

THE PROGNOSIS OF SUDDEN
CORONARY OCCLUSION
AS SEEN IN GENERAL PRACTICE

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I N T R O D U C T I O N .

It is now some four years ago that I began to realise that sudden occlusion of the coronary arteries is not merely a matter of academic interest to the general practitioner.

I had always been interested in coronary occlusion as a cause of death:- while a house physician in hospital it chanced that I saw two patients die suddenly from this cause, just as I was passing their beds, - but it was during the winter of 1933-4, when I was making daily and twice daily visits to three patients at the same time, who were all acutely ill as the result of coronary occlusion, that I realised that it must be a common occurrence in general practice. When six months later I watched my own father die in an acute attack (he had advanced and rapidly progressive arterial disease) my interest was further stimulated, and I began to consider that a study of acute occlusion as seen in general practice might make a good subject for a thesis.

Another attraction of this disease as a subject for the general practitioner is the fact that its identification and description as an entity, diagnosable and treatable during life, have been made possible by purely clinical methods, and this in a period when laboratory methods seem to be becoming essential to the simplest diagnosis. I personally am convinced that the future of medicine lies along more and more scientific paths, but a subject like coronary occlusion is a refreshing reminder that bedside diagnosis is at least equally important. The only aid to the diagnosis of this condition not applicable by the general practitioner is the electrocardiogram, and careful clinical observation makes its aid very seldom necessary.

I pride myself on the fact that I had diagnosed from first principles a cardiac infarction, in a patient who survived, before I had read anything about the clinical features associated with these attacks.

About nine months ago I was browsing through the Lancet's symposium on Prognosis, and read in Bedford's article on Prognosis in Coronary Thrombosis (7) "If all cases in which the diagnosis can be established with reasonable certainty are included, the immediate mortality is probably not more than 25 per cent. These figures..... exclude sudden deaths at the onset." and later "the average duration of life in those that recover from coronary thrombosis is not yet known, our own statistics show an average survival of six months in thirty patients who died and thirteen months in 68 still living." I thought that surely the results in general practice were better than that, and thereupon decided that the Prognosis

of Coronary Occlusion would be the subject for my thesis, and that I would follow up every patient known to have suffered from coronary occlusion who had been seen in our practice since I joined it in 1929.

I have collected 41 cases. No case has been included in which I consider the diagnosis was not either certain on good clinical grounds, or confirmed by post-mortem - with the possible exception of Case 18, where I considered the attack to be due to embolus of a coronary artery, and - despite the adverse opinion of a cardiologist - having watched the case through, I feel convinced that nothing else could account for his illness.

This case also raises the question of nomenclature. After much thought I have decided to use the term coronary "occlusion" instead of "thrombosis" or "cardiac infarction." It is true that occlusion may occur very slowly and I have therefore qualified the term in the title to "sudden" occlusion. Without doubt, the majority of coronary occlusions are due to thrombosis, (23 of Levine's 35 cases where the point of occlusion could be determined (41)). Clark, Graef and Chasis (11) suggest that more careful examination microscopically would reveal thrombosis more often than this; Murray Lyon (55) found intravascular clotting in most cases of recent infarct, but some have no clotting and some are due to embolus.

Also it has been shown experimentally by Feil, Katz Moore and Scott (22) that the typical changes in the R-T segment of the electrocardiogram are due to ischaemia, in the production of which coronary occlusion is one factor, and that some other factor must be present (in their experiments venous obstruction). Wiggers also has shown that heart pain due to oxygen lack may give an electrocardiogram resembling that of occlusion. (82)

The term "cardiac infarction" is open to the objection that there is still apparently some doubt as to the invariable occurrence of infarction in non-fatal cases, diagnosed as coronary thrombosis or occlusion. An excellent correlation of clinical, pathological and electrocardiographic findings by Saphir, Priest, Hamburger & Katz (69) shows that "in the present state of our knowledge it seems impossible to differentiate clinically between myocardial infarction brought about by coronary thrombosis and that following arterio-sclerotic narrowing or occlusion of the coronary arteries.....also clinically.....the characteristic picture may occur in the absence of thrombosis and infarction".

Certainly later post-mortem examination may show completely occluded large branches of coronary arteries, with no visible lesion of the heart wall, as in my Case 1, Moritz and Beck (54) give examples of cases where "old complete occlusion of the major coronary artery was not associated with corresponding infarction", and experimental

evidence is available (8) that temporary cardiac ischaemia may result in lasting electrocardiographic changes, hitherto believed to be characteristic of infarction, in the absence of any discernible histological evidence of necrosis.

Gross (25) also points out that "in the determination of infarct formation besides the factors of size of the obliterated vessel, its location, the duration and rapidity of the obliteration, the condition of the general circulation and that of the heart musculature, another very important one must be added - namely the age of the individual".

Whatever name is used the syndrome which we are attempting to label is by now a well defined one, and the main factor in its production is undoubtedly sudden coronary occlusion.

At first, I meant to confine this paper wholly to prognosis but, as it is quite impossible to consider prognosis without first considering the diagnosis, I have given as full a description as possible of the actual attack in each case. If in some cases a much fuller description of the attack seems to have been given than is necessary to justify the diagnosis, it is because certain considerations, which have arisen during my study of the cases and the necessary reading, have led me to include in the thesis a discussion of certain other features, namely the cause and rôle of the fall of blood-pressure which is so common in the acute attacks, the question of collateral circulation and blood supply, and the relationship of coronary occlusion to angina pectoris as seen in general practice. This last discussion has required a last-minute search through our practice for cases of angina pectoris without sudden coronary occlusion in their history, but only the total number of such cases has been considered.

Two other things I have tried to do:- firstly, in order to avoid as far as possible the turgidity and obscurity which mars so much medical literature, I have tried never to use a technical term where a good English word will convey the necessary meaning, and I have been sorely tempted to include "H. W. FOWLER, Dictionary of Modern English Usage, 1926 Oxford, Clarendon Press," in my bibliography, but I feel that perhaps the acknowledgment is more fitting here: secondly, I have tried in describing the cases to get away from the "case" atmosphere, and to treat them as human beings, and to keep alive some at least of the drama from which an attack of coronary occlusion in real life is inseparable.

H I S T O R I C A L.

The natural starting point of the history of coronary occlusion would seem to be in 1786 with Heberden's "Account of a disorder of the breast" (28) (which disorder later came to be called angina pectoris) in which he describes a series of 100 patients suffering from recurrent precordial pain coming on at first only on exertion, going on in time to pain without exertion and after years to prolonged attacks of pain and to death. It is probable that the attacks without exertion, and the prolonged attacks, were frequently attacks of sudden occlusion of a coronary artery. Heberden mentions death occurring in early attacks, but says that "once only the very first attack continued "the whole night". This last was almost certainly an attack of sudden occlusion.

Heberden's description seems to have dominated the minds of the medical profession for nearly 150 years, and it certainly is substantially the same as the description taught to me as a medical student. My experience in general practice however has been that the slow development of anginal symptoms is rare, and that more or less severe attacks of occlusion usually appear early in the development of coronary disease. This question is discussed later in the paper.

The first important steps in the clinical separation of coronary occlusion from angina pectoris came much later, in 1878, when Hammer (27) described a case recognised ante-mortem. In 1884 Leyden (46) described coronary thrombosis both from clinical and pathological standpoints. R. Marie's Thèse de Paris in 1896 (52) showed that coronary occlusion was a frequent occurrence, and that Marie recognised its symptoms - and Dock (24) in the same year reported the first case diagnosed in life and confirmed at post-mortem.

Huchard in a pathological study in 1899 (34) of deaths from angina pectoris, showed how frequent was thrombosis of a coronary artery. Krehl (39) in 1901 seems to have been the first to suggest that recovery from an attack of occlusion might be possible. The next important contribution was that of Obratzow and Straschesko in 1910 (57) who described three cases, two of which were diagnosed ante-mortem, and gave an excellent description of the clinical features. Herrick in 1912 (29) was the first to show with any certainty that there was any possible favourable prognosis, that is that an attack of occlusion need not end fatally. He made no suggestion of the possibility of a long life after, and it is interesting in view of modern treatment to read his remark that "the importance of absolute rest in bed for several days is clear" (the underlining is mine). Not much attention was paid to either of these contributions for some years until Herrick's further paper (30), and in the same year Levine and Tranter (43) described the condition diagnosed ante-mortem in a case suspected of being an acute abdomen, and also a case operated on in error which died. Thereafter papers describing the clinical features of the disease followed thick and fast. The occurrence of electrocardiographic changes was noted by Pardee (62) who pointed out notching of QRS, left

ventricular preponderance, and the absence of the normal iso-electric stretch between R and T, and also inversion of T₂; and by Wearn (74) who described diminished amplitude of the waves.

Most interest was centred in the clinical features of the attack and the immediate prognosis till about 1928, when Parkinson and Bedford (64) published a series of 100 clinical and 82 post-mortem cases in the Lancet, and the following year when Levine's (41) excellent monograph on coronary thrombosis appeared as Medicine Monograph no. XVI.

One of the earliest papers devoted more especially to the subject of this thesis, the prognosis of coronary occlusion, was that of Paul White (76) in 1926 which sounds a restrained note of hope when he says "it may be said that patients often survive for years in good or fair condition the average duration of life after the attack in this group is close to two years". Parkinson and Bedford (64) said "a hopeful prognosis is often justified, for many survive and are able to lead useful lives sometimes for years". Levine in his Monograph (41) said "One can merely predict at present that when the final outcome of the entire series is known the average length of life will probably be about three years" i.e. in those who get over the acute attack.

From then onwards papers devoted more or less exclusively to prognosis and containing follow-up series have appeared, by Conner and Holt in 1930 (13) (287 cases of whom 117 were alive). White and Bland (79) in 1931, (200 cases, 101 dead, having lived on an average 1.5 years), Willius and Barnes (87A) and Carey Coombs (15) in 1932, Clark in 1933, (10) a discussion in the Proceedings of the Royal Society of Medicine led by Hay in 1934, (26) Bedford (6), Cooksey (14), Padilla and Cossio (58), and Middleton (53) in 1935, Cowan (17), Strong (72) and Willius (85) in 1936, and Palmer (61) in 1937. These contributions will be discussed more fully later in this paper in comparison with my own cases, suffice it for the moment to say that more optimism is heard in the later papers.

In the last year or two the effect of special features such as the size of the heart and the height of the blood-pressure in influencing the prognosis has been given more attention, they have been stressed in Cowan and Ritchie's textbook, 1935 edition (18) and Palmer has devoted three papers (59, 60, 61) to these aspects of the disease.

Coronary occlusion does not seem to have received the same attention in foreign literature that it has in the English and American literature, despite the early paper of Obratzow and Straschesko from Kiev in 1910 (57) already mentioned. The next important contribution would appear to be that from Moscow by Jegorow (36) (assistant to poor Pletnew who is figuring so uncomfortably in the news at the moment) who is obviously teaching something new, as he insists that "the diagnosis in life presents no great difficulty and only exceptionally can not be made". Eight out of his 17 cases came to autopsy. There is no record of the others after they left the clinic. No attempt at a follow-up is made.

Delrous (20) analysed 100 well-founded cases from French literature in 1932, in an "Etude clinique et "electrocardiographique".

In 1935 a paper devoted to prognosis appeared in the Argentine by Padilla and Cossio (28).

Hochrein published his book "Der Myokardinfarkt" in 1937. He devotes a chapter to prognosis of 287 cases "sufficiently investigated clinically". (31)

All the English and American follow-ups seem to be from hospital or consulting practice, and no real idea of the prognosis of coronary occlusion as seen by the general practitioner seems to be available. Even though this report is of too small a series to give anything like reliable figures I feel it is justifiable in that it should give an impression of how these cases go on from day to day and month to month, under the eye of the general practitioner, after their attack of what, at the very mildest, is a terrifying illness from the patient's point of view.

In a general practice such as ours, too, we see every kind of case, the mild cases, the severe cases and the sudden deaths, and we follow them right through their illness either in their homes, or in Nursing Homes, or in the local Cottage Hospital. Only one case in this series (Case No. 4) went into a large General Hospital as an in-patient at any time.

CASES FROM OUR PRACTICE.

I have included, to the best of my knowledge, every patient seen by myself or my partners in the last eight years who had, or had had an attack of coronary occlusion, in which the diagnosis was confirmed by post-mortem or by undoubted clinical evidence, sometimes confirmed by electrocardiography. It is only in the last year or two that the development of the portable electrocardiograph has made it a possible aid to diagnosis in general practice, but despite the willingness and helpfulness of our local consultants it has not always been possible, even since portable machines have become available, to have every case electrographed sufficiently soon after the acute attack to get typical pictures.

All available electrocardiograms have been included with the case reports, and as this paper is mostly a follow-up study, I have done my utmost to get an electrogram of every surviving patient as long as possible after the attack.

At the end of the description of some of the cases I have made a few notes which refer to the "discussion" which is to follow.

I have classified all the surviving patients in three groups,

- (1) Those who lead to all intents and purposes a normal life for their age.
- (2) Those who lead a restricted life, and
- (3) Those who are complete invalids.

In my case-notes it will be noticed that blood-pressure readings almost invariably end in 0; this is not because our patients are always sufficiently obliging to have their blood-pressures in round figures, but because, having taken something like one thousand blood-pressure readings annually for eight years, I consider that to pretend any greater accuracy of reading than to the nearest 10 mm. of mercury is sheer scientific pedantry; where doubt has arisen as to whether say 120 or 130 was the nearest reading 125 has been taken as the figure.

Case	Name	Age	Sex	Date of attack	Date of death	Period of sur- vival in those who died	Duration of previous angina	Angina after reove	Heart enlarged	Previous High IS	Subsequent	Status after reove
1	Mrs.O.	62	F	1927	Oct. 1937	10 yrs	0	+	+	+	?	1
2	B.L.	61	M	Sept 1930	1930	4 wks	5 yrs		?	?		
3	Miss F.D.	70	F	17 Jan 1932	23 Jan 1932	6 dys	?		?	?		
4	Mrs. E.A.G.	52	F	1 Sept 1932			3½ yrs	+	+	+	+	3
5	T.A.W.	48	M	6 Sept 1932			0	-	+	?	-	1
6	J.C.	69	M	11 Sept 1932			0	-	+	+	+	1
7	W.H.	58	M	Nov. 1932	Nov. 1932	0	2 wks	+	+			
8	A.E.T.	57	F	April 1933			? 2 yrs	-	+	+	+	1
9	H.B.	46	M	1 Dec 1933	25 Mar 1937	3 yrs 4 mos	2-3 yrs	+	+	?	-	2
10	Mrs.J.	76	F	3 Dec 1933			0	-	+	+	-	1
11	Sir A.P.	74	M	15 Jan 1934	11 Feb 1937	3 yrs 1 mo.	2-3 yrs	-	+	+	+	1
12	Mrs.K.	67	F	12 Feb 1934	8 Apr 1934	2 mos.	10 yrs	+	+			
13	F.B.	61	M	7 Apr 1934			0	-	-	-	-	1
14	H.W.	65	M	1934	1934	0	?		?	?		
15	Mrs.C.	57	F	2 May 1934			0	+	+	?	-	2
16	Mr.K.	53	M	6 Aug 1934			0	-	+	-	-	1
17	Canon E.	54	M	5 Nov 1934			2 mos.	-	+	?	-	1
18	J.H.H.	36	M	27 Nov 1934			0	-	?	-	-	1

1. Leading a practically normal life for age.
2. Leading a restricted life.
3. Complete invalidism.

Case	Name	Age	Sex	Date of attack	Date of death	Period of sur- vival in those who died	Duration of previous angina	Angina after recov	Heart enlarged	Previous High R	Subsequent	Status after recov
19	Mrs. A.M.	76	F	26 Apr 1935			4-5 yrs	+	+	+	+	3
20	T.G.	72	M	30 Aug 1935	30 Aug 1935	0	3 yrs 4 mos.		+	+		
21	Mr.H.	67	M	3 Oct 1935			1 wk	-	+	+	+	1
22	J.H.	30	F	7 Oct 1935	7 Oct 1935	0	2 yrs		-	-		
23	Mrs.T.	66	F	16 Nov 1935			1 wk	-	?	+	-	1
24	N.B.	58	M	26 Nov 1935	10 Dec 1935	2 wks	12 mos.		?	+		
25	Mr.W.	70	M	1936	1936	0	?		?	?		
26	Mr.L.	79	M	2 May 1936	2 May 1936	0	0		?	+		
27	Dr.K.	68	M	24 May 1936			10 dys	-	+	-	-	1
28	Mrs.W.	65	F	13 Jun 1936			13 yrs	+	+	+	+	2
29	A.W.P.	32	M	16 Dec 1936			16 yrs	-	+	+	+	1
30	M.K.	62	M	14 Mar 1937			2-3 yrs	-	+	+	+	1
31	M.L.	56	M	Apr 1937			0	-	-	-	-	1
32	Mrs.W.	59	F	1 Jun 1937			4 yrs	+	+	+	+	2
33	Mrs.F.	50	F	19 Aug 1937			6 wks	Still ill	+	-		Still ill
34	M.G.	58	M	7 Sep 1937			0	-	-	-	-	1
35	Mrs.L.	74	F	Oct 1937	25 Feb 1938	5 mos.	1 mo.		+	+		
36	Mrs.L.	77	F	22 Dec 1937	22 Dec 1937	0	Many yrs		+	+		

1. Leading a practically normal life for age.
2. Leading a restricted life.
3. Complete invalidism.

<u>Case</u>	<u>Name</u>	<u>Age</u>	<u>Sex</u>	<u>Date</u> <u>of</u> <u>attack</u>	<u>Date</u> <u>of</u> <u>death</u>	<u>Period</u> <u>of</u> <u>sur-</u> <u>vival</u> <u>in</u> <u>those</u> <u>who</u> <u>died</u>	<u>Duration</u> <u>of</u> <u>previous</u> <u>angina</u>	<u>Angina</u> <u>after</u> <u>recovery</u>	<u>Heart</u> <u>enlarged</u>	<u>Previous</u> <u>High</u> <u>Subsequent</u>	<u>Status</u> <u>after</u> <u>recovery</u>
37	E.J.Y.	59	M	5 Jan 1938			3 mos.	-	-	? -	1
38	A.J.B.	66	M	27 Jan 1938	29 Jan 1938	2 dys	3½ yrs	+	+		
39	T.W.	88	M	3 Feb 1938	3 Feb 1938	0	0	?	?		
40	Mrs.H.	72	F	16 Feb 1938	16 Feb 1938	1 dy	0	-	?		
41	W.T.	77	M	22 Feb 1938	22 Feb 1938	0	?	+	?		

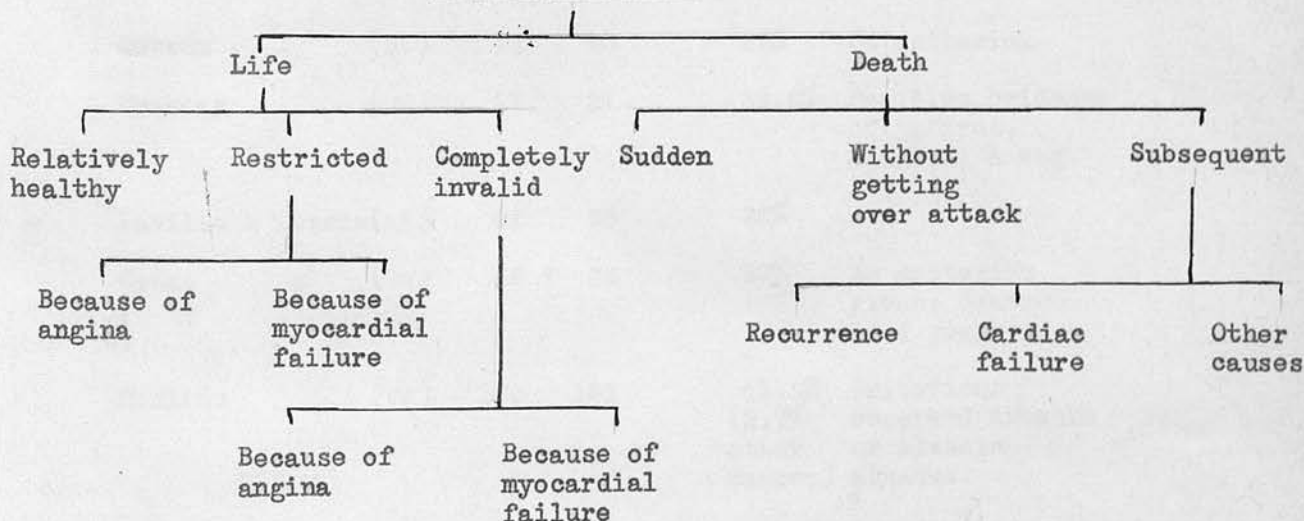
1. Leading a practically normal life for age.
2. Leading a restricted life.
3. Complete invalidism.

DISCUSSION: CHIEFLY OF PROGNOSIS.

With such a small series of cases it is misleading to subdivide them into different categories such as sex, age-periods, and so on; nor is it fair to draw conclusions from the occurrence of less usual symptoms such as glycosuria, various arrhythmias, friction, etc., otherwise one might be found remarking on the remarkable prevalence of this disease among patients whose names begin with H, K and L (13 cases: 32%); but I think it is quite worth while treating them as one group, and comparing the mortality and so on with that reported by various authorities. It is a remarkable fact that while I was preparing the material for this paper no fewer than three sudden deaths occurred, and two deaths within 48 hours of onset, all of which I felt compelled to include, even though we may not have another sudden death for another two years.

The prognosis of coronary occlusion like that of other diseases, can be reviewed from so many different aspects that I have decided to make a table of the various aspects and then consider them - thus:-

P R O G N O S I S .



Life or death: With regard to the first and most obviously fallacious aspect of the prognosis (because it depends so largely on how long the series has been observed) the question of total mortality, it is perhaps best to give a table of the figures reported by various authorities:-

T O T A L M O R T A L I T Y.

<u>Authority.</u>	<u>Refce.</u>	<u>No. of Cases.</u>	<u>Deaths.</u>	<u>Percentage.</u>	<u>Criterion of attacks; and remarks.</u>
White in 1926	(76)	62	32	52%	
Parkinson & Bedford (1928)	(64)	100	31	31%	Criterion: ?
Levine (1929)	(41)	143	101	71%	53% immediate rest subsequent
Conner & Holt	(13)	287	142	49%	All doubtful cases excluded
White & Bland	(79)	200	101	50.5%	Criterion: no clinical doubt
Carey Coombs	(15)	144	81	56%	Only certain diagnosis included
Delrous	(20)	100	100 in 3 yrs	100%	
Cotton	(26)	73	20	27%	No criterion
Cooksey	(14)	53	21	39.6%	Positive evidence of infarct, clinical & ecg.
Padilla & Cossio (58)		92	35	38%	
Cowan	(17)	66 ?	33	50%	No criterion given; deaths in 2 years
Willius	(35)	370	191	51.6% (2.7% other causes)	Criterion: observed attacks or classic attacks.
Master, Jaffe & Dack	(50)	243		21.2%	Immediate deaths only, certain diagnosis.
Strong	(72)	120	75	63%	Well defined clinical features, surviving initial attack.
Palmer	(61)	212	65	31%	Only "recovered" cases; all had cardiac pain for at least 1 hour.

<u>Authority</u>	<u>Refce.</u>	<u>No. of Cases</u>	<u>Deaths</u>	<u>Percentage</u>	<u>Criterion of attacks; and remarks.</u>
Hochrein	(31)	287	175	61%	sufficiently investigated clinically
Freeman	(23)	26	11	42%	Criterion ?
Self		41	19	46%	Including sudden deaths.
		32	10	31%	Excluding sudden deaths.

SUDDEN DEATH.

As far as I can make out all these authorities exclude cases of sudden death absolutely from their calculations. Levine (41) considers that sudden deaths are offset in calculating the mortality by slight cases which go unrecognized, and which do not die. Strong (72) on the other hand believes that 50% of all people suffering from coronary occlusion die before medical attention can be obtained. I think my own figures in this respect are really illuminating, as we see all the cases of sudden death that occur among our patients, or at least we know of them. In this series there are nine cases who died suddenly, of what I think undoubtedly was coronary occlusion. Of these, three have occurred during the last month or two, which is abnormal. The brother and sister of case No. 3 were both found dead; the brother had suffered from attacks of cardiac decompensation; the sister had suffered from recurrent bronchitis; both were diabetic. It is probable that these also were deaths from coronary occlusion, and to err on the safe side, if we add these on to the nine cases already reported, we get eleven sudden deaths out of a total of 43 cases, giving a percentage sudden mortality of 25%. As I have indicated, this is probably on the high side.

It seems to be established that it is the degree of coronary insufficiency which determines whether sudden death, or infarct of the heart wall, will result, whether this insufficiency be due wholly to blockage of an artery, or partly to other factors. I have therefore included, as being due to coronary occlusion, all cases of sudden death where the symptoms and history suggest that coronary artery disease was the fundamental cause. Most of them provide in addition other evidence of occlusion.

Levy (44) discussing sudden deaths from coronary disease says, "The microscopic appearance of the thrombus.....indicated that it had formed at least several hours or days before....." (cf case 41) "thrombosis is rarely the immediate cause of sudden death. It increases the liability.....the cessation

"of the heart beat is caused by acute coronary insufficiency".

The actual mechanism of sudden death is probably ventricular fibrillation, first suggested apparently by MacWilliam in 1889 (47), as a cause of sudden death, especially in cases of angina pectoris. Levine (42) quotes a case of a patient who died in an attack of angina pectoris which came on while he was being electrographed, and where the tracing showed ventricular fibrillation.

Deaths before recovery from attack: but not sudden.

This is the class of death which has received most attention in most of the published work, those who do not die at the onset, but succumb before their heart is healed, from later sudden death, or from further infarction, or from rupture of the heart, or from cardiac failure, or from secondary embolism and so on.

In this class various authorities find:-

<u>Reference</u>		<u>Cases</u>	<u>Deaths</u>	<u>Percentage</u>	<u>Remarks</u>
White (in 1926)	(76)	62	18	29%	Within 1 year
Parkinson & Bedford	(64)	100	(12 23)	12% 23%	" 1 month " 6 months
Levine	(41)	143	76	53%	Before recovery
Commer & Holt	(13)	287	46	16.2%	Immediate mortality in 1st attack
Carey Coombs	(15)	144	49	34%	Within or shortly after attack
Delrous	(20)			33%	Within 1st 4 weeks
Cooksey	(14)	53	19	38%	Within 2 months
Cowan & Ritchie	(18)	100	48	48%	Within 1 month
Bedford	(6)			25%	Within 6 - 8 weeks
Padilla & Cossio	(58)	92	25	28%	Within 3 months
Moritz & Beck	(52)	94	14	15%	

<u>Reference</u>	<u>Cases</u>	<u>Deaths</u>	<u>Percentage</u>	<u>Remarks</u>
Master, Jaffe & Dack (50)			(8% in 1st attacks) (16.5% in all attacks)	See below
Hochrein (31)	287	156	58%	Within 4 weeks
Self	32	7	22%	Without getting up, all except one within 2 months.

Master, Jaffe & Dack claim that only cases of certain diagnosis were included and attribute the very good results to prolonged rest and a low calorie diet (800 calories per day).

There are obvious and surprising differences in these figures of immediate mortality, even allowing for the different methods of estimating them, and reading the original papers does not help one to understand the differences.

"SUBSEQUENT" DEATHS.

Much the most interesting aspect of the mortality to me is the subsequent mortality of those who survive, as it is in this respect chiefly that the results in this practice seem to be so much better than those of most published series.

There are equally varying views and figures about this aspect i.e. deaths occurring among those who recover from the original attack. It is quite impossible to give accurate mortality figures at various intervals for "subsequent" deaths, as no series of cases has yet been followed to the death of all the cases, but general impressions can be given from the figures of various authors.

Parkinson and Bedford found that of 100 cases, twelve died in one month, eleven died between one and six months, and seven between six and 24 months. (64)

The average life of 19 of Levine's cases (41) who died after recovery from the attack was 22.6 months. He predicts an average life of three years for the entire series. In 1935, (42) however, he says "the average duration of life after recovery is not much longer than two years".

White and Bland (79) had 80 patients who lived over one month who died after an average life of 1.9 years, mainly cardiac deaths.

Carey Coombs (15) says one-third of those who survive the attack die within the year.

None at all of Delrous' 100 cases (20) survived over three years.

Hay (26) says "those who survive are sorely crippled and "it is only too true that sooner rather than later they will "die a cardiac death either from congestive failure or a "further coronary thrombosis".

Cooksey (14) reports 53 cases of whom 21 are dead; of these 21, 19 died within two months, and only two after two months; yet he has eleven patients alive more than six years after their attack, and all the 32 survivors have lived over one year. His experience is much more like ours. All his patients were private cases.

Padilla and Cossio (58) had seven subsequent deaths among 64 patients surviving over six months, two within the year, two in four years, one in six years and two after six years.

Cowan (17) had 66 patients who lived over six months. Of these, seven died in first year, six in second year, five in third year, three in fourth year, three in fifth year and so on, 33 altogether are known to have died, 24 of cardiac failure, 14 slowly and 10 suddenly.

Freeman (23) says "The only conclusion to which I am forced "is that when angina pectoris begins death is not far away".

Hochrein (31) had 19 cases die subsequently ("in the "following months and years") out of 131 who survived four weeks.

The experience of Cooksey, of Padilla and Cossio and of Hochrein is much closer to that in this practice, although we seem to have been singularly fortunate in having no early deaths at all subsequent to recovery from the attack. So far there has not been a single death from any cause under three years after recovery.

Of 25 patients who survived the initial attack, only three have died, one of cancer, none of them under three years from the date of the first attack. Of thirteen patients who have lived over three years after an attack, two have died between the third and fourth year: six patients have survived over four years, and four over five years, only one of these is dead, ten years after the attack.

The cause of subsequent deaths may be recurrence of occlusion, or cardiac failure, or some cause unrelated to the heart. Our subsequent deaths are too few to compare with others;—three deaths, two from recurrence, one from cancer: but it is of interest to see the likelihood of the various causes in the experience of others.

White and Bland (79) found that in only six, out of 101 cases who died, was death not due to the heart. They do not distinguish between immediate and subsequent deaths.

