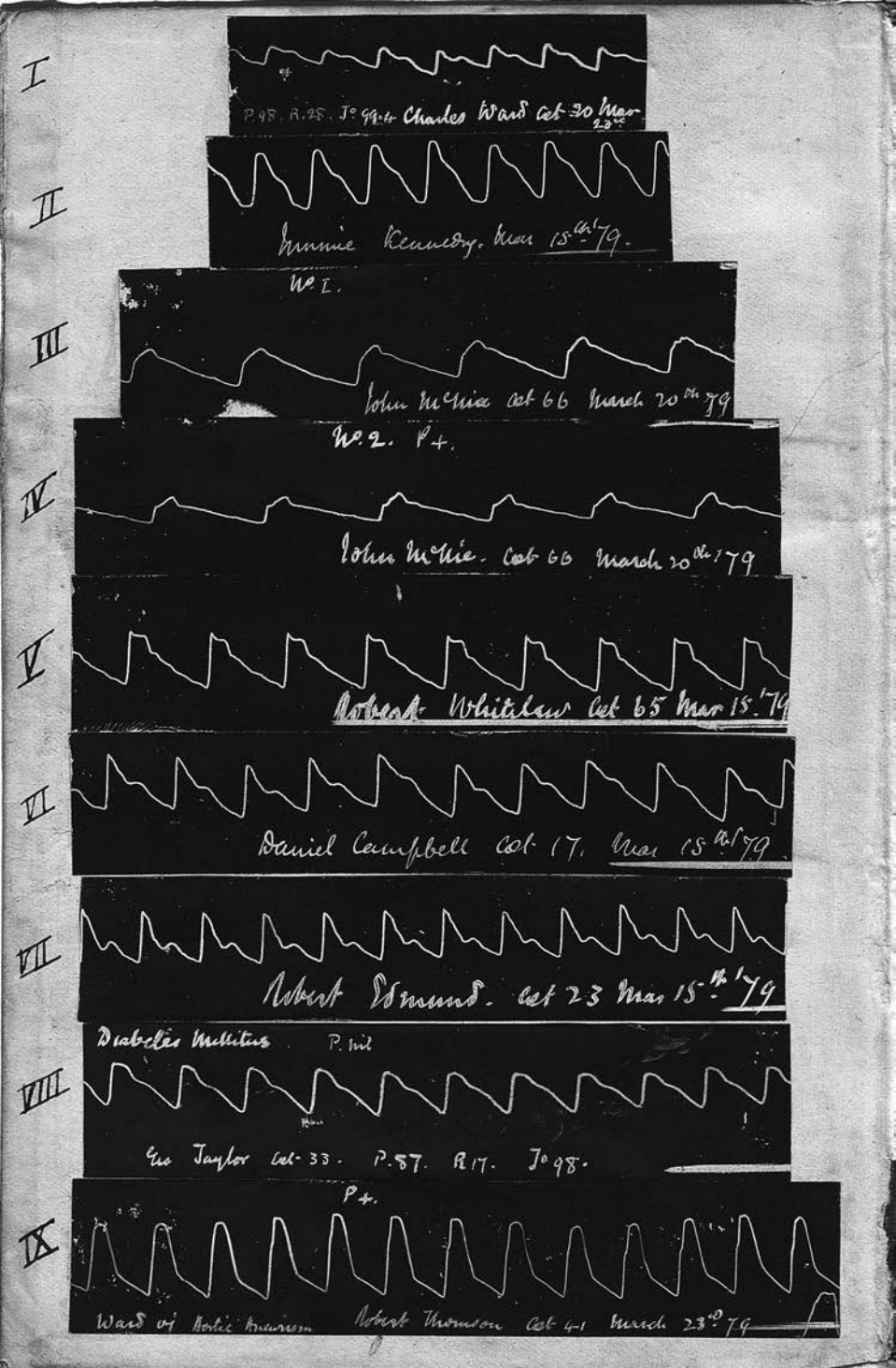
Contents

I. Personal observations on the frequency of Precordial murmurs in 89 cases of children's diseases

II Notes of fifteen cases, adults + children met with in Hospitals & Private practice along with charts.

III Discussion of these cases & inferences


I
P.98. R.28. Jo 99.4 Charles Ward Cet 30 Mar
II
Minnie Kennedy. Mar 15th 79.
III
No. I.
John McNeil Cet 66 March 20th 79
IV
No. 2. P4.
John McNeil. Cet 66 March 20th 79
V
Robert Whitelaw Cet 65 Mar 15. 79
VI
Daniel Campbell Cet 17. Mar 15th 79
VII
Robert Edmund. Cet 23 Mar 15th 79
VIII
Diabetes Mellitus. P. nil
Geo Taylor Cet 33. P.87. R17. Jo 98.
IX
P4.
Ward of Aortic Aneurism Robert Thomson Cet 41 March 23d 79

I Phthisis pulmonalis – waxy degeneration of liver & Spleen & albumen in the urine. Pleurisy with effusion on Left side. Cardiac impulses in three intercostal spaces on right side of sternum & dullness almost to right nipple line. Heart sounds normal.

II Chronic Bronchitis & Emphysema of several years standing – with markedly barrel-shaped chest. Heart sounds almost normal – accentuated second.

III & IV. Tracings of the same pulse – IV being under double the pressure of III.

Aortic obstruction & Mitral regurgitation with Dilatation of aorta and Atheromatous disease of the Blood vessels.

V. Atheromatous arteries with slight Mitral Regurgitation & Dilatation of the left side of the Heart. Oedema of legs.

VI. Phthisis pulmonalis with waxy degeneration of liver. Spleen and albumen in the urine.
Heart sounds normal.

VII. An aortic diastolic murmur was always heard and sometimes an aortic systolic and a Mitral presystolic.

VIII.

IX. Systolic & Diastolic aortic murmurs.

1888

It is now fully three years since I first observed the comparative frequence of a transient precordial murmur, more especially in children and as a rule in patients seeking advice for other ailments than those referable to the heart. It was while resident in the Leith General Hospital that I first observed it in an out patient. The number of juvenile patients admitted to the house is not great but the Dispensary Journal records a yearly average attendance of four & a half thousand a large number of whom are children. Subsequently at the Royal Hospital for Sick Children I confirmed my observations, and since I have been in attendance at the Royal Public Dispensary I have met with one or two instances in support of my thesis, and these always in children.

This murmur being detected so often in the hurried examination to which out patients are subjected, led me to argue that it must mean something

although its presence was a cause of no suffering to the patient and that as a rule it disappeared, without specially directed treatment, and sometimes before he ceased attending. I thought this must be capable of some explanation and ought not to be dismissed with the trite remark "a functional murmur', or 'a murmur of debility'.

How though fully convinced myself of the importance of these murmurs; the want of definite and carefully ascertained histories, prevents my quoting the Out Patient cases in support of the views contained in this paper. In my citation of cases therefore, I have confined myself to those admitted to hospital, where careful & continuous observations alone were possible. These are numerous enough for purposes of illustration. Of the frequency of precordial murmurs in Dispensary practice I can give no statement. Any such average, seeing it would embrace more

adults than children would loose in significance for the adult heart is not so easily influenced as the heart of the child, and it was in children I first made the observation, and the first and most important part of my paper applies to them.

In the case of Hospital patients however the following note may be of interest. Examining my notes of patients admitted to the Royal Hospital for Sick Children I find that in 89 consecutive cases — some of which were admitted for surgical diseases — heart murmurs were detected in 20. i.e. 22.4 per cent. 5 of these 20 were admitted for heart diseases; so if we exclude these we have murmurs occurring in 15 out of 84 patients admitted for other diseases i.e. in 17.8 per cent. This is a very high percentage.

In 2 of the 20 cases there was a personal history of acute or Sub-acute Rheumatism.

In 3 there was a history of Rheumatism

either in the Father or Mother.
In 2, we have in one case a history of Chorea & in the other a doubtful Rheumatic history, and
In 1 there was a history of Scarlatina.
In the remaining 12 (two of which were admitted for heart disease); we have no history either of Acute, Subacute Rheumatism or Scarlatina.
In all these cases the history was carefully taken by myself, and the family histories & previous illnesses as accurately ascertained as possible, It is impossible I think to overestimate the value of these family histories as it is always obtained from the parents who not infrequently can give valuable information for one, sometimes two generations back.
Some of these remaing 12 had had measles, but the other exanthems only occasionally lead to endocarditis [Goodhart, Diseases of Childrn page 528]
Endocardial changes following Measles must be very rare. I am not aware

of any book which records accurate observations on this point. Walsh, Bartholow, ~~nor~~ Eustace Smith nor Balfour even mention it as a possibility. Goodhart & Brantwell allude to it "among other causes" but neither records a single case.

Allowing for the possibility of a slight attack of Scarlet fever or Rheumatism having passed unnoticed the latter of which as Goodhart says is frequently unobserved; considerable faith may be placed in the histories, more especially if the murmur disappears during the observation of the case, the heart in children seldom completely recovering if once affected in an attack of Acute or Subacute Rheumatism. It is said, that in the Rheumatism associated with Scarlet fever "the endocardial murmur is prone to disappear, though this must not be taken to indicate that the bruit has been of a 'Functional' nature and unassociated with endocarditis" [Diseases of Children, Goodhart page 5-20]. This does not agree with my experience.

In the few cases of Rheumatism following Scarlatina, which I have met with, the murmur did not disappear while the patient was under observation. Moreover I have notes of several heart cases which date from an attack of Scarlet fever. This is the experience of Eustace Smith (Diseases of Children pg. 38). The absence of cyanosis, clubbing of fingers &c. excludes Congenital Malformation, while the transientness of the murmur excludes intrauterine endocarditis.

If I then consider those murmurs which have disappeared as not having been due to organic changes in the valves I think I cannot be accused of assuming too much, for the vanishing of cardiac murmurs if organic is decidedly rare; and the presence of organic changes in the heart, without a murmur, still rarer. (Balfour, Diseases of the Heart, page 220 et seq).

I have recorded these Statistics to show how frequent a precordial murmur is in children & it is my impression

that the percentage I have given is under the real figure. If every child admitted to the Royal Hosp. for Sick Children, during one year, had its heart carefully examined I feel sure the precentage of those with murmurs would be about 25. and that the majority of these on being discharged would have normal heart sounds. Some of these would be due to anaemia & debility, but some too, would I feel sure, be classified along with the cases I shall record.

I have now to consider a series of cases in which a precordial murmur was observed Antecedent to the development of serious disease in other organs. A brief record of these is necessary. I shall give them consecutively & thereafter ~~make a few~~ discuss them shortly.

Case I.

J. P. aet 10. admitted to R. H. S. C. on 7th May 1886. She complained of slight cough and pain in her side but an examination of her lungs revealed

Name James Philbin Age 7 Disease Diarrhoea Result Cured.

June	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	1886.
Pulse m			56	60	58	48		60	60			48				50			50			Pulse m
Pulse 6				49	48				48											44		Pulse 6
Resp. m				20	24	30		24	24			20				20			20			Resp. m
Resp. 6			16	26	20				20											24.		Resp. 6
Motions		2	2	1	3	3	2	2	2	2	2	2	2	1	1	1	2	1	2		1	Motions
Urine, ozs.		clear Straw																				Urine, ozs.
Sp. Gr.		1030																				Sp. Gr.
Reaction		acid																				Reaction
~~Chlorides~~		Mucous cloud. no albumen began a bite.																				Chlorides
Albumen		trace																				Albumen
Day of Dis.																						Day of Dis.

Weight 33 1/2 lbs.

