's rest.

He had hard vessels and very intermittent pulses—64 to a minute.

His heart dulness was increased and the sounds very distant with a soft aortic systolic murmur.

There was an occasional rise in the lungs but the breathing was not at all hurried.

I only such a case of aortic incompetence as I described when speaking of that affection—severe chest pain with dyspnoea, and in going over Dr. Balfour's illustrative cases of apania in "The Senile Heart" I find five of this class, two of them pure cases of aortic reflux.

The old lady, confined to bed and attacked with sudden pain over the heart and extreme asystolia and anxiety. The pain extended in a few hours and the pulse resumed its rhythm by
the following day. The arteries were atheromatous and the condition was suggestive of coronary mischief. Such cases must be fairly common to most practitioners—sharp heart pain on exertion; pain local and reflex on a markedly senile heart embarrassed for the time; the pain in a senile heart with aortic lesion, and, lastly, the pain of a plugged coronary, analogous to the blocking of the middle cerebral.

But, as I have said, amongst our old patients the symptoms are modified, perhaps largely on account of two things, the more widespread arteriosclerosis which is central as well as cardiac, and the lessened general reaction to a local lesion which characterizes old age. Most frequently it is one of the associated aneurous appearances that we have to deal with.

A syncope aneurosa is a pretty frequent occurrence among the senile with advanced atheroma and it may follow true attacks of angina pectoris (Broadbent).
It may pass into attacks of pseudo-apoplexy, the Adams Stokes syndrome.

Then there is the angina pectoris due to Gardner, a term that might often be applied to the symptoms of acute dilatation in the aged.

Such a heart feeling of a very disturbing kind undoubtedly occurs, apart from pain, and as Sir Balfour says "most if not all fatal cases are of this character; so far as my experience goes by far the greater number of fatal seizures have been apparently painless."

And then, there is cardiac asthma, as it is termed, a very common feature of heart trouble and a very terrible one sometimes.

The typical type is a breathlessness on exertion which often dates in the patient's mind from a bronchial or influenza attack or after some extra exertion when the cardiac reserve was overstressed.

Then we have a persistent dyspnoea accompanying some valvular disease. In aged hearts with atheromatous vessels it is often the last phase of disease.
There is a respiratory hernia which
condemns the sufferer to death
with hard labour and relief is only
obtained when the medullary centres
become numbed and then may
be preceded by respiration of the Cheyne-Stokes type.

I once attended an old lady of
77 who had constant dyspnoea for
two months before her death and
throughout her fatal illness her
heart beat about 130 times a
minute; occasionally it was quite
uncountable. The heart sounds
were foetal in character, best
heard in the back, and it was
impossible to say whether there was
any increase of the heart dulness.
Her vessels were achromatous,
though her breath was humane
withstanding her dyspnoe.

Dropsy of the left leg and then the
right developed about a fortnight
before she died, but there did not
appear to be any interference from
fluid in the thorax.

Then again, more especially
in connection with valvular lesions
we get paroxysmal attacks of
dyspnoea. Such a case I remember
in a very acute woman of 56.
Her asthenia was not evident. She had a good deal of breathlessness on very slight exertion, a compressed pulse of 96, no heart impulse and the heart sound at the apex very faint. During the month or so that I attended her she gradually developed a systolic bruit, heard best around the pulmonary area. The lips became purple and she had numerous rales and crepitant sounds about the bases of the lungs. I was called to her one night for a sudden cyanosis of dyspnoea. I found her unconscious, clammy, blue and almost pulseless and was told that she had become so after a violent struggle for breath. I concluded that she was moribund but on the following morning she was comparatively comfortable and beyond a little hurry in the breathing was much better. Such attacks recurred at intervals for about a week before she died. I have found her almost black and gasping for breath and in an hour or two she would be better in every way.
But the attacks became more frequent and she died after some hours of unconsciousness.

On the whole I did not consider that the attempts to relieve the distress, digitalis, nitroglycerine, or the free use of oxygen made the slightest difference in her case.

Independent of fluid effusions, the lung congestion and lung oedema and iron bathing must play an important part in cases of failure of the left ventricle with increase of auricular pressure, and former points out that it is a respiratory insufficiency apart from rapid or deep breathing that characterizes it. The "expirations salutary" or lung ability is limited in disproportion to the work it is driven to do.