Of Willius' (85) 370 cases, 191 died of heart disease (again there is no separation of immediate and subsequent),

36.6% of coronary thrombosis, 51.9% of failure, 11.5% suddenly.

Cowan (17) had 24 of his survivors who died of cardiac failure, 14 slowly and ten suddenly.

Padilla and Cossio (58) consider that from fifteen days to one year after the attack death is more likely to be due to congestive failure, after one year it is usually due to recurrence.

Hochrein (31) gives five cases dying of recurrence after the twelfth week, and eleven of failure.

PROGNOSIS AS TO LIFE.

Much more interesting to us, and more important from the patient's point of view than his chances of death, are his prospects of life and health.

RECURRENCE.

Those who survive an attack of coronary thrombosis are first of all always liable to a recurrence. This does not seem now so probable or so certain as was once believed.

Jegorow's seventeen patients (34) had all except one had previous attacks of "status anginosus". Although he gives no follow-up of the cases after the "described" attacks, and doesn't discuss the subsequent prognosis, he points out that eight had lived over a year since their first "status anginosus", two over ten years, and one over fifteen years.

Of Conner & Holt's (13) 117 "survivors", 38 died in second attacks, 24% of his cases had two attacks, 4% three attacks, 5% from four to seven attacks.

Cotton had five cases, out of 73, die of recurrence.

White & Bland (79) had 20 cases who survived a second attack and eight who survived two attacks.

Willius (85) reports two attacks in 17%, three in 22% and four in 0.5%.

Half of Master, Jaffe & Dack's (30) cases had had previous attacks.

Of Palmer's (61) 212 cases 28% recurred at least once, 5% had a third attack, 0.9% a fourth.

Bourne & Scott (9) report 32 cases, of whom five had more than one attack.

In the present series of cases out of the 25 "survivors" of the attacks six, or 24%, have had further attacks since, one (case 4) has had three recurrences (and still lives), and a possible attack prior to the first established one; two have

had two recurrences, of whom one died in the second (case 9), one survived the second to die of cancer (case 11), three have had one recurrence of whom one died (case 1). In addition cases 12, 18 and 29 may have had previous attacks, case 12 would then have died of a recurrence.

It is not fair to draw conclusions as to mortality in recurrent attacks from only six, or at most nine, cases, but most authorities report a much higher mortality rate in recurrences, Padilla and Cossio (58) 60%, Master, Jaffe & Dack (50) 16.5% in all attacks, only 8% in first attacks. Willius (85) 69.8% in second attacks, 75% in third. Palmer (61) "20% in subsequent attacks".

LONGEVITY.

Apart from the liability to recurrence we have to consider what prospect of life and health lies before our patients who survive the initial attack. I do not think there is much advantage in considering the figures of average length of life following recovery shown by various authors, as of course none of the series (except Delrous!) have yet been carried to the end of life in all the cases;—although the average survival for my 24 "recovered" cases is even now three years $1\frac{1}{2}$ months, and, if we discard cases who had their attack less than a year ago, it is three years eight months, including the three cases who have died; if we discard these the average is three years seven months for all cases. This compares with Paul White's $24\frac{1}{2}$ months in 1926 (76), White and Bland's 3.2 years (79), Parkinson and Bedford (64) under 11 months, Bedford (6) thirteen months, Willius (85) 5.0 years after one attack, 2.5 years after four attacks. Palmer (61) 4.2 years in 65 dead cases, Levine (42) in 1935 said "average duration of life after "recovery is not much more than two years". None of Delrous' cases survived over three years!

There is however ample and adequate evidence of the possibility of considerable longevity after even several attacks, John Hunter according to Ryle (68) lived twenty years after his first infarct, Paul White had (in 1932 (77)) a parson alive and active after twenty years and three attacks; and another patient alive after $17\frac{1}{2}$ years, who climbed mountains at 72, and at 80 years of age could walk five miles; this case died of apoplexy and a firm scar was found in the heart post-mortem.

Conner and Holt (13) also quote a case of survival for 17 years.

Jegorow (34) had a case whose first "status anginosus" was fifteen years previously. 5.2% of Palmer's "survivors" lived over ten years (61).

The longest survival in my cases was ten years, and the patient died in a second attack (case 1). This case of course was discovered post-mortem and the history of the first attack obtained afterwards. I have three other cases alive, two well,

more than five years after attack, and one alive and well four years and eleven months after.

STATE OF HEALTH OF SURVIVORS.

Of the 24 cases here reported who recovered from the attack no fewer than eighteen (75%) come in the category of practically normal life for age after recovery. Of these two are dead, one from recurrence and one from cancer; Four (16.7%) come in the restricted life class, of whom three were equally restricted before, and the fourth died of a recurrence; Two (8.3%) come in the complete invalid class, both were very restricted before, and one of these (case 19) I do not consider the occurrence of coronary occlusion has hastened her lapse into invalidism at all. Thus in all the "surviving" cases where one might have hoped at all for a good recovery, only one has failed to obtain it, and he ultimately died.

These figures compare very favourably with those of others.

Parkinson and Bedford (64) in 1928 had eight cases leading a normal life, 33 restricted, and 25 completely invalid from pain or failure.

Cooksey (14) has 32 "survivors" of whom 78% are restored to their occupations, 24 have no symptoms, eight have symptoms.

Cowan and Ritchie (18) say "The occurrence of an infarct is, "as a rule, followed by symptoms of cardiac failure, and less frequently by attacks of angina.....an infarct if the patient "survives almost always limits the cardiac reserve" Cowan (17) gives the numbers as good health 16, fair 12, poor 4.

Willius (85) reports 169 "survivors" 72 (42.6%) in good health, 39 (23.1%) restricted, 49 (28.9%) anginal, six (3.6%) suffering from congestive failure and in bed, three (1.8%) suffering from cerebral vascular accidents.

Of Strong's (72) "survivors", 55% had a normal life and 45% are in poor health, (17 cases of whom twelve have angina and five dyspnoea).

Palmer (64) of 212 "survivors", says one in four lead active lives, the rest have angina or dyspnoea in equal numbers.

Of Hochrein's (31) 95 recovered cases 36 (38%) are in full work, 38 (40%) are in restricted work, and 21 (22%) are unable to work but as he points out 21 of the cases in the second and third categories are over 65, and would hardly be expected to do full work anyway.

EFFECT OF VARIOUS SIGNS AND SYMPTOMS IN ESTIMATING

PROGNOSIS.

HEART SIZE.

Enlargement occurred in 26 out of the 32 cases in which the size was known. Of these ten have died - five who died suddenly, two in attacks and three "subsequently", of whom two were well till death, and one had a restricted life. Thus all the three "recovered" cases of this series who died later had enlarged hearts.

Sixteen cases with enlarged hearts are alive, of these eleven are well (in three of whom enlargement is only apparent on X-ray examination), two lead restricted lives, and three are invalids, (one being not yet completely recovered from her attack).

Six cases have, or had, apparently no enlargement, this is confirmed by X-ray in two and post-mortem in one.

Thus only three cases out of 32 showed a proven normal-sized heart.

Two out of six, who had probably normal-sized hearts, are dead - both suddenly, (i.e. one-third), all the others are well. Ten out of 24 enlarged heart cases are dead.

Of cases recovering from the attack, eighteen had enlarged hearts, and four normal-sized hearts.

The number of cases with normal-sized hearts is too small for accuracy, but there is no evidence here that they are less liable to die early than the cases with enlargement, though they are possibly less likely to die subsequently.

Of the 26 cases with enlarged hearts eighteen were known to have previous hypertension, and we know that three had no previous hypertension, in the other five it is not known. Eight of the cases with previous high blood-pressure are dead; none of those without are dead; of the doubtful cases two are dead. In two of the cases without previous high blood-pressure the enlargement was diagnosed by radiology. So far as we know none of the cases with normal heart size had previous or subsequent high blood-pressure.

The incidence of enlarged heart is much less in published series. Palmer (59) found it in 64% of "recovered" cases. He also classes unconfirmed cases as of normal size. He points out that congestive failure is commoner with enlargement, recurrence is also commoner, and that enlargement is important in cases with restricted lives.

Cowan and Ritchie (18) report 46 hearts enlarged afterwards in 100 "recovered" cases.

Cowan (17) quotes 57 recovered cases in whom the heart size was known, 34 were enlarged, 23 normal. Of the 34 enlarged ten lived less than two years. Of 23 not enlarged only three lived less than two years.

White and Bland (79) found enlargement in 26 of 33 cases living less than one year, and only in seventeen of 33 cases living over four years after the attack, and believe "cardiac enlargement of considerable degree is a somewhat unfavourable sign".

Levine (1935) in "Clinical Heart Disease" says of prognosis in heart diseases in general, "the larger the heart, other things being equal, the poorer the prognosis".

With regard to the cause of the enlargement, Palmer (59) found hypertension "to be by far the most important, and held it to be the single predominant cause in more than 80%". (My figure is 85%). Coronary artery disease alone led to increase in size in eleven cases (8.6%) of whom four had aneurysm of the heart, three bundle-branch lesions and four had enlargement due probably to chronic ischaemia alone.

Parkinson and Bedford (64) say that "coronary disease alone can cause some hypertrophy, especially of the left ventricle, but unless there is high blood-pressure or V.D.H. in addition it is not a feature".

Nemet and Gross in a discussion of arterio-sclerotic heart disease (56) believe that "in the majority of cases generalized and advanced cardiac hypertrophy is caused by hypertension, present or antecedent, and not by vascular and myocardial damage".

Parkinson (41) in a recent contribution to the Lancet, which discusses various theories and views as to the cause of enlargement of the heart, says "this haemic or toxic origin of hypertrophy is not merely of academic interest, for it has recently gained prominence among those interested in the question of cardiac hypertrophy in coronary lesions" and later:- "Evidence has been collected that hypertrophy may be produced by interference with the proper blood-supply of the myocardium. If that is so, there is theoretically no reason why the same result should not follow coronary obstruction. Cardiac enlargement is known to occur in experimental and clinical anaemia; in cases where an anomalous coronary artery arises from the coronary sinus; and in deficient coronary flow. That coronary disease is actually a cause of hypertrophy has not been conclusively proved!"

Thus in my series the incidence of enlargement of the heart is greater than in other series, but this symptom does not seem to affect the mortality in my cases as much as in those of others. Hypertension would appear to be the

principal cause of heart enlargement in my cases as in others. Three of my cases (16, 27 and 33) would appear to have enlargement due to coronary disease without previous high blood-pressure, in two this has been confirmed radiologically.

BLOOD PRESSURE.

Eighteen of these 41 cases were known to have had hypertension (by which I mean a systolic blood-pressure over 150) before their occlusion; in two more cases 21 and 30 the subsequent blood-pressure was so high as to make it certain that they had previous high blood-pressure; and in another (case 32) the blood-pressure the first time I saw her, a few days after the attack, was 160/100 and subsequently fell further. I think it is fair to include all these as cases of previous high blood-pressure making 21 in all. Of these twenty-one, eight died from the attack, and two subsequently. Of the twelve who got over the immediate attack nine made a good recovery, two are invalids and one leads a restricted life. Two lost their hypertension after their attack.

In twelve cases the previous blood-pressure is quite unknown. Of these, seven died of the attack and five had no hypertension after, of whom one died from recurrence after a restricted life, two lead restricted lives and two have made good recoveries.

Of eight cases known to have had no previous hypertension, one died in the attack (suddenly, age 30, case 22) the others have no hypertension now, all have made good recoveries except one who is still ill.

After recovery from the attack ten out of twenty-five cases had high blood-pressure, thirteen had low or normal blood-pressure, one is still ill, and in one (case 1) no reading exists. Of the thirteen without hypertension afterwards, one died subsequently, two lead restricted lives, and the rest are well. Of the ten with high blood-pressure afterwards, one died subsequently, two are completely invalid, and one leads a restricted life.

Thus the mortality is much higher in cases with previous hypertension or doubtful cases, than in cases known not to have had hypertension. On the other hand hypertension after recovery does not seem to affect the state of health to any great extent. Case 21 who has made one of the best recoveries, has a pressure of 200/110.

Levine (41) found that previous hypertension did not affect the mortality in his series, the mortality-rate in 58 hypertensives was the same as for the whole series.

Conner and Holt (13) found antecedent hypertension in 34% (93 out of 274) of cases, more commonly in the older cases.

Cowan and Ritchie found high blood-pressure (over 150 systolic) in 28 out of 58 cases after infarct.

White and Bland (79) found hypertension past or present, in 50 of 200 cases, of whom 30 are dead after an average life of 2.4 years and 20 alive, with again an average life of 2.4 years. High blood-pressure was only slightly more frequent in cases dying under one year than in cases living over four years. It did not seem to affect the prognosis in his series.

Hay (26) cannot satisfy himself that any reliable inference can be drawn from the blood-pressure findings.

Willius (85) found 74 cases out of 370 where the blood-pressure was hypertensive after recovery. (He gives no figures of how many recovered).

Howard (33) found previous hypertension in 28% of 165 cases.

Cowan (17) found high blood-pressure in eleven out of thirteen cases who survived less than two years, and in twenty-four out of 44 cases who survived more than four years, and considers the prognosis better if the blood-pressure is not unduly high.

Master, Jaffe & Dack (50) say that hypertension, which preceded the attack in 66% of cases, did not directly influence the prognosis.

Palmer (60) analysed in detail the blood-pressure findings in 212 patients who had recovered from coronary thrombosis. He gives a table of the percentage of hypertensives in various series, but some of these are figures for preceding hypertension (e.g. Levine) and some for subsequent hypertension (e.g. Cowan and Ritchie) and they vary from 25% to 75%. His own figure in "recovered cases" is 73% for hypertension "determined by readings made before or at any "time after the attack".

The figure for my series is 52% of cases before occlusion, 40% afterwards. Of the cases without previous blood-pressure readings five had enlarged hearts, one before and four after attack, and if we put these down as hypertensive the figure for my series is 63%.

Palmer (61) suggests in another paper that hypertensive cases are more favourable, probably because the capacity of their coronary system is greater, and quotes Russow as showing that the lumen of the coronary system is greater proportionately to heart weight in hypertensives.

In his paper on "Blood-pressure following coronary thrombosis" (60) he says "the incidence of high blood-pressure in cases destined to get coronary thrombosis is probably not "different from that in coronary arterio-sclerosis as a "whole".

P R E V I O U S A N G I N A .

Of these 41 cases thirteen are known definitely to have had no previous angina, of four it is not known for certain whether they had or not. Twenty-four had previous discomfort of some sort which I have classed in the table as angina pectoris.

Of these twenty-four there were eight where the previous angina was a recent symptom, of not more than three months duration. In all these eight cases it was a true angina of effort, of the eight one died suddenly, one died without recovery from attack, one is still ill from the attack, and not doing too well; the other five were cured of their angina after the attack.

In the case of the sixteen cases with long standing previous discomfort I think it is of some interest to examine them in greater detail. All had at least one year's history.

In case 2 there was a five years' history of genuine angina of effort. He died in the attack.

In case 4 the discomfort was epigastric for over three years, and only four months before the proved attack do we get any complaint of "cardiac" pain. The onset of the epigastric pain was sudden, lasted two days and there was no abdominal tenderness. This may have been a previous attack of occlusion. She still has angina and has had several recurrences of occlusion.

Case 8 had what was probably real effort angina between the time I treated her for "blood-pressure" in 1931 and her occlusion in 1933. She certainly was having angina of effort a few days before her attack of occlusion - but she is not a "good witness" and doesn't give a clear history. After her occlusion she had angina which improved, and she is now practically free from it.

In case 9 the discomfort was of the nature of a "tightness" of the chest on exertion, and was diagnosed by me as bronchitic in nature. It was of two or three years' duration. He died of a recurrence and had angina in between.

In case 11 the discomfort (also of two or three years' duration) was again considered to be due to the patient's chronic bronchitis, it was definitely "tightness on effort." His occlusion cured his discomfort on effort.

Case 12, who had at least ten years of previous angina, almost certainly had had an occlusion ten years before. It is possible, however, that she had angina even before this first suspected occlusion.

Case 19 had, and has, true but not typical angina of effort, both before and since her occlusion, contributed to by a very much weakened myocardium.

In case 20 the relatives give a vague history of pain in his heart region, and of his looking ill and grey on exertion, dating from the time I attended him for a so-called Menieres attack in 1932, till his sudden death in 1935.

In case 22 I have classified as previous angina for two years an irregular dyspepsia, of which no one could find the cause or nature, which was only doubtfully related to exertion, but I don't think the question of its relation to exertion was ever raised until after her sudden death.

Case 24 had twelve months of true effort angina, and died four weeks after the occurrence of occlusion.

Case 28 had a long history of cardiac pain which I have classed as angina, but she had an old rheumatic carditis, and I am not sure that her first real effort angina was not just two months before her occlusion. She is now free from angina.

Case 29 had a real if not very typical angina of effort for sixteen years before his occlusion of 1936. An electrocardiogram in 1932 suggests he may have had a previous occlusion. Since his last occlusion he is better and free from angina, with a lower blood-pressure level.

Case 30 also had real effort angina for two or three years before, and has also been more or less cured by an occlusion and the attendant rest.

Case 32 had the typical Heberden angina pectoris for four years before, and still has it nine months after, occlusion.

Case 36 had a typical long-standing heart-kidney-blood vessel syndrome, with true angina and also other vascular effects. She died of her occlusion.

Case 38 was another of the real Heberdens, his first diagnosed occlusion was his last.

Thus out of the sixteen cases that have been classed as having previous angina of long-standing, eleven cases had undoubted true effort angina, while in five cases the nature of the discomfort is doubtful. In three cases it is possible from the history that the angina originated in a previous undiagnosed occlusion. One other case probably had a previous occlusion, not at the onset of the angina.

No fewer than five of the sixteen long-standing anginas were cured of their angina by the attack of occlusion, one after sixteen years.

Of the thirteen cases without previous angina three died of their occlusion, nine were free from angina after recovery,

(one-case 31 - had angina for months but was cured by later adequate rest), and only one has lasting angina after the attack (case 15).

All except three of the nineteen cases with or without previous angina, who were free from angina afterwards, had what would now be considered adequate rest; of these one (case 1) seems to have been lucky for she had only a few days' rest, case 5 had several weeks' rest in spells, case 31 had no rest for four months, and got angina, but when he did seek medical advice and take adequate rest he was cured again.

ANGINA AFTER RECOVERY.

Only five cases had lasting angina after recovery from occlusion, of these one was undiagnosed at the time but had adequate rest, she was an invalid beforehand (case 4); cases 15, 19 and 32 were also invalids beforehand, leading very restricted lives because of cardiac weakness, leading very (No. 9) of all the cases where a cure of previous angina seemed at all feasible failed to be cured despite adequate rest.

What all this means, briefly, is that in our experience:-

1. Previous undoubted true angina of long-standing occurred in only eleven cases out of 41 having occlusion, of whom five (45%) died of the attack.
2. Undoubted angina of recent origin occurred in eight cases, of whom two (25%) died of the attack.
3. Thus 46% (19 cases) had undoubted previous angina.
4. A further five cases had previous cardiac discomfort, or discomfort that may have been of cardiac origin, not necessarily true angina; and two died of the attack.
5. Of all these twenty-four cases with previous pain or discomfort, nine, (34%), died of the attack and only four had angina after, of whom three were leading very restricted lives beforehand.
6. There was no previous angina in thirteen out of 41 cases of occlusion, of these three died, (23%) and only one had angina after, and she was an invalid before.

Therefore, in our experience the existence of previous angina seems to make the prognosis as to life worse only if it is of long standing, and doesn't seem to influence much the liability to angina afterwards; if patients recover at all they are not very likely to have angina after, especially if their previous angina was of recent origin.

Also, in my experience, occlusion occurs frequently soon after the origin of angina, (in eight cases within three months, and in a further three cases of previous true angina there was possibly a previous occlusion at or about the onset of angina).

When we consider the figures of previous and subsequent angina given by various authors an extraordinary variety of findings is apparent.

All but one of Jegorow's seventeen cases (36) had not only previous angina, but at least one previous attack of "status anginosus".

Paul White in 1926 had 32 anginals in 62 cases of coronary thrombosis. (76)

Parkinson and Bedford (64) found previous typical angina in 45 cases, atypical in eight, "prodromal" angina in nine, (62% altogether), 38, (38%), had no previous angina.

Levine (41) in 1929, said that angina pectoris generally precedes coronary thrombosis, and in 1935 (42) that the great majority of cases of coronary thrombosis had previous angina pectoris of short or more often long duration.

Conner and Holt (43) had 64 cases out of 287 with preceding angina of effort, and 105 with antecedent symptoms of some sort. Twenty-two cases with previous angina made a good recovery and eight of them had no further pain.

125 of White and Bland's 200 cases (79) had angina, not necessarily antecedent. (Angina existed beforehand in 95 of 200 cases (78)), They decide that neither the previous occurrence of angina or its duration afterwards has seemed to matter in the outlook. Angina doesn't affect the prognosis of coronary thrombosis; and vice versa: coronary thrombosis doesn't affect the prognosis of angina.

Carey Coombs (15) who found previous angina in 65-6% of "recovered" cases, considers that it makes little difference to the outlook.

In another, earlier, paper (16) he says that not more than one-third of the patients who develop coronary thrombosis have had previous experience of cardiac pain.

Cotton (26) reports angina following occlusion in 33 out of 73 cases, (45%), and says he has no concrete evidence that the prognosis is graver in angina following occlusion than in other angina, but he suspects that it is. He has never seen a good functional result in angina after occlusion. (but I have - case 31).

Angina occurred before occlusion in 18 out of 57 of Cowan's cases (17), and didn't apparently affect the prognosis.

Cowan and Ritchie (18) show in 100 cases of infarct eight in perfect health without previous angina, twelve more with no cardiac disorder, 80 were frail or had cardiac insufficiency.

Willius (95) reports angina of from two weeks to fifteen years' duration before occlusion in 83 out of 370 cases (24%), 167 had angina afterwards (45%), in 120 it developed afterwards for the first time; 27 cases were cured of angina by occlusion.

Palmer (61) in his 212 "recovered" cases found angina in 39% before, 53% after. Under 10% were cured of angina by occlusion.

I think it is fair to say that most authorities find preceding angina in from one-quarter to over one-half of the cases, and my figures fit in with this. When, however, we come

to consider the incidence of angina afterwards it is present in only five of my twenty-five recovered cases (20%) and four of these were seriously invalid beforehand. This is much less than in other series. Moreover nine out of the nineteen undoubted anginas, and out of twenty-four patients with previous discomfort of some sort, have been "cured" by the attack of occlusion. This is much better than the figures of others.

The reason that the results in this respect are so much better in my series is, in my opinion, twofold, (apart from the possibility that the small size of the series makes it somewhat fallacious). In the first place our practice is mainly residential, and fairly "well-to-do", and so the opportunities of adequate treatment by rest of body and mind are much greater than in some of the published series. In the second place we possibly see a different type of patient suffering from occlusion than do those who draw their cases from consultant and hospital practice. These are not necessarily milder cases, (for it cannot be said that mild cases form a large proportion of this series), but cases which in the normal course of events would not have occasion to have a "second opinion" either in hospital or private, and who, I may say without boasting, might not receive adequate treatment by rest under other circumstances.

THE ELECTROCARDIOGRAMS.

It is as well to consider the changes which are believed to be characteristic of coronary occlusion, before making an analysis of the electrocardiograms of this series.

"The characteristic changes appearing a day or two after the obstruction are as follows: The QRS group is usually notched in at least two leads, and usually shows left ventricular predominance. The T wave does not start from the zero level of the record in either Lead I or Lead III though, perhaps, from a level not far removed from it, and in this lead quickly turns away from its starting point in a sharp curve, without the short straight stretch which is so evident in normal records preceding the peak of the T wave. The T wave is usually of larger size than customary and accordingly shows a somewhat sharper peak. The T wave is usually turned downward in Lead II and in one other lead. Not all of these changes are to be found in every record, but enough of them are present to give it a "characteristic appearance". Pardee (62)

The occurrence of low voltage electrocardiograms was described by Wearn (74).

The development of these changes described by Pardee leads, in the course of a few weeks, in many if not most cases, to electrocardiograms of two main types: T1 and T3, where T is inverted in leads I and II, or II and III and the RS-T interval is displaced in the opposite direction to T. Parkinson & Bedford (64)

These authors do not note alterations in the initial ventricular deviations; a prominent Q3 is often labelled S in their figures.

"In the course of time - it may be months or years - T slowly changes in the direction of normal, and even complete return to a normal upright T in each lead has been recorded". Gilchrist & Ritchie (24)

The presence of a deep Q3 would appear to have been first noted by Levine (41).

This occurred in four cases, and R-T deviation in 52 of 93 cases from the literature, in which post-mortem examinations were made. The three standard leads do not give reliable information as to the site of infarction. Gilchrist & Ritchie (24)

Distinctive changes in the initial ventricular deflections often accompany the characteristic RS-T, and T-wave alterations. In the T1 type there may be a marked Q1 and large R2 and R3, and so there is a resemblance to left preponderance.

In the T3 type there is usually a large Q2 and Q3. In precordial leads there is a large Q, and negative T.

As a rule these QRS changes are more persistent than RS-T and T changes, and may aid diagnosis a long time after infarction. Wilson et al. (87)

Chest leads are of most value in diagnosing infarctions which do not give much evidence in the standard leads especially in the first hours of the attack, and in some anterior infarcts. Wilson (76) Levine (42)

Electrograms taken in four dimensions are universally agreed to be most valuable, and indeed almost essential, if they are to help in sudden or chronic coronary occlusion. Unfortunately it has not always been possible to obtain serial electrograms in these cases of mine.

Electrographic abnormalities may have been present before infarction - subsequent alterations in T may be due to fibrosis, secondary to chronic occlusion. Gilchrist & Ritchie (24)

R-T deviation points to active spread of necrosis, return of the T wave towards normal indicates recovery of function, explained by the development of collateral circulation, etc. Parkinson and Bedford (65)

The possibility of the occurrence of characteristic electrocardiographic changes, especially in R-T segment, in the absence of infarction, has already been noted (8) (22) (82) (69)

Normal electrocardiographic records were found in 26 out of 151 patients who had had coronary occlusion, by Conner & Holt (13) of whom fourteen were alive, seven dead and five lost.

T-wave changes occurred in 109, of whom 37 were alive, 52 dead, and 20 lost. They do not, however, consider the electrocardiogram of much value in prognosis, those with normal records on the whole did best.

Hay (26) considered electrocardiograms of more value in diagnosis than in prognosis - low voltage is unsatisfactory and uncommon in survivors.

23 cases had abnormal QRS, 20 inverted T1, and nineteen inverted T3 out of 45 cases electrographed after attack, quoted by Cowan and Ritchie (18).

Three cases were normal out of thirty cases electrographed by Cooksey (14) at the end of a follow-up study, 27 showed evidence of coronary disease; R-T changes in seventeen, T-wave changes in 25, Q3 in five, low voltage in two.

In fifty cases seen after attack, 32 out of 38 living over two years had abnormal electrocardiograms. In eleven out of twelve living under two years, T1 was inverted in fifteen, T2 in seventeen, T1,2&3 in one. The prognosis is better in the presence of a normal electrocardiogram. Cowan (17)

Mortality was greater in Padilla and Cossio's series (58) in those without typical electrocardiographic changes.

Twenty-four of my cases were electrographed, eleven more than once. In four cases there are electrocardiograms made before the reputed first attack.

In seventeen cases there were changes usually considered characteristic of infarction, or alteration of the changes in successive electrocardiograms. Five more had only a single electrocardiogram. Only one case electrocardiographed in the fourth dimension (i.e. repeatedly) failed to show characteristic alteration.

Changes in QRS occurred in twenty-one cases; in many there was only slight slurring; in ten cases Q waves occurred in one or other lead at some time. Three of these ten are dead, and two lead restricted lives.

Definite R-T shift occurred in eight cases at some time, one is permanently ill and one leads a restricted life.

Thirteen cases had T-wave alterations, -either significant in one electrocardiogram, or changing in serial electrocardiograms. Of these one is permanently ill, and one restricted.

Four cases had R-T shift in one or other lead more than a year after recovery. One of these (case 29) had a greater R-T shift $3\frac{1}{2}$ years before his attack, possibly from an undiagnosed attack. One of these four is restricted, the rest are well.

The only change of prognostic significance would appear to be the occurrence of a Q wave. Half of these patients have had a bad result. On the whole electrocardiograms have not helped much in prognosis.

"The mental status of the patient with coronary occlusion takes unusual prominence in evaluating the prognosis. Early apprehension is the rule. Then having weathered the initial storm the physician must be alert to the reaction. Too frequently the fear of the early phase gives way to a remarkable euphoria, but it must never be misleading, since it is often the calm before the delayed storm. Not infrequently the mental anxiety persists and one is taxed to learn whether it is cause or effect. Certainly it is not unusual to have recurrent thrombosis in this group of patients. Delusionary, hallucinatory and illusionary states may complicate the mental picture and render the outlook more dangerous." Middleton (§3)

This is the only author who, as far as I can find, has referred to this side of these cases at all, and his experience does not agree with mine.

I propose to give where possible a brief note of the mental state of each patient who recovered from the attack.

- Case 4. Cheerful despite complete invalidism and three recurrences.
- Case 5. Cheerful, interested in his heart, but unconcerned.
- Case 6. Quite bright, but careful. More worried about his blood-pressure than his heart.
- Case 8. Definitely over-optimistic.
- Case 9. Quite bright until death, despite recurrence.
- Case 10. Cerebral dementia.
- Case 11. Anxious and unconsolable until death, from cancer.
- Case 13. Quite normal.
- Case 15. Diabetic: cheerful but intolerant of restrictions especially diet.
- Case 16. Cheerful but careful, not worried.
- Case 17. Man of high level of intelligence, mentally very alert, yet apparently quite unconcerned about future possibilities.
- Case 18. Bronchiectatic, a little apprehensive, more about lungs than heart.
- Case 19. Quite bright, though complete invalid.
- Case 21. Quite bright and unconcerned about heart; like case 6 more concerned about blood-pressure.

Case 23. Quite happy and unconcerned.

Case 27. Wonders whether we were not wrong about his heart after all!! Rather worried if any slightest symptom arises, but completely unconcerned while he feels well.

Case 28. Calm and bright, careful.