Precordial mitral murmur always present. Loudest to the L. of the sternum from the 3rd – 4th inter-costal spaces but audible also in the mitral area & slightly prolonged towards the axilla.

The temp was very carefully taken but showed not the slightest evidence of rebrile. This must have been imminent for in less than two months from his discharge he was readmitted with evidences of the disease advanced a considerable distance & the temp high & remittent.

TEMPERATURE FAHRENHEIT'S SCALE. 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°

TEMPERATURE CENTIGRADE SCALE. 41° 40° 39° 38° 37° 36°

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nothing. She had a soft blowing murmur audible in the mitral area but most distinct in the 4th L. intercostal space. On the second day the physical signs of a Pleurisy were detected at the left base posteriorly and in the L. axilla. On the 20th the Lungs are reported quite normal. She was discharged on the 24th cured, Heart sounds normal.

Case II J. P. act 7. admitted to R. H. S. C. on June 9th-86. This was a fat somewhat flabby boy with diarrhoea I have noted in his case a murmur in the Mitral area prolonged towards the axilla, but best heard in the 3rd & 4th Left. intercostal spaces. Otherwise his systems were normal. The diarrhoea was stopped by means of Bismuth. & careful dietary and he was discharged the heart murmur however still being present. The Pulse was always very slow for a child but regular and of good tension. vid, tracings on next page. & chart ~~number~~ opposite.

DISEASE.

Pleurisy

Notes of Case.

Name { Ann Alexander

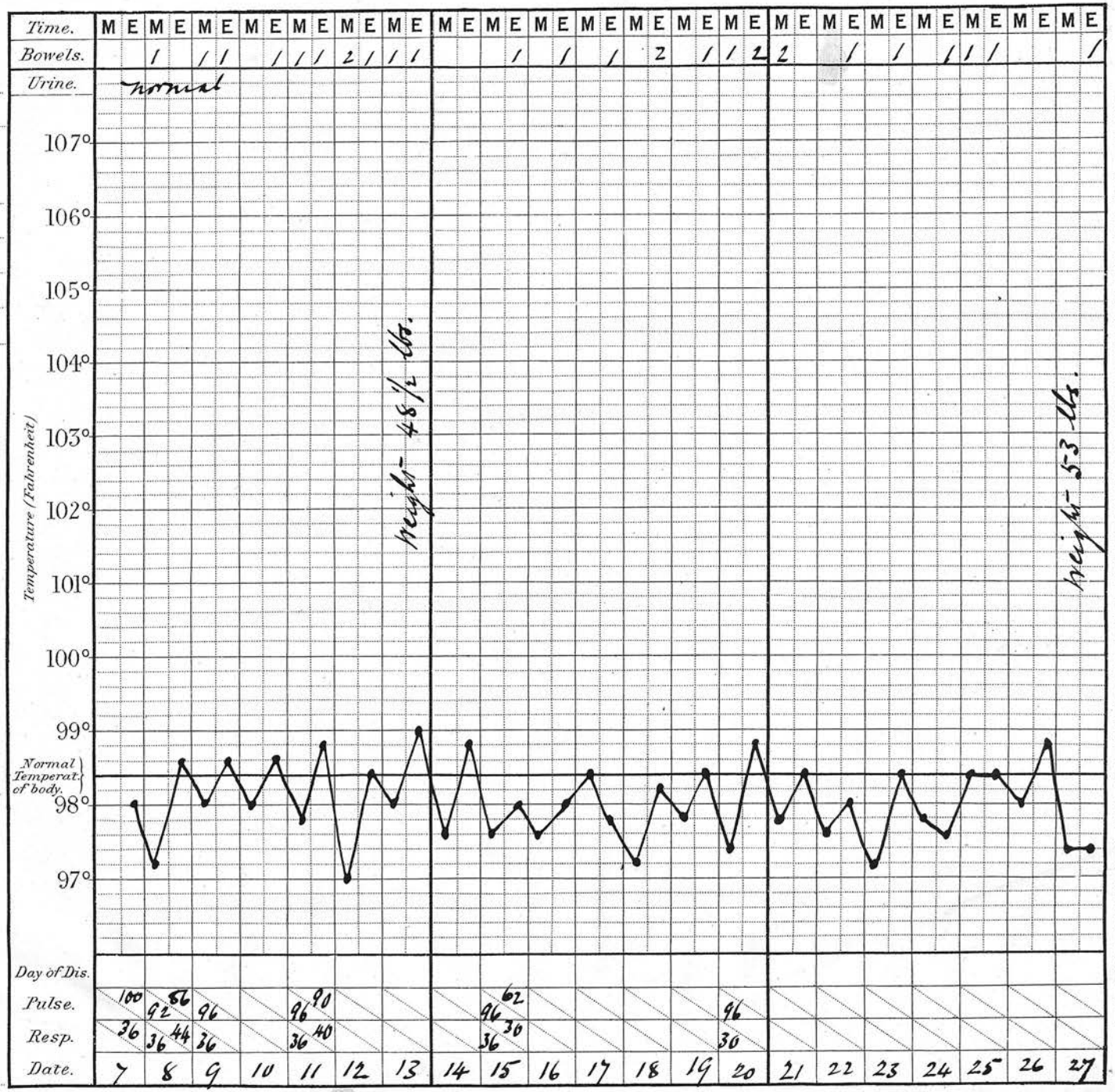
Age 10

Diet

Case Book No.

N.B.

Chart inserted to shew that no information could be obtained from the temperature regarding the condition in the ~~Chest~~ pleura.



Date of admission.

7/7/86

Result Cured

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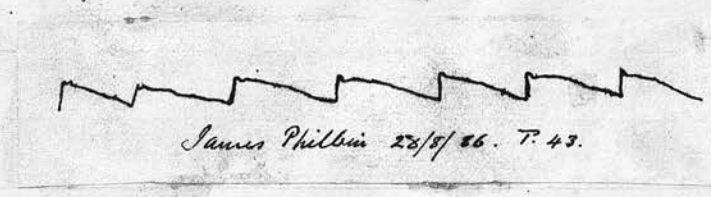
July

James Philbin 9/7/86 P. 52.

August.



James Philbin 28/8/86. P. 43.

These are typical of his pulse all the time he was in the house.

Some two months later he was readmitted to Dr. Carmichael's ward with Phthisis at both apices. Every symptom of tubercle was ~~detected~~ present but no heart murmur was detected. He died but unfortunately no P. M. could be obtained.

Chart opposite.

Case III. A. A. aet 10 first came under observation on the 5th July 1886. She was complaining but showed nothing definite. She had a systolic murmur over the base of the heart & the first sound in the mitral area had a ringing character. She was admitted to the R. H. S. C on the 7th with a Pleurisy

DISEASE.

Pleurisy

Notes of Case.

Name { Richard Chisholm

Age 10

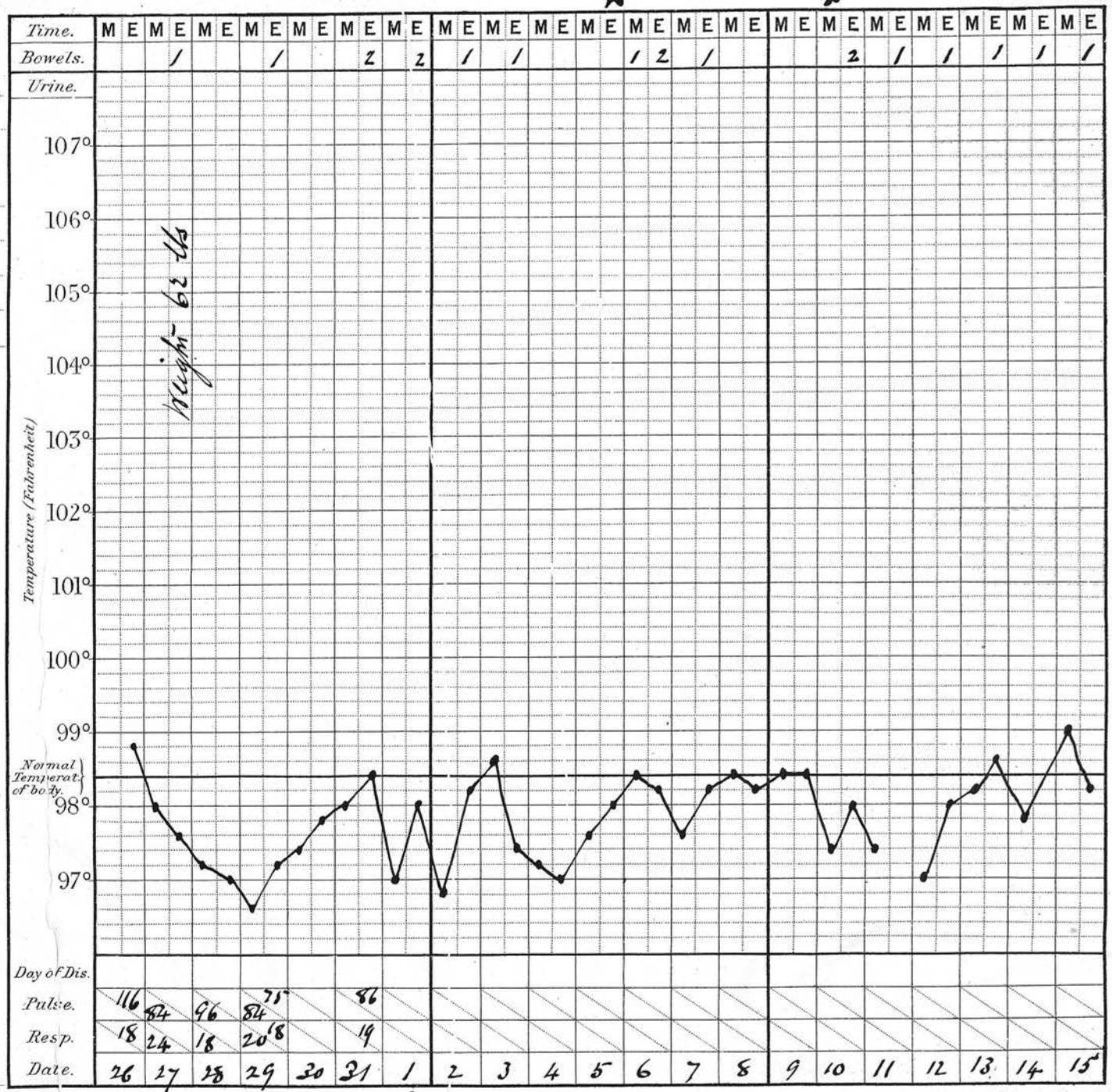
Diet

Case Book No.

N.B.
chart inserted to shew that no information could be gathered from the temperature regarding the pleurisy.

Date of admission.
July 26 1886

Result Cured



Entered at Stationers Hall. Printed and published by Wodderspoon & Co, 7, Serle Street, Lincolns Inn Gould's Clinical Chart.