Case 29. Seems quite unconcerned.

Case 30. A little dull-witted, (normally), but quite unconcerned.

Case 31. Although a very nervous man, in other ways is quite unconcerned.

Case 33. Still ill from attack, but quite happy.

Case 34. Normal.

Case 32. Excessively cheerful, despite severe restriction of life.

Case 37. Garrulous and unconcerned.

Thus it will be seen that a very large proportion of the patients are very bright, a feature which has always struck me most forcibly. It seems to be of similar nature to the "spes phthisica".

Only two cases have been at all unhappy since their recovery. One is Case 11 who was never happy before his attack, and whose heart afterwards was merely, for him, a further peg on which to hang his worries, for the time being; and the other Case 18, who had always been worried about his "chest", which he will never agree to have X-rayed. He has forgotten his heart.

This happy state of most of the patients is I am sure of great aid in preventing recurrence, and aiding recovery.

BLOOD PRESSURE DURING RECOVERY FROM ATTACK.

Much more interesting, and, I think, important than the state of the blood-pressure before and after the attack, is its behaviour in the attack and in the period immediately following, before recovery. It is my experience, and my partners agree with me, that the fall of blood-pressure which follows occlusion of a coronary artery after a longer or shorter interval, is a beneficent feature; and is accompanied usually by improvement in the condition of the patient, and especially by loss of pain. Furthermore, a premature rise towards its previous level of a lowered blood-pressure is accompanied, in my opinion, by deterioration of the patient, by return of pain, and sometimes actually by fresh infarction. This will be illustrated later.

This conflicts with Palmer's opinion (60) that "the behaviour of the blood-pressure during the first month is without ultimate significance," with Hay's opinion already quoted that "no reliable inference can be drawn from blood-pressure findings. If anything, those with lower pressures fare worse": and with that of Middleton (53) who considers maintenance of a low blood-pressure particularly ominous.

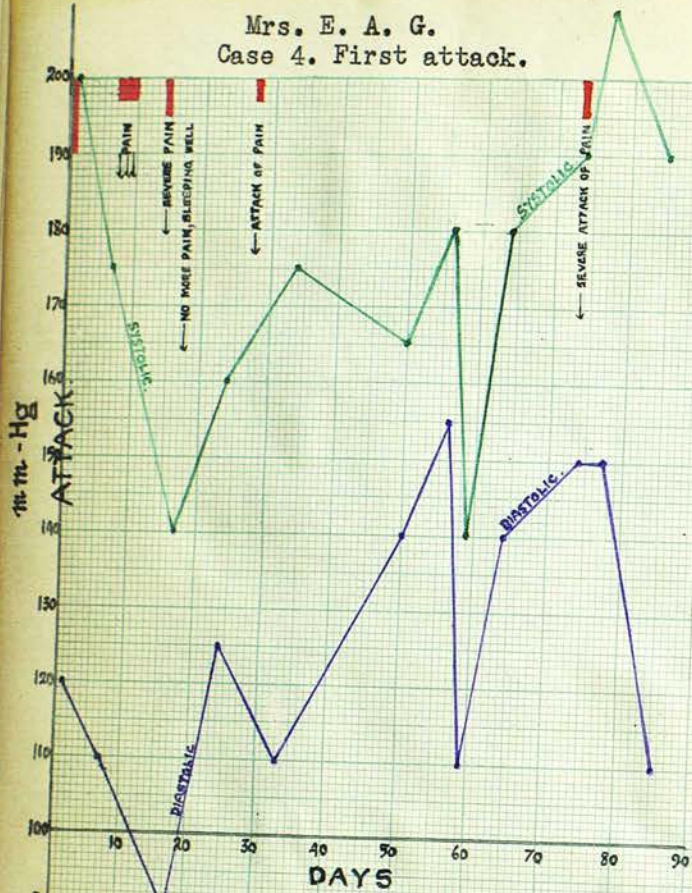
Levine (41) on the other hand says "it follows that a marked fall of blood-pressure is a welcomed sign if it does not rise again to the previous level."

That this fall of blood-pressure is a gradual and delayed phenomenon has long been familiar to me, and I thought it was so to everyone not until I began reading for this thesis did I find otherwise.

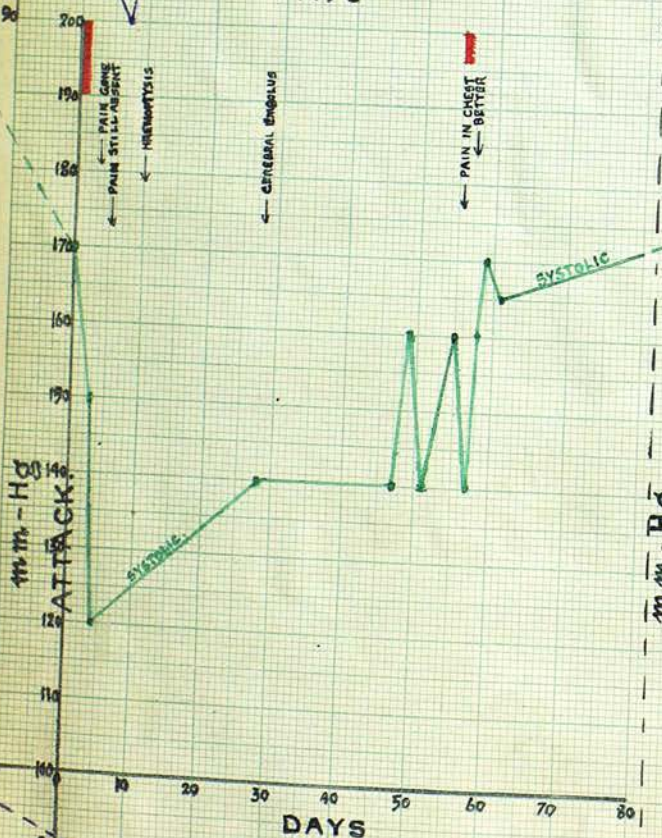
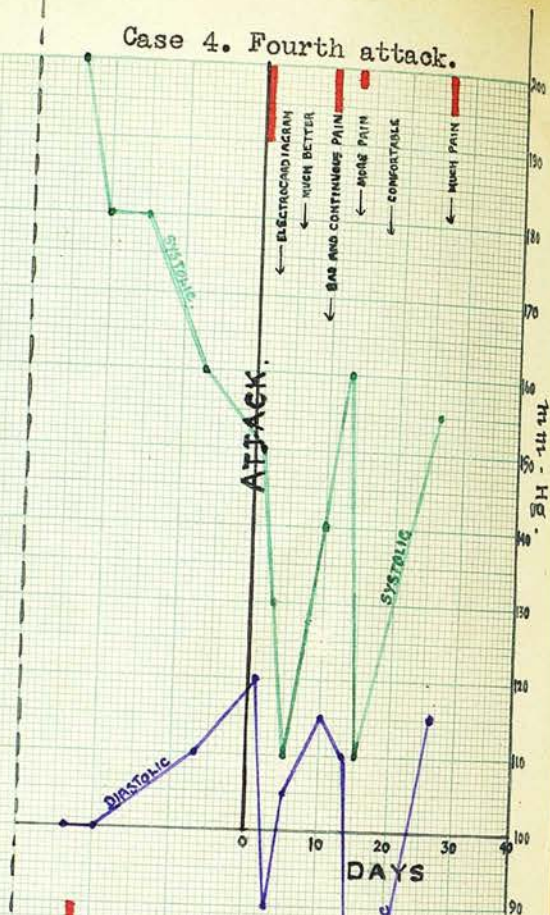
That this fall, when it occurs, is rapid, and due to circulatory weakness and shock seems to be taken for granted by most authors. That this is so in a few cases is undoubted. Levine in 1929 (41) talks of a "gradual fall occurring in several hours" and "when death does not occur.....the fall in pressure may either take place immediately,.....or may gradually fall during the first twelve to twenty-four hours". He does say that "the patients who have done best showed a marked fall".

As recently as 1933 we find Wood saying (88) "In certain of our patients the blood-pressure has been quite high for the first twelve hours, and a drop has not appeared until next day. This phenomenon is probably well known to those who see a number of such patients, but it has not been sufficiently emphasised in the literature".

Mrs. E. A. G.
Case 4. First attack.

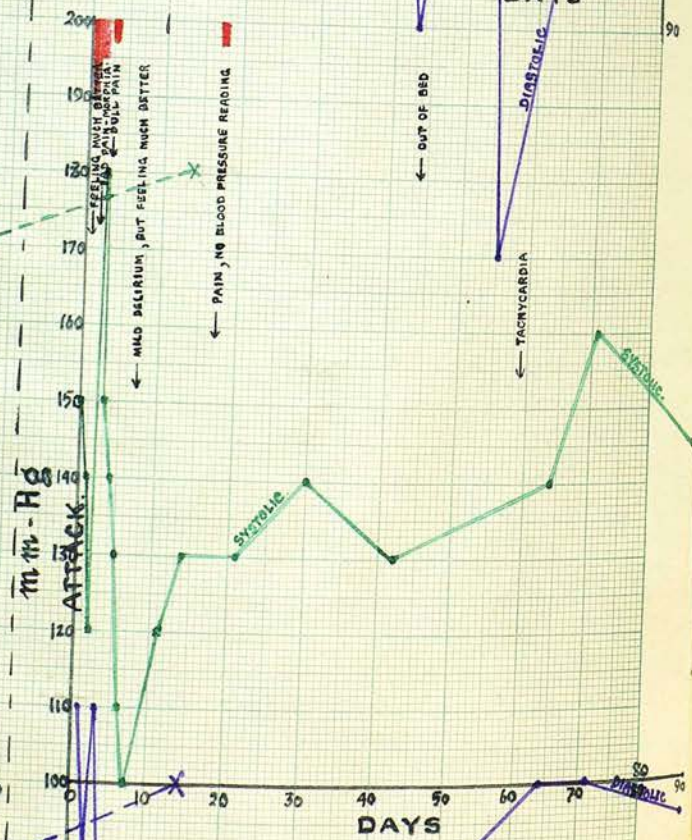


Case 4. Fourth attack.



Case 11.

Sir A. P.



Case 17. Canon E.

Cowan and Ritchie (18) say "In some cases we have observed that the blood-pressure maintains or even exceeds its usual level for a day or two after the occurrence of an infarct. As a rule the blood-pressure falls at once, often to a new level".

Levine in 1935 (in "Clinical Heart Disease") says "The fall occurs in varying relation to the onset of the attack, sometimes the blood-pressure is very low right away, sometimes it is still elevated during the first few hours, and falls subsequently. In the typical case pressure gradually falls although this change may take place over several days". The underlining is mine.

That a high-blood-pressure frequently appears in the early hours after an attack, is maintained for several hours, and then soon gives place to hypotonia is Hochrein's opinion (31)

In a good many of the cases in this series frequent blood-pressure readings were taken. It is interesting to consider them, as I feel that these readings give a clue to the factors which influence recovery and its degree.

These cases also illustrate to some extent the association of return of pain with rise of blood-pressure. It should be remembered that I had not realised this association until the end of 1935, and although many blood-pressure readings are available, there are not so many notes as to the time of occurrence of pain before this date, or indeed for some time after. Much can be gleaned by referring to the old notes, but there are many occasions on which pain is mentioned, yet no blood-pressure was taken on that day - presumably because the patient was in pain. I had taken it for granted that return of pain, while the patient was still in bed, was due to nervous influences, or to further damage to the heart, - which latter in cases where it is followed by a further fall of blood-pressure, and perhaps some rise of temperature, is probably true, - but there were numerous occasions where one realised that there was no fresh damage to the heart.

The first and fourth attacks of case 4 have been set out in the form of a graph of the blood-pressure, with annotations. The course of affairs between her attacks is also interesting. Between the first and second attacks her blood-pressure ran round 200/100, and she had frequent pain. After her second attack the blood-pressure level was from 140-160/100-120 and she had much less pain for six months or so. Then the blood-pressure started to rise again, and after a further five months she had her third attack. Thereafter for seven or eight months she was practically free from pain with a blood-pressure of 160-170/100-110, then her systolic pressure rose to 200 and she had pain again in both arms. This was short-lived, however, and she had another seven or eight months of lower pressure (160/100) - then a rise to 200/100 occurred, with pain, leading, as is shown on the graph, to the fourth attack.

Case 11 is also shown in the form of a graph.

Case 12, a few months before the attack, had a blood-pressure of 180/100. The day after the attack it was 150/80. She had repeated attacks of pain and the blood-pressure gradually fell to 100/60 without relief of pain, and the result was bad.

Case 13 had his attack in the night. At 9 a.m. his blood-pressure was 130/80, and at 11.15 a.m. the same day 90/70, and his pain was gone. In this case the fall was sudden, and the result good.

In case 16 within an hour or so of the attack the blood-pressure was 125/90, in a few days it fell to 90/60. He had pain, however, for about ten days, and he was very ill indeed. A recent electrocardiogram shows marked residual signs, Q1, deep Q3, inverted T1. His blood-pressure is now 115/70, and he has made a very good recovery.

Case 17 is also represented on a graph.

In case 18 the blood-pressure on the first day was 120/80, on the second day 115/80, and the same till the ninth day, when it fell to 90/60. He was quite free from pain after the original attack.

In case 19 the previous blood-pressure was 210/100, on the second day of the attack it was 150/90, and the pain was gone. A month later it had risen to 230/110 and she was having frequent pain, although still in bed. Nitrites relieved this and brought the blood-pressure down to 190/80 and a week later to 180/80.

Case 21 on the day after the attack, had a blood-pressure of 160/100 with no pain. In two or three days it was 120/90, in three months it was 220/210.

In case 23 the blood-pressure fell over two or three days from 180/100 to 140/90. In the succeeding days whenever the blood-pressure tended to rise above this level she had pain. She has made a good recovery and her blood-pressure is now 150/80.

Case 24 when he came to see me the day after his attack had a blood-pressure of 140/90, and was comfortable. The next day his blood-pressure was 170/130, and he was in very severe pain. The next day it was 100/90, and his pain was worse. On the fifth day it was 140/100, and he had bad pain, which did not benefit from trinitrin. He was in almost continuous pain until his death despite large doses of morphia. His lowest blood-pressure was on the fourth day. I should think he had a spreading infarct of the heart.

Case 33 is another very instructive one. Her previous blood-pressure was 140-150/80-90. On the day of her attack,

which was painless, it was 150/80. On the fourth day it was 160/80, and she had much pain on the fifth and sixth days. On the seventh day it was 125/70, and on the eighth day 115/70, and she was more comfortable till the end of a month.

She then had an attack of "status anginosus", on the third day of which the blood-pressure was 155/100. Ten days later came another attack of pain with a blood-pressure of 160/100. Next day her blood-pressure was only 100/80, and the pain was better. Thereafter the blood-pressure remained low. She probably had a fresh infarct here. A low level was maintained for two months then the pressure rose to 130/80, and she had further pain, and a subsequent fall to 95/65.

Thereafter there was a gradual rise till nearly three months had passed from the onset, when it was 140/90, and she had a further painful seizure, with a fall next day to 110/80.

More than four months from the onset her pressure was again up to 140/90, and two days later she had yet another onslaught followed by a fall to 115/70.

THE CAUSE AND ROLE OF THE FALL OF BLOOD-PRESSURE.

The cause of this fall of blood-pressure, which is such a striking and, I believe, beneficial feature of most attacks of sudden coronary occlusion, is the question which naturally arises here.

Most authors, as already mentioned, seem to take it for granted that the fall is a symptom of shock, or of weakness of the heart action; only Levine "welcomes" it. My experience, and that of my partners confirms Levine's view, except in certain rare and very unfavourable cases (such as possibly cases 12 and 34), in which the fall does not lead to relief.

A brief consideration of the physiological considerations involved may help us.

"Stimulation of the vagus, application of cold or heat to the pacemaker, or the injection of any cardiac poison, causes the blood-pressure to fall at once. Clinically we obtain a low blood-pressure for similar reasons when the cardiac musculature is poisoned by the toxins of disease, depressed by cold, or impaired by deficient action of the valves or by fatty degeneration". (McDowall (49)).

"At each beat of the heart impulses apparently pass up from the carotid sinus via the glosso-pharyngeal nerve and from the arch of the aorta via the depressor fibres of the vagus to the vagus centre, which is stimulated, and to the vaso-motor centre, which is inhibited.....The depressor mechanism may be looked upon as a means of safety whereby the heart is relieved should the pressure against which the left ventricle has to pump become suddenly excessive". (loc. cit.)

Whether, after occlusion, the injury to the heart causes reflex stimulation of the vagus, or the poisons formed in the damaged area cause the fall of blood-pressure, or whether the fall is merely due to weakness of the damaged pump cannot be stated without further investigation, experimental and clinical, but my suggestion is that it may well be a beneficial defensive reaction - however brought about - and that it is not due to shock or weakness of the heart.

The disappearance of pain with the fall of blood-pressure, and its return with premature rise may again be of the same nature as the disappearance of angina pectoris when cardiac failure supervenes.

"The explanation" (of this frequently observed feature in cases of angina pectoris) "as Mackenzie has stated, is undoubtedly due to the fact that when myocardial failure develops in these patients, their activities are so restricted on account of dyspnoea and other disabilities that they are unable to exert themselves sufficiently to bring on pain". Keefer & Resnik (38).

This is certainly not true of cases of sudden occlusion as seen by us. After the fall of blood-pressure the patients are certainly not usually "unable to exert themselves" and have frequently to be restrained (sometimes unsuccessfully) from so doing,- the disappearance of pain is not due to inability to bring it on. These facts have always suggested to me that the relief of pain might in some way be due to improvement in the blood supply to the heart, which is just the opposite of what we might expect from first principles.

Conversely the occurrence of pain on restoration of blood-pressure in my cases, (and the occurrence of attacks of coronary occlusion in sleep), might be accounted for by the changes similar to those described by MacWilliam (48) who says "disturbed sleep may be attended by remarkable elevation of blood-pressure.....In view of the rapid development of such changes in sleep.....it is evident that a formidable strain may be thrown on the weak points of the circulatory system, whether these be cardiac with susceptibility to anginal attacks or to ventricular fibrillation and sudden death.....Such rises of general pressure, and pressure in the head would bring into action various normal regulating mechanisms" - (chiefly through the vagus). MacWilliam (48).

In this connection Anrep & Segall (4) later showed that rise of pressure in the head in an innervated heart lung preparation causes decrease in coronary flow.

PHYSIOLOGY OF BLOOD-PRESSURE AND CORONARY CIRCULATION.

Consideration of various views on the physiology of coronary circulation may help us to find the effect of these changes of blood-pressure on the coronary circulation. Earlier work seemed to show that the coronary flow was more or less proportional to blood-pressure, and heart-rate, but independent of cardiac output. Anrep (1)

Later work however shows (1) that a fall in cerebral blood-pressure causes a marked increase of coronary flow, (2) that in the denervated heart the blood-pressure is the only factor which determines coronary circulation, but (3) that "in an innervated heart-lung preparation it is improbable on the basis of these experiments that the increase in the coronary flow accompanying increased cardiac output can be explained by any rise in systolic pressure", (3) that acceleration of the heart rate has no effect on the coronary flow and (4) that there exists a reflex mechanism by which the coronary flow can be adapted to the demands of the heart. Anrep & Segall (4), Anrep, Cruikshank, Downing and Subba Rau in a later paper (2) confirmed that changes in systolic pressure had no effect on the coronary circulation.

Experiments also show that considerable variations in the systolic pressure have very much less effect on coronary flow than small variations in diastolic pressure. A rise of diastolic pressure causes an increase of coronary flow. The maximum flow to the heart through the coronaries is in diastole. (These experiments are chiefly concerned with the time relationship of the coronary flow (systolic or diastolic) and seem to be independent of any reflex effects.) Anrep, Davis & Volhard (3).

Not only do cerebral pressure changes produce these effects, but low blood-pressure in the carotid sinus causes a general depressor effect. Increase in pressure in the carotid sinus causes a fall of aortic blood-pressure, and reduced coronary flow: but if the diminution of aortic pressure and of heart rate is prevented the coronary outflow is still diminished. Changes in the coronary circular similar to those produced by Anrep & Segall (4) can be produced by pressure changes in the carotid sinus. Stella (7)

Hochrein & Keller (32) also showed that reduction of sinus caroticus pressure, and section of the sinus nerve augment coronary flow.

When the heart output is increased by lowering the temperature of the body coronary flow increases regardless of changes in aortic pressure - apparently due to vagal reflexes, as the reactions failed after vagotomy. Rein (67)

Both constriction and dilatation of coronary arteries can be evoked by reflex influences, according to experiments, but the general reactions are in the direction of dilatation.

Wiggers (31) says:- "In fact, to judge from such experimental work the coronary vessels must be kept continuously dilated under the exigencies of everyday life. Teleologically it is difficult to understand the purpose of such somatic and visceral reflexes unless it represents a provision for reducing coronary resistance under conditions (Pain, injury) that tend to lower blood pressures, and which would otherwise reduce the flow through the heart".

Hochrein in "Der Myokardinfarkt" (31) also discusses the physiology of the coronary circulation and says that "From our own experiments all factors influencing blood flow and resistance in the coronary circulation under physiological conditions work together, so that the coronary flow adapts itself immediately to heart output". And also:- "Heart weakness does not, as Anrep assumed, lessen coronary flow under biological conditions, but coronary flow increases with rising output". He claims in conjunction with Keller to have first established the dependence of coronary flow on heart output in the whole animal.

Most experimental work is designed to ascertain the effect on coronary flow of increased demands on the heart, none is on the effect of conditions such as occur after coronary occlusion, but there seems to be sufficient indication in the work quoted that it is possible that reflex influences may lead to an improved coronary circulation in the presence of a marked fall of systolic with a lesser fall of diastolic blood-pressure, such as we get in coronary occlusion.

This may partly account for the improvement of these patients as the lowering of the blood-pressure level occurs, although it is possible, or, even likely, that this improvement is entirely due to the lessened work demanded of the heart in these circumstances.

Conversely, with the rising of blood-pressure in subsequent days, without, perhaps, increased heart output, the coronary circulation may be reduced, and the work demanded of the heart increased and so the recurrence of pain may be explained - for it has been suggested that if you get increased work of the heart leading to increased and unsatisfied demand for blood-supply, pain results, because of anoxaemia of the heart muscle (Danielopolu (19), Katz (37)), brought about by the accumulation of a metabolic product, acid in nature. Katz, (37)

Wenckebach holds that an attack of angina is the result of delayed dilatation of the peripheral vessels from an unduly delayed depressor reflex action (quoted by Cowan & Ritchie (18)). In this case a failure in fall of blood-pressure would produce similar effects to a rise with which the damaged heart cannot cope.

FACTORS DETERMINING PROGNOSIS.

In a consideration of prognosis I feel it is important to discuss the factors which determine what the outlook is to be, and to see if any information can be gleaned from the case-records as to what developments contribute to these factors.

The actual factors which determine the prognosis are probably firstly the satisfactory healing of the infarcted area with formation of a good firm scar; and secondly, probably, the development of a satisfactory collateral circulation to the area affected, but not quite killed off, by the occlusion.

SCAR-FORMATION. Little need be said on the question of scar-formation. No evidence except perhaps that of the sedimentation-rate, is available during life to tell how the process is progressing. It is possible that this is the sole method of healing after infarction, and in cases who lose their angina after infarction "it is logical to assume that the infarction and subsequent scarring has thrown out of function, beyond doubt, the area of myocardium previously responsible for the pain". Keefer & Resnik (38).

"The area of infarction turns into a firmly healed scar and they resume their customary activities". Levy (44)

Gross (25) says however "Nevertheless when vascular obliteration takes place, a certain amount of compensation does occur, so that the infarcted area is smaller than the region supplied by the obliterated vessel".

NORMAL COLLATERAL CIRCULATION. It is equally likely to my mind that following infarction a collateral circulation may develop which improves the nourishment of the fringes of the area affected by the occlusion, and that this improved blood supply, together with the scar, leads to loss of pain.

Of course, whether the development of collateral circulation occurred or not, or was even possible, was for long a hotly-debated question. Recent work seems to have settled the matter in favour of the possibility.

For a long time it was believed that the coronary arteries were true end-arteries, "for when plugged by emboli or thrombi in man or when artificially occluded in animals an infarct results. The rapid necrosis of cardiac tissue could scarcely occur were adequate anastomoses present". Wiggers (81)

Also experimental occlusion of a coronary artery leads to cessation of contraction in the area supplied within one minute. Tennant & Wiggers (73)

In 1901, W. T. Porter (66) wrote as follows: "The objection that one of the coronary arteries can be injected from another, and that therefore they are not terminal, is based on the incorrect premise that terminal arteries cannot be thus injected, and has no weight against the positive evidence of the complete failure of nutrition following closure. The passage of a fine injection-mass from one vascular area to another proves nothing concerning the possibility of the one area receiving its blood-supply from the other. Such supply is impossible if the resistance in the communicating vessels is greater than the blood-pressure in the smallest branches of the artery through which the supply must come. It is the fact of this high resistance, due to the small size of the communicating branches, which makes the artery 'terminal'. This condition of high resistance is really present during life, or infarction could not take place."

Gross in a monograph on "The Blood Supply to the Heart" (25) based on very pretty and delicate injection experiments says "it can be concluded that in the ordinary course of events, and in the average young adult's heart the intricate system of anastomoses are all in active function and are not prepared to act suddenly as entirely adequate compensatory agents."

Gross also discusses very fully the history of varying views with regard to the existence of inter-coronary and extra-coronary anastomoses and decides with his experimental evidence overwhelmingly in their favour.

Wearn (75) reviews the whole subject of collateral channels from the anatomical standpoint. He discusses the existence of extracardiac communications with vasa vasorum of aorta, mediastinal, pericardial and diaphragmatic vessels, and vessels in the hilum of the lung, and especially his own theories of collateral supply from the Thebesian vessels through capillaries and sinusoids to the coronary arterioles.

When critically reviewed, the bulk of experimental evidence supports the view of pathologists that the coronary branches are essentially terminal and that anastomoses are normally of no functional value. Wiggers (81)

These discussions of course refer almost entirely to the healthy heart.

COLLATERAL CIRCULATION IN CORONARY SCLEROSIS.

All the preceding observations apply to normal hearts.

"It must be emphasized, however, that these different experimental proofs that collateral supplies are negligible in normal hearts does not preclude an increase in their caliber in disease or the formation of new systems of vessels which furnish adequate nourishment for the myocardium under pathological conditions. Such assumptions seemed to be necessary to account for absence of degeneration or infarction in instances where one or several coronary orifices were found completely occluded".
Wiggers (21)

"When vascular obliteration takes place, a certain amount of compensation does occur, so that the infarcted area is smaller than the region supplied by the obliterated vessel, the remaining portion receiving sufficient nutrition from the anastomoses. Moreover, if the obliteration is gradual and the circulation good, sufficient dilatation of the anastomosing vessels can occur to preserve considerable, if not all, of the musculature. When the obliteration occurs in a relatively older individual's heart, the patent and free anastomoses, as well as the well-developed arteriae telae adiposae, can often amply supply the affected area so that the myocardium can be completely spared.

"An old heart is therefore much more prepared to receive the brunt of a sudden obliteration of a nutrient vessel".
Gross (25)

"Hearts were examined in which old complete occlusion of the major coronary artery was not associated with corresponding infarction. In other hearts, large cicatrized infarcts indicated remote major trunk occlusion, but canalization of the thrombus and enlargement of collateral channels obscured the original site of obstruction".
Moritz & Beck (22)

The existence of a developed collateral supply does not seem to have been sought for in many of the pathological studies of coronary disease; but Gross (25) describes and illustrates a case with almost completely obliterated right circumflex artery, which had "an ample and abundant anastomosis of large patent rami interventriculares and.....of rami telae adiposae on the right ventricular surface".

The absence of infarction could be explained solely by existing anastomoses between the coronary arteries, in Saphir's pathological series (69). That two main branches at least of the coronary arteries were involved when infarcts were present supported this view.

"It is generally asserted that collateral circuits develop in regions of diminished blood supply because a need for more blood exists. This is a truism but not an explanation. It

"does not follow ipso facto, any more than the need for more money automatically places more in one's pocket. Some other mechanism is concerned in the realization of such results. We believe that the establishment of larger pressure gradients after partial or complete occlusion of a main vessel, at least mechanically, distends normally useless vessels and that such mechanical widening may be the initial step in establishment of collateral flow. This of course is followed by biotactic reactions that lead to development of "new vessels". Wiggers (51)

This conflicts with the experimental results of Beck & Tichy (5) whose experiments showed that vascularization of the myocardium from a collateral bed was slight, and in some experiments almost completely absent, if the coronary circulation was normal. In other words, the blood vessels grow into the myocardium when the latter has need for more blood. These experiments, however, with a prepared vascular bed, have no real relation to what occurs in disease.

Experiments by Wiggers (80) have shown that if the pressure within a coronary branch decreases "the pressure curve changes its contour. The pressure gradients are then such that blood tends to move from the ventricular cavity to the coronary vessels during systole. The pressure gradients are also favorable for a continuous flow from extracoronary to coronary vessels during systole, as well as diastole, but the magnitude of the pressure differences are less during diastole. This also offers a fair explanation for the frequent communications discovered at necropsy in human hearts in which some narrowing of the main arteries apparently exists.

"Summarizing, when pressures in a coronary branch are reduced by narrowing, the pressure gradients are favorable for development of flow through intercoronary or extracoronary anastomoses throughout the cycle but a flow from the ventricular cavity to the artery could occur during systolic ejection only.

"When a main coronary branch is completely occluded, the same pressure relations exist, except that no pressure gradient exists between coronary branches and the ventricular cavity during diastole".

That an increased coronary flow at lower pressure develops after the occurrence of coronary occlusion has been suggested by the study of my cases, and of the known physiology of the coronary circulation.

I wish to suggest, very tentatively, that this reaction may not only improve the blood supply to the heart muscle immediately, but may aid the process described above by which collateral circulation is established.

It is, of course, quite wrong to take results from experiments made under one set of conditions, and apply them to other, and quite different, conditions. The above is the merest suggestion. Further investigation would appear to be badly required.

THE RELATIVE INCIDENCE OF ANGINA PECTORIS AND SUDDEN
CORONARY OCCLUSION.

I have already drawn attention to the relative infrequency in this practice of angina after occlusion, and have suggested reasons for it.

In this connection I should like to point out two facts that have struck both myself and my partners. One is the comparative disappearance of angina pectoris from general practice in recent years. The other is the greater frequency with which we are diagnosing coronary occlusion (on, I believe, good and adequate clinical grounds).

One cannot fail to associate these two opposite tendencies in two undoubtedly related diseases. This leads, naturally, to a consideration of their relationship in general, and in our practice in particular. There is also possibly a relationship to the fact that we have a much lower incidence of angina after occlusion, and a much higher percentage of cases losing their preceding angina on recovery, although we have just as high a proportion of patients who give a history of angina beforehand.

First of all let us consider the relative incidence of these two diseases, or two phases of one disease, in the experience of others.

Paul White in 1926 (76) analysed the 160 cases of so-called angina pectoris in Mackenzie's book on that disease, and considers that eighteen had definite coronary occlusion.

The proportion is 201 anginas to 86 cardiac infarcts in Carey Coomb's experience (1929) (16).

In 1931 White and Bland (79) report a follow-up of 500 cases of angina pectoris and 200 of coronary thrombosis, 130 of the 500 anginas had coronary thrombosis as well, and are included in the series of coronary thrombosis, so the proportion is 370 to 200. (75 more cases of angina might have had coronary thrombosis, which would make the proportion 295 to 275).

127 anginas to 73 coronary thrombosis is the proportion given by Cotton. (26)

An electrocardiographic analysis of 2,000 cases by Willius (84) gives 1,720 anginas and 282 healed infarcts.

Cowan and Ritchie's (18) textbook discusses 200 cases of angina, and 100 of infarct.

The grand total of these series is 2,778 cases of angina and 579 cases of coronary thrombosis, occlusion, or cardiac infarction:- call it what you will.

In the last two months I have tried to find the total number of patients in this practice who are suffering, or have suffered, from angina pectoris in addition to any who are already in this series, while I have been here. It is an extraordinarily difficult matter to find more than a few. The type of patient who goes about with amyl nitrite, or chocolate chewing tablets of trinitrin, in his pocket is fast becoming extinct.

There are, or have been, eleven cases whom I think it fair to class as angina pectoris, one of whom had an undoubted tobacco angina. (Two incidentally have been cured by adequate rest). There are two more who might be added, - one who is a hypochondriac hypertensive who complains of his heart and has a "coronary" electrocardiogram, but who has not typical effort angina by any means; and another who had one isolated attack that was almost certainly anginal. This makes thirteen.

To make the list as nearly comparable as possible to what might be seen in hospital or consulting practice I have extracted from the series of occlusion all cases who had angina and died of their first occlusion, and propose to add them to the list of anginas who have not, so far as we know, had an occlusion.

There are nine such cases of whom one had angina for only two weeks, two had discomfort of doubtful nature, and one possibly, and one probably, had a previous occlusion.

Thus by stretching our figures to the very utmost we get 22 cases of angina pectoris without occlusion, to 32 cases of coronary occlusion.

What is the reason for this extraordinary discrepancy with published figures? In my opinion there are two factors. Firstly, patients with angina of effort are far more likely to seek consultant advice, privately or in hospital, than patients who have an occlusion, very often diagnosed as indigestion, gastritis, pneumonia and so on. Secondly, general practitioners who are on the look out for cases of sudden occlusion have a much better opportunity of watching cases of angina throughout their illness and of diagnosing the sudden occlusions that occur. These are often an early event in the disease and would be missed under other circumstances. I also believe that by diagnosing, and giving adequate treatment to, these occlusions one can "cure" or prevent many cases of angina pectoris, and that we do so.

One case in our practice (case 29) had fifteen years of previous angina pectoris, and possibly a previous missed occlusion, and yet is free from angina since his occlusion and rest.

We have five cases who have made a complete recovery after a sudden occlusion out of eight who had only a short spell of previous angina. My belief is that some at least of these would in earlier days have become typical Heberden anginals - Case 31 started with an undoubted although undiagnosed occlusion, developed along typical Heberden lines and was finally cured, when he did seek medical advice, by adequate rest.

In no fewer than five cases in this small series I found evidence suggestive of a previous occlusion while collecting material for this thesis.

These were:-

Case 1 where I found what I considered conclusive evidence at post-mortem of a previous attack, later confirmed by history.

Case 4 where notes and an electrocardiogram obtained from a teaching hospital together with practice notes, gave conclusive evidence of an attack prior to the one I was investigating at the time. This discovery has of course been treated as the attack for the purposes of this paper. She may even have had one before this.

In case 12 history and notes revealed a probable attack, which may have initiated angina pectoris, ten years before.

In case 20 rather scrappy notes of my own together with the post-mortem findings suggest a possible previous attack, initiating angina pectoris, three years before.

In case 29 there is evidence from notes and electrocardiogram of a possible previous attack nearly four years earlier, not however in this case, an initial attack.

Histories given by the patients in these cases can be amazingly misleading but I feel certain that, if more careful observation in the light of modern knowledge were made of all suspicious cases in general practice, it would be found that angina pectoris, in a large proportion of cases, arises from, or leads very soon to, an attack of the syndrome which we are discussing, and which I have labelled sudden coronary occlusion. The attack is frequently fatal, but if the patient survives and if he is properly treated most if not all cases may have for a time at least complete freedom from angina pectoris.

How does this belief of mine tally with the accepted views as to the cause, pathology etc. of angina pectoris?

THE CAUSE OF ANGINA OF EFFORT AND ITS RELATIONSHIP
TO CORONARY OCCLUSION.

The aortic and nervous theories of the causation of angina of effort need hardly be considered.

"The study of coronary thrombosis has nailed angina "for ever to the tree of decaying coronary arteries". So wrote Parkinson and Bedford in 1928 (64) and nothing that has been written or discovered since has done anything to refute the view that an overwhelming majority of cases of angina pectoris have a "coronary" pathology.

That local or general anoxaemia of the heart muscle, however caused, is the physiological mechanism which produces angina pectoris is the theory that has attracted most attention of recent years. (37) (38) (82) This of course resolves itself in most cases into deficient blood-supply, due to coronary disease, although it accounts also for angina in pernicious anaemia, aortic incompetence and so on.

Angina is due to disproportion between coronary blood flow and the work of the heart, as was first propounded by Danielopolu, (19) who considered that angina might even arise in a normal heart with normal arteries if the demands on the heart were great enough.

This disproportion leads to using-up of the available oxygen by the affected portion of heart muscle. This local anoxaemia explains every characteristic of angina, including the likelihood of sudden death (due to ventricular fibrillation). Keefer & Resnik (38)

"In every case of genuine angina pectoris there is a "lesion that may produce anoxaemia of the myocardium, and "this may account for all the manifestations". (ibid).

"In angina the anoxaemia is relative and transient. "It disappears when the increased work of the heart disappears. "In acute coronary occlusion the blood supply is shut off, and "effort is no longer necessary to bring on anoxaemia" (and pain) (ibid).

Cowan and Ritchie (18) agree with this "There is no "essential difference in the causes of angina and infarct; "it is much a matter of degree, in angina there is "limitation of blood-supply, in infarct, stoppage".

"Coronary thrombosis is related to angina pectoris in "much the same way as an occlusion of a vessel of the leg, "with gangrene, is related to intermittent claudication", wrote Levine in 1936 (42). In 1929 he had said "Coronary "thrombosis is the end result of angina pectoris".



"The relationship is, however, not quite as clear as
"this, for not more than one-third of the patients who
"develop coronary thrombosis have had previous experience
"of cardiac pain; and it is only a minority of those who
"suffer from anginal attacks in whom thrombosis ultimately
"develops". Carey Coombs (16)

However "coronary thrombosis may initiate angina
"pectoris, complicate its course, or prove the fatal
"termination. It is increasingly clear, both on pathological
"and clinical grounds, that many patients hitherto considered
"simply as cases of angina pectoris are actually suffering
"from a past coronary thrombosis. In other words, we are
"witnessing a splitting up of angina pectoris into two
"component parts, which clarifies its pathology. First, the
"group in which coronary occlusion is a gradual process from
"atheroma or syphilis and leads to scattered myocardial
"fibrosis, and in which the clinical history is of anginal
"pain on exertion becoming more frequent and more easily
"induced with the passage of time. Secondly, the group in
"which, at the onset or during the course, an acute infarction
"occurs, usually from coronary thrombosis, and in which the
"history includes one or more of the outstanding attacks we
"have described". Parkinson and Bedford (64)

Out of 112 cases of angina of effort "coronary thrombosis
"was proved to have initiated the condition in two cases, and
"a typical history suggesting that it had done so was present
"in fifteen others. The clinical history of angina of effort,
"in so far as this condition is related to coronary thrombosis,
"may be of one of three types. A coronary thrombosis may
"occur and may be followed directly by angina of effort, which
"may subside, with complete recovery from cardiac pain;
"alternatively, the angina of effort may be persistent.
"Another train of events may be as follows. A coronary
"thrombosis is succeeded by angina of effort which disappears,
"but a subsequent attack of illness of another type - for
"instance, influenza - may result in a recrudescence of the
"angina of effort symptoms. In other cases coronary thrombosis
"may be followed by angina of effort which, although it
"improves very gradually up to a point, never completely
"disappears". Bourne and Scott (9)

I would go further and say that angina pectoris as we
see it is much more often the result of coronary thrombosis.

The conclusion to which I have come after studying our
own cases, and the subject matter of the foregoing discussion
is embodied in the following paragraphs:-

In coronary sclerosis angina may appear as the blood
supply to an area of heart muscle becomes restricted, slowly
or suddenly. The syndrome of sudden occlusion occurs when
more than one source of supply becomes cut off, (Saphir et al
(69)) which happens sooner or later (and usually much sooner).

"When vascular obliteration takes place, a certain amount
"of compensation does occur, so that the infarcted area is
"smaller than the region supplied by the obliterated vessel,
"the remaining portion receiving sufficient nourishment from
"the anastomoses". Gross (25)

After this event the fall of blood-pressure aids in the supply of blood to the damaged muscle, and possibly in the development of collateral circulation, from intercoronary branches and Thebesian veins.

Thereafter one of two things may happen (1) angina of effort results if there is not a satisfactory development of collateral circulation because of the existence of a chronically insufficient blood-supply to a portion of heart muscle surrounding the scar. (2) If there is a satisfactory development of collateral circulation we get the type of case of which Keefer and Resnik (38) said "following the obstruction with its accompanying pain, the angina disappears. Here it is logical to assume that the infarction and subsequent scarring has thrown out of function, beyond doubt, the area of myocardium previously responsible for the pain".

And I think it is equally logical to assume that favourable circumstances - which may include adequate rest - have led to restoration of satisfactory blood-supply to the muscle around the scar, which was not sufficiently damaged to die.

Thus cure of the angina results.

Even before sudden occlusion has occurred adequate rest may produce cure of angina at least temporarily, as it has done in two cases of mine with typical histories. Nevertheless, cure of angina is probably more likely after sudden occlusion with its attendant reactions, provided the patient survives.

Once cure of angina is established the patient is probably safe for a varying period of time as "the processes of growth may conceivably underly the occurrence of occlusion", and "the ramus descendens posterior may, and usually does, lag behind the ramus descendens anterior by a period of ten years; the rate of growth among these vessels varies". Cohn (12)

So not until another area of muscle has its blood supply cut off do we get further symptoms.

S U M M A R Y.

A series is presented of 41 cases of sudden coronary occlusion seen in a general practice of residential type.

The prognosis is discussed.

It appears that the immediate prognosis is similar to that in various published series, but the prognosis in cases that recover from the attack would appear to be better both as regards life and health.

The effect of various factors in estimating the prognosis is discussed.

It appears that the size of the heart did not matter in this series, but that hypertension before attack made the outlook less favourable. These findings are directly opposed to the findings of some others.

The incidence of angina pectoris before occlusion would appear to be of a similar degree to that in other series: the frequency of angina of very short prior duration is pointed out.

Angina Pectoris following occlusion, however, is much less common in this series than in others.

It is also pointed out that angina pectoris apart from occlusion is becoming very rare in this practice.

An effort is made to find the reasons for these apparent anomalies.

A discussion of certain features of the cases is correlated with known physiological and pathological findings and with the findings of other authors.

A suggestion is advanced very tentatively: that with more careful investigation of history, and observation of cases, angina pectoris and the syndrome called here "sudden coronary occlusion" will be found to stand frequently in a relationship to each other, opposite to that which is usually accepted.

It is also suggested that angina of effort might often be prevented or cured by early recognition of attacks of occlusion.

CASE HISTORIES

AND ILLUSTRATIONS.

CASE No. 1.

Mrs. O. Aged 62 at time of attack, 1927.

This lady was a farmer's wife, taking an active part in the running of the farm, when one day in 1927, while apparently in perfect health and having had no illness of any kind for years before, she was seized by a sudden pain in the chest which was very severe. She was so bad that she fell unconscious to the floor. She was seen soon after by her doctor and put to bed for several days. This doctor has now retired from practice but from what he remembers of the case she had a high blood-pressure then.

Thereafter she never complained of any pain or breathlessness until a few days, or a week or two, before her death, when she began to have "rheumatism" across both shoulders and the front of the chest. This was not severe enough to prevent her doing her full work in the house and garden, but in the afternoon of October 28th 1937, she said to her husband that her rheumatism was worse and asked him to go to a nearby shop for a tin of mustard to make a plaster for it, and "to be sure to make it a "large tin". Her husband was gone some ten minutes and on his return found her sitting back in her chair gasping for breath, and she died in a few seconds without speaking to him.

I was summoned and later made a post-mortem examination. The pericardial sac contained an excess of blood-stained fluid. The heart was, to my impression, moderately enlarged. No scar was seen in the myocardium, and no signs of recent infarction, although these findings may be evidence not so much of a satisfactory collateral circulation as of my lack of skill as a pathologist. The coronary arteries were all somewhat sclerosed and the left anterior descending branch was completely obliterated and converted into a fibrous cord for a distance of about one inch from its origin. In the absence of the necessary skill (and the necessary finer instruments) I was unable to reveal a recent thrombus but I had no hesitation in attributing death to sudden occlusion of a coronary artery. It seems certain that the obliteration of the left anterior descending branch dated from the attack ten years before, and that death occurred too soon in the last attack for signs of infarction to develop.

A point of passing interest in this case is that her pain was attributed to "rheumatism" rather than to the more usual "indigestion".

CASE No. 2.

Mr. B. L. Aged 61 at time of attack, September 1930.

This patient was a master builder, who for years had had typical angina of effort which was attributed to "indigestion". Twelve months before, he woke up with a pain in his right arm, and was seen next day by a doctor who could find nothing wrong with his heart, who said his arteries were excellent and made no remark about his blood-pressure, which was taken. The remark was made that if it had been his left arm he (the doctor) would have been more worried. For a few months before his last illness he had had a sensation like an iron band round his chest and a stifling feeling, when he hurried.

On the 12th of September 1930 he had an attack of acute retention of urine. He was admitted to the local Cottage Hospital and a catheter tied in on the 14th. On the 15th his blood-pressure was 150/85, he was very uncomfortable and was bleeding. On the 17th his prostate was removed under a general anaesthetic, and he was apparently doing well when on the 21st he had pain in the right loin and coughed up some blood-stained sputum, and his temperature rose to 99°. Next day the base of his right lung was dull and his temperature was 100°, and he obviously had an infarct of the lung. A few days later he had symptoms suggestive of a cerebral embolus.

On 29th September he had a sudden pain under the middle of his sternum at 4.30 a.m., with breathlessness and very bad colour, and was given oxygen. After this attack of sub-sternal pain which had all the appearance of a coronary occlusion, further questioning elicited the above-mentioned history of "indigestion", coming on in the chest on exertion and after meals, and eased by rest. It then became obvious that the occurrence of emboli on both sides of the circulation was secondary to infarction of the interventricular septum. This infarction had probably occurred during the operation.

He had a further attack of sternal pain and some delirium on 3rd October; on the 6th October he developed a femoral vein thrombosis in his left leg, and on the 17th of October was seized by sudden dyspnoea just as I arrived to visit him and died in a few minutes.

In connection with this case it is of some interest that Professor Mackey of Birmingham in a personal communication tells me he has seen six cases of coronary occlusion after prostate operations.

CASE No. 3.

Miss F. D. Aged 70 at date of attack, 17th January 1932.

This patient was a deaf mute who had never complained of any pain or indigestion before 17th January 1932, although her disability may lessen the significance of this. On that date she had an attack of very severe epigastric pain and when I saw her I diagnosed acute cholecystitis and called in a surgeon who concurred but advised no operation. The pain improved in the next day or two but her temperature rose and she became very ill and by the 22nd her heart showed gallop-rhythm, she was cyanosed, and had oedema of the base of the lungs. I called in a cardiologist in consultation and by the time he saw her she was in auricular fibrillation with a loud systolic murmur all over her heart, her hands and feet were cold, and she was pulseless. He agreed that this was a toxic heart condition secondary to acute cholecystitis and a few hours later the patient died.

I now realise that this was an undoubted example of a coronary occlusion simulating an acute abdomen, especially as her brother and one of her sisters subsequently died suddenly. I have not included them in my series for lack of proof.

CASE No. 4.

Mrs. E. A. G. female. Aged 53 at date of attack, 1st September 1932.

PREVIOUS HISTORY. This patient has been a source of profit to our practice for many years, and is classed generally as a hopeless neurotic. The most important features in a mass of early records are:-

1920, Vertigo, blood-pressure 180/100.

1925, Vertigo, blood-pressure 180/100.

1926, Neuritis left arm and shoulder and side, and cystitis.

1927, Gall-bladder suspected. Investigated in General Hospital, Birmingham, gall-bladder exonerated, aortic systolic murmur then, Wassermann was negative.

1929, 31st January: For two days severe pain across upper segment of abdomen, temperature 100^o, abdomen soft and no tenderness in right iliac fossa or for the matter of that in gall-bladder region.

1929, November 14th, epigastric pain.

1930, July 25th, epigastric pain unaffected by food, marked systolic murmur.

1930, August 13th, blood-pressure 150/120.

1931, April, blood-pressure 200/110,

1931, May, blood-pressure 170/90 (after nitrites).

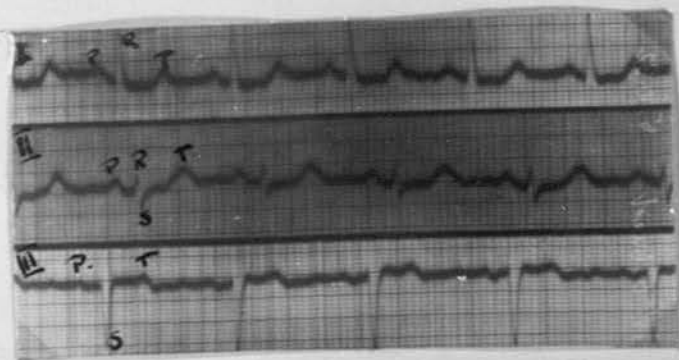
1932, January, blood-pressure 190/100.

1932, April 5th, has been having severe pain in cardiac region, lasts about 10 minutes, pulse 86.

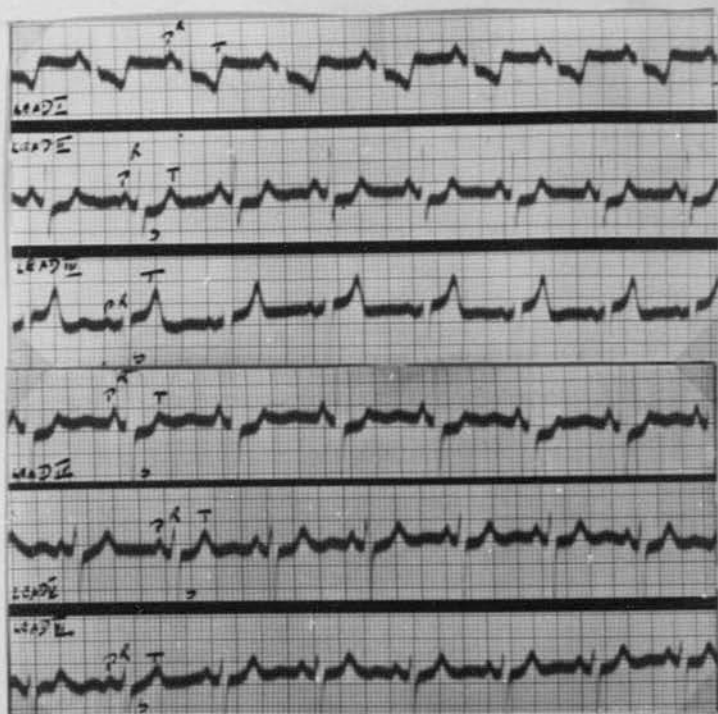
THE ATTACK. On 1st September 1932, patient complained of "sudden pain in sternal region, goes straight into back, in "flexures of arms and palms of both hands". The heart was slightly enlarged, with a systolic murmur conducted over the sternum, blood-pressure 200/120, urine normal. On the 7th, 8th and 9th she had attacks of pain, blood-pressure on 7th was 175/110. On the 15th (two weeks from onset) she had severe pain for 20 minutes going into arms and hands: two days later her blood-pressure was 140/90, pulse 80, heart murmur less. Three weeks from the onset she was seen in consultation by a Birmingham physician and admitted to a large teaching hospital. In her hospital record I find:

24th September	Blood-pressure 160/125, pulse 84.
28th September	Attack of pain.
2nd October	Blood-pressure 174/110.

CASE NO 4. MRS E.A.G.



FIVE WEEKS AFTER FIRST ATTACK.



5½ YEARS AFTER FIRST ATTACK,
2 DAYS AFTER LAST ATTACK.



2½ YEARS AFTER FIRST ATTACK.

5th October	Electrocardiogram (five weeks after attack) shows left preponderance, slurred R1 and S3, depressed R-T1, low origin of S-T2 and characteristic shape of T2, raised S-T3, inverted T3, (opp.)
20th October	Blood-pressure 165/140.
26th "	180/155 (?)
29th "	140/110.
3rd November	180/140.
13th "	190/150 severe attack of pain.
16th "	210/150
24th "	190/110.

She went to a convalescent home and came home in January 1933; on the 25th January she had "much pain in the cardiac region again", and on 27th blood-pressure was 140/100. By 12th April blood-pressure was 200/130.

AFTER HISTORY. Thereafter there is a long story of attacks of pain, of heart attacks, and especially of flatulence. She was almost constantly in bed.

1934: In March 1934 we find "acute attack of angina last night, feet swollen, blood-pressure 200/120" - in April the remark "heart not definably enlarged" - in June, pain - in October, pain.

1935. The tragic story continues through the notes; in March 1935, "four days ago bad sternal pain, 190/100, systolic murmur back again" - in April she has swollen feet and is having "salyrgan". In May of this year (1935) an X-ray shows "dilated heart, enlarged atheromatous aorta" (opp.)

SECOND ATTACK. On 30th May "severe pain in chest all day yesterday radiating down arms and up neck, 140/100, pulse 68, "pale and ill, no pulse at wrist", next day "160/100, much better".

Thereafter blood-pressure ranged; 140-160/100-120 with very few complaints of cardiac pain, although she took to having cystitis.

1936. In 1936 her blood-pressure started to rise again, and on 27th January she had a very severe attack of pain. THIRD ATTACK. On 15th May she had severe pain in precordium, blood-pressure 170/90, and on 20th May there is an entry "? has had coronary thrombosis", this was the first time this diagnosis had been suggested. At this stage my senior partner, whose patient she was, had his own coronary occlusion (24th May 1936 - case No. 27) and I took charge. I also thought that she had had a recent occlusion, her blood-pressure fell to 140/90, and continued thereabouts as long as I looked after her (till September). She was never febrile - nor is there any record of fever in previous attacks. I had her in bed for a further month and then got her up and about. By the time my partner returned to work in September she was going to the pictures for the first time in years.

1936-7. The first time my partner saw her again on his return to work (4th September 1936) her blood-pressure was

200/110!! but thereafter it ran about 160-170/100-110 until May 10th 1937, when there is a note "blood-pressure 200/? "attack this a.m. both arms". For the rest of 1937 blood-pressure was about 160/100 and she was getting about a little all the year.

1938. On 10th January blood-pressure was 200/100 with cardiac pain, on 14th 180/100.

FOURTH ATTACK. On February 3rd she had a severe attack of pain lasting eight hours which started in the night and woke her from sleep, and when seen she was very ill indeed. By the 3rd day blood-pressure was 130/90. On this day an electrocardiogram was made (p. 59). It shows "normal sinus rhythm, rate 83. The limb leads show left ventricular preponderance. The R-T interval is depressed in leads I & II, & slightly raised in lead III. It is also slightly depressed in leads IV & V. The tracing, taken together with the clinical history, suggests the presence of an infarct on the posterior surface of the heart".

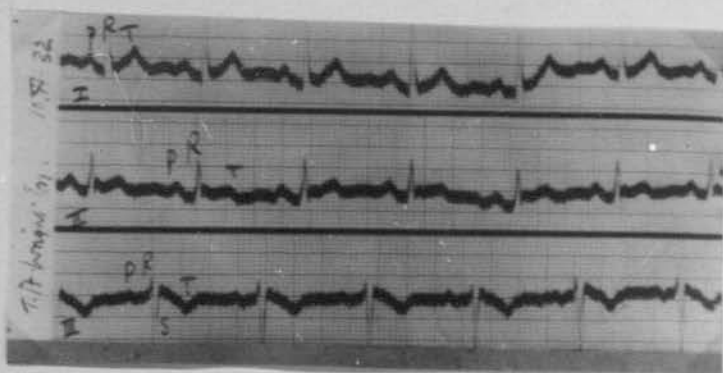
By the fifth day blood-pressure readings were 110/105 and 115/110, heart sounds were almost inaudible, heart very dilated, apex almost in mid-axilla. As on previous occasions no rise of temperature occurred. On the tenth day blood-pressure had risen to 140/115, and she had very bad and continuous pain. Sodium nitrite brought relief. Three days later (16th February) blood-pressure was 160/110, pain recurred. On 18th 110/60, and on 21st blood-pressure unobtainable but patient comfortable. The nitrite was stopped, and on 1st March 1938 blood-pressure was 155/115 with much pain.

There we leave her, very much in the balance, undoubtedly in category 3, a complete invalid.

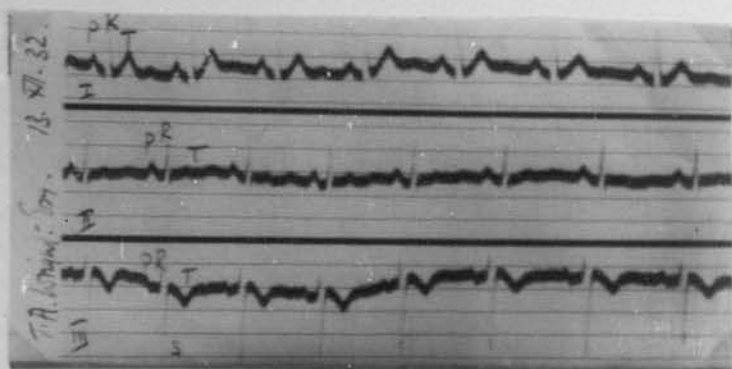
I have given rather full notes of this case because it presents so many points of interest. The earliest mention of pain which might have been cardiac (if we exclude her ? gall-bladder in 1927) is the attack in 1929 of epigastric pain with no tenderness. This may have been an occlusion. It is surprising that as recently as 1932 both my partner - who is a first-rate doctor - and a prominent consultant physician and the staff of a big teaching hospital should all have missed what would now at any rate appear to be an obvious diagnosis.

I would draw attention to the occurrence of pain as the blood-pressure rises, both during recovery from attacks and between attacks.

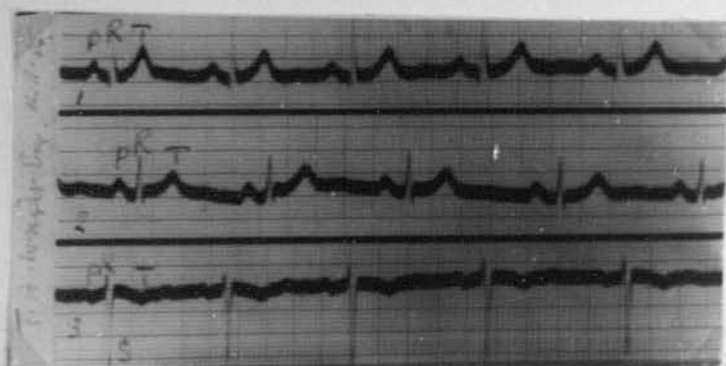
CASE NO. 5 - T.A.W.



ONE MONTH AFTER ATTACK.



THREE MONTHS AFTER ATTACK.



SEVENTEEN MONTHS AFTER ATTACK.



5 1/2 YEARS AFTER ATTACK.

CASE No. 5.

T. A. W. male. Aged 48 at date of attack, 6th September 1932.

This patient is an accountant who prior to his attack had never been ill since he was fourteen years old, but had led an extremely busy and active life up to that time.

On the 6th of September 1932 while on holiday in the Isle of Man he played golf on a cold day, and during the round he became conscious of a pain in his larynx. He continued his game, but the pain persisted, and that evening began to extend down towards his chest. By 11 p.m. his pain had become distressingly severe and he called in a doctor who was on holiday in the same hotel. His description is as follows:-

"The patient had severe pain wholly confined to below both clavicles and upper part of sternum, worse on the right side. He was most restless, sitting up in bed and tossing from side to side. Pulse 70 and reasonably good, heart sounds faint, temperature normal, anxious expression".

Angina pectoris was diagnosed and the doctor gave chlorodyne and whisky and stayed by him until 1.30 a.m. when he was somewhat better.

The patient played tennis the next day, went home in a few days and at his golf club met his own doctor who felt his pulse and reports "he had such a severe fibrillation that I was afraid to let him go home". On the 21st, two weeks after the attack he saw Professor Wynn who found pulse 68, blood-pressure 120/80, no murmur or friction; and ordered one month in bed. He obeyed for three weeks then got up and as he was still feeling seedy he was sent to Professor Wilkinson on 10th November 1932 who reports:- "Apex not felt, heart a little enlarged, first sound soft all over, aortic second sound accentuated, blood-pressure 145/100. I suspect he has had a high blood-pressure, which is borne out by his electrocardiogram which shows left preponderance. He has early retinal arterio-sclerosis, his radials are soft". This electrocardiogram two months after attack, is the first of three of which copies are shown. It shows some upward shift of R-T segment, and is of the T3 type of Parkinson and Bedford (65).