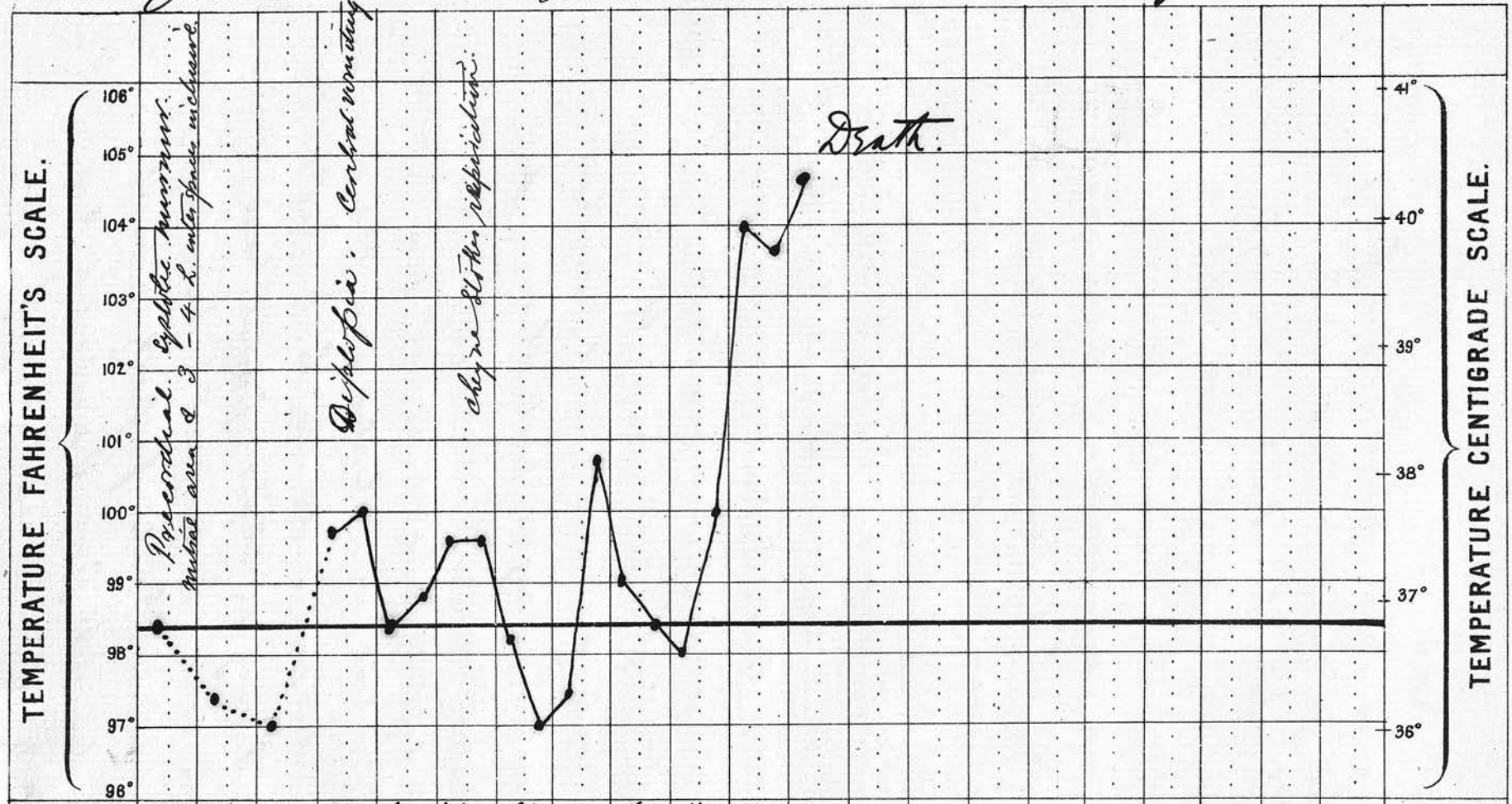
of the Right side. She was under observation a month and was discharg-ed cured the murmur having dis-appeard.

Chart opposite.

Case IV R. C. aet 10. When first seen 22 July 86 he was heavy looking & apathetic complaining of a pain in his side but with no definite or indicating symptom. He had a systolic basic murmur and a also on the aortic & Pulmonary areas. On 26 the was admitted to the R.H.S.C. with friction on the R base. The other signs of a Pleurisy with effusion Shewed themselves soon after. He was discharged in abmt a month with the pleuritic condition cured & with normal heart sounds.

Case V. B. G. aet. 12 admitted to the R. H. S. C. on the 18th Sept. 1886. re-ported to be suffering from fainting at-tacks & complaining of Precordial pain. She had an accentuated & impure first sound the impurity at times

Name J. Wilson Age 6 Disease Tubercular Meningitis Result Death.



Pulse	m	80	76	84	84	60	110	92	112	82	120	156	172	?						Pulse
	6				86	60	120	108	108	124	120	168	?							6
Resp.	m						54	63		30	46	52	50	?						Resp.
	6						74	60	66	54	54	36	?							6
~~Motions~~		Bowels constipated.																		Motions
Urine, ozs.																				Urine, ozs.
Sp. Gr.																				Sp. Gr.
Reaction																				Reaction
Chlorides																				Chlorides
Albumen																				Albumen
Day of ~~Dis~~ Visit		1	2	3	4	5	6	7	8	9	10	11	12							Day of Dis.

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amounting to a murmur. After being in hospital for some weeks she had a severe Epileptic fit. She had several then & for sometime after took them frequently. The precordial pain was not constant occurring only before the seizures at which times also the murmur was distinct

Case VI J. W. aet 6. Willow Bank Row Newhaven. First seen in August. 87. His mother stated that he had been ailing for some time back; - 'dwining'. - & apparently without cause. He was pale but not anaemic, well nourished & well grown. Tongue quite clean. pulse 80. Temp normal. (at time of visit). He had a syst. systolic murmur heard over the Mitral area & also over the 3rd & 4th interspaces to the L. of the Sternum. Saving a slight frontal head-ache (wh was not constant) he was free from pain. Joints perfectly normal Urine do. do. At first I suspected Acute Rheumatism with the heart affection ap-

Name H. McNeil Age 13 m. Disease Lat. Pneumonia (?) Result Death.

	21	22	23	24	25
Pulse m &	100			174	
Resp. m &				80-90	
Motions					
Urine, ozs.					
Sp. Gr.					
Reaction					
Chlorides					
Albumen					
Day of Dis.					

TEMPERATURE FAHRENHEIT'S SCALE.

106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°

TEMPERATURE CENTIGRADE SCALE.

41° 40° 39° 38° 37° 36°

Paracentesis thoracis

9 p.m.

12 P.M.

Death 2 a.m.

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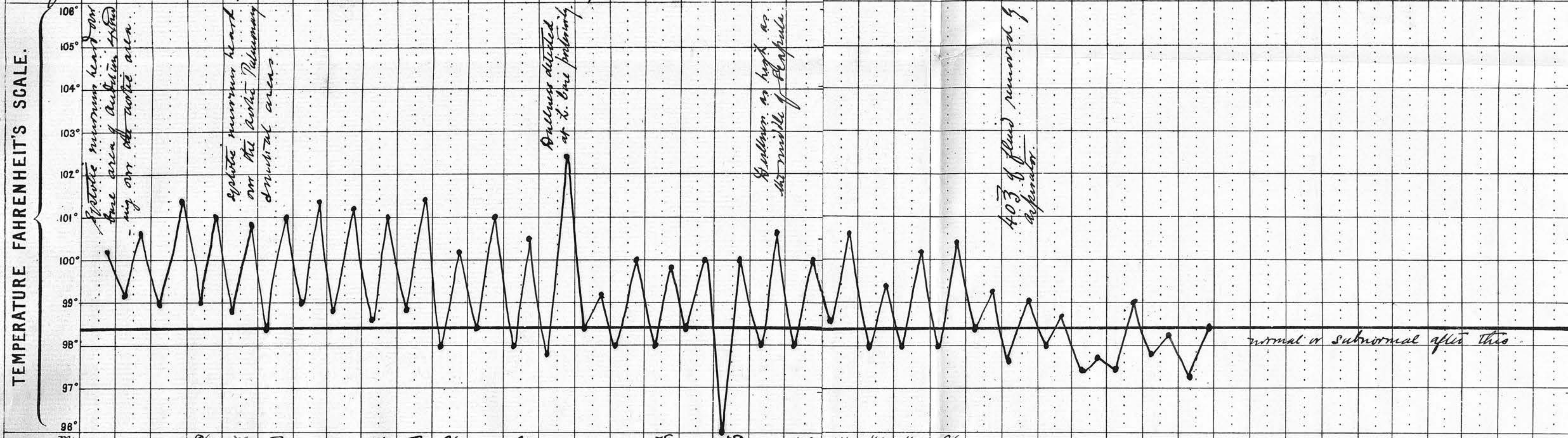
-pearing before the joint troubles, but his tongue was clean, & he had neither fever nor sweating. He remained in this state for the next two days. On the 4th day his mother told me he had said, he had "seen two mothers". That day he had cerebral vomiting & the hydrocephalic cry & soon after Cheyne-Stokes breathing. The case terminated fatally in less than a fortnight from its commencement.

Case VII H. McN. aet 13 m. Seen on 13 March 1888. He had had several spasmodic seizures attributed to teething. The gum over the two upper central incisors was cut & he continued well thereafter, no fits occurring. I saw him again on the 16th. He was still well & lively. On the 21st he appeared well also but had an occasional cough. He was stripped & examined both back & front but no dullness nor tubular breathing was detected anywhere. His pulse was 100 & on his heart a soft systolic was audible to the L of the Sternum in the 3rd & 4th interspaces. Breathing was not accelerated

Had I not been in the house it would not have been thought necessary for a doctor to see the child & it was at my own suggestion that the child was sounded. Of course I assured the mother that the child was perfectly well & free from chest disease. I further intimated that further visiting on my part was unnecessary. On the 24th I was called at 9 P.M. to see the child. It had then a temp of 104° Pulse 174. Resp. 80 - 90. The greater part of the R lung was consolidated, breathing tubular throughout, accompd numerous & dry rales. There were also areas of tubular breathing on the L. side but dullness on percussion was not satisfactorily made out. It became ill the early evening of the 23d. It died at 2 AM on the 26th 20 P.M. This is one of the most important cases I have to record & it occurring while I was writing my thesis gave it a special interest. Unfortunately however I failed to observe the advice I would recommend to others. I confess I was unsuspicious of impending disease & being a private patient

Name *Jemima Wilson* Age *9 years* Disease *Scabies & Pleurisy* Result *Cured.* Age Disease Result



May	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31
Pulse m				96	72	84			102	84	96		90				78		78
Pulse e	96	114																	
Resp. m									24	20	24		26				24		22
Resp. e	20	18		24	24	26													
Motions			1			2	2	2	1	3	1		1	1	2	2	2	2	1
Urine, ozs.																			
Sp. Gr.																			
Reaction																			
Chlorides																			
Albumen																			
Day of Dis.																			

June	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Pulse m		102	114	102	120	96																	
Pulse e			90																				
Resp. m		32.	38	20	30	31																	
Resp. e			30																				
Motions		2	1	1	1	1	2		1		1												
Urine, ozs.																							
Sp. Gr.																							
Reaction																							
Chlorides																							
Albumen																							
Day of Dis.																							

Urine, Sp. Gr., Reaction, Chlorides, Albumen: *perfectly normal throughout.*

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I felt indisposed to continue visiting when the patient seemed to all, in excellent health. This little patient was under my care in the autumn of last year. At this time developing hydrocephalus I was subject to convulsive seizures. At this time I examined him carefully & his heart sounds were quite pure. I can only regret I did not watch him for there by I might have detected the premonitory change, & tried, while it was possible to hope for success, to avert the unfortunate issue.