The patient had a further two weeks in bed, and saw Professor Wilkinson again on 13th December 1932, three months after his attack, when he was much better but still giddy on sudden rising from a chair. The electrocardiogram was better, the R-T shift having gone; T2 was flatter.

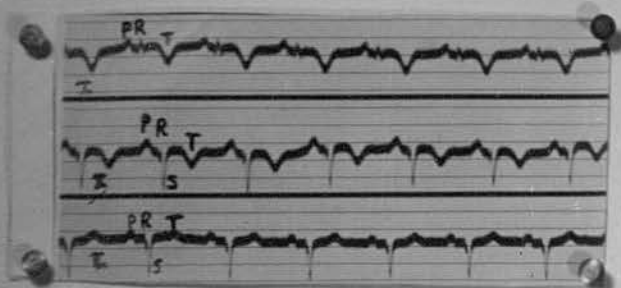
Since his recovery from the immediate effects of his attack this patient has felt (and looks) perfectly well, except for occasional spells of slight fatigue. He takes life more easily and does not work after the afternoon. If tired he stops work, but he plays golf and has a full social life. He has never had any breathlessness or pain or oedema since his attack. He had another electrocardiogram done in December 1934 as a precaution, this shows return of T2 to the upright position and flattening

of the previously inverted T3.

In August 1937 he felt a little off colour and tired, but his heart was apparently normal to auscultation and percussion and his blood-pressure 140/100, his ankles were possibly just a shade puffy, his urine was normal, exercise tolerance was good.

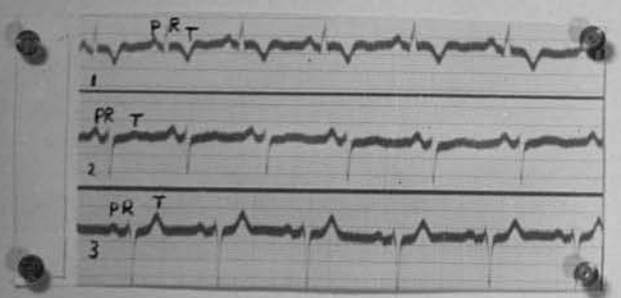
On 18th February 1938 I examined him and found him feeling well and apparently in perfect health. His heart sounds were normal, his apex was impalpable, I made the left border $3\frac{1}{2}$ inches in 5th space on percussion, blood-pressure 150/100, I made him step up on a chair six times which raised his pulse from 60 to 72 but it returned to 60 in 20 seconds. I asked him to have his heart X-rayed and as will be seen from the photograph it is slightly enlarged. (p. 61)

I place this patient in my first class, as to all intents leading a normal life for his age. The cardiac enlargement is interesting, - either he had a previous hypertension and is one of the cases whose hypertension is cured by a coronary occlusion (cf Levine (41)) or else he is one of the unusual class of patients described by Palmer (59) who have enlargement of the heart as a result of coronary occlusion. The only evidence of hypertension is his left preponderance but Pardee (62) describes this as "characteristic of changes appearing a day or two after "the obstruction".

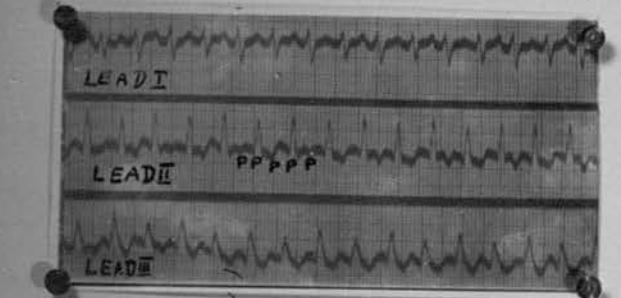


SIX
WEEKS
AFTER
ATTACK

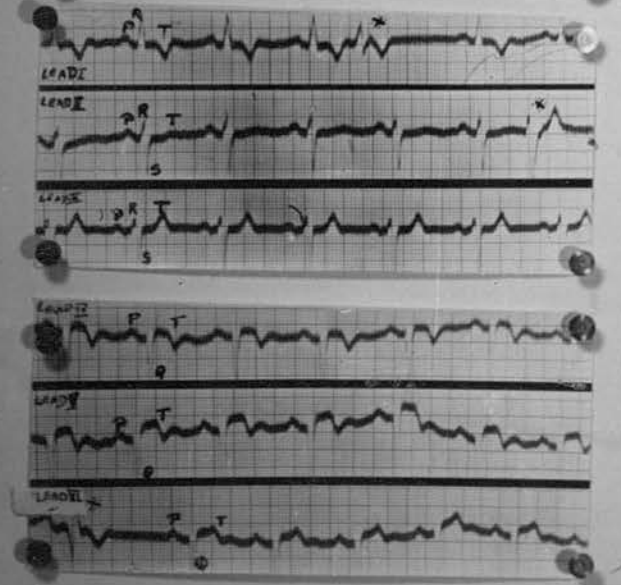
CASE No 6
J.C.



1 1/2 YEARS
AFTER
ATTACK



3 1/4 YEARS
AFTER
ATTACK
(FLUTTER)



5 YEARS
AFTER
ATTACK



CASE No. 6.

J. C. male. Aged 69 at date of attack, 11th September 1932.

In 1910 this patient, a lawyer, had been advised to go easy because of high blood-pressure. This he had done ever since, only going to his office for half days and so on. On the 11th of September 1932 he climbed a ladder to adjust a rhone-pipe on his house, which had come loose. He was seized with violent pain across the front of his chest, and he came gingerly down the ladder and went indoors and lay down. The pain did not go so I was sent for. When I saw him he looked extremely ill with a harassed expression, and I fetched the district nurse and with her assistance carried him to the bed on which I ordered that he should lie undisturbed for three weeks. His blood-pressure was 140/90. I made a vague diagnosis of a "heart attack", but next day his temperature was 99.4° and on the analogy of infarction of the lung I said he had a clot in a coronary artery with death of a part of his heart. His heart apex was then visible beating under his left nipple, in the 4th space, 4 inches from the mid-line, he had gallop-rhythm and a diastolic murmur at the apex. Next day his temperature was 100° and remained about there for 3 or 4 days. After three weeks in bed his systolic blood-pressure was 170, and he was allowed up and got about gradually with very little distress. On 21st October 1932, six weeks after the attack, I sent him to a cardiologist. His blood-pressure then was 150/100 and he still had a diastolic murmur. His electrocardiogram then is of the T1 type with notched and slurred low voltage QRS1 and the cardiologist reports "severe myocardial defect and a bad prognosis".

He went on very well until December when he had a slight set-back more, I thought, nervous than real, but thereafter he required no medical attention until February 1934, up till which time he had felt well but done very little, and he wanted to know if he could do more. His blood-pressure had risen a bit but his heart sounds were closed, and I sent him for another electrocardiogram. This shows considerable improvement (or at least regression of signs) QRS1 is bigger, but still notched, T2 is now small and upright, T3 is higher and more pointed.

He was told to do more and again went for nearly two years without medical advice, attending to business part-time, but avoiding physical exertion. On 6th January 1936 feeling that a new year had started and that he had completely regained his youth he was misguided enough to get into bed with his wife, and immediately started the most violent palpitation which refused to yield to rest and he had to send for me. When I saw him he was obviously in auricular flutter with a ventricle rate of 180.

I sent for a consultant whom I had recently found to own a portable electrocardiograph, and the diagnosis was confirmed.

The attack failed to yield to massive doses of digoxin, pressure in the neck had no effect, quinidine was tried, then intravenous digoxin, various consultants were called in, but despite all efforts the ventricle rate was never below 180. At last I advised his people that nothing more could be done and apart from giving him 0.25 mgm. of digoxin each day in the faint hope of his heart responding some day he was best left alone. He gradually sank, his lung bases became moist, he lapsed into a state of semi-coma with muttering delirium and I said that even if his heart did revert to normal rhythm his brain would be irretrievably damaged. Day after day I went to visit him (with the death certificate more or less in my pocket) and day after day as I entered the house I heard the low running mutter of delirium upstairs, until at last 9 weeks from the onset I went in one morning and there was quiet. I went upstairs "to view the body" and found my patient sleeping quietly and peacefully with a pulse of 70 per minute.

He woke up sufficiently to be told that he had been near to death and had miraculously been cured and then went off to sleep again. Next day when I called his right side was paralysed and he couldn't speak.

His cerebral lesion fortunately cleared up in a few days, and in a few weeks he was off my books again.

Since then he has had two brief attacks of flutter, in September 1936 and July 1937, which yielded rapidly to digoxin (or got better on their own despite digoxin) but otherwise he has been remarkably well considering his history. Of course he is, and looks, a frail old man who makes no physical exertion at all, but he goes to business and takes short walks and lies down when he is tired, and never gets any pain.

In November last he sent for me because he had been having some giddiness and faintness and when I saw him (6th November 1937) his blood-pressure was 180/105, his heart could be felt at $3\frac{1}{2}$ inches from the mid-line in the 5th space, and at 5 inches in the 4th space, he had an accentuated second sound all over, his pulse was 72, he had no dropsy. His arteries were much thickened. He was having slight breathlessness on exertion. He was going to business.

I took the opportunity of sending him for a further electrocardiogram. His limb leads are substantially the same as in 1934. The chest leads show a Q-T shift, and prominent Q-waves suggesting recent fresh myocardial damage, but he didn't show any other signs of this and has gone on well since. An X-ray at this date shows aortic sclerosis, a photograph of an orthodiagram made then is shown. (p. 63).

I made a further examination on 15th February 1938, and found blood-pressure 185/100, and heart apex in anterior axillary line (5 inches) in 4th and 5th space - so perhaps he had further myocardial damage after all, but he seemed well, with no pain or breathlessness.

This patient seems to me a perfect demonstration of the amount of insult the human organism, and especially the heart, will submit to without succumbing. I place him in class two, leading a restricted life.

CASE No. 7.

W. H. male. Aged 58 at date of attack, November 1932.

One cold morning in November 1932 I was called urgently from my round to the local Hall, and on arrival I found the chauffeur, W. H. lying dead on the garage floor. He had come down to the garage from his house upstairs immediately after breakfast, pulling on his coat as he came down the stairs, and had fallen dead with his coat still half on.

Two or three weeks previously he had consulted one of my partners because of some pain in his chest, angina pectoris had been diagnosed and he had been warned to go carefully. His blood-pressure then was 175/100.

On post-mortem examination I found a moderately enlarged heart showing no myocardial fibrosis or degeneration but the aortic valves were thickened and the aorta showed typical syphilitic aortitis. The orifices of both coronary arteries were almost completely occluded.

CASE No. 8.

A. E. T. female. Aged 57 at date of attack, April 1933.

PREVIOUS HISTORY. On the 26th February 1931 this patient who was a cook, consulted me because of palpitation. She had a persistent tachycardia, her blood-pressure was 170/90, her heart very fast, and she had a systolic apical murmur. She had three weeks rest in bed and was much better thereafter.

THE ATTACK. On the 30th of March 1933 she consulted me again because of precordial pain on exertion. Her heart then showed a curious triple-rhythm, her blood-pressure was 160/80. The urine was normal. I arranged for her to leave her situation and go to a friend's house for several weeks in bed. A few days later when I called on my daily visit I was told she had had a very bad attack of pain in her chest in the night, which had made her sit up in bed and roll about. I diagnosed coronary thrombosis and admitted her to the local Cottage Hospital. Unfortunately the hospital records have been lost but I know that her blood-pressure fell, that she had a rise of temperature for about a week, that her pain was very persistent, that she had also persistent tachycardia, and that I kept her in bed a full two months, and that she was ten weeks altogether in hospital.

AFTER HISTORY. Four months after the attack (14th August 1933) she had still tachycardia, with blood-pressure 180/100 and unable to undertake any exertion. Two months later, 4th October 1933, blood-pressure was 170/100, the heart sounds were normal, the urine contained a haze of albumin but no sugar and she was still seriously incapacitated. I told her she would never work again, and advised that she go to live with a relative on the other side of Birmingham.

When I decided to write this thesis I wrote to her, having heard nothing of her in the meantime. I was pleased and surprised to get a reply saying "I will willingly let you examine me any time you like I have not been to the Doctor's for eighteen months, I go out to work every morning I am much better than I was when you saw me last".

On 24th October 1937 I wrote again saying I should call to see her in a few days, and the next day which was bright and sunny she decided to call on me, and I was delighted to see her walk into my surgery.

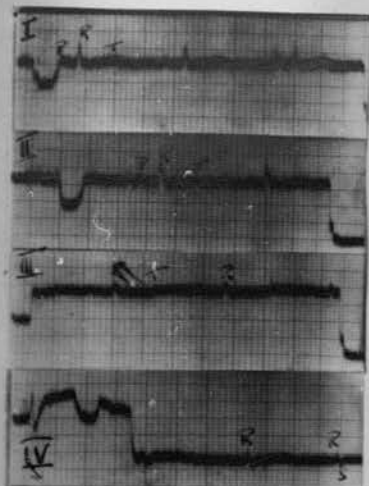
She told me she had steadily improved since she left the district and had been going out "charring" every morning for two years. She got some slight breathlessness and pain across her chest, and occasional palpitation - only with violent exertion, which she avoided.

Her blood-pressure was 210/120, pulse 80, the heart apex was palpable 4 inches from the mid-line in the 5th space, and the first sound was reduplicated in mitral and tricuspid areas. Her retinal arteries showed slight sclerosis and radials marked

thickening. The urine showed a very slight haze of albumin, no sugar, and microscopically a few hyaline casts were found.

On 21st January 1938 at my request she saw Dr. Brenner, a cardiologist, at his hospital, he suggested that her pain now was not typical of angina as it occurred apart from exertion. X-ray of the heart showed slight general enlargement, the electrogram showed myocardial defect, low voltage and flat T in limb leads. (see p. 67)

This case is of interest as showing the great degree of progressive restoration of function possible in a heart severely damaged by infarction, even many months or years after the original attack.

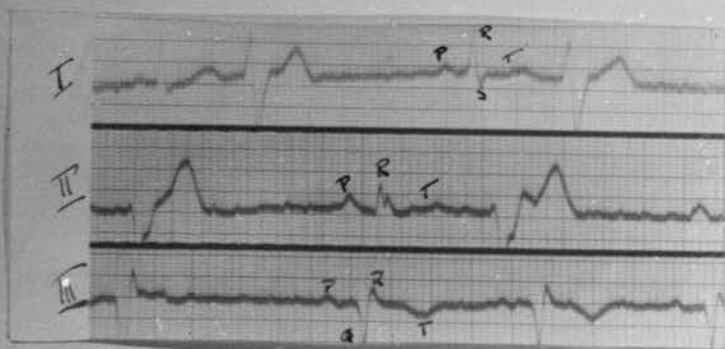


Case No. 8.
A. E. T.



4 1/2 Years After Attack.

Case No 9 H.B.



10 Weeks After Attack.



2 1/2 Years After Attack.

CASE No. 9.

H. B. male. Aged 46 at date of attack, 1st December 1933.
Occupation, Golf Club Steward.

PREVIOUS HISTORY. Patient had had symptoms which I attributed to a mild degree of chronic asthma associated with infected antra.

THE ATTACK. On 1st December 1933 he was shopping in Sutton Coldfield when he was seized by a severe pain in the chest, the pain continued for several hours but he went home and carried on with his work, and as pain was still present in the evening I was sent for. I am afraid I missed the diagnosis completely at first. I attributed it to indigestion and kept him in bed, and when in a few days he had a temperature of 102° I was considering the possibility of typhoid when the correct diagnosis was suggested by a partner whom I called in. His blood-pressure was then 140/90. He was kept in bed for six weeks, and then allowed up and sent about a week later for examination by a cardiologist, who confirmed the diagnosis. His blood-pressure was then 150/100, the electrocardiogram showed, as can be seen, flat T1 and T2, inverted T3, notched QRS2, low origin of S-T1 and high origin of R-T2. (ppp.) His Wassermann reaction was negative.

AFTER HISTORY. Thereafter patient took things very quietly for the rest of the year. In November 1934 nearly a year after his attack he was electrocardiographed again. I have no copy of this but it showed improvement and he was allowed to start a little putting. He had felt fairly fit in the interval, and he now started to do more. I saw him casually in February 1935 and he was having occasional pain on exertion, and carried trinitrin tablets, but was doing all his work.

In October 1935 nearly two years after his attack he played in a golf competition and won first prize, although he had pain during the round and had to stop several times - and was very tired afterwards.

In March 1936, 2 1/3rd years after his attack, he had a severe illness which came on while I was away, and which was apparently either pneumonia or a pulmonary infarct, and was associated with spitting of blood. A week after the onset he had a severe pain in the chest. When he got up again he had attacks of pain in the chest and down the left arm, especially after meals, which were relieved by trinitrin. On 18th May 1936 I had a cardiologist out to see him. His heart was then enlarged, blood-pressure 150/100, an electrocardiogram showed prominent Q3, broad and notched R2, S-T1 and S-T2 slightly depressed. Q4 prominent broad slurred and notched, no R4 or S4 - myocardial damage, chiefly due to the old infarct, no certain evidence of a recent infarct. (ppp.)

Thereafter the patient always had occasional precordial pain radiating down the left arm, especially after meals; he always carried trinitrin tablets and used them most days.

In August 1936 he had another slight haemoptysis and was kept in bed for a few days. He was X-rayed in October and general enlargement of heart with an atheromatous aorta revealed.

On 25th March 1937 he was suddenly seized with a severe pain in his chest. I saw him inside half an hour and he was obviously desperately ill with coronary occlusion. I gave morphia and three hours after went back to see how he was and arrived just in time to see him die.

When this patient was well he led a practically normal life but he had two illnesses which kept him in bed during his three years and four months of life after his first attack.

CASE No. 10.

Mrs. J. female. Aged 76 at date of attack, 3rd December 1933.

PREVIOUS HISTORY. During the early part of 1933 I attended this lady because of symptoms such as dizziness due, I considered, to hyperpiesis. Her blood-pressure at this time was 180-200/80-90, the heart was enlarged, she had no angina. Her memory was failing.

THE ATTACK. On the afternoon of 3rd December 1933 I was summoned in a great hurry and on arriving at the house was told that the patient had been seized by a very severe pain in the chest, and had fallen to the floor and become unconscious. She was coming round when I saw her and I helped to get her to bed, - the pain was a little easier. I made a diagnosis of coronary thrombosis and gave her morphia. The diagnosis was amply confirmed when in the next few days her blood-pressure had fallen to 120/80, and the temperature had risen to 99.5°, to about which level it rose nearly every evening for a week.

By the end of the week she was desperately ill with congestive failure, with oedema of the lungs and of her legs and lower part of her back. She went gradually into a state of muttering delirium, and when everyone had decided that she was going to die she began as gradually to improve, and by the end of two months was getting out of bed and wandering round the room whenever the nurse's back was turned for a moment. The blood-pressure rose to 140/80, she was very confused mentally and difficult to handle, and at the end of February 1934 she was allowed up officially.

AFTER HISTORY. I was gradually able to decrease the frequency of my visits until I was seeing her only weekly and then monthly. She had, however, to have a companion constantly with her.

Her blood-pressure remained about this new lower level 140-150/80, and she became more and more difficult in memory and in temper and no one at all could control her paroxysms of rage. During 1935 and 1936 I was in constant and regular attendance, both on the patient and on a succession of companions, acting both as doctor and as mediator. Despite her colossal rages she never seemed to have any cardiac pain.

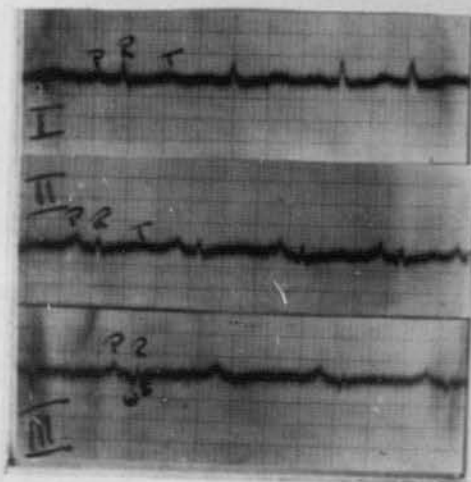
In the spring of 1936 her husband died, and, as her family all lived at some distance and didn't want her, I had her admitted to a home for elderly invalids. There she lives now, with practically no memory, apparently quite happy and only very seldom in a rage. I visit her monthly. She gets about well and goes upstairs as briskly as a woman of 40, without any breathlessness. When, occasionally, she runs away from her nurse in the garden she gets some shortness of breath but no pain. On 3rd August 1937 when she was 80 years old her blood-pressure was 140/80 pulse 72, heart apex 4 inches from mid-line in 5th space. She had slight oedema of the feet. She had had two slight attacks of chest pain in about six months. The urine was normal.

Case No 10 MRS. J.



4½ Years After Attack.

Case No 11. Sir A.P.



4½ Months After Attack.

On the 2nd February 1938 her blood-pressure was 150/80 and heart apex $4\frac{5}{4}$ inches out in 5th space. On 7th February 1938 I had an electrocardiogram done which shows, as will be seen, normal sinus rhythm, rate 75, QRS slurred in all leads. T2 and T3 inverted and of "coronary" type. (opp.) The cardiologist suggests that this probably indicates an old infarct on the posterior aspect of the heart.

The most interesting points in this case are the amazing degree of functional recovery of the heart after such a severe attack, and the re-establishment of a previously hypertensive blood-pressure at a new and non-hypertensive level.

CASE No. 11.

Sir A. P. Aged 74 at date of attack, 15th January 1934.

PREVIOUS HISTORY. As far back as 1924 this patient, a Black Country industrialist, had breathlessness on exertion, was overweight, and had a blood-pressure of 200/100, a faint first sound of the heart, and no palpable apex-beat. Thereafter he had constant hypertension, in 1927 he had epigastric discomfort, his blood-pressure was 220/100, the heart sounds were faint and heart dullness 2 inches outside nipple. In May 1931 his pressure was 200/80, and "he was able to play golf better than for a year". During the next few years he began to have tightness across his chest "when he walked up the hill to the second hole", his blood-pressure was never below 200/100, he had an attack of bronchitis each November and had constant crepitation at the base of his lungs.

All his life he had had "bilious attacks" for which he used to take calomel.

In January 1934 the poor man was beset on all sides by troubles, the slump was affecting his business very badly, his only son was rapidly sinking into melancholia, because his wife had left him and was bringing a nullity suit against him, and one of his daughters was divorcing her husband and had come with her son to live with him. It was not surprising, therefore, that something should give way.

THE ATTACK. On the 16th January 1934 when I called at his house to see his grandson I was told that "father had a bad attack of indigestion last night with pain all across his chest, he was sick but he took some calomel and he was all right this morning and went to business". Next day I was sent for and found him lying in his chair by the fire in great pain and looking pale and anxious. He had been to business and the pain had come on again on the way home. He had been sick. His systolic pressure was 170. I put him to bed with morphia and a tentative diagnosis of coronary occlusion, to be confirmed by events. By the morning of the 19th (four days after onset) his temperature had risen to 99.5°, and his blood-pressure was 150/90 and the diagnosis seemed certain. His pain was gone and he felt much better but I asked for a second opinion. He was very ill indeed that night when the consultant came, with pulse 100, temperature 100°, blood-pressure of 120/80, the heart sounds were almost inaudible, there was a pericardial rub in 3rd and 4th spaces beside the sternum, he had profuse basal creps. He was able to talk, and his pain was still absent.

His blood-pressure remained low, and his temperature stayed over normal for four days, but he slowly recovered. On the tenth day he spat up some blood and had a rise of temperature to 99° for two days. During the second week he showed also various degrees of heart block, pulsus bigeminus, trigeminus etc. A month from the onset (February 14th) he had a cerebral embolus, he was aphasic, and later in the day lapsed into a low delirium; his

blood-pressure was 140/90. Next day his temperature had risen again to 99° and he was better; and his speech was completely recovered in two or three days.

His heart sounds were always faint and the heart size couldn't be made out because of emphysema. His arteries were much thickened.

During March his blood-pressure began to rise again, on the 5th it was 140/60, pulse 80, 7th 160/70, 9th 140/80, 13th 160/80 with a note that he was having occasional pain in chest, 15th 140/? pulse 80 feeling better, 16th 160/90, 17th 170/90, 19th 175/80.

AFTER HISTORY. About this time he was getting up for an hour or so (two months from onset). Thereafter he gradually got about, his blood-pressure rose to 180/100, and by May he was walking half a mile to a mile a day, and having no chest pain; his legs stopped him before his heart did!! (intermittent claudication). On May 31st I took him into Birmingham to see Dr. Brenner who found blood-pressure 230/100, pulse 100, (this increase was due to sheer funk!) and made an electrogram which showed low voltage ventricular complexes, and T flat in all leads. (p. 72)

During the summer he became able to lead quite a varied quiet life, and to go to business several mornings a week, but his mental state was pitiable; he required constant reassurance that he was improving and not in danger of his life and he seemed quite incapable of making any decisions about anything.

On 20th August 1934 he was seized by pain in the upper abdomen and lower chest, was sick twice, and in the next day or two his blood-pressure fell again to 130/80 and temperature rose to 99.5° for two days. At the request of his family and because of his mental anxiety I attributed his attack to biliousness and with great difficulty managed to keep him for a month in bed.

However the results were good, and by October 3rd he was able to go with me to Birmingham to see Professor Parsons again. His blood-pressure then was 170/110, and Dr. Parsons thought him remarkably well, and agreed that he might be allowed to take on some of his County Council committee work, but forbade his other hobby - which was golf.

During this winter (1934-5) he went to business four (and when I wasn't looking, five or six) days a week, and once or twice a month went over his beloved main roads and bridges with the county surveyor. He had no pain or tightness across his chest but occasional breathlessness, his heart was seldom audible, his blood-pressure lay always between 160 and 180 systolic - never more, and 90-100 diastolic. He was happier but still always a little frightened.

He continued in much the same state throughout 1935, requiring constant medical reassurance, which was somewhat trying, even if profitable. Despite my protestations his blood-pressure must be taken at each visit, even though I never told him the answer and

blood-pressure was 140/90. Next day his temperature had risen again to 99° and he was better; and his speech was completely recovered in two or three days.

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He continued in much the same state throughout 1935, requiring constant medical reassurance, which was somewhat trying, even if profitable. Despite my protestations his blood-pressure must be taken at each visit, even though I never told him the answer and

he wouldn't have believed me if I had! Actually if I took it before he rose in the morning (usually on Sunday) it was always 160-170/90, and if I saw him after a morning at business it was 170-180/100. His one constant plaint was "if there is nothing wrong with me why won't you let me play golf?" To which my invariable reply was that I had never said there was nothing wrong with him, but that he was quite as fit to lead a normal, quiet and useful life as many men of his age - which was true.

On December 31st 1935 he had a third attack, similar in every way to the second but if anything slighter. This time I refused to tell lies, and told him he had had another and slighter attack of his old trouble and that there was no reason at all why he should not make as complete a recovery. He took it very well indeed and seemed happier and more at peace afterwards than he had been for two years. He stayed in bed for a month without any fuss and during this period I noticed that when his blood-pressure first rose to 140-150 it was associated with a return of precordial pain - this made me refer to his old notes when I realised a similar thing had happened after his first attack (*vide ante*). I have noticed this in other cases and it is discussed later.

After this attack he was very much better in every way, fitter than he had been since I got to know him (six years before) and in summer of 1936 he played mild golf without detriment. He still required frequent visits and blood-pressure readings however, and the latter were always as before, 160-180/90-100. In the early winter however his anxieties and miseries began to return, he lost all his appetite and his weight began to go down. His heart condition seemed unchanged, and finally in December a lump appeared under the ribs on the right side in the gall-bladder region. From then on he went steadily downhill, developed signs of cerebral degeneration and died on 11th February 1937, three years and a month after his first attack of coronary occlusion - from carcinoma of the gall-bladder.

He never had any angina after his first attack, and before it his "effort angina" was more tightness across chest than pain. The pain during recovery associated with rise of blood-pressure is interesting, as was the establishment of a new and lower blood-pressure level after his first attack.

CASE No. 12.

Mrs. K. female. Aged 67 at date of attack, 16th February 1934.

This lady, the wife of my senior partner, had had a nephrectomy twenty-five years before and had been a chronic invalid ever since, largely because of the presence of residual infection with discharging sinuses.

In June 1923 she was overweight, had a blood-pressure of 165/88, and a slightly deficient urea concentration, albuminuria, and pyuria.

In February 1924 after an attack of angina-like pain her blood-pressure was 130/85. Thereafter blood-pressure slowly rose until in 1930 it was always about 180/100. In October 1931 she had epistaxis and blood-pressure 200/110 and had developed a mitral murmur, in June 1933 blood-pressure was 180/100. She had sugar in her urine for a short while in 1929.

During all these years she was having angina of effort, and her companion-nurse always carried amyl nitrite.

THE ATTACK. On 11th February 1934 she went into the bathroom and was seized with violent substernal pain and thought she was going to die. The pain only lasted a few minutes. For the next few days she went about having pain at intervals. On the night of the 15th pain came on very severely in bed and lasted all night. Next day her temperature had gone up to 99°, her pulse was 86, blood-pressure 150/80, thereafter as the days passed she had repeated pain, and the blood-pressure fell to 100/60 and temperature rose to 101.4° on 21st. She was more or less febrile thereafter and had repeated attacks of pain. At the end of March congestive failure appeared, and on 6th April she developed pneumonia with a steadily rising temperature and died on 8th April - two months after the attack.

Professor Wynn, who had looked after her for many years, on looking back suggests that she had had a slight coronary thrombosis in 1924. Of interest is the delay in fall to its lowest level of the blood-pressure, although in this case there was continued pain, and a bad result.

CASE No. 13.