Case VIII J. W. aet 9. Admitted to the R. H. S. C. on 13th May 1887. Complaining of a skin eruption. In the course of the routine Systematic exam. a Systolic murmur was detected over the base audible also over the aortic area. In the evening it was also heard in the Pulmonary Every other System was perfectly normal. On the 17th a murmur was heard over the Aortic Pulmonary & mitral areas. A "Cardiac pulsation is visible in 2nd 3rd 4th 5th Space to L. of Sternum." Her temp had been a little

elevated for a few days but always fell to normal in the morning. Dr. Rayfair noted on the 21st. "Temp this continues abov normal + + Child runs perfectly well Cardiac symptoms unchanged. Pulmonary System carefully examined to day nothing detected." Although daily examined nothing was found till the 26th (i.e. 13 days after admission) when dullness was found over the L base posteriorly. On the 29th the lung condition remained much the same & the "Temp seems to be getting more settled". On the 30th the note on the heart is "No heart murmur is heard in any area". On the 1st June dullness had increased to middle of scapula. It increased & on the 8th 40 ℥ of fluid was removed by the aspirator. She improved after this & was sent out on the 30th perfectly well without any heart murmur.

I have detailed this case more than the others for it was supposed by case to be a case of Rheumatism - & it had the slight elevation of temp in favour of this diagnosis. For 13 days though

examined in bed daily — sometimes twice a day — it defied detection. The skin eruption was scabies & of course had nothing to do with the case.

In recording these cases I have stated nothing beyond the facts. I wish to emphasise Notes of Pulse rate Temp &c. would merely have added to their bulk. References to the charts wh. have been added where necessary will show these more satisfactorily than writing them out.

It is necessary that I once for all make it clearly understood that the family histories in all these cases was carefully taken & all the systems thoroughly & regularly examined by myself. In none was there any other system found affected save that, or those noted in the case.

In case I the child had had scarlatina.

In case III. there was a history of a former slight attack of chorea. but there was not the slightest evidence of it when she was in hospital.

Here then are notes of 8 cases of important diseases occurring in my own

experience, all of which presented difficulties in diagnosis when first seen. In none of them did anaemia or debility ~~seem~~ seem satisfactory explanations of the existing condition the probability of such being the cause was easily discarded on examination. I hold that a child with a healthy flesh coloured skin, red gums, lips, & conjunctivae is not anaemic & all these cases were such, with perhaps the exception of Case II where there was a suspicion of it. His skin was very white but mere whiteness of skin does not constitute anaemia. Nor do I feel inclined to attribute a murmur to debility where the child has been in robust health up till the day or so before he comes under observation.

I consider that the heart in these 8 ~~cases~~ cases was not affected otherwise than functionally, for at, & sometimes before, their termination it was found sound & healthy as far as our present means of diagnosis warrant us in giving such an opinion. This precludes the possibility of Case I having been organic

disease dating from the scarlatinal attack, & I do not presume too much if I refuse to regard the murmurs in case III as choice for the child at the time had not a single symptom. It is true survival had measles but measles even more rarely than scarlatina leaves organic disease behind, besides the same reasoning which precludes the possibility of Case I's having been organic will equally exclude organic disease in the cases which had measles.

Eight cases then in none of which was there a rheumatic or other history which would reasonably account for a murmur present; present themselves for advice complaining of no symptoms which would lead me to suspect a heart affection but generally rather vague feelings classifyable under the term "Malaise". In the course of the systematic examination these soft murmurs inconstant in situation but systolic in time is discovered but all other systems appear normal. In a short time symptoms of grave lesions appear in other organs & in the child

† This is hardly correct. When visited me he had dearborn's only and not the slightest evidence of Tubercle in the lungs. It developed soon after however. See case on page 8.

being discharged the heart is found normal. What is one to conclude? I have already given reasons why I refuse to consider this murmur either as haemic or due to debility; & from the histories of the patients ~~that~~ it is highly improbable they could be organic. We are therefore compelled to classify these murmurs as functional, unless we consider the cases I have called "pleurisy" as "Rheumatism" a plausible suggestion only in ~~so~~ so far as Rheumatism in children seldome reveals itself by much joint pain. If this sometimes affirmed, it is absent altogether, but in these cases the probability is that the joint pains have disappeared before bring the patient for treatment for the graver condition. Now had these murmurs been observed only in cases of Pleurisy I would not have been so certain that they might not be organic. But I have given notes of a case of Subacute Meningitis, of Epilepsy, of + Tubercle of the lungs (as the lungs being the organs chiefly affected) & of Pneumonia in all of which before the correct diagnosis was

Name Johan Law Age 27 Disease Typhus Result cured

Feb.	24	25	26	27	28	March 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Pulse m	120		90	120			108	120	120					132	120	130	113		100	120	96
Pulse e				128	108	108									120	144	128	128	72	96	96
Resp. m				30				40	40						40	36	36		48	36	36
Resp. e	30		30	32	28	32									36	48	28	48	30	24	24
Motions		—		—																	
Urine, ozs.																					
Sp. Gr.																					
Reaction																					
Chlorides																					
Albumen																					
Day of Dis.	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28

TEMPERATURE FAHRENHEIT'S SCALE. 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°

TEMPERATURE CENTIGRADE SCALE. 41° 40° 39° 38° 37° 36°

Pneumonia

Abortive Crisis

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made a precordial murmur was detected. It is worthy of note however that the majority of cases are cases of Pleurisy a fact I shall remark upon further on.

I wish now briefly to draw attention to four other cases – all adults – in whom a murmur shewed itself in the course of the disease.

Case IX. J. L. aet 27. Admitted to Leith Hospital on Feb 24 1886. Suffering from an attack of Typhus. Her temp was 103. P. 120 Resp. 30. Eruption copious & dusky. She was a millworker. Hygienic conditions bad. Tongue moist but fur very thick & of a dirty yellow colour. Her case was severe. On the 14th day a soft systolic was heard over the base & there was a suspicion of a diastolic over the pulmonary. Temp this evening 104°. Next morning it fell to 101°. & she took as if the crisis had occurred but in the evening her face looked heavy & dusky & her respirations were rapid. Temp 102·4. On examination it was now found that she had a Pneumonia of both bases. Her temp did

Name Mary Lindsay Age 23 Disease Typhus Result Cured.

April	30	May 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	
Pulse m		120	108		120	132	132	120														Pulse m
Pulse e	108	120	96		108		140															Pulse e
Resp. m		30	30		30	36	38	30														Resp. m
Resp. e	36	32	34		36		30															Resp. e
Motions																						Motions
Urine, ozs.																						Urine, ozs.
Sp. Gr.																						Sp. Gr.
Reaction																						Reaction
Chlorides																						Chlorides
Albumen																						Albumen
Day of Dis.	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	Day of Dis.

TEMPERATURE FAHRENHEIT'S SCALE. 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°

TEMPERATURE CENTIGRADE SCALE. 41° 40° 39° 38° 37° 36°

Abortive Crisis

Subsultus Tendinum Retention of Urine

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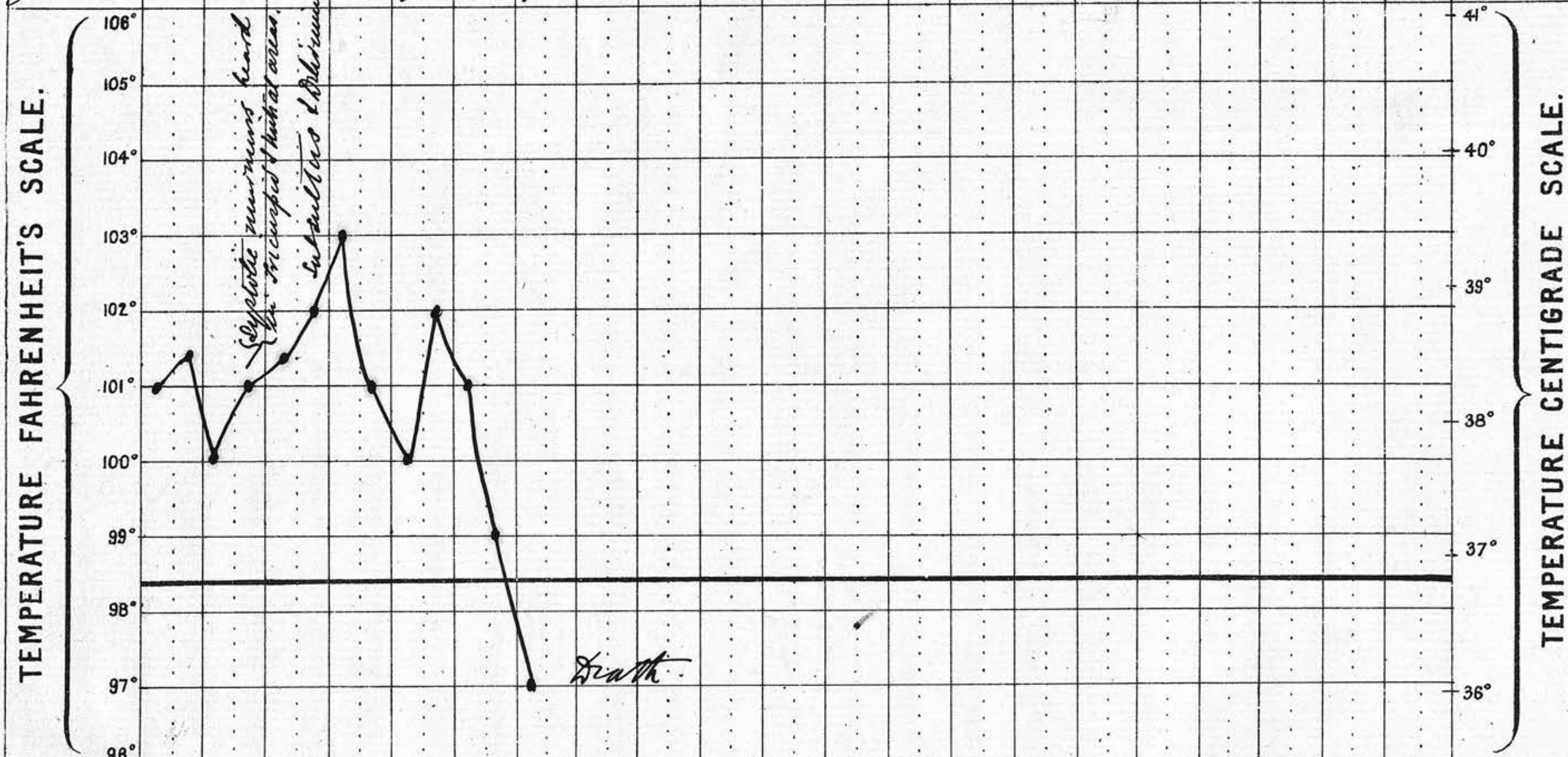
not touch the normal for 13 days. during which time her chance of recovery seemed very small indeed. Convalescence unlike the simple Typhus was very tardy. She eventually did well & was discharged cured the murmur having disappeared. 7th April - 85

Case X. Mary Lindsay. aet 23. admitted to L. H. on 30/4/85. Suffering from an attack of Typhus. She had been ill for a fortnight. When first seen her temp was 105° P. 108 R. 36. Rash slight & fading, but general condition very weak due probably to her having been going about part of the time. The discrepancy between Temp & pulse rate was due to some pulse waves failing to reach the radial. She had a Systolic murmur but having an occasional rhonchus had no further complication. She lay awake all night but was not delirious but next day had symptoms of cerebral effusion & slight "picking of the bedclothes". She now also had retention of urine. She recovered by lysis The murmur disappeared soon after her temperature became normal. Discharged 2nd June.

Name *Peter Brown* Age *60* Disease *Typhus* Result *Death.*

June	3	4	5	6	7	8	9.		



Pulse m	100	105	112	140	?	?			
Pulse e	120	128	120	108	?	?			
Resp. m	28	28	32	40	Irregular				
Resp. e	27	30	32	46	Irregular				
Motions									
Urine, ozs.									
Sp. Gr.									
Reaction									
Chlorides									
Albumen									
Day of Dis.	?7	8	9	10	11	12	13	14	15

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Case ~~xi~~ Peter Brown aet 60. Admitted to L. H. on June 3rd. 1883. Suffering from an attack of Typhus. He had been a drinker all his life & never lived properly. When first seen the attack did not seem very severe. The rash was slight. His temperature however was low, his pulse feeble & somewhat irregular. On the evening of the 4th a systolic murmur was heard over both mitral & tricuspid areas. He passed his water in bed. On the evening of the 5th he became delirious. His tongue appeared like a firm ball, dry & black. Subsultus tendinum very pronounced. He became comatose on the 8th. Lung falling & died early on the morning of the 9th.

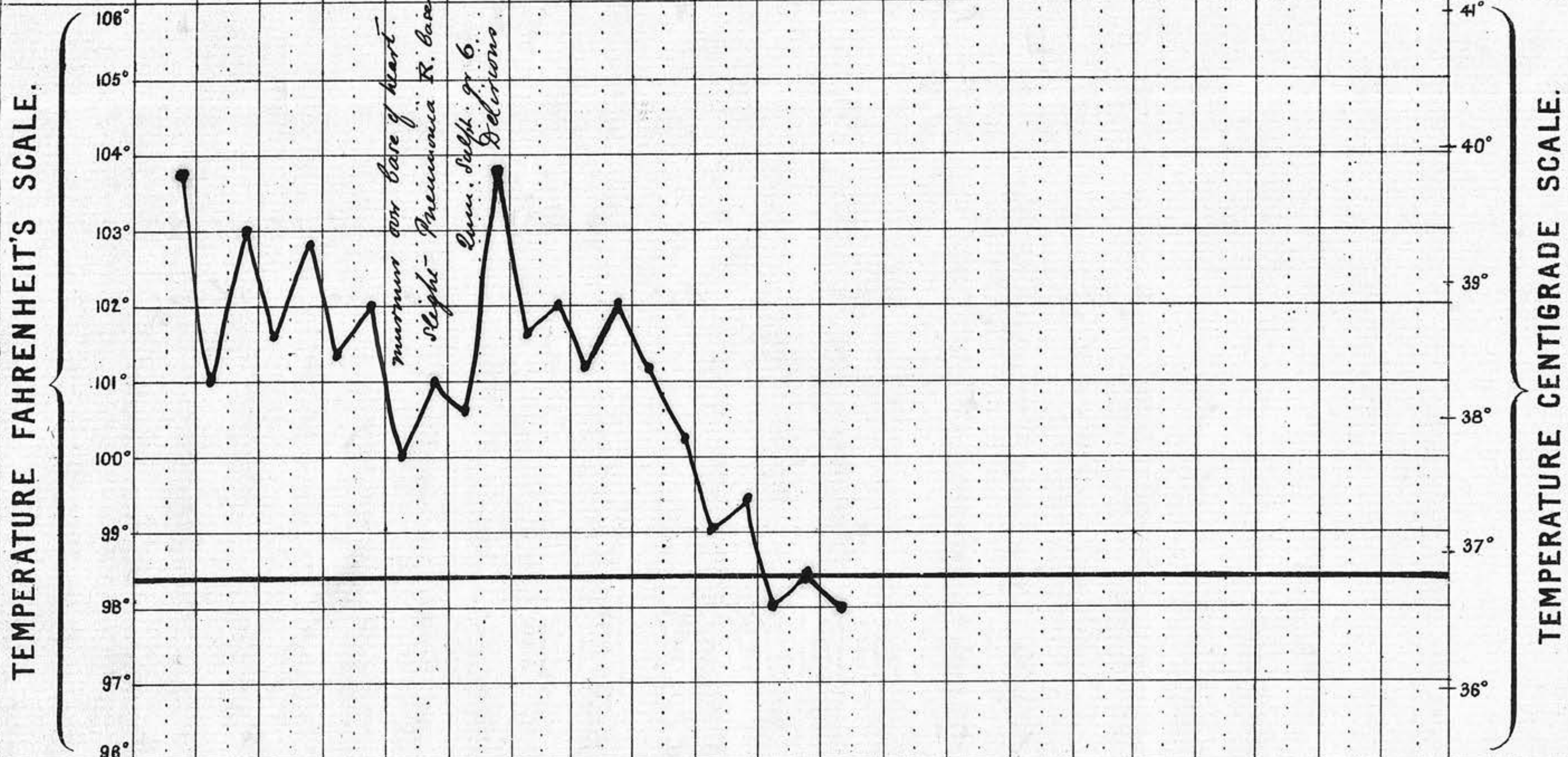
P.M. Pericardial sac contained 3i of reddish serum. Heart soft. & distended with blood, black & fluid. In the R auricle one or two small fragments of red clots were seen. Valves healthy.

Lungs Some old adhesions over both lungs. Lungs somewhat congested post - eriorly otherwise healthy.

Spleen enlarged soft & flabby.

Name *George Thomson* Age *30* Disease *Erysipelas of head.* Result *Cured*

March 28 29 30 31 *April* 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 *1883*



	28	29	30	31	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	
Pulse m		108	84	84	60	96	96	96	64	72	72											Pulse m
Pulse 6			120	108	72	108	100	96														Pulse 6
Resp. m			20	28	46	40	40	24	20	18	18											Resp. m
Resp. 6		30	30	46	48	30	46	24														Resp. 6
Motions																						Motions
Urine, ozs.																						Urine, ozs.
Sp. Gr.																						Sp. Gr.
Reaction																						Reaction
Chlorides																						Chlorides
Albumen		trace																				Albumen
Day of Dis.	2	3	4	5	6	7	8	9	10	11	12	13	14									Day of Dis.

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Lungs & Kidneys congested & somewhat cirrhotic.

Bladder nearly full

Brain. Great congestion of veins on hemispheres & cerebral fluid in excess. Sinuses full of black blood. Ventricles distended with fluid. Brain substance &c. It is chiefly the post mortem condition of the heart to which I wish to direct attention.

Case XII. George Thomson act 30. admitted to L. H. on 28th March. 1883. Suffering from Erysipelas of the face. His case presented nothing noteworthy till the morning of the 6th day when a murmur was observed over the base of the heart systolic in time & the Pulmonary 2nd seemed to close imperfectly. His temperature was 100° having been 102° the evening before & his pulse had fallen from 108 to 60. His respirations however it will be noticed did not fall correspondingly. They had been 46 the night before & they remained 46. His lungs were examined but nothing was detected. At the evening

Name Susan Reid Age 83. Disease Scarlatina Result Cured.

March	21	April 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20

TEMPERATURE FAHRENHEIT'S SCALE. 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°

TEMPERATURE CENTIGRADE SCALE. 41° 40° 39° 38° 37° 36°

Desquamation commencing on upper lip & cheeks.

Natural Pyrexia resumed.

Pulse m		108	108	100		96																Pulse m
Pulse 6	108	120	108	105	108																	Pulse 6
Resp. m		24	24	20		16																Resp. m
Resp. 6	26	26	20	20	22	1																Resp. 6
Motions																						Motions
Urine, ozs.																		20	25	16	20	Urine, ozs.
Sp. Gr.																						Sp. Gr.
Reaction																						Reaction
Chlorides																						Chlorides
Albumen	—	—	—	—	—	—	—	—		—		—		—		—		Trace	1/6	1/2.	1/2.	Albumen
Day of Dis.	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22.	Day of Dis.

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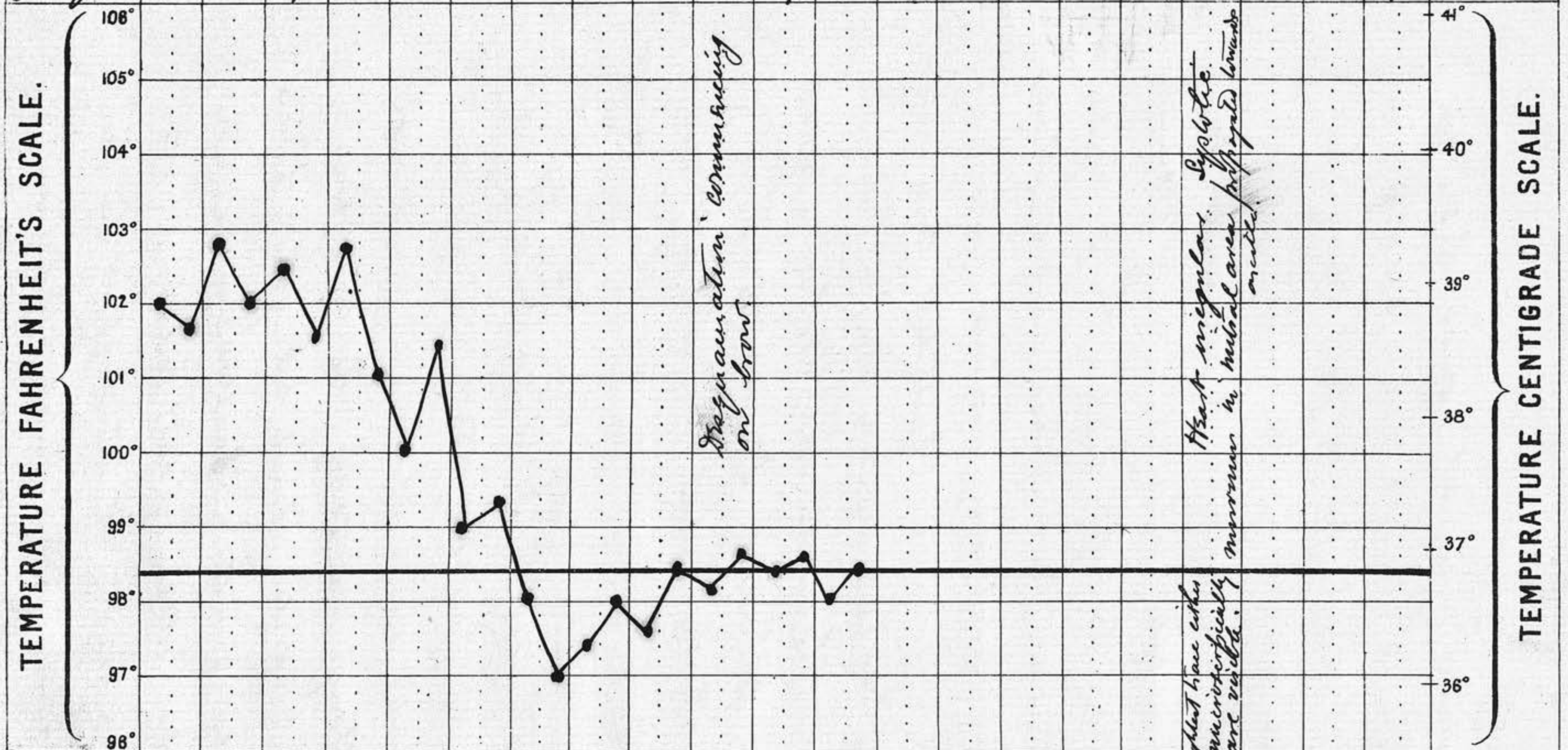
visit
x he was again overhauled & patches of pneumonia were discovered both at the apex & base of the R lung. Next day towards evening he became delirious — (the erysipelatous condition had subsided, though the face was still swollen) — with a temperature of 103°.8. He got 6 grs of Quinine & fell next morning to 101°.6 but did not become normal till the 10th day. His heart murmur disappeared before he was discharged on the 24th April-83.