F. B. male. Aged 61 at date of attack, 7th April 1934.

This patient, a company director, had never had any illness of any consequence until 1934. On the night of 6th April 1934 he was awakened by severe pain in chest radiating to both shoulders. When seen at 9 a.m. he had distant heart sounds, blood-pressure 130/80, pulse 100, no rise of temperature, moist sounds at the base of the left lung. At 11.15 the same day his blood-pressure had fallen to 90/70, and his pain had gone.

Next day he had pain on deep breathing only, blood-pressure 125/85, heart sounds faint, pulse faster when he moved.

Three weeks after the attack his blood-pressure was 135/85, pulse 80, liver not enlarged, heart normal in size, no pain, moist sounds both bases. A week later blood-pressure was 140/85 (3rd May 1934). On 27th May, seven weeks after the attack, he got up; his blood-pressure was 120/80. A month later it was 120/80, first heart sound was soft, heart not enlarged. On 8th July blood-pressure was 140/80, he still had râles at both bases, he was allowed to play a little golf, exercise tolerance was very good.

Since then he has been perfectly fit, does a hard days work, spends full time at business, plays a full round of golf. His urine has never shown any abnormality.

In January 1938 his heart was still not enlarged clinically, blood-pressure 140/70, pulse normal, no myocardial insufficiency, no pain.

I put this patient in the first class, living a normal life for his age.

CASE No. 14.

H. W. male, aged 65 at date of attack ? 1934.

This patient, who had not complained previously, began to have pain in the chest one afternoon while at business, which continued all afternoon and evening (he was working late). He went home at night by train in continuous pain, but was able to walk to his house from the station, a distance of a mile. When he got home he went to fetch some brandy, and fell dead on the floor.

Post-mortem showed widespread aortic and coronary atheroma, and thrombosis of the main trunk of the left coronary artery.

He had not consulted a doctor previously. He had one brother under constant medical care because of high blood-pressure and nephritis, and another brother who has symptomless high blood-pressure.

CASE No. 15.

Mrs. C. Aged 57 at date of attack, 2nd May 1934.

PREVIOUS HISTORY. Patient had her gall-bladder drained in 1926, and again in 1931. At the time of the second operation sugar was found in her urine, and while in the nursing home she had a very severe attack which nearly killed her. This I gather was an embolus of the lung, as the lower lobe of her left lung is permanently moist all over. She had breathlessness and pain on exertion ever since.

THE ATTACK. I was called from a cinema on the night of 2nd May 1934 to see this patient, whom I had never seen before. She had been seized by very severe pain across the chest and in the right arm and left hand, and had vomited, without relief to her pain. She looked extremely ill, pale, with anxious expression and very restless and demanding relief. She reeked of acetone, her urine was loaded with sugar. Her heart sounds were very faint and her heart much enlarged. Blood-pressure 135/90, pulse 130.

I installed a nurse and put her on copious carbohydrates and insulin, and she made a rapid recovery. Slight exertion in the early weeks used to cause pain in the left hand. I kept her six weeks in bed by which time her urine was sugar-free, and her blood-pressure was 150/100 and she was allowed up. A week later blood-pressure was 130/70, heart much enlarged, breathless on exertion.

AFTER HISTORY. About the end of July, $2\frac{1}{2}$ months after the attack I allowed her to go on holiday, although she was still unable to undertake more exertion than a short walk of half a mile. While away she had an attack of cardiac asthma and was very ill indeed, and as soon as she could travel was brought home in an ambulance and kept in bed at home. Again she made a good immediate recovery and got about gradually, and I visited her occasionally throughout the winter of 1934-5. She steadfastly refused to graduate her activities, and would do far too much one day and be laid up for days after, and as result her heart reserve remained very small, and she could only take short walks without breathlessness and pain.

In the summer of 1935 however, she did improve, and was able to walk a mile or two, and to do a morning's shopping in Birmingham. During 1936 and early 1937 I saw nothing of her.

In August 1937 she was away on holiday and had an attack of pain which was diagnosed as gall-bladder trouble. Her urine contained sugar and she was put on a low carbohydrate diet, with the result that by the time she was brought home on 15th August she was desperately ill, full of acetone, no sugar in her urine, and in more or less constant precordial pain. Her blood-pressure was 130/80 and she had severe pain in both hands. I again got a nurse in and filled her with glucose and insulin. She did well and got about again. She had one

recurrence of severe pain one afternoon when the minister called in the afternoon and delayed her tea. She had had a large dose of insulin before lunch and I considered the attack of angina to have been brought on by hypoglycaemia (cf. Smith (70)).

In October 1937 she had a further severe attack of epigastric pain while I was on holiday, and this was again attributed to gall-stone colic, but I think was angina. She soon got over it however, and was off my books by the beginning of November.

At the end of November however, she began to have sciatica badly and sent for me on 23rd November 1937. She had been breaking diet and her urine was full of sugar, her blood-pressure was 120/80, heart apex $5\frac{1}{2}$ inches out in 5th space, no oedema, left lung as usual full of creps. She was able to walk a mile without precordial distress, although of course her sciatic nerve complained.

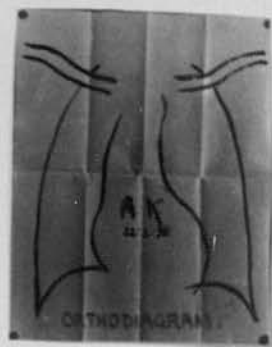
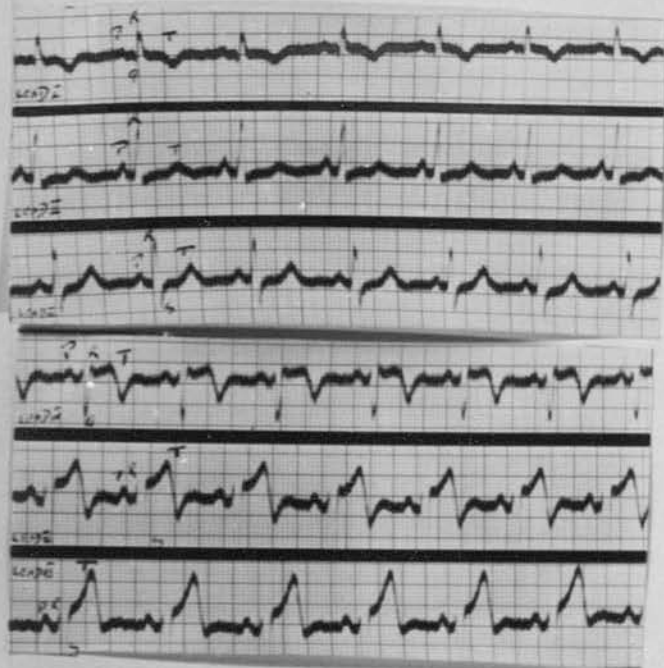
Diet and small doses of insulin cured her sciatica and she has dieted carefully since with the result that on 14th February 1938 she looked and felt remarkably well, and was able to walk several miles. Her heart was smaller, apex $4\frac{1}{2}$ inches in 5th space, soft first sound, blood-pressure 125/75.

This patient goes in category 2, leading a restricted life. She gets no real angina of effort now. Her activity is limited rather by breathlessness.

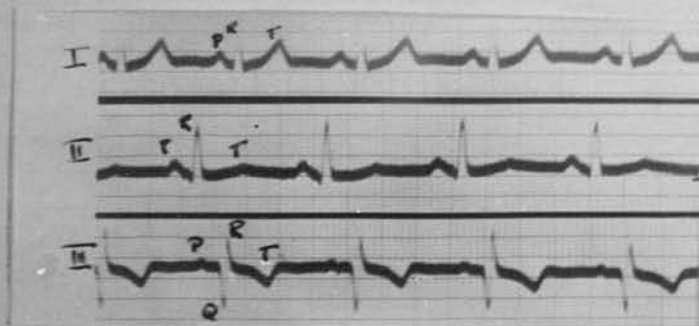
She has steadfastly refused to have her chest X-rayed and I have never dared suggest an electrocardiogram!

CASE NO. 16 - A.K.

3½ YEARS AFTER ATTACK.



CASE NO. 17. - CANON E.



3½ MONTHS
AFTER ATTACK.



2 YEARS
AFTER ATTACK.

CASE No. 16.

A. K. male, aged 53 at date of attack, 6th August 1934.

PREVIOUS HISTORY. Was entirely negative. He had never had any pain or indigestion before his attack. He had never had any more serious illness than influenza. Twelve months before he was passed as an A 1 life for insurance purposes and presumably therefore did not have high blood-pressure then.

THE ATTACK. By a curious coincidence, the next time I went to the same cinema as in the previous case I was again called out, this time to this patient. He had been playing tennis in a competition when he started having a pain in his chest, this became so severe that he had to stop playing, although he tried to finish his match, and he had quite a difficulty in getting to his house which was only a short distance away. When I saw him he was very pale and anxious looking and was rolling about on the bed, complaining of severe pain across his chest radiating into both arms. His heart sounds were very faint. His blood-pressure was 135/90 and I made an immediate diagnosis of coronary thrombosis. This was confirmed during the next few days when his blood-pressure fell to 90/60 and he ran a temperature, never above 100°. He was more or less in pain for 11 days and was in bed altogether for 9 weeks. When he first got up he used to have pain coming on in spasms brought on especially by emotion, but this soon improved. For a further 6 months he had slight pains after meals or on exertion, but since then he has been very well indeed, and now only gets occasional pain after a meal and does not get pain on exertion - although he never undertakes any violent exertion. He does, however, cut the lawn and clean his own car.

On the 13th December 1937 I examined him, his blood-pressure was 115/70, I felt his heart apex in the 4th space 3 inches from the mid-line, the first sound was short and soft, otherwise his heart sounds were normal. His exercise tolerance was very good; after getting up on a chair six times his pulse rose from 72 to 96 and fell again to 84 in 20 seconds and 72 in a minute. His radial arteries show slight sclerosis, his retinal arteries are normal. He has a curious appearance in that the region of the edge of his liver is marked by numerous small veins; this was certainly not present at the time of his acute attack and rather suggests that some collateral circulation is developing in this region.

On the 22nd February 1938 an electrocardiogram shows normal sinus rhythm, rate 75. There is slurring of the QRS complex in all leads. A Q wave is present in lead I, and there is a very deep and slurred Q in lead IV. The T wave in lead I is inverted and of the "coronary" type. The origin of the T wave is slightly raised in all the chest leads. In lead IV, the T wave is inverted, in lead V it is diphasic. These changes are

characteristic of an old infarct on the anterior aspect of the left ventricle. The orthodiagram shows a long narrow chest, with a low diaphragm. There is marked increase in the curvature of the left lower arc, indicating the presence of considerable left ventricular enlargement.

This patient obviously goes in category 1 leading a normal life.

CASE No. 17.

Canon E. aged 54 at date of attack, 6th November 1934.

This patient holds a very important post in the Church of England and during the latter part of 1934 was having an extremely trying and strenuous time, as in addition to administering his own very large charge, he was doing a large part of the work of his Bishop who had broken down in health. With regard to events leading up to his attack, which he had whilst staying with friends in this district, I cannot do better than quote from the diary which he himself prepared for me.

"In November 1933 a medical examination for insurance disclosed sugar in the urine, which the examining physician thought was serious. I was at once put on a diet without any sugar or starch at all, and in a few days (I think four days) the sugar had completely disappeared. After that I had frequent tests and the results varied from nil to considerable quantities. My own doctor decided on a diet from which sugar and starch were almost entirely eliminated, and I observed this diet fairly strict during 1934.

"In August 1934 I had a holiday by the sea, and was more than usually active physically, swimming a good deal and walking.

"Very soon after this holiday, i.e. in September 1934, I began to have noticeable pains in the left arm spasmodically. At first they came on in the evenings, or after I went to bed. By lying on my left side I got almost immediate relief and was able to sleep. The pains, however, increased in frequency and intensity, and I remember particularly that during the Leeds Musical Festival in the second week of October the pain was practically continuous. About October 15th I was examined by my doctor, who thought the pain in the left arm was neuritic. The blood-pressure was low - I believe the figure was 90" (actually his doctor tells me it was 130/90-G.R.M.) "and he ascribed this to fatigue and under-nourishment, and suggested that I should have a fuller diet.

"Immediate preliminaries to thrombosis.

"Sunday, Nov. 4. Had pain in chest after mid-day meal (hot pork). Thought of indigestion.

"Preached in the evening: went to bed usual time (10.30). Had sharp pain in chest in bed, got up and had a whisky and water, went to bed and went fast to sleep.

"Monday, Nov. 5. Had fairly full day till evening. Attended Luncheon Club and ate pork again. Had no pain during the day, but had meanwhile arranged with ----- that I should come down

"on Tuesday, November 6th, for a few days' rest. Had return of the pain, similar to the previous night, after I had gone to bed. Again drank whisky and water and went to sleep.

"Tuesday, Nov. 6. Went by train to Endwood. No pain. Ate usual dinner in the evening, went to bed about 10.45 and slept immediately. Woke up some time between 12.30 midnight and 1 a.m. with very acute pain in chest. Not wishing to disturb the household, I spent some five hours in a vain attempt to get relief from the pain, lying in various positions, getting up and walking about, went downstairs three times during this period to try and find whisky. Not finding any, I took some brandy (not much) and returning to my room presently fell into an exhausted sleep and was still sleeping when I was called at 7.30 a.m. Woke without any pain at all, but feeling a little exhausted.

"Wednesday, Nov. 7. Got up for lunch, feeling free from pain until about 3.30 then dull pain for about an hour, felt feverish, doctor came at 6.30, said almost sure thrombosis, gave injection to give a good night's sleep. No pain during night due to drug?" (His blood-pressure on this evening was 150/110, the injection was morphine gr. $\frac{1}{4}$.—G.R.M.)

"Thursday, Nov. 8. Got up for lunch, dull pain again in afternoon but felt better and went out in car for two hours" (against my wishes.—G.R.M.) "very fatigued and drowsy. Doctor came in evening, very worried because temperature was only up to 98.4° but he felt sure in spite of that, that it was heart and not indigestion." (On this date blood-pressure was 130/90 in morning but 180/110 in evening.—G.R.M.)

"Friday, Nov. 9. Due to return to Leeds. Slight pain in back and chest in early morning, before early tea. Got up for lunch, had difficulty to get dressed after bath, was very fatigued and in a sweat when I came downstairs, after packing bags. Doctor came to give consent to journey or otherwise - temperature up to 99.6° so said definitely thrombosis - went to bed, lying flat, lunch from feeding cup etc. very fatigued, slept during afternoon $1\frac{1}{2}$ hours. Specialist at night." (Blood-pressure was 150/110 and temperature 100.2° in evening.—G.R.M.)

On the 10th his blood-pressure had fallen to 140/90, on the 11th it was 130/80 in the morning, and in the evening 110/85, his temperature was 99.2° and on this day sugar appeared in the urine. On the 12th (five days after his attack) his blood-pressure was 100/70, his temperature was 99.2° and he had two short spells of mild delirium, but felt much better. Thereafter he improved. On the 17th his blood-pressure was 120/80 and his temperature 98°, 20th his blood-pressure was 130/80. On the 25th he had some pain in the evening and his temperature was 98.8°, on the 27th blood-pressure 130/90. He never had any pain after this. On 5th December blood-pressure 140/80, 17th December 130/80. On the 19th December he got out of bed. On the 4th January 1935 he had an attack of tachycardia. On the 9th his blood-pressure was 140/100 and on the 16th 160/100.

He stayed on at Streetly under my care, and on the 21st February, $3\frac{1}{2}$ months after the attack, he had an electrocardiogram done, which showed tall T1, flat T2, deep Q3, inverted T3 with slightly raised origin of R-T3. (The T3 type of Parkinson and Bedford (65)). (See p. 81) His blood-pressure on this date was 145/95.

He took things very quietly until April and went back to work six months after his attack. In the following winter, the gentleman with whom he had been staying when he turned ill, died, leaving a large and complicated estate, and the Canon, being one of his Executors, had a great deal of extra work to do in this connection. In addition to his ordinary Church work he undertook it all willingly and cheerfully and seemed to be none the worse. During 1936 he continued to do the work of two or three men until on December 13th 1936 I was asked to go to Leeds to see him as he had had some chest pain on the previous day. I agreed to see him in consultation with his doctor in Leeds, and when I examined him could find nothing abnormal clinically, his blood-pressure was 140/90, his pulse was a little fast, 90 per minute, his pain had been short-lived although it had come on without exertion. I advised caution, that he should stay in bed and that a local consultant should be asked to take an electrocardiogram. The following day his doctor decided that he was so much better that this was unnecessary and he came South again to Streetly for a rest, and I had an electrocardiogram done while he was here. This shows no evidence of recent damage, but still a deep Q3, flat T2, T3 is still inverted but flatter, T1 is less tall, (see p. 81)

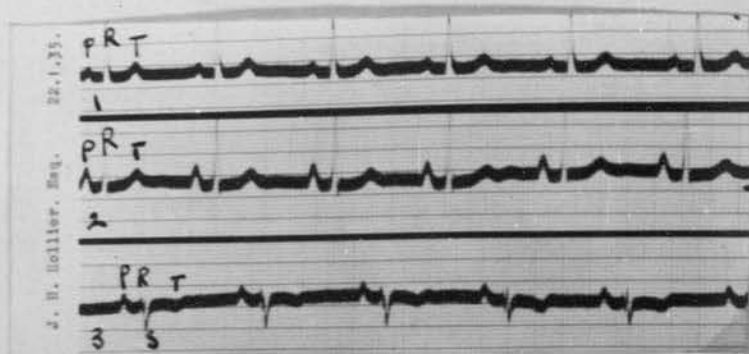
He had a good holiday after this and then went back to his overwork. I examined him on the 13th November 1937 when he was still very well indeed, blood-pressure 130/80, heart apex 4 inches in the 5th space, heart sounds normal, rate 72, liver not enlarged, lungs normal, retinal arteries normal, radial arteries soft, getting up on a chair six times raised his pulse-rate from 72 to 108, but it returned to 72 in one minute and 40 seconds.

This patient is leading a more than normal life for his age.

In this case I would like once more to call attention to the return of pain as the blood-pressure starts to rise again. Unfortunately, at the time I had not realised this association and did not take his pressure on the actual day of pain. Also of note is the fact that blood-pressure fell progressively till the sixth day.

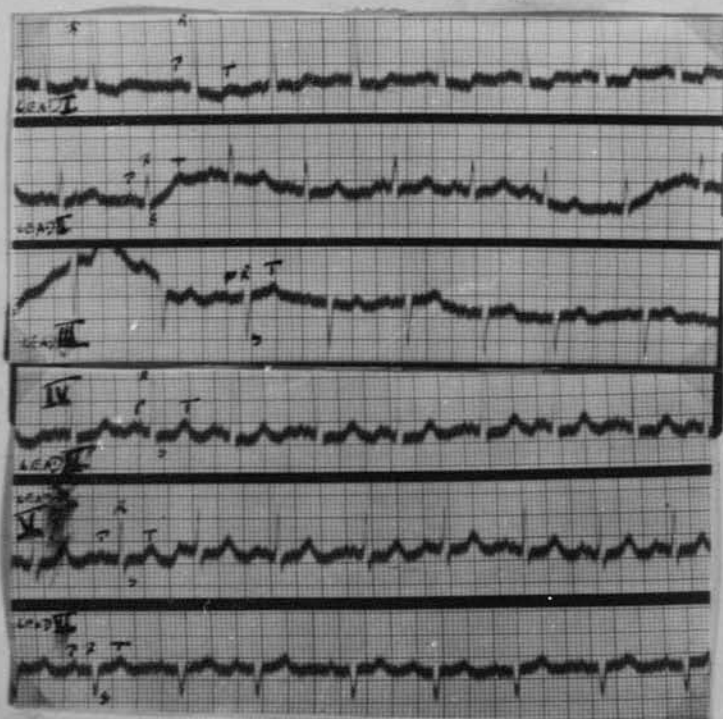
I cannot say whether his pain two months before the attack was angina or diabetic neuritis, probably it was the latter. I should think he had hypertension before his attack. I doubt if the reading of 130/90 one month before the attack is accurate. I have not, however, classed him as a case with previous hypertension.

CASE NO. 18 - J.H.H.



THREE MONTHS AFTER ATTACK.

CASE NO. 19 - MRS A.M.



THREE YEARS AFTER ATTACK.

CASE No. 18.

J. H. H. male, aged 36 at date of attack, 27th November 1934.

PREVIOUS HISTORY. Patient is an old bronchiectatic who had been invalided out of the army because of haemoptysis. He has had several attacks of haemoptysis in the last 15 years but tubercle bacilli have never been found in his sputum, and he has steadfastly and constantly refused to have his chest X-rayed.

He had an attack of pain similar to that about to be described in December 1933, but I did not consider coronary occlusion at all then as a cause and never took his blood-pressure! I had tried to explain that attack (which came on like indigestion as he left a picture house one night) on a basis of lung disease and the nearest I could get was a localised pneumothorax.

THE ATTACK. At 3 a.m. on 27th November 1934 when apparently in his usual health he was awakened by a pain on the left side of the chest, round the nipple which made him get up and walk about in search of relief. He says the pain was "not like pleurisy," which he has had, but came and went in a pulsating manner with the breathing, and was eased by lying on his right side. He has never (except for the one occasion mentioned above) had a similar pain, before or since. He had never had pain on exertion.

On examination there were no abnormal signs in the front of the chest, no friction, or hyperresonance on percussion, or altered breath sounds. The heart sounds appeared normal. The blood-pressure was 120/80.

Next day the temperature was 98.6° and it went up higher each evening till the 30th (4th day) when it was 100°. Thereafter it ran at 99° - 100° till 4th December. The blood-pressure on the second day was 115/80 and remained about that level until 5th December (9 days) when it fell to 90/60 and thereafter remained low.

He was in bed until early in January (sixth week) feeling quite well and rather restive, and then he was got up gradually and sent on 22nd January 1935 to a cardiologist, who says "the fall in blood-pressure is very suggestive but it took so long to come down, and I have never seen a man with this fall in blood-pressure as a result of coronary occlusion recover".- To which my reply is that I have (case 16). The electrocardiogram then showed only slight abnormalities, R slurred at end, T1 and T2 rather flat, T3 biphasic (mostly inverted), QRS3 notched.

AFTER HISTORY. Since then I have never seen the patient professionally, he has kept very fit except for slight bronchitis each winter. He has no pain or dyspnoea on exertion, and works hard.

After much hesitation he agreed to be examined on 22nd February 1938, and I found heart apex impalpable, on percussion left border 3 inches in 5th space. Blood-pressure 110/80, radial arteries good, retinals show slight arterio-sclerosis. There are a few creps at the base of each lung with prolonged expiratory murmur.

I did not dare suggest an electrocardiogram or X-ray!!

This case is I think, the only one in which any doubt can be cast on the diagnosis. I am convinced personally that the only fitting explanation of his attack is a coronary embolus from a bronchiectatic lung. If this is so the occlusion was not an incident in progressive coronary disease, which may account for the anomalous clinical and electrocardiogram findings.

This patient, despite his chest, leads a normal life for his age.

CASE No. 19.

Mrs. A. M. female, aged 76 at date of attack, 26th April 1935.

PREVIOUS HISTORY. I had attended this patient off and on since 1931 because of breathlessness on exertion, due to cardiac insufficiency as a result of high blood-pressure. Her blood-pressure used to be 180-220/110-120. She had steadily increasing disability as the years passed. In April 1933 she had an attack of decompensation during which her heart apex went to 5 inches in the 5th space. In November 1934 she had an attack of cardiac asthma following by persistent crepitation at the bases of both lungs.

THE ATTACK. In April 1935 I had put her to bed because of anginal pain. Her blood-pressure then was 210/100. On the 26th of April she had a very severe pain across the chest which lasted all night. In two days her blood-pressure fell to 150/90, and she ran a temperature up to 99° for several days. She was not very ill, and free from pain after the first day: gradually her pressure rose until a month from the attack it was 230/110, and she was having frequent heart pain although still in bed, and had a very intermittent pulse. Under the influence of nitrites her pressure fell again to 190/80 and her pain went. On 20th June (8 weeks from the attack) her blood-pressure was 190/80, and a week later, when it was 180/80, and heart $4\frac{1}{2}$ inches she started to get up for a short while.

AFTER HISTORY. She has never been able to do much since. (She was leading a very restricted life before). When she comes downstairs she cannot get up again without severe pain, and she will not sleep on the ground floor, so for a year now she has lived entirely on the first floor.

In December 1935 she had an attack of congestive failure with some congestion at the lung bases.

In April 1936 her angina, from which she had never been free since the occlusion, became much worse and I kept her entirely in bed again for some weeks, during which she developed a femoral thrombosis, which of course prolonged her stay in bed. Thereafter she required frequent trinitrin tablets to get about at all, and only got about for a few hours a day. That summer (1936) she was only outside on a few occasions.

Early in 1937 she seemed to have improved somewhat so I tried the effect of making her chew trinitrin tablets before undertaking any exertion. She tried climbing stairs on this basis but it was a failure.

In the summer of 1937 she had another slight coronary occlusion, and later developed signs of cerebral arteriosclerosis, -she had one attack in which she fell unconscious, after which she had transient weakness on one side with a Babinski response. She became rather "difficult", insisted on doing things for herself (and of course precipitating attacks of pain), and took to locking herself in the bathroom and having

a bath without assistance, and so in desperation I insisted on her being confined entirely to bed.

Since then she has been "officially" in bed, which means she gets up and potters round a very little bit occasionally, without permission (but with my knowledge), and as a result she has been very much better, with only very rare pain, and mentally she is alert and moderately happy and contented. She has two Theominal tablets every day and her blood-pressure runs about 170-190/100.

On 29th January 1938 her blood-pressure was 180/100, her heart apex was 5 inches out in 5th space. On 7th February 1938 an electrocardiogram showed "Normal sinus rhythm, rate 93. There is a single auricular extrasystole in lead I. There is slight slurring of the QRS complex in all leads. The limb leads (I, II, III) show left ventricular preponderance, and a depressed R-T interval in lead I. The complexes in the chest leads are of rather lower voltage than usual. The S wave in lead IV and the R wave in lead VI are very inconspicuous". (p. 86)

This case is another which illustrates the return of breast pain with the restoration of blood-pressure.

She was a cardiac invalid before her occlusion and I don't think the occlusion has hastened her increase of disability very much.

She is in the class of complete invalids.

CASE No. 20.

T. G. male, aged 72 at date of attack, 30th August 1935.

PREVIOUS HISTORY. Before I knew him he had many extrasystoles and "fluffy" heart sounds and had been warned against too strenuous exercise. His urine was normal and blood-pressure normal.

On the 27th April 1932 the patient was weeding in his garden when he suddenly went giddy, he sat on a barrow feeling very unsafe for half an hour and then went into the house feeling ill and looking very pale. He had no nausea or sickness, no change in voice or speech, no loss of power or sensation and no staggering. His heart had been irregular for years especially at night (this was due to extra-systoles). His only illnesses had been influenza in 1910 and 1916. I kept him in bed for several days after this, but have no notes of any examination until the 6th May 1932, when he had extreme pallor, his heart was slightly enlarged, and the aortic second sound accentuated. His blood vessels were thickened especially the retinals, his blood-pressure was 200/100 (which I remember surprised me). He had wax in his left ear in which he was complaining of slight deafness, and removal of the wax restored his hearing. An electrocardiogram taken on this date is shown (p.92) It shows only minor abnormalities, left preponderance, slurred S2 and S3, slightly low take-off of S-T1 and S-T2, slight Q1.

I did not see him again professionally, but I am told that after this turn in 1932 he had pain in his heart at intervals, especially when overdone, and on occasion looked grey and ill; and that he changed in character and became difficult and miserly.

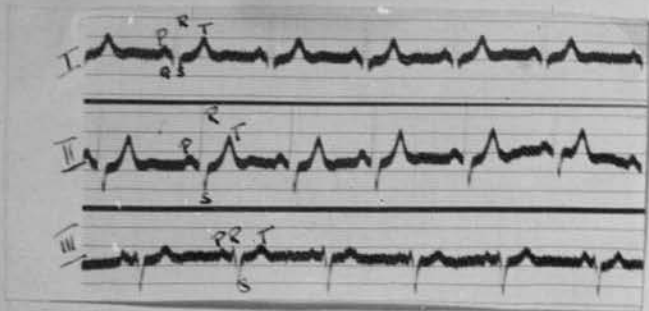
At the time this attack was diagnosed as Menière's disease, but the electrocardiogram is slightly suggestive of coronary trouble, and with the possibility of giddiness as a "substitute symptom" of angina (Levy page 208 (44)), and with his later mode of death I should think this attack was certainly of coronary origin.

THE ATTACK. On the 30th August 1935 he was walking down New Street, Birmingham when he was seen to fall to the ground. When picked up he was dead.

The post-mortem report taken from the Coroner's records in Birmingham is as follows:- "Heart enlarged and ventricles slightly dilated. There was relative incompetence of the tricuspid valve. Mitral and aortic valves showed senile degenerative thickening. The coronary arteries were the seat of advanced atheroma which had caused partial occlusion of the lumen. The anterior descending branch on the left side was almost completely obliterated. The myocardium showed a

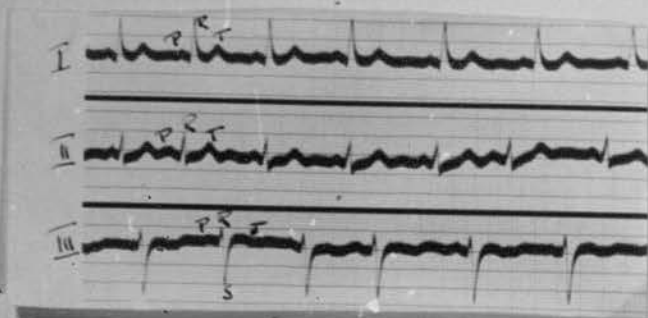
"fine diffuse fibrosis and was pale and soft.

"No significant changes were seen in the brain or cerebral blood-vessels."



CASE NO 20
T.G.
3 YEARS
BEFORE ATTACK.

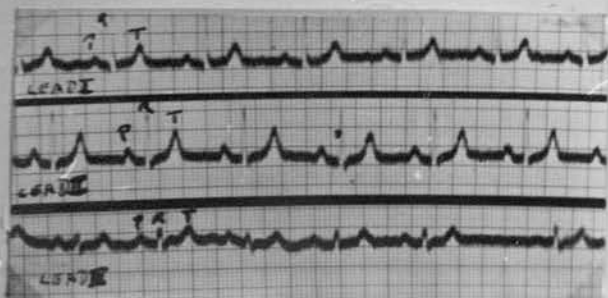
CASE NO 21 - JHWH.