I have yet a few other cases to record in which a distinct ~~murmur~~ murmur was present for a considerable time; but in all it first appeared after the acute illness was past for which the patients was admitted to hospital.

Case XIII. Susan Reid act 33. admitted to Leith Fever Hospital on March 31st. 1883. suffering from Scarlatina. Rash distinct. Throat red & inflamed & swollen. Submaxillary glands enlarged. Desquamation began on the 9th day. Urine diminished in amount somewhat opaque but free from albumen:

Name *Helen Stephen* Age Disease *Scarlatina* Result *Cured*



July	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	
Pulse m	120	136	124	140	108	84		80														Pulse m
Pulse e	126	136	136	112	116		76															Pulse e
Resp. m	30	32	32	36	24																	Resp. m
Resp. e	32	32	36	28	20		16															Resp. e
Motions																						Motions
Urine, ozs.																						Urine, ozs.
Sp. Gr.																			1026	10.24	10.25	Sp. Gr.
Reaction																			Acid	Acid	Acid	Reaction
Chlorides																						Chlorides
Albumen	—	—	—	—	—														Trace?	distinct	1/3	Albumen
Day of Dis.	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	Day of Dis.

Urine did not give the slightest trace either of albumin or blood but microscopically a few red corpuscles are visible. (written across the lower rows, July 24)

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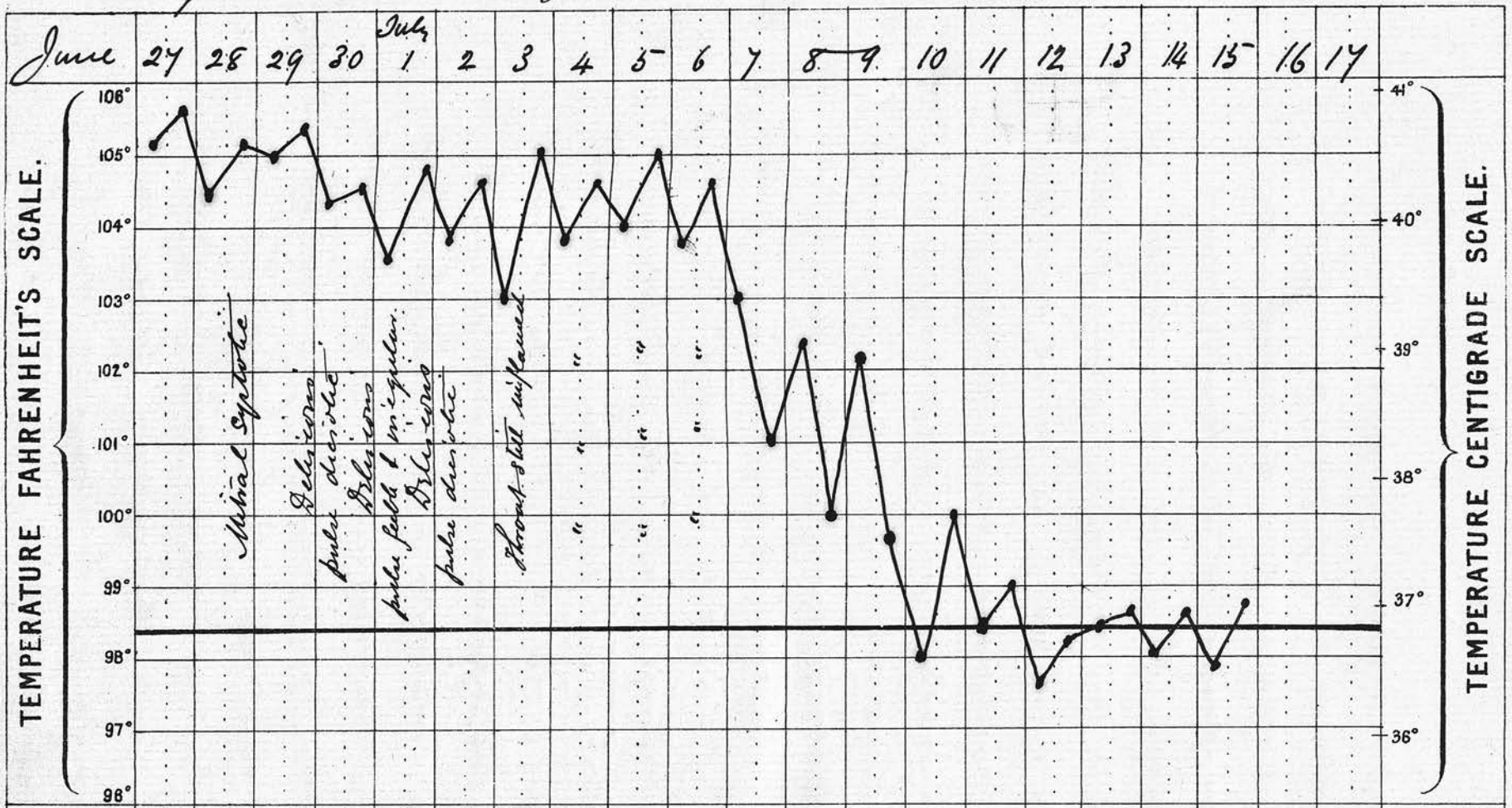
Her temperature remained normal & she did well. On the 15th day a mitral systolic murmur was observed for the first time, but nothing else was noted. She had no joint pain & the urine was normal & remained so till the 4 days after when a trace of albumin was detected. Quantity 3XX. Pulse regular & of good tension. No displacement of apex beat. The quantity of albumin increased & the urine diminished. Granular Epithelial casts were observed. Desquamation was slow in being completed. The urine did not become normal till after a fortnight. She was discharged cured, with no mitral murmur on May 18th 83.

Case XIV Helen Stephen admitted to L. F. H. on July 7th 1883. This patient had a mild attack of Scarlet fever. Rash distinct though not severe. The throat was pretty severely affected. Tonsils enlarged, palate and uvula swollen. Swallowing caused pain. Submaxillary glands enlarged, as also the lymphatics at the angle of the jaw over the Parotid. Urine tested depositing

urates, but quite free from albumen. On the 12th day desquamation began over the brow. Some days later the pulse tension increased considerably & a tendency to irregularity was observed. The apex beat was distinct & firm. On the 20th day, the irregularity still continuing, a systolic mitral murmur was observed propagated towards the axilla. The note on the urine for that day is "not the slightest trace of albumen (with cold nitric acid) nor blood (with the guaiacic test) but microscopically a few red corpuscles are visible". Next day there was a doubtful trace of albumen, but the day following it was quite distinct & a few casts - epithelial - were detected. On the 6th day after the first appearance of the kidney complications the urine was "quite normal". She was discharged cured on the 14th August the murmur having disappeared.

Case XV. Agnes Grant admitted to L. H. on June 27th 1883. Suffering from scarlet fever. This was a very severe case. Temp - evening very high 105°.2 P. 156 Resp. 40.

Name Agnes Grant. Age Disease Scarlet Fever (+ Diphtheria ?) Result Cured



Pulse m	156	168	166	2	2	2	148	156															Pulse m
Pulse 6	170	156	178	.	.	.	172	160		148	110	120		96									Pulse 6
Resp. m	40	46	66	60			48	46															Resp. m
Resp. 6	46	48	72	68			56	48		56	36	36		28									Resp. 6
Motions																							Motions
Urine, ozs.	Small quantity						10	16	19	26	24												Urine, ozs.
Sp. Gr.	1030					?	1030	1028	1029	1020	1020												Sp. Gr.
Reaction	acid					acid	acid	acid	acid	acid	acid												Reaction
~~Chlorides~~						Epithelial Casts						Fragments of casts											Chlorides
Albumen	trace					1/2	1/2	1/2	1/6	1/6	1/6	trace	trace	trace	1/6	trace							Albumen
Day of Dis.	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22		Day of Dis.

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Throat deeply congested & covered with a stringy yellowish looking material. Slight purulent discharge from the nostrils. Her urine was passed in very small quantity & was of high Sp. gr. but did not contain albumen: On the 3rd day. (i.e. dating from the appearance of the rash). a mitral systolic murmur was detected & the cardiac pulsation was much fuller & felt over a wider area than normal. Next day she was delirious Pulse very rapid & irregular & respirations very fast. There was no inflammation of the lungs as far as I could detect & I was inclined to think meningitis had set in. The cold cork was kept on her head constantly pouring iced water & her body was frequently sponged. She remained delirious for three days. The throat was treated with Borogycerid The urine was examined whenever it could be obtained viz on the 7th day. Only a small quantity was passed, which was turbid & contained 1/2 albumen besides numerous epithelial casts & granular debris. Her temp. remained up between 104° & 105° till the 12th day when a crisis occurred

This keeping up of the temperature is explained by the severe inflammation of the throat & tonsils. The urine became normal on the 18th day. ~~about~~. The heart regained its normal about a week after this & she was discharged cured on August the 14th. I have not been able to make up my mind as to whether Diphtheria complicated this case or not. The discharge from the nostrils certainly was suspicious but the appearance of the throat was not like it. There were no patches, but a good deal of stringy purulent material on the tonsils & post. wall of the pharynx. Then the tonsils ulcerated. The early appearance of nephritis cannot be held as proving it diphtheritic for it (the nephritis) "may occur any time between the 2nd & 21st day" in Scarlet fever. (Eustace Smith's Diseases of Children pg 38). & Goodhart (Diseases of children pg 139) says he "has seen a severe nephritis begin suddenly as early as the 3rd or 4th day". Then further the fact that she recovered heightens the improbability of its being Diphtheria for "these cases usually die" Goodhart (ibid page 145.).

I shall now discuss the preceding portion of my paper, stating as briefly as possible what deductions I think may legitimately be drawn therefrom

As I have recorded these cases, it will be observed I have adopted a sort of classification viz

1st. Those in which a precordial murmur preceed a serious lesion in some organ

2ly. Those in which during the acute process of some disease a murmur developed immediately before some organ became diseased and

3ly. Those in which after the acute stage of a definite disease was past a murmur developed in the heart shortly before an organ became diseased.

I admit the classification is a little artificial & that few of the cases agree entirely in the presence of a "Functional" murmur" Yet this fact of itself necessitates an analysis of the facts. As Dr Latham (Collected works pg 40) (while discussing a precordial murmur which he considered as having

a relation to Phthisis) with remarks "
there is a practical usefulness in the
mere experience of coincident facts, though
their pathological relation be not yet understood.
Thus where from my direct examination of the
lungs I cannot get beyond a suspicion of
tubercular disease, the murmur in the space
indicated must always contribute to confirm
it."

In my first three of cases Eight in number
I find that in 6 of these the murmur
preceded some lung trouble. Case II page 8
was a case of diarrhoea, but be it observed
the heart murmur which was present when he was
first in the house & which were distinct when
he went away was not present on his re-
turn some two months later. But you may ask,
why do I consider this murmur as having any
relation whatever to the Tubercle of the lungs
for which he was readmitted? Well; there are
the following facts in its favour 1st. the evidence
that a functional murmur may shortly precede organic
disease in another organ as in Cases I, III, IV
V, VI, & VIII. (This looks like a case of Petitio
Quaesiti, but I am really bringing a fact

Although I say "the amount of anaemia necessary to cause a haemic murmur" I dont wish it to be misunderstood that I believe the changes noted in the blood of anaemics are by any means satisfactory explanations of, or can reasonably account for, a murmur observed. When a murmur disappears in an anaemic after treatment I think it due to a toning up of the heart muscle & the muscles generally over the body

in support of a conclusion that has been questioned) 2ly If there was 'a degree of anæmia' as was supposed to exist in this case; is it likely that this anæmia would disappear to such an extent immediately before a phthisis developed, as to render a murmur formerly very distinct quite inaudible? Trousseau in his Clinical lectures in discussing anæmia Leucocythæmia ~~&c~~ says that after the administration of iron & the cure of an anæmia a consumption may suddenly develope & he in fact adds that the first case of Gallopping consumption he ever saw occurred in a girl whom he had rapidly cured of anæmia by exhibiting iron & then concludes with the emphatic statement that administration of iron in anæmia, & the attempt to cure such persons with a questionable history must roundly be condemned. Now I know for a fact that iron was not given this child, & if Trousseaus statement is correct it cannot have been due to that. There may have been a slight improvement in the state of the blood (not having examined it I cannot say) & this may have been sufficient to dispel the murmur for I often think that the amount of anæmia necessary to cause a haemic murmur differs very greatly in individuals, some be-

- ing deathly white, without any cardiac murmur 2 others having loud murmurs 2 ypt only a slight degree of anæmia. 3°. Though observed a considerable time before the Phthisis we must not forget that in case VIII pg. 14 the murmur preceded, to my knowledge by 13 the Pleurisy days. To return then, - 6 of these 8 cases show murmurs preceding lung disease; one a murmur before Tubercular Meningitis & one before Epilepsy. Let us consider the epilepsy first. A murmur has been observed before in this disease for Shapter (Diseases of the heart page 182) says that "murmurs are met with which apparently depend on functional disorder in the actions of the heart itself. The murmurs heard occasionally in Chorea, Epilepsy & after violent exercise are of this nature." Walshe (A practical treatise on diseases of Lungs & heart pg 617.) also refers to murmurs (dynamic) "not only in chorea but in other nervous diseases". But I have not met with any reference to a murmur such as is recorded in case V page 10. The murmur "was not constant" but "preceded the fits" It is also stated that she felt "precordial pain" before the fit & knew it

was imminent. It was her "aura", but the murmur was the sign which told us when it threatened. It therefore could not be due to the violence of the muscular contractions & must probably have some nervous cause. Because however I shall not try to explain. I merely record the fact that it regularly preceded the fit, as reliably foretelling us as the pain did her. I cannot see why the impending perturbation in the central nervous system should not manifest itself in this way, for we know how easily our hearts are influenced by our emotions. The degree to which a heart may be influenced must necessarily vary with the constitution of the individual; but it must occasionally be very great for Latham (op. cit pg 37) records the case of a young woman where after an attack of poignant grief, he discovered that "her heart was contracting with excessive force and with the loudest possible bellows murmur. The next day the heart was beating quietly & the murmur was gone".

I shall now take up the 6 cases wherein a murmur was detected before disease of the lungs manifested itself in other ways.

I have already discussed the improbability of the murmurs in these cases being due to organic changes at the valves. If this is not granted they then fall into the rare class where organic murmurs entirely disappear occasionally for considerable intervals. (I have only seen one case of organic heart disease, a boy of 11 with mitral stenosis; in which the murmur disappeared. Some days it was present, some days it was not, but at any time it could be made very distinct by making him run up stairs.) I can find very little information on the subject of intra uterine endocarditis but what I have seen leads me to conclude that writers on diseases of children seem to think it may account for (a) some of the malformations met with & (b) for some of those heart cases where the child dies early, never having suffered from any known exciting cause since birth. It is at once evident that my cases cannot fall under this class, so the only remaining question is; were they produced artificially? I feel satisfied myself that this was not the case. Sir T. Watson (Principles & Practice of Physic pg 254 vol II) says "You may even

make a temporary bellows sound by forcibly pressing your stethescope on the praecordia, especially in children in whom the ribs are pliable & yielding And you may fall into errors of diagnosis if you are not aware of this." Is this really a fact? I cannot say I have found it so; & Latham (op. cit pg 38) talking of the possibility of so producing a murmur; records one case, but adds "never indeed, the chest being not deformed, never but in this single instance have I produced a murmur simulating that of valvular disease" & he repeats this statement again on the same page (38). (Forcible pressure with the stethescope does produce a "bellows sound" but it emanates from the larynx & not the heart & is attributable to quite another cause.) You can by "forcible pressure" make the pulsations of your own carotid, self audible & sometimes if out of sorts, they are so prominent as to render fallacious all observations for the time being, but how a normal chest pressure could produce a murmur I cannot understand, So useless have I found firm pressure that I have never yet been able to decide by this means, in doubtful cases of Pleurisy though it is recorded in text books as an important means in differential diagnosis.

It is not surprising that so many of my cases were of lung affections considering the intimacy that exists between the two. J. C. Williams (Practical Remarks on Palpitation & other Functional Diseases of the heart pg 65) says "The reciprocal influences of the heart & other organs or systems of the body are extremely numerous ... Hence we can readily understand how any disturbance of the circulating fluid in any important organ may by disturbing the balance disarrange the function of the reservoir. & from this circumstance we may be prepared to foresee that many derangements of the heart's function which at first sight appear merely sympathetic are virtually produced by the disturbance of the circulation at some point more or less remote". Let me examine the four cases of Pleurisy first. When one considers the great extent of surface affected by the inflammation we cannot doubt that this peripheral irritation must have a definite reflex expression. (Dr Todd in his work on pain has enumerated many beautiful instances in all parts of the body) Besides clinical experience of inflammations of other serous sacs shows they profoundly affect the heart. The pulse in peritonitis is characteristic of that disease alone, and in

Bright's disease the alteration in the pulse is frequently the first symptom that warns the practitioner of a suddenly developed Pericarditis. I mention these two instances to shew that serous inflammations undoubtedly influence the heart very directly. Now the Pleura is a large serous surface which when inflamed causes a series of very definite changes in the lung & chest wall. These are briefly as follows; whenever the pain is felt, the chest is fixed to reduce the irritation to a minimum. Absolute fixation is impossible but is attained as nearly as possible. Now if a pleurisy is carefully watched from the first, it will be found that the chest is kept unnaturally full of air. i.e. the desired fixed position is Inspiration (I am talking of Pleurisy without effusion). The reason of this is not far to seek. Could the patient voluntarily stop using one lung he would do so, & free himself from pain by avoiding friction; but the increase in temperature, meaning an increased metabolism & therefore an increased waste, necessitates an increased activity in the part of the lungs; therefore that state of the lungs which exposes the greatest surface of epithelium to the air, through which an escape of waste matter may be effected

is that involuntarily chosen & that position is Inspiration. Once the position is attained, it is kept as far as possible — (for the reason afore given, viz the diminution of pain) — & future respiratory acts are small & chest expansion is reduced to its lowest. This is further possible, for by fixing the chest as nearly as possible in Inspiration, we have an increase in the amount of residual air, through which exchange of waste material is to take place. But this alone will ~~not~~ not square matters. Additional work has to be done & the consequence is the rate is increased. The fixing (or nearly so) of the chest then in Inspiration, brings two new factors into play, each affecting the heart directly. Primarily we have an increase in the suction action of the thorax when the chest is raised, & when this position is maintained the great veins at the upper part of the thorax have more room to expand & so become overfull. Secondarily we have lost the elastic recoil both of the chest wall & of the lung, two factors which materially aid the return of blood to the Left Auricle. This means that the lung on the affected side becomes hyperaemic & the Right side of the heart overfull.

TABLE OF CASES

SHEWING

Diseases, Murmurs, Situation, Propagation. &c

No. of Case	Disease	Murmur Situation and Propagation	Other Facts.
I	Pleurisy	Systolic Mitral having its point of maximum intensity in the 4th L. interspace. Propagated for one inch from Apex towards axilla.	Left side of chest affected.
III	Pleurisy	Systolic over base. 1st sound in mitral area ringing	Right " " " " .
IV.	Pleurisy.	Systolic over base, also audible over Aortic and Pulmonary areas.	Right " " " "
VIII	Pleurisy	Systolic over base area of audition extends over Aortic area. Hard Cats or in case over Pulmonary & mitral as well.	Left " " " "
VII	Pneumonia	Systolic to Left of Sternum in 3 & 4th. interspaces	Right " " " "
II	Tubercolosis of Lungs.	Mitral Systolic propagated a considerable distance towards axilla but not heard in 3 & 4th. interspaces to L. of Sternum.	Both Apices.
V	Epilepsy.	Mitral Systolic	Murmur only distinct immediately before & for a short time after the fit.
VI	Tubercular Meningitis	Mitral Systolic but not heard in 3rd & 4th. interspace to L. of Sternum.	
IX	Typhus.	Soft systolic over base of heart & a suspicion of a diastolic over the Pulmonary	Pneumonia of both Bases.
X	Typhus.	Mitral Systolic.	Cerebral effusion Paralysis of Bladder.
XI	Typhus.	Systolic murmur over Mitral & Tricuspid areas.	Paralysis of constrictor fibres at neck of bladder. Cerebral Effusion.
XII.	Erysipelas of face.	Systolic over the Base. 2nd Sound over Pulmonary but not close properly.	Pneumonia at apex & Base of Right Lung.
XIII.	Scarlatina	Mitral Systolic	Slight Attack of nephritis
XIV	Scarlatina	Mitral Systolic	Very Slight Nephritis.
XV	Scarlatina (very severe).	Mitral Systolic	Delirium for 3 days Sharp attack of nephritis

As a result of this we have an increase in the safety valve action of the Tricuspid. Now when I first observed these murmurs I felt disposed to look upon them as Tricuspid regurgitant murmurs, but on further thinking them over it seemed improbable that the mechanical explanation though very simple was correct. It presupposed an obstruction in the pulmonary circulation of some magnitude & one would have expected, that such obstruction would have been detected by the methods employed in physical examination & this as I have shewn was not the case. Further, if this murmur is purely dynamical there ought to be the other evidence of an accentuated Pulmonary Second Sound for as G. W. Balfour has pointed out (Edinbr. Medical Journal June 1874), this accompanies disease of the lungs & if there is no heart disease, may be interpreted, if found, as pointing to lung disease. Upon looking over my notes of the first eight cases (for it these I am discussing at present) I find that in one only was the 2nd sound accentuated, though in another it was reduplicated. If it be held that this hyperæmia in the lung corresponding to a Pleurisy — a hyperæmia which is more passive than active —, I