3 MONTHS
AFTER ATTACK.



2 1/2 YEARS
AFTER ATTACK.



CASE NO. 23.
MRS. T.
4 MONTHS
AFTER ATTACK.

CASE No. 21.

J. H. W. H. male, aged 67 at date of attack, 3rd October 1935.

PREVIOUS HISTORY. He had an attack of "dyspepsia" in 1933 which I thought was of gall-bladder origin, but an X-ray was negative. He never had any pain on exertion until a week before the attack.

THE ATTACK. At the end of September 1935 he had, for about a week, pain in his chest which he called indigestion, which came on when he got up from his chair after a meal. In the afternoon of 3rd October the pain went right across his chest. He went out for a walk but the pain continued, and on his return it was very much worse and I was sent for. I put him to bed and on examination could find nothing much in his heart. His temperature was normal and his blood-pressure was 160/100. I told him that the diagnosis would depend on what happened in the next few days, that his temperature and blood-pressure would have to be watched, and that I was going on holiday the next day but that one of my partners would watch him. His blood-pressure actually fell in a day or two to 120/90 and his temperature rose to 99.5°. He had no more pain after the original attack and felt perfectly well. He was six weeks in bed and then got up gradually and went about slowly. On the 7th January 1936 (three months after his attack) I sent him to a heart specialist. His report is as follows:- "Blood-pressure 220/110, pulse 72, frequent extrasystoles "heart not very enlarged, no dyspnoea. The heart sounds are good "but he has probably considerable aortic atheroma and peripheral "arterio-sclerosis, his retinals and radials are both thickened, "his myocardium is good. He has a dead tooth which should be "removed. He appears to have made a very good recovery".

An electrocardiogram on this date shows:- left preponderance, slurred QRS all leads, curved R-T1 and S-T3, flat T3 inverted, ? T3 type of Parkinson and Bedford (65) late stage. (opp.)

AFTER HISTORY. Since then this patient has had all his dead teeth removed and has felt very well indeed, he has had no effort angina, no breathlessness and feels now that he could run upstairs if he cared to - which he does not. He has never had to consult a doctor since his attack. He is somewhat overweight. On the 10th December 1937 his blood-pressure was 190/120, his heart apex was $4\frac{1}{2}$ inches in the 5th space, and 5 inches in the 6th space, his pulse was 72 per minute, getting up on a chair six times caused an increase to 84, but it had returned to normal in 45 seconds. I thought his radial and retinal arteries at this date were not too bad. I examined him again on the 24th February this year, and his blood-pressure was 200/110, his heart apex was impalpable, and on percussion I made the left border $5\frac{1}{2}$ inches in the 5th space. He was electrocardiographed and radiographed on the 2nd March. On that date "His arteries "were thickened. His blood-pressure was 240/130. The edge of "his liver was palpable about 2 inches below his costal margin. "X-ray screening of the chest showed marked left ventricular "enlargement, the left lower arc being very prominent and curved.

"The left ventricular curve also projected markedly backwards
"in the left anterior oblique position. There was no
"prominence of the left middle arc formed by the pulmonary
"artery and conus of the left ventricle, nor, in the right
"anterior oblique view of the left angle. The aortic shadow
"was unusually dense, as in atheroma. The picture was that of
"pure left ventricular enlargement with aortic sclerosis. I
"enclose a tracing of the antero-posterior orthodiagram showing
"the size and shape of the heart. (See p.92) The
"electrocardiogram, of which I enclose prints, shows normal
"sinus rhythm, rate 88. The irregularity in lead III is due to
"the patient moving. There is marked left ventricular
"preponderance. The QRS complex is slurred in all leads. The
"T wave in lead III is inverted. The origin of the T wave is
"very slightly raised above the base line in leads V and VI.
"The chief differences in the limb leads from those in the
"previous tracing are the presence of an S wave in lead II, the
"slightly less prominent T in lead II, and the greater degree of
"inversion of T in lead III". (p.92)

This patient is of the "normal life for age" class.

CASE No. 22.

Miss J. H. female, aged 30 at date of attack, 13th October 1935.

On my return from the holiday, which started on the day of the attack of the previous patient, I was called on the telephone by one of my partners and asked to do a post-mortem the next morning on this case. The previous history showed indigestion of vague type occurring chiefly in the morning for the previous two years. She had seen several consultants both in London and in Birmingham and had been examined in every conceivable way, except apparently electrocardiographically, and no cause found for her dyspepsia. She used to complain of fulness in the stomach and constipation, and apparently the pain was not particularly affected by exertion. She used to walk long distances and played golf most days. Her heart was normal in size and in sounds, and her blood-pressure was normal.

THE ATTACK. On the morning of 13th October 1935 when the maid went to call her with her morning tea she found her lying dead in bed.

At post-mortem I found some blood-stained fluid in the pericardial sac, her heart was not enlarged, there was some reddish staining under the endocardium of the left ventricle especially on the septum, such as had been pointed out to me by Dr. Gilbert Miller in the Post-mortem Room at Edinburgh just a few days before as being a fairly characteristic finding in cases of sudden death from coronary occlusion. But all her organs appeared very congested and rather pink in colour, and so I sent them for examination to the County Pathologist; he had no hesitation in saying that death was due to coronary occlusion and considered that her coronary arteries were thicker than they should have been at her age. He said that the congested appearance of the organs was also not unusual in such cases.

CASE No. 23.

Mrs. T. female, aged 66 at date of attack, 16th November 1935.

PREVIOUS HISTORY. Patient had been fairly well for a great many years except for aches and pains secondary to a marked scoliosis, for which she was fitted with a spinal corset.

THE ATTACK. She had had for about a week before the attack some pain across the chest (described as indigestion) on exertion. This increased in intensity and came on more and more easily during the week until on 16th November 1935 she got a pain which continued, although not very severe, without ceasing for two or three days during which her blood-pressure fell from 180/100 to 140/90. She had no fever. She was kept in bed and watched carefully, and at first every time the blood-pressure tended to rise she had pain. She was in bed altogether for five weeks and by that time was free from pain, and she was allowed up.

AFTER HISTORY. In February (three months after the attack) she began to have vague pains in the region of her heart and in the epigastrium, not related to effort, so I sent her to a cardiologist for an opinion. He thought the heart was slightly enlarged, but of course her scoliosis prevented an accurate estimate. Her blood-pressure was then 160/95. He thought her pain was not of cardiac origin but due to her spinal trouble. An electrocardiogram was more or less normal (see p. 92). He agreed she must have had a coronary occlusion in November, probably quite small. Since then she has never had any pain on effort. She climbs stairs slowly and takes life easily, not because of pain but because she thinks it wiser to be careful.

On the 5th February 1938 I examined her. Her blood-pressure was 150/80, so far as I could tell clinically her heart was not enlarged, she had an accentuated second sound all over the heart. The urine was normal.

This patient leads a normal life for her age. She is apparently an example of re-establishment of blood-pressure at a lower level.

CASE No. 24.

N. B. male, aged 58 at date of attack, 25th November 1935.

PREVIOUS HISTORY. This patient had had some chest pain for the previous twelve months, which used to make him stop and rest while playing golf. He was a confirmed alcoholic and a heavy smoker.

THE ATTACK. He came to the surgery one evening when I was on duty (26th November 1935) with the story that on the previous day he had had rather a severe pain in the chest, which had passed off after an hour or so. He had attributed it to indigestion, and he had had a little return of it that afternoon, and thought he had better see the doctor.

I examined him very thoroughly. Apart from the fact that he had a blood-pressure of 140/90 when from his appearance I should have expected 200/100 (he was the florid, stocky type) I could find no signs. However, I told him that his pain might be of grave import,- at which he was inclined to scoff,- that he should go to bed, and allow my partner who was his usual attendant to watch his blood-pressure and temperature for a few days before any decision was made, and that on the whole I thought it unwise for him to drive himself home. He had his wife in the car and I went out and communicated my fears to her and told her to drive.

My partner saw him next day and found blood-pressure 170/130, and the patient in very severe pain which had come on during the night in the front of his chest, and round his ribs, and down his left arm. The next day (three days from onset) his pain was worse and his blood-pressure was 100/90. On the fourth day he was still having attacks of pain, which were not relieved by trinitrin, his heart sounds were normal, blood-pressure 140/100. His temperature had not risen.

From then onwards he was better some days, but the pain always returned, by the end of a week he was having as much as 2/3rds grain of morphia in one dose without his pain being relieved. About ten days from the onset his heart began to fail and he had oedema, but his pain persisted and he was continually under morphia. Towards the end he became delirious and died on December 10th, two weeks from the original attack.

The lowest blood-pressure in this case also was some days after the onset of the attack. This was a case where the fall in blood-pressure was not associated with relief of symptoms, and he died.

CASE No. 25.

Mr. W. male, aged 70 at date of attack, ? 1936.

This is a case which was seen by one of my partners after death. We have no notes. He had had a pain in his chest all one afternoon while at business. He came home by train, walked from the station to his house and fell dead as he entered the door. No post-mortem was made.

The history and mode of death leave one in no doubt that this was an attack of coronary occlusion. The coroner was apparently satisfied with the evidence!!

CASE No. 26.

Mr. L. aged 79 at date of attack, 2nd May 1936.

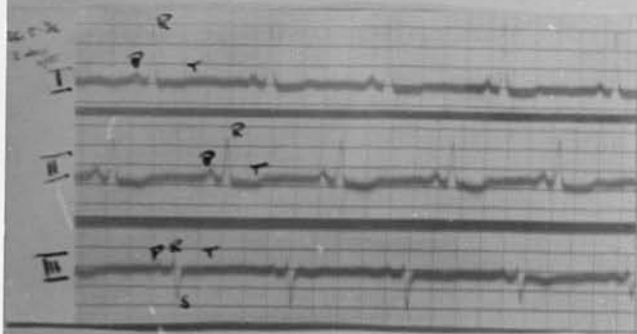
PREVIOUS HISTORY. In September 1935 he had an attack of asthma which I considered to be of cardiac origin. His blood-pressure at that time was high (varying round 180/100) and his heart was enlarged. He had no pain. He was three weeks in bed.

In November 1935 he developed a femoral thrombosis and was in bed for three months, and under my care for a further month. I have no record of any blood-pressure readings during this illness and probably took none.

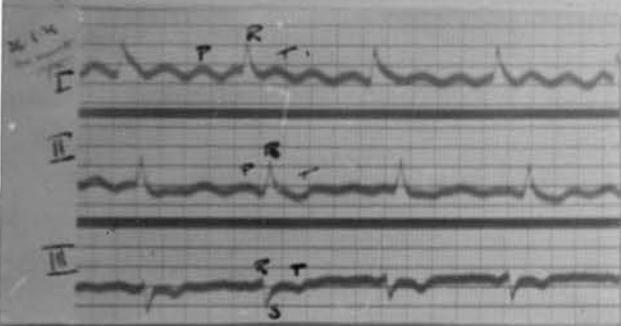
THE ATTACK. I had not seen him since March when on May 2nd 1936 his wife sent for me. He had been seized by pain under the sternum while working in the garden (against advice!) and had come into the house and lain on the couch looking very grey and ill. When I saw him he was pale, drawn and anxious, in great pain, restless and demanding relief. I had no doubt whatever what was wrong with him and gave morphine gr. $\frac{1}{4}$, and with the aid of the district nurse got him into bed. He was easier for a little and then was again seized with pain, and looked desperately bad. I injected some coramin without effect and he died in a few minutes.

This case might quite easily have come into the category of sudden death without benefit of medical attendance, but for the chance of my being immediately available.

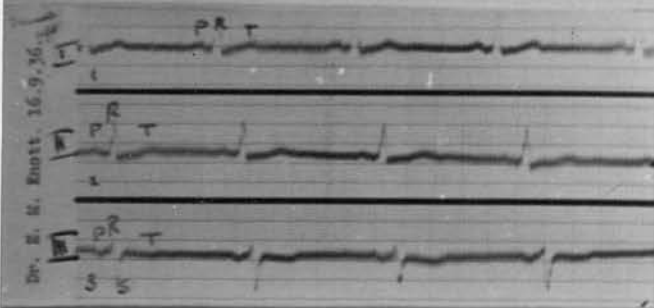
Case No 27
Dr K



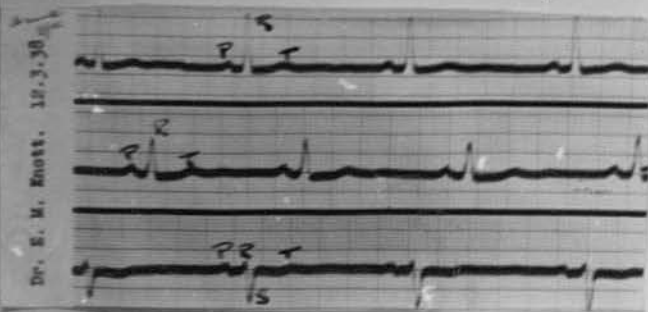
2 Days After Attack



1 Month After Attack
(Bad Connection)



3 Months After Attack



22 Months After Attack



21 Months After Attack

CASE No. 27.

Dr. E. M. K. aged 68 at date of attack, 24th May 1936.

PREVIOUS HISTORY: Patient, who is my senior partner, has always been a remarkably fit and active man, his worst troubles were occasional colds (in which he always has basal creps) and in November 1933 an attack of "chill on the gall-bladder" with slight temperature which may or may not be significant. He had some very bad teeth, and has chronically infected antra which cause him little trouble. He had always had a low blood-pressure. He is of the short, stocky type.

THE ATTACK. In the middle of May 1936 he began to have pain in the middle of his chest on exertion. He first noticed it while out fishing, after eating lunch, when he had to walk over a field in heavy boots. The pain made him sit down till it passed. Later in the day the pain came again when he hurried back to his car, more severe this time. That evening he tried to take his own blood-pressure and made it 180 systolic. The following day he went to a funeral and got the pain when he hurried up a hill to the church.

He consulted me and I could find no signs. I made his blood-pressure 120/80. I told him I thought he had a true angina of effort and called in a consultant. The consultant concurred, and we decided that he should have a few teeth out, and then take a quiet holiday. During these few days the pain was coming on with slighter provocation and taking longer to pass off.

The dental extraction was all set for the 25th of May, with a consultant anaesthetist to give the anaesthetic and so on, but on the night before he was awakened by pain which was fairly severe, and after sticking it for four hours he rang me up at 4 a.m. I went to see him and found him restless and anxious, with blood-pressure 115/90, and gave him morphia gr. $\frac{1}{4}$. This produced relief and next day, instead of his dental operation, we had a consultant with an electrocardiograph. His blood-pressure was then 105/80 and temperature 99°, pulse 66. The electrocardiogram (opp.) showed depressed R-T1 and R-T2, flat and inverted T3, flat T1 and T2. (opp.)

He had no more pain, and his blood-pressure rose to 115/90, and temperature fell to normal (i.e. below 98°) in two days. He stayed six weeks in bed. A second electrocardiogram a month after the first was not too good because of a bad contact, but T2 is inverted. (opp.)

AFTER HISTORY. He had a month at home after getting up, -going quietly, -and then went on a month's holiday, during which he fished and walked in moderation. He had no pain except on the day before his return when he climbed a steep hill, and then only a little. On his return his blood-pressure was 115/80 and an electrocardiogram showed T1 and T2 flattish but T2 is now upright, T3 inverted, slightly widened QRS in all leads and slight slurring QRS2 (3 months after attack). (opp.)

Since then he has led a more than normal life for his age. He fishes or shoots two or three days a week, and does all his practice on the other days! He looks and feels very fit indeed, and has married again.

Apart from a little attack of discomfort over the gall-bladder in April 1937 for which he had two days in bed, he has been perfectly well since. He never gets any pain on normal activities. He does get a slight discomfort if he walks up a steep hill immediately after lunch in shooting boots - but he is 70 years old after all. His blood-pressure remained at 115-120/80 until recently. On 6th February 1938 however, it was 140/80.

On 16th February 1938 an X-ray showed some general enlargement of the heart. An electrocardiogram on 12th March 1938 is practically the same as the last (cf. September 1936) - (p. 99)

He is of the "normal life for age" class. Of interest is the enlarged heart in a case whose blood-pressure (except for an obviously fallacious reading of his own) has always been low both before and after the attack.

CASE No. 28.

Mrs. W. aged 65 at date of attack, 13th June 1936.

PREVIOUS HISTORY. This patient had rheumatism as a girl and rheumatic fever as a young woman and as a result has an enlarged heart with severe mitral valve damage.

In 1914. Pulse was intermittent and irregular, rate 84, apex beat below nipple, short loud systolic murmur and presystolic murmur.

1919. Pain in left arm - always in night - ? neuritis.

1922. Heart very dilated, 1 inch outside nipple line, systolic murmur conducted to axilla, rough first sound - long spell in bed after which pulse was 80, lately has had pain in left axilla and down arm. Apex 2 inches outside nipple line.

1923. Pain on left when lies down.

1930. Pain left breast, heart 1 inch outside nipple line.

1931. In hospital for rest, blood-pressure 140/90.

1932 September. Pain in peroneal muscles left leg, giddy, blood-pressure 200/120, apex 2 inches out, systolic murmur - she had rest and nitrites and in two months we find blood-pressure 160/90, apex 2 inches out.

1933. Blood-pressure varied 160/80 - 180/115 - 140/90.

1936. In this year she began to have pain which was probably effort angina, which used to start in a tooth on hurrying. (cf. Levy (*ibid*) p. 207). On 26th April she went for a long walk which brought on a pain in her chest in the sternal region. Blood-pressure was 180/100, apex 2 inches out, systolic murmur conducted to axilla, liver not enlarged. Urine shows haze of albumen - no sugar. She was given nitrites and kept in bed and on 6th May blood-pressure was 160/60 and she had no pain. She continued thus till 19th May when she got up and had pain in her chest whenever she moved about, blood-pressure was 140/80, heart $5\frac{5}{4}$ inches, pulse 80, regular, systolic murmur, albumin in urine. She improved however, and early in June went to London to look for a house where she might live with her daughter who had got a job in that city.

THE ATTACK! On Sunday morning, the 13th June 1936, she got up and started to do her work, she had sat down to rest when a pain came on very badly in her tooth and spread down the right side. This pain was similar to what she had suffered on previous occasions, but then it had only lasted for a minute. This time the pain continued and went into her neck and shoulders and chest on both sides. She vomited and with assistance got upstairs and into bed. She telephoned to my senior partner, her doctor, who was then lying in bed recovering from his own occlusion. He

rang me up and told me that from her story he thought she had a coronary thrombosis. I went to see her immediately. The pain then had been on for nearly an hour; I agreed with the diagnosis and gave morphia. As she was then living by herself I got her into the local Cottage Hospital.

Her temperature rose gradually for three days to 99.8° on 16th, then settled gradually, but from 24th onward was up to 98.8° or 99° each evening till 27th June (two weeks from onset). The pulse was 80 on admission, and about that for a week after, by 3rd July it was 64. She was in bed for eight weeks and in hospital for nine weeks. Her blood-pressure was round 140/90 all the time she was there, and she had no heart pain, but she had some discomfort which she described as indigestion and which was relieved by a mixture containing rhubarb, soda and cardamoms. This was probably actually of cardiac origin.

On September 25th, a month after leaving hospital, she walked too far one day and as a result her heart apex was 3 inches beyond nipple line.

Shortly after this she went to Hove.

She was under the care of a doctor there who tells me that on 15th December 1936, she had pain in the centre of the chest going down the left arm "due to walking too much", her pulse was then 80, blood-pressure 185/130, heart apex $3\frac{3}{4}$ inches in 5th space. Loud mitral systolic murmur at apex, liver not enlarged, no oedema.

In January 1937 blood-pressure was 160/115, and she was much better.

In February 1937 she had a little pain while walking against the wind. Apex inside mid-clavicular line.

By May 1937 she could walk half a mile without breathlessness or pain and her blood-pressure was 160/125. Her heart was slightly enlarged to the left.

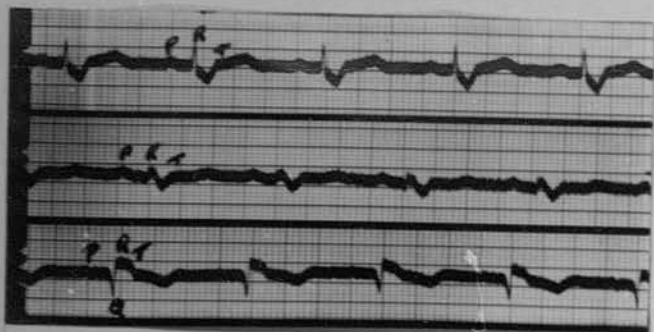
Since her attack she has only had pain once or twice, chiefly when she is "nervous". She has had no pain on effort, only on cold days she gets a "sore" feeling in chest.

4th March 1938. Blood-pressure 190/110, heart apex in anterior axillary line, loud systolic murmur, radial arteries soft, retinals show slight arterio-sclerosis. She looks and feels very well, but does no work from choice. She lies down every afternoon.

I had an electrocardiogram done on 12th March 1938 which showed depressed R-T1, raised R-T2 and R-T3, inverted T2 and T3, marked Q3 present.

I put her in class 2, leading a restricted life.

Of interest is the absence of any severe angina since complete recovery from the occlusion, (she had some during the first year but has had none for many months) and also the question of the nature of the pain before occlusion; I think that only in the last few months before occlusion was the pain of the nature of true angina. She is unusual also, in that coronary disease is unusual in people with old rheumatic carditis, but the electrocardiogram leaves no doubt of her coronary disease.



21 Months After Attack.

Case No 28

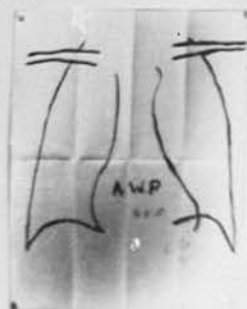
Mrs W



3 1/2 Years Before Attack

Case No 29

D W P.



← 16 Months After Attack

CASE No. 29.

A. W. P. male, aged 72 at date of attack, 15th December 1936.

PREVIOUS HISTORY. In 1920 he had giddy turns and used to look white after smoking in hot rooms. His systolic blood-pressure then was 190. His heart was 1 inch outside nipple line, and he had some slight cardiac pain.

In 1921 he again had slight cardiac pain, systolic pressure was 165, pulse 84.

In 1927 he had for a week pain in his left arm and in the region of his heart. His apex was in the sixth space, below the nipple, blood-pressure 160/80, pulse 96.

In 1928 blood-pressure was 150/60.

In 1929 blood-pressure was 160/90.

In 1931 he had a "feeling of falling every now and then", his blood-pressure was 160/80, pulse 96.

In January 1932 blood-pressure was 160/80, pulse 100, and in March he was having definite substernal anginal pain after meals and on exertion. The pain went into the back but not into his arms or neck. His blood-pressure was 190/105, pulse 100, his radial arteries were thickened, and he had retinal sclerosis. His apex was in 6th space. He was kept in bed for three weeks, and blood-pressure fell to 165/80, pulse 82. During this attack sugar appeared in the urine. He got up early in April and by the end of the month was having pain again. He was sent to a cardiologist who found blood-pressure 160/95, heart apparently not enlarged (?), regular, 84 per minute. Electrocardiogram taken then shows (to my reading) depressed R-T1, and R-T2. (opp.)

In June his blood-pressure was 170/90 and he was still having pain. In August he had been free from pain for some time. In November his heart became irregular and he was having "wind" in the upper part of sternum, heart dullness and apex were both in nipple line. Blood-pressure was 180/90.

In 1933 blood-pressure was 150/85.

In 1934 blood-pressure was 170/105.

Early in 1936 blood-pressure was 180/95, in April sugar appeared in urine again.

THE ATTACK. On the afternoon of December 15th 1936 he was feeling perfectly well when he began to have a stifling feeling in his chest while in the street in Birmingham. This eased when he got into the train but came on again before he got home. He couldn't get rid of it and it continued all evening and got

worse, although he was able to go out and visit some friends. During the night the feeling became a definite pain in the centre of his chest which lasted all night. The doctor was summoned in the morning and found his blood-pressure 160/80 and heart very irregular, due to extrasystoles. His pain was gone in two days. Three weeks from the onset his blood-pressure was 130/90, pulse 80, still numerous extrasystoles, his heart dullness was 2 inches out. He got up after five weeks feeling and looking very much better. Two months after attack his blood-pressure was 160/100.

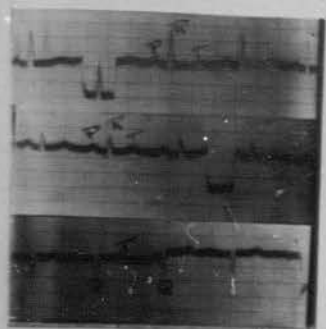
AFTER HISTORY. Since this attack he has required no medical attention. He is much better than before it and can now climb steps from the station without pain. These steps used to cause him pain and breathlessness. He has only chewed two trinitrin tablets since his recovery. I saw him on the 24th February 1938 and found - heart impalpable, on percussion left border 5 inches in 5th space, heart sounds closed, second sound accentuated in mitral area and at base, blood-pressure 165/105.

On 9th March 1938 he saw a cardiologist who reports:-
 "The electrocardiogram shows normal sinus rhythm, rate 90. An "auricular extrasystole, marked with an asterisk, is present in lead IV. The chief differences in the limb leads from the previous tracing are the somewhat lower voltage, particularly in lead III, and the return of the S-T segment in leads I and II to the iso-electric level. X-ray screening showed clear lung fields. The chest and the heart with it, was rather long and narrow. There was some enlargement of the left ventricle both in the antero-posterior and in the left anterior oblique views. The aortic shadow was rather dense, as in atheroma."

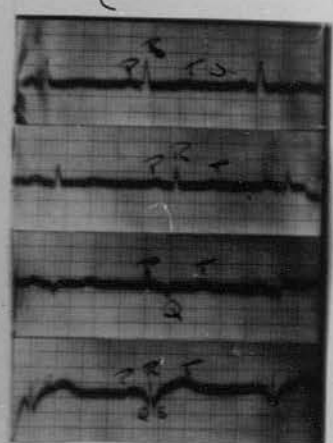
This case goes into class 1, leading a normal life for his age; which is all the more interesting when one remembers that he had had cardiac pain of some kind for over fifteen years, and that he had had definite angina of effort and an abnormal electrocardiogram $4\frac{1}{2}$ years before. The question arises whether he had not had a "silent" occlusion in 1932, he can now remember no "attack" of any severity at that time.

He is one of the cases described by Levine (41) who have had previous hypertension and angina, who establish a lower level of blood-pressure after occlusion, and are then free from angina. I think it is fair to say that his blood-pressure now is lower than in the months before attack.

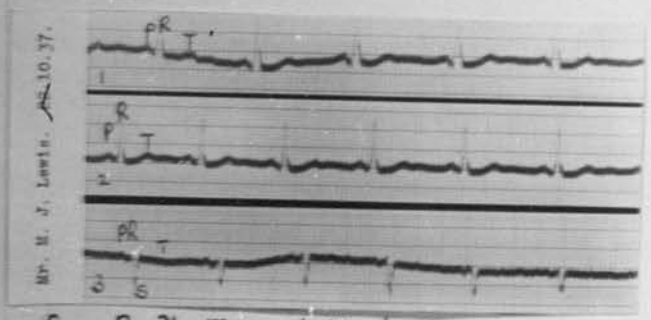
Case No 30. M.K.



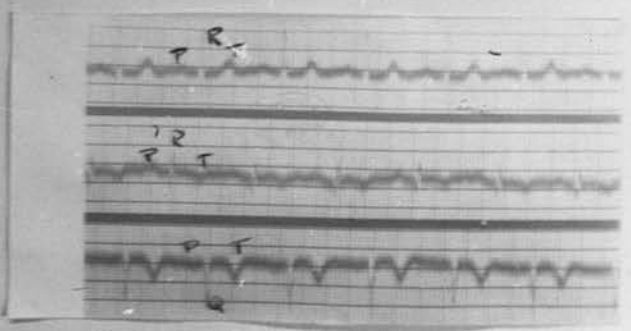
3 Months After Attack.



1 Year After Attack.



Case No 31. M.L. - 6 Months After Attack.



Case No 34 M.G. 7 Weeks After Attack.

CASE No. 30.

M. K. male aged 62 at date of attack, 14th March 1937.

PREVIOUS HISTORY. This patient is a gardener of the heavy stocky type. For two or three years before his attack he had had some pain behind the sternum and across the chest on exertion, especially when riding his bicycle and sometimes after meals, especially his evening meal. This pain on exertion eased off when he stopped the exercise concerned, and he was frequently forced to rest until the pain went.

THE ATTACK. On the 14th March 1937 there had been quite a heavy snowfall and he was working hard clearing the snow from some bushes in the drive at his place of employment when his usual pain came on in the centre of his chest, and this time did not go when he rested. This pain continued all afternoon and evening, not very severely but at night when he went to bed it increased until it was agonising for 20 minutes or so, and he was very breathless. I saw him the following day when his heart sounds were faint, the size of his heart could not be made out, his blood-pressure was 140/100. He looked well then but his wife told me that he had looked extremely ill when the pain was on. He was free from pain. Next day his blood-pressure was 120/80 and the temperature 99°. I prescribed eight weeks in bed and this was carried out. After the first few days he was entirely free from pain and felt perfectly well. His blood-pressure while he was in bed rose gradually to 140 or 150/100. When he got up he had some return of pain on exertion and his feet swelled quite a bit.

AFTER HISTORY. On the 11th June (three months after his attack) I sent him to a cardiologist at hospital. He reported his blood-pressure that day 200/120, said that his arteries were not bad but that X-ray revealed an enlarged heart. An electrocardiogram showed slurred QRS in all leads, left preponderance and flat T1 (opp.). He remarked that the patient was very much overweight, and took blood for a Wassermann which was negative.

Thereafter the patient gradually increased his exercise and chewed trinitrin tablets when the pain came on, or if he knew he was likely to get pain. In another two months, however, he was using these very seldom. His blood-pressure was 175/90, pulse 75. His heart was $3\frac{1}{2}$ inches in the 5th space on percussion. He had slight breathlessness on exertion, very slight oedema of the feet, and the only time he got pain was when he cycled to his work in the morning (he had then just started work).