A. It is almost unnecessary to say this murmur did not vindicate Principia Studies

Leg, if it be held that this should cause accentuation of the Pulmonary 2° how much more ought the active congestion of a commencing Pneumonia be expected to occasion it. Now this was not the ~~case~~ fact in the case of Pneumonia I have recorded. If this discrepancy — the want of accentuation of the Pulmonary second sound and Tricuspid regurgitation — could be explained away; there are other difficulties to account for. Let us examine the murmurs & their situation (See table facing page 39). Look at the first four cases, all Pleurisys. In the first the murmur is Mitral, in the next 3 it is over the base. A In the first it is loudest in the 4-5 L. intercostal space & is propagated a little way towards the axilla. In three heard over the base, in one it was not conducted at all in another its area of audition extended over both aortic & pulmonary areas & in another it extended over the aortic, then pulmonary & Mitral in the case could be heard over every valvular area except the Tricuspid. In two instances the Right Pleura was affected & in two instances the left. The case of Pneumonia was heard in the Left 3rd a systolic interspaces. (When I use 3rd interspace I mean to include costal cartilage as well). This might be

regarded as indicating Tricuspid regurgitation.
In the case of Tuberculous of the Lungs I suppose we must conclude that there was regurgitation through both Tricuspid & Mitral orifices, and if so the murmur heard in the case of Tubercular meningitis must be similarly accounted for. The murmur in the Epileptic was a simple Mitral Systolic, appearing along with precordial pain shortly before the fit & disappearing soon after it had passed. With such a record of facts one cannot with justice speak positively & it is not right to theorise on so few cases. Many of the murmurs occurred in the 'region of Romance', and to my mind are not satisfactorily explained by either Balfour's or Russell's theory, if we adopt the purely dynamical explanation. Firstly how is the Basic murmur (that explained in 3 of the cases of Pleurisy? The murmur in the first case with some show of reason might be explained by Tricuspid regurgitation, but if Tricuspid regurgitation is to account for the other three, it can only be done by supposing them conducted upwards by the Sternum. Perhaps this is crediting the 'sounding board Sternum' with too musical qualities; perhaps not, for Tricuspid regurgitant murmurs are conducted generally across

the sternum & it may be that in some conditions of the heart & thoracic wall, it may be conducted upwards instead. I have no minute notes of the physical conditions in my cases. But even supposing these murmurs are not Presystolic in origin they cannot be accounted for by the Pulmonary Valve or aortic. I have already shown (pg. 37 & 38) how the R. side of the heart might become overfull through a mechanical disadvantage, but this very mechanical imperfection while overfilling the Right, underfills the L side. Now it is necessary to suppose a "dilated L. auricle" sufficient to cause a slight amount of "constriction" of the Pulmonary to explain a basic murmur by Dr. Russell's theory; and this is a condition we have not got Dr. G. W. Balfour explains this Basic murmur by attributing it to mitral regurgitation, conducted to the anterior wall of the chest by the dilated appendix of the L. auricle. It may be due to these causes but we cannot obtain the necessary conditions by the mechanical way I have described. We are now in the following position. Four cases of Pleurisy have been described in all of which before the disease could be diagnosed a systolic murmur, variable in

A. Balfour supposes that "the fluid veins formed in the early stage of chlorotic regurgitation are of low tension & but little force hence the vibrations they originate are but slightly propagated to the L. auricle, only with difficulty from it to the chest wall in the mitral area, where they are heard as an impure first sound. But on the other hand these vibrations are readily communicated to the wall of the auricle, on which these fluid veins impinge & are easily transmitted to the chest wall with which the auricular appendix is in contact, becoming audible in the auricular area as a distinct murmur." If this be so (and there are many authorities who agree with Balfour) why cannot the same reasoning be applied to the R. side. The R auricle & appendix are just behind the body of the sternum. A slight amount of tricuspid regurgitation could then be only distinctly audible over the base of the heart, and a greater regurgitation would be audible in the tricuspid area. If this be allowed it is possible to account for the murmurs observed in the cases I have recorded

position & functional in nature, was detected. In one of the cases it seems possible to account for it by Tricuspid Regurg. occasioned simply by mechanical obstruction in the lungs, but in all the other cases this will not account for the murmur found. It therefore ~~seems~~ appears that the mechanical theory must be set aside, or least so far as it causes the Incompetence. And if we discard this we are left with a defective or altered innervation as the cause. If we suppose a dilatation of the L. Ventricle, A & R to which succeeds a widening of the Mitral & Tricuspid orifices & consequent regurgitation this possible to account for most of the XV cases, although the exciting cause of this altered innervation & subsequent dilation must differ materially in many of the cases. It must be different in the Pleurisy the Pneumonia, in the Pneumonia & other the Tubercular affections, Typhus or Scarlet Fever. Admitted then that the dilatation theory is the more rational of the two whether is Balfour's or Russell's theory most correct? The one supposes a 'dilated auricle', 'constricting' the pulmonary the other a 'dilated appendix coming in contact with the chest wall. There is anatomical evidence

that the auricular appendix, frequently shews itself very slightly in health & it is possible to suppose it coming easily in contact with the chest wall & from its anatomical structure one would expect it to dilate sooner under pressure than the auricle itself. Further I have seen cases of empyema & pleurisy with such cardiac displacement that the apex beat was felt beyond the R nipple & yet the most careful auscultation revealed nothing unusual in the heart sounds how in these Empyemas there must have been that straightening of the Pulmonary artery which Dr. Russell supposes & that too to a much greater extent than a slight dilatation of the left auricle is likely to occasion. So far I have not been able to decide which is the more correct. There is a good deal of theory in both; but if I attempt to get any help from my cases I am almost compelled to adopt Balfour's explanation. I say almost, for I have still some difficulty in understanding why mitral regurgitation should manifest itself at one time by a systolic murmur in the mitral area & at another by a purely basic systolic. Still though this is not very clear, it seems more

possible (& therefore probable) than Russells, for in his ~~case~~ theory there is a Physiological fact to disprove before it could be accepted. Thus to my mind he has not explained. In Foster's Physiology (3rd Ed.) Page 150 a description of the passage of the blood through the heart is given a description with which all physiologists & physicians agree. It is briefly this, that while the ventricle is contracting — during which interval a systolic murmur must occur — the auricle is only dilating. Now Russell would require to have both auricle & ventricle distended with blood at the same time & further, that the auricle should remain sufficiently large to allow the calibre of the Pulmonary artery, a physiological impossibility. On this account Balfour's theory seems the more probable of the two. Though I discussed anaemic murmurs as a possible cause in an earlier portion of this paper, I have no belief in them if it is attempted to explain them by an altered condition of the blood. We have no evidence that the blood even in the most depraved conditions differs in the slightest degree on one side of the heart from that on the other, otherwise than that it contains more oxygen on the one & more carb-

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Foster's Physiology. 3rd Ed. pg. 309 "The fundamental difference between venous & arterial blood is in the relative proportion of the oxygen & carbonic acid gases contained in each + + + pg 310. This is the real differential character of the two bloods all other differences are either, as we shall see to be the case with the colour, dependent on this, or are unimportant and fluctuating. From 100 vols. may be obtained

	of oxygen	of carbonic acid	of nitrogen
of Arterial blood	20 vols	39 vols	1-2 vols
" Venous "	8-12 "	46 "	1-2 "

B. My clinical experience of "functional" murmurs in anaemics is given at the top of page 32 & bottom of page 31; and supports the statement I have just made. I have at present under my care a young woman, large & flabby, who has systolic murmurs audible over any valve, not all at one time but occasionally one & then another & yet the blood in this girl does not seem unnatural in the slightest degree. She menstruates regularly, is pale with a brunette complexion & not any evidence of organic disease, nor has she any history of predisposition nor of diseases which cause heart affections.

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-onic acid on the other. X ~~If~~ But supposing this does change the character of the blood, which-ever side of the heart we are examining has only a pure current running through it & is always the same. If we had a mixture I could suppose the addition of oxygen were sufficient to change the density of the blood from that containing excess of carbonic acid then we might account for these fluid veins or currents which are supposed to be the cause of all murmurs. But seeing that the blood on either side of the heart consists only of one kind it is different to explain how fluid veins can originate, in what, I may for the moment be allowed to call, a

B homogeneous fluid, unless there is some alteration in the containing cavities & orifices through which it has to pass. Anaemic murmurs must, I think, be explained by heart changes and I think the theory which supposes any incompetence from yielding of heart wall is at present the most satisfactory. In anaemics this is due partly to impaired muscular nutrition & partly to defective innervation, but in the cases of pleurisy, pneumonia, tubercular meningitis & epilepsy I believe it is purely

nervous. In the case of Epilepsy, none will doubt this. It occurs before the fit, along with the subjective aura, precordial pain. Had it occurred after one would have supposed it partly due to the violent muscular contractions but muscular contractions & general convulsions cannot occasion a murmur before they occur. I have shown how pericarditis & pneumonitis affect the heart & that very early sometimes; why should not pleurisy & meningitis do the same.?

I shall now briefly discuss the murmurs which developed in the Erysipelas & three Typhus cases I have recorded, and am prepared for the statement that all there are simply murmurs of debility. It is universally stated that murmurs heard in the course of acute fevers are due to debility, excluding of course those which are occasioned by organic changes as in Rheumatism. This may be so in many cases, but I think not in all. During my 15 months residency in Leith Hospital I had considerable opportunity of observing fevers for the city fever Hospital is under the care of the Leith Hospital medical staff. The cases I saw were chiefly Typhus & Typhoid but there were also a good many Scarlatinas & some Measles

I also saw 2 cases of Ague but in none of them was anything found wrong in the heart. I cannot say that systolic murmurs are very common. Such is not my experience. A muffling & general want of definition in the sounds is very common, in fact in an average case is generally present in Typhus for a day or so before the crisis & in Typhoid any time after the first week, but what could conscientiously be called a murmur is not very common. I think there is a good deal of truth in what Shapples says:- (Diseases of the heart page 30) "A naturally weak heart, or a heart in which the ventricles are hypertrophied will frequently in agitation generate sounds or so modify the valvular sounds that these are to a certain extent masked, the sounds become hurried & intensified & confused & might in a hasty examination be regarded as being a murmur. In agitation, is an important point for fever patients unless unconscious are very easily excited, more especially if women, and in making the above statement regarding the frequency of murmurs in fevers there must be borne in mind. Further the severity of the attack should be considered. The statement I have made above is simply my own experience.

The cases were numerous, some mild & some severe, & were as occasionally to prove fatal. In recording these cases the points I wish to emphasise, are 1st the sudden occurrence of what, without scruple, one could call a murmur & 2nd the relation as regards time that this murmur had to some intercurrent complication. In case IX pg. 20 a systolic murmur was observed over the base on the 14th day. On the morning of the 15th there is a slight fall in temperature, suggesting the expected crisis, but it rises again thus evening & a double pneumonia is discovered. In case X there was a systolic mitral & in case XI there was a systolic over both mitral & pulmonic areas & in each case it preceded cerebral effusion. Case XI died & the P.M. showed a soft flabby heart, but free from valvular disease. (page 22). Case XII pg. 23. had a basic Systolic preceding a pneumonia. The presence of a heart murmur (unmistakeable I mean) in either Typhus or Typhoid makes the prognosis much graver. But you may say; this still does not make the murmurs you have observed other than debility murmurs. True, it indicates debility but more than that. The debility it betrays

was due to the accession of an intercurrent complication. If we look at the charts at pages 20, 21, & 23, it will be observed that the temperature fell disarming suspicion of any untoward development. My impression is that the heart murmur in these cases is a result but a cause of the complication & that when a murmur develops in the course of an acute illness, more especially if it does so suddenly it will frequently be found the precursor of some important complication. As is shown in these cases may manifest itself before the other diagnostic signs of the special disease are detectable. It will be seen, therefore, that I regard these murmurs as reflex. I believe they are instances of impaired innervation, of cardiac paresis, & this is possible for loss of power was observed in other viscera in these cases, by the two cases in which paralysis of the bladder was observed.

I have still to consider the Scarlatinal cases. I believe that these were all instances of mitral regurgitation but whether they are to be explained by the increased blood pressure which occurs in disease of the kidney or by a supposed dilatation of the ventricular wall & consequent incompetence I would not like to say. Had it

A. There is no mistake about the mitral regurgitation in the Kidney cases. The only difficulty is to account for the murmurs heard in the chest cases. The second part of the note opposite page 43 would explain them. The absence of venous pulsation may be said to disprove tricuspid regurgitation, but it is only present in well marked cases; being absent in slight degrees of incompetence (Hilton Fagge)

been due to pressure one would have expected ~~either~~ an accentuation of the aortic 2° sound. This was not observed. In discussing Scarlatina Goodhart (Diseases of Children pg 141) says with regard to the onset of nephritis "the evidence of cardiac disturbance is indeed often striking. + + + + + + + + the first sound may be thick & murmurous or accompanied