A month later he was doing light work with comfort and only got a little pain when he cycled up a hill. He had no oedema, his blood-pressure was 160/90 and his heart $3\frac{1}{2}$ inches in the 5th space.

On the 21st February 1938 he came to see me at my request. He told me that he only got the very slightest pain now when cycling to work, not sufficient to make him stop, he gets a little breathless when he cycles or walks fast but never while at work. He has been doing quite heavy digging but has not been doing lawn mowing. His blood-pressure was 170/80, his heart apex beat was palpable at $3\frac{1}{2}$ inches in the 5th space. He had no oedema of the feet, his lungs were dry, and his liver not enlarged.

I had another electrocardiogram and X-ray done on 1938 (p.106), the X-ray shows the heart to be smaller than nine months ago, the electrocardiogram shows:- Similar appearances to the first. In the first electrocardiogram there was a marked Q3 which is now less marked, there was also a slight Q1 in the first electrocardiogram. In this second electrocardiogram QRS3 is W shaped, a fourth lead was taken which shows a low origin of S-T4.

This patient has made a very good recovery so far and goes in class 1, living a more or less normal life for his age.

CASE No. 31.

M. L., male, aged 56 at the date of attack, April 1937.

This patient is a golf professional, thick-set and florid, with a very red face.

PREVIOUS HISTORY. He had had no previous angina of effort, his blood-pressure had always been normal. He had always had excellent health.

THE ATTACK. He had been very busy during the spring of 1937 getting his course into trim for the Daily Mail Professional Competition, and was feeling somewhat tired and overdone. At the end of April 1937 he was playing a shot from the rough at the 10th when he had a sudden shoot of pain in his left breast which made him fall to the ground. He got up again and managed to play on, although still in pain. The pain lasted through the rest of the round and until he got back to the club house. It was present for a good hour. During the next few days he felt "very low" and did not sleep at nights as he had to sit up in bed to get his breath. In fact this went on right until June, and the astonishing thing is that he never consulted a doctor.

AFTER HISTORY. In June he began to have angina of effort which steadily became worse. He called it indigestion. The pain came on across his chest and down the left arm and went away when he stopped the effort which was causing it. The pain got steadily worse and was produced more and more easily. In August he went on holiday to Westward Ho and while there he coughed up some blood, but he waited until his return home before he consulted his doctor, one of my partners, early in September 1937.

He then had pain and breathlessness on exertion and a blood-pressure of 145/90. His heart size could not be made out. He was told to go to bed and did not, but a few days later the pain had become so bad that he was forced to stay in bed, and during the earlier part of his stay in bed the slightest movement such as reaching for a book caused the pain to come on. He was in bed for six weeks and his blood-pressure never went above 145/90 but always stayed about that figure. By that time he had been free from pain for a few weeks, and when he got up although conscious of his heart he had no real angina of effort.

He saw a cardiologist on the 22nd October 1937 who X-rayed him and said his heart was within normal limits for a man of his size. His electrocardiogram (p. 106) shows left preponderance, QRS widened and slurred in all leads, R-T1 and R-T2 slightly depressed, T3 flat.

Two or three weeks later he started playing a little mild golf. Even now (February 1938) he still only plays with iron clubs, but hopes to use his woods in a week or two. He has not had any heart pain since September, except a faint ache in the chest when he gets home at night if he is tired. On the 24th February 1938 he walked twice round his golf course, and when I examined him on the same evening he seemed very well indeed. He had lost a stone in weight but was still overweight; his blood-pressure was 140/80, his pulse was 70, his heart on percussion was 4 inches in the 5th space. I thought the condition of his arteries excellent.

This, to my mind, is a typical case of angina of effort coming on after a coronary occlusion and relieved by adequate rest later. He comes in class 1, leading a normal life for his age.

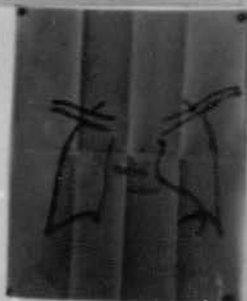
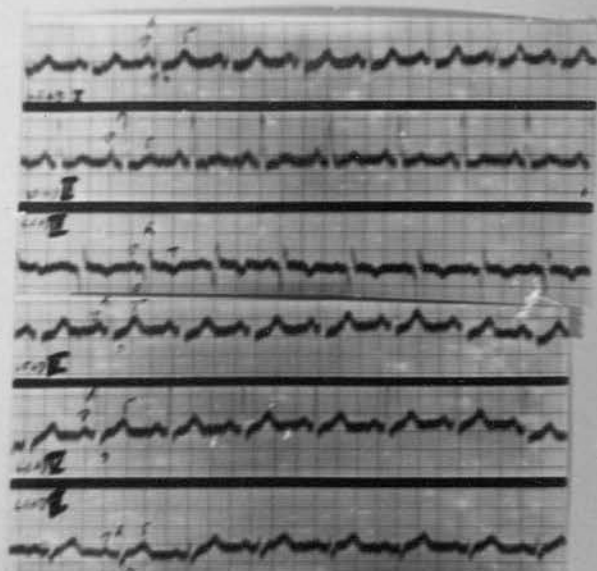
CASE No. 32.

Mrs. W. female, aged 58 at date of attack, 1st June 1937.

PREVIOUS HISTORY. For four years this patient had had pain in her right arm, which came on at first only when she walked up a hill. In the early stages she only had to stop once while walking into the village where she lived. She consulted a doctor there who said that her heart was all right. As the months and the years passed she gradually got worse, and the pain came on with less and less effort. She came to live in Streetly, and lived by herself, and despite her disability used to go about a fair amount. She tells me that there was a certain brick on the railway bridge about half way between the village and her house which was quite shiny because of the number of times she had rested her elbow against it on her way home. At last she got so bad that even knitting would bring on the pain.

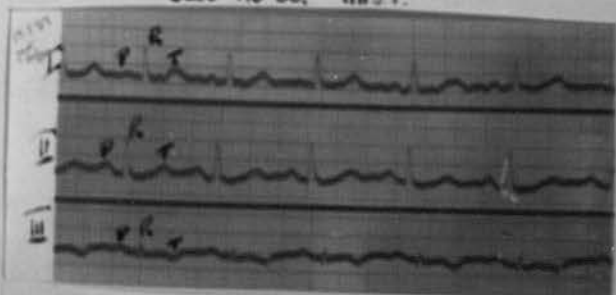
THE ATTACK. On the 1st June 1937 she was tying up sweet peas in her garden when the pain came on, and got worse and worse all day, and would not go away as it had previously done. She could get no relief neither on sitting nor lying, and felt as if she would choke. The pain by the evening was right across her chest, and in the night it went across into her left arm as well. She thought that she was dying, and reflected that in any case her affairs were in order, and she had left the window open so that when her neighbours missed her they would be able to get in to find her body. It did occur to her that there would have to be an inquest which would be rather a nuisance to her relatives, but then she philosophically thought that as she would not be there it would not matter to her. However, after many hours of pain she fell asleep and in the morning woke free from pain. She did not get up at all that day, and lay in bed all by herself. She spent most of the time for three days in bed and the neighbours did not notice. On the fourth day she got up and went to a Whist Drive, although in considerable pain and distress all the time. On Saturday (five days after the attack) she went shopping in a nearby town and the pain came on very badly, and she did not think she could get home. However, she did; and she actually walked a distance of about a mile to my surgery on Monday, the 7th June, to see me. She was then extremely breathless on the slightest exertion. Her blood-pressure was 160/100, her heart was enlarged, there was a soft blowing systolic mitral murmur. I sent her straight home and tried to make arrangements for someone to look after her. She was in bed at home for four days, and, as I could not get her efficient help, I got an ambulance and had her removed to her daughter's house in Birmingham. I sent a letter to her daughter's doctor saying "her history of angina for some time culminating in a terrifying and prolonged attack of pain coming on while at rest in bed, together with the fact that her blood-pressure has fallen from 160 to 120 while I have been observing her, seemed to me to make the diagnosis of coronary thrombosis an absolute certainty".

Case No 32. Mrs W

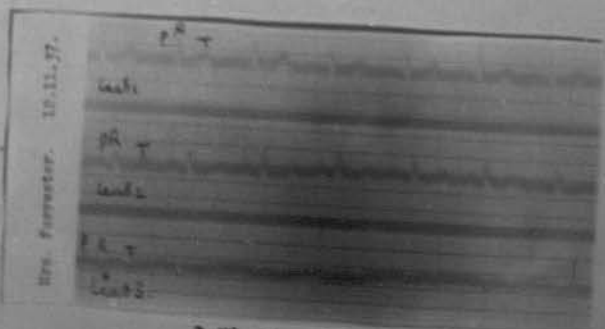


9 Months After Attack.

Case No 33. Mrs F.



Immediately Before Attack.



3 Months After Attack.

She had her eight weeks absolute rest in bed at her daughter's and then gradually resumed her usual life.

AFTER HISTORY. She had some mild anginal pains occasionally in the early months, but never any serious attack. She was in her bedroom for a further two or three weeks in a chair and then came downstairs for a few hours each day. In September she went by motor car to Weston-super-Mare and was there wheeled about in a chair for a fortnight. Since then she has been getting about moderately and having more or less angina of effort. I went to see her in Birmingham on the 18th February 1938. She is grossly overweight, weighing 15 stone. Her arteries were very much thickened, pulse rate 90, blood-pressure 170/90, her heart size could not be made out because of her fat. An X-ray revealed a considerable enlargement (opp.). She had slight breathlessness even while at rest and some oedeme of her feet.

At my request she went to see a cardiologist on the 24th February:- "The electrocardiogram shows normal sinus rhythm, rate 93. There is some slurring of the ventricular complexes in the limb leads. There is a very prominent Q in lead III, and the T wave in the same lead is inverted. The chest leads show no definite abnormality. Taken together with her clinical history, I think that these findings indicate the presence of an old infarct on the posterior aspect of the heart, due to occlusion of the right coronary artery or of the circumflex branch of the left. The orthodiagram shows a short, broad chest, with a high position of the diaphragm due to abdominal adiposity. The heart in consequence lies transversely, but is no doubt also enlarged to the left". (opp.)

CASE No. 33.

Mrs. F. female, aged 50 at date of attack, 19th August 1937.

PREVIOUS HISTORY. She was a fit woman except for occasional bronchitis, prior to the attack her blood-pressure was 140-150/80-90, her heart was normal in size and sounds, her arteries were normal.

Two months prior to her first pain she was very tired for two weeks. In the middle of July 1937 she began to have pain in the middle of her chest on walking, which stopped when she slowed down. Within a few days she was seized one day while walking in the Park with a "heavy" and severe pain in the left arm: slowing down her walk did not relieve it so she "carried her arm home", and sat down, and the pain went off. This pain made her look quite pale. Thereafter she was more or less confined to her house and garden, pottering there gave her no pain, but her walks had to be more and more curtailed. By the middle of August going for a drive in the car was sufficient to bring on the pain. She saw a consultant who arranged for her to be electrographed in one of the large hospitals.

THE ATTACK. She went to the hospital to have this done on 19th August 1937. This electrocardiogram shows:- Low voltage especially lead III, inverted T3, "coronary" shape of R-T1 and R-T2 (see p. 111). Immediately after she had been electrocardiographed, she was suddenly seized by palpitation and extreme faintness and collapsed completely, "going from one faint into another". She had no pain. She was brought out by ambulance to a nursing home (where she has lain in bed ever since). Her doctor sat with her all night. She was in a state of extreme prostration most of the time. Her blood-pressure when she was well enough for it to be taken was 150/80, pulse 94, temperature 98.8°. Her urine was normal.

On the next day and the next the temperature rose to 99°. On the fourth day the blood-pressure was 160/80. She had bad pain on the fifth and sixth days, requiring morphia. Seven days after the attack her blood-pressure was 125/70, the temperature which had been up to 99°, didn't go above 98° this day. On the eighth day blood-pressure had fallen further to 115/70.

Thereafter she was more comfortable till the end of a month from the attack, when she had an attack of pain without effort which continued more or less for three days and went across her chest, into the neck and down both arms, and into the abdomen. On the second day of this attack the temperature was 99°; and 100° on the third and fourth days, with pulse 100. The blood-pressure on the third day was 155/100.

Ten days after this attack she had a third (on 25th September) her blood-pressure on that day was 160/100. Next day her blood-pressure had fallen to 100/80; later in the

day it was 130/90, her temperature had risen again to 99° and her pain was better. The next day was similar:— blood-pressure 110/80, temperature 99°; on 28th blood-pressure was 115/80 and pulse 86.

From then on her blood-pressure remained low, on 3rd October it was 110/80, pulse 112. On the 11th October the blood-pressure was 130/80, pulse 98, and on the 12th and 13th October, two months from her first occlusion she had a fourth attack. The blood-pressure on the 13th fell to 95/65, temperature 99.2°, pulse 104.

The blood-pressure rose again gradually: on October 22nd it was 105/80; 26th, 120/80; November 3rd, 140/90, pulse 88; and on that day she had a fifth attack. Next day the blood-pressure was 110/80, pulse 96, temperature 98.8°. She had a further electrocardiogram done on 11th November which shows low voltage in all leads, T2 very flat, T3 negative, high origin of R-T2, "coronary" shape of R-T1 (see p. 111).

Throughout the rest of November she was fairly comfortable, Her blood-pressure rose to 135/85 by the 17th and stayed about there till December 9th when it was 140/90. On the 11th December she had more pain and her blood-pressure fell to 115/70.

She had a further, less severe attack on Christmas Day. Since then she has been all the time in bed, with occasional pain.

On 14th February 1938 her blood-pressure was 135/85, pulse 80. Heart $4\frac{1}{2}$ inches in 5th space on percussion. The first sound was very soft, and sounds showed tic-tac rhythm. Her arteries were soft. Her Wassermann taken previously was negative.

This attack and the subsequent weeks have been described rather fully because of the close association between re-establishment of blood-pressure and attacks, not only of pain, but apparently of further infarction.

CASE No. 34.

M. G. male, aged 58 at date of attack, 7th September 1937.

PREVIOUS HISTORY. Two years ago he was complaining of not feeling very fit and was thoroughly overhauled. The physical examination was quite negative, the heart was not enlarged, the sounds were normal, the blood-pressure was 140/80, the arteries were in keeping with his years, he had some heavily filled teeth which were removed and he was cured. He had no previous angina.

THE ATTACK. On Tuesday, the 7th September 1937 he was on holiday in Bournemouth, and while he was having dinner he felt that he was eating too quickly as he got some discomfort in his chest. However, he finished his meal and went out to put away his car. The discomfort in the lower part of his chest increased while he was doing this, and shortly became a terrible pain right across the chest radiating down both arms, and up the neck, and into the left side of his jaw. He vomited, perspired profusely, became breathless, and felt utterly helpless. He crouched against the wall for awhile and then crawled into the hotel and sat down and had two brandies. He managed to crawl up to bed and was sick again. The pain continued all night, and he was rolling in agony. A doctor was called in in the morning who found that his systolic blood-pressure was only 90, diagnosed coronary thrombosis, and removed him to hospital. The pain had gone in 24 hours, in 48 hours the blood-pressure had risen and he was febrile. Thereafter his progress was uneventful for two weeks and he was brought home in an ambulance. On his return his systolic blood-pressure was 120 and the first sound was very soft, otherwise there were no physical signs. The Wassermann was negative and the urine normal. At the end of the third week he had a large infarct of the lung from which he nearly died. However, he did not and the infarction cleared up.

AFTER HISTORY. Seven weeks from the attack he started to get up gradually, and began to have pain in his chest and in both his thumbs which lasted for long periods and was unrelated to exertion, although relieved by trinitrin. He was seen in consultation by a cardiologist on 27th October 1937 who found that his blood-pressure was 130/90 and he had slight sacral oedema, his heart sounds were distant, the first sound at the apex particularly being soft. An electrocardiogram taken then shows:- left preponderance, inverted "coronary" T3, and corresponds to the T3 type of Parkinson and Bedford (65) the initial ventricular deflection in lead III is probably a Q wave, there is a Q2, there is no R3. The cardiologist agreed that his pain now was mostly functional. He was given luminal and encouraged to get about with the result that he is now at work, and apparently perfectly fit. The size of his heart has been normal throughout and is still normal on clinical examination. He is in category 3 leading a normal life for his age.

CASE No. 35.

Mrs. L. female, aged 74 at date of attack, October 1937.

PREVIOUS HISTORY. Patient had glycosuria since 1934, albuminuria since 1935. She has had slowly-developing paralysis agitans for years.

In 1935 her blood-pressure was 160/80.

In 1936 it was 130/80 and the heart was found to be enlarged. In September of that year she had an attack of cholecystitis with a tender palpable gall-bladder.

In July 1937 blood-pressure was 145/100 and heart sounds were very faint.

THE ATTACK. In September 1937 she began to have angina while on holiday and was electrographed on her return. This shows left preponderance, low voltage 2, slurred and sloped R1 and S3 (see p. 117). At this time her blood-pressure was 165/100.

Early in October 1937 she had a severe attack of pain lasting for two hours, going into the left arm. She had no fever. On the 10th October her blood-pressure was 110/70 and later rose to 140/90. She had no real pain after that but was conscious of her heart.

She had no further pain thereafter but she was a complete invalid. She got up for a short while on Christmas Day and had "a heart attack" in which she became blue and cold. After this she was never out of bed and became gradually weaker and weaker and died on 25th February 1938.

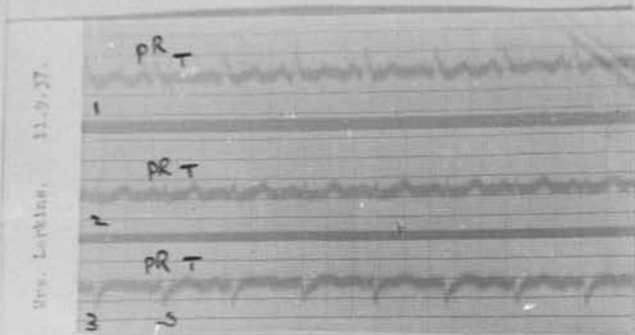
This occlusion was an incident in a general break up and she really never recovered from the attack.

CASE No. 36.

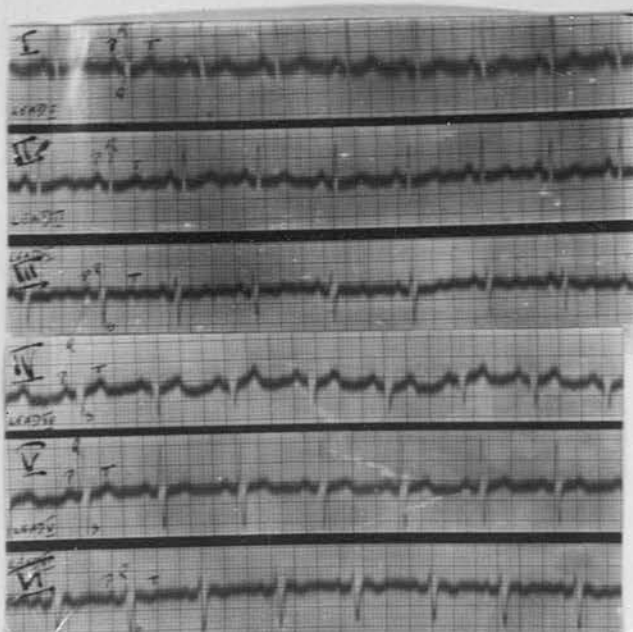
Mrs. L. female aged 77 at the date of attack, 22nd December 1937.

PREVIOUS HISTORY. This patient had suffered from hyperpiesis and arterio-sclerosis for many years (at least ten), her blood-pressure was constantly over 200 systolic. Some 18 months before her death she began to have attacks of cardiac asthma with oedema of the lungs. She had albumin, blood, and casts in the urine. During the early part of last winter she developed congestive failure with cerebral degeneration, her systolic blood-pressure was 300, although it sometimes fell to 120 during her attacks of cardiac asthma. Twelve weeks before her death she developed a femoral thrombosis. She had a colossal heart with the apex in the posterior axillary line.

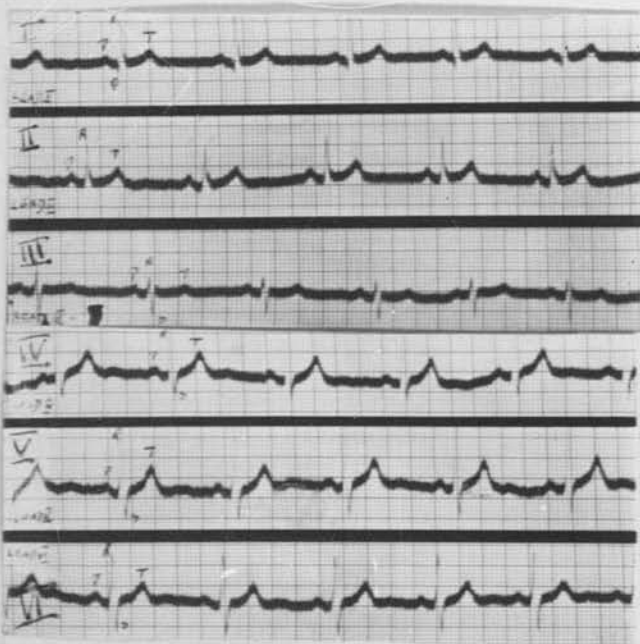
THE ATTACK. On the 22nd December 1937 she did not appear to be any worse than she had been for some days, but quite suddenly fell back dead in bed.



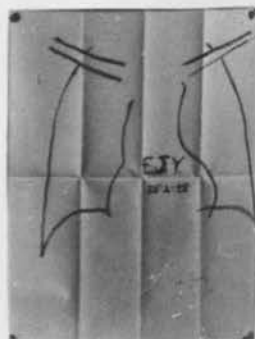
Case No. 35. Mrs. L.
One Month Before Attack



Case No. 37. E. J. Y.
Day After Attack



7 Weeks After Attack



A

CASE No. 37.

E. G. Y. male, aged 59 at the date of attack, 5th January 1938.

PREVIOUS HISTORY. In March and April 1935 he had had three short spells of temperature of uncertain origin, and on the 12th April had an attack of intense epigastric pain which lasted for several days. Nothing abnormal was found then and the attack was put down to acute gastritis. In the following month he was operated on for cholecystitis. An operative diagnosis of pancreatitis was made, and the gall-bladder was drained. So far as can be ascertained at this time his blood-pressure was normal, but his wife says that he was, at this time, suffering from considerable breathlessness on exertion. After his operation he was very much better for some time, but during the latter half of 1937 he began to have tightness and pain across his chest, going into the armpits, especially on the left side, when he went out on cold days and when he climbed up hills. His wife said that, when he had these uncomfortable feelings in his chest, he used to look grey and ill, and although no doctor was consulted she was quite sure his heart was bad.

THE ATTACK. On the 5th January 1938 while in a theatre he felt sick and ill, He looked so bad that his wife thought he would not be able to drive home, and drove herself. He was put straight to bed and in the night a pain came on severely in his chest, radiating into the armpits. For one hour it was very bad and for the rest of the night not so bad. He sent for me in the morning (6th January 1938). When I saw him there were very few physical signs, his blood-pressure was 130/90, his heart did not appear to be enlarged, and the sounds, although distant, seemed quite normal. I made a diagnosis of coronary thrombosis on the history, and had him removed to a nursing home in Birmingham. That evening I had a cardiologist to see him and take an electrocardiogram, his blood-pressure then was 155/90, temperature 98.4°. The electrocardiogram (opp.) showed:- very little abnormal, small Q1 present.

The next day his blood-pressure was 130/90, pulse 60 and he had 10,000 white blood cells per c.mm. The next day the blood-pressure was 120/70, pulse 70, the next day 120/65, pulse 64 and it remained around the 120/70-80 level during his stay in the nursing home. On the second day after his attack I noticed for the first time a reduplication of the second sound in the aortic area.

On the 20th February ($6\frac{1}{2}$ weeks after his attack) his blood-pressure was 140/90, and the left border of his heart was $3\frac{1}{2}$ inches in the 5th space, he still had the reduplicated second sound. On the 22nd February he left the nursing home and came to his brother's house at Streetly. His blood-pressure was then 125/75.

On the 27th February (nearly two months after the attack) the cardiologist "could find no abnormality on

"examination except for a reduplicated second sound at
"the base, which I do not think is significant. X-ray
"screening of the chest showed the left dome of the
"diaphragm to be raised by a large gas bubble in the
"stomach. The heart in consequence lay rather transversely.
"It is of full size, but not definitely enlarged. The aorta
"was rather dense, indicating the presence of atheroma. I
"enclose a tracing of the orthodiagram. The electrocardiogram,
"of which I enclose prints, shows normal sinus rhythm, rate 60.
"The limb leads are practically identical with the previous
"tracing. In the chest leads, Lead VI has a somewhat
"different shape from that in the previous tracing, the R
"wave being taller and the S smaller. It is probable that
"this is chiefly due to a somewhat different position of the
"electrode". T in chest leads is taller, T6 formerly
inverted is now tall and upright. (See p. 117).

This patient who is now able to live a practically normal
life, was cured of his previous angina after a sudden
occlusion.

CASE No. 38.

A. J. B. male, aged 66 at the date of attack, 27th January 1938.

PREVIOUS HISTORY. As far back as 1929 this patient was having pains in the epigastrium of no very certain cause, but definite angina seems to have first been noted in 1934, when there is a note that he had chest pain, and was eased by trinitrin. In September 1934 he was being much troubled by angina of effort, and had an aortic systolic murmur. The pain was not bad enough to stop him but it radiated down both arms, his blood-pressure was 160/90, the heart was enlarged, the apex being in the nipple line. He had no evidence of cardiac failure. The Wassermann was negative. In October 1934 the pain was not coming so easily, his blood-pressure was 140/70, his pulse 96, the heart still enlarged. In April 1935 the blood-pressure had risen again a little and was 160/95. He had continued like this during the last three years, always carrying trinitrin tablets, and using them with great regularity. It was his regular practice to start chewing his trinitrin tablet as he passed the doctor's house on his way to the station in the morning.

THE ATTACK. On the 27th January 1936 he came to the surgery because he found that for the last few days he needed to chew his tablet before he reached the doctor's house - the pain was coming on more easily, his blood-pressure then was 140/100, he had a systolic murmur at the apex. The next day, the 28th January, he sent for the doctor because he was having a severe attack of pain which had lasted 40 minutes before the doctor arrived, and which was not relieved by trinitrin. His blood-pressure was 180/100 and pulse 94. He was given morphine and a diagnosis of coronary thrombosis was made. He was easier and went to sleep and had a fair night, but the next morning he suddenly got a very severe pain which lasted for 30 minutes, at the end of which time he was sick and suddenly died.

CASE No. 39.

T. W. male, aged 88 at the date of attack, 3rd February 1938.

This patient was a chronic alcoholic.

PREVIOUS HISTORY. A year ago this patient had an attack of diarrhoea for which he did not consult the doctor. Ten days before his death he again had diarrhoea, very much worse this time. Three days before death he had intense thirst. On the afternoon of his death he asked for a cup of tea, and as he started to drink it he was seized with severe pain in the chest which radiated down both arms and up into the jaw, and he was dead in a few minutes. No post-mortem was performed. He had had no previous angina, and the suggestion was that he had become so dehydrated that it had led to a thrombosis in a coronary artery.

There can be little doubt that his death was due to a coronary occlusion.

CASE No. 40.

Mrs. H. female, aged 72 at the date of attack, 16th February 1938.

This case occurred while I was busy preparing this paper. She was living in her daughter's house. Her daughter was ill with an infection of the antrum, and her grandson had just a day or two before been sent to hospital with double otitis media. She had been helping with the nursing and was a little tired, although on the previous day I had actually taken a good look at her to see how she was standing up to the strain, and she seemed not too bad. On the evening of the 15th February 1938 she began to have a pain in the left side of her chest which increased in intensity. In the night she started to cough and the pain continued; in the morning she felt better and got up, but she looked bad. When she coughed she noticed that there was some blood in her sputum, and so went back to bed. When I came to the house I was asked to look at her. She was obviously very ill with profuse blood-stained sputum and rattling in her throat. The bases of the lungs were full of crepitations. Her temperature was sub-normal and the pulse 110, her heart sounds were moderately good and the heart was not enlarged, She was very pale. The idea that she had an acute cardiac failure secondary to a coronary occlusion crossed my mind only to be dismissed because it was so obviously a projection of my recent reading and writing. Because of the presence of an acute infection in the house I thought she was starting a very acute pneumonia. I managed to get a nurse straight away. In the afternoon she had a sudden attack which the nurse described as being "like a heart attack" because she suddenly collapsed and became pale. She came round, however, in a few minutes. I saw her in the evening, her temperature was 102.4°, pulse 120 respirations 44, there were profuse crepitations at the base of both lungs especially the left. About an hour after I had visited her she suddenly fell back dead in bed.

Because of the attack in the afternoon and the mode of death, I now feel sure that my original suspicion was right and that she had a coronary occlusion.

CASE No. 41.

W. T. male, aged 72 at the date of attack, 22nd February 1938.

This patient had not been ill for many years, or at least not sufficiently ill to consult a doctor. He used to be a little breathless because of "bronchitis", but this never "pulled him up".

On the night of the 22nd February 1938 he was found lying dead in the lavatory. At post-mortem examination the heart was much enlarged and very fat, and the muscle very flabby. The left anterior descending branch of the left coronary artery felt like a firm cartilaginous cord. At a point about 2 inches from its origin, where a large branch leaves the artery, there was pink staining of the arterial wall and surrounding tissue. On opening the heart the myocardium seemed to be rather brown in colour, the mitral valve showed senile thickening, the heart muscle at the apex of the heart was soft and friable with some infiltration and staining with blood. On opening the left coronary artery it was found to be very sclerotic, and a clot was found in the anterior descending branch at the site of the pink staining noted from outside.

This patient had apparently had an unrecognised attack of occlusion several days before which had led to his sudden death on the 22nd. Levine (41) states that the softening of the muscle is greatest from the 5th to the 14th day after infarction, so it was probable that this attack had occurred at least five days before.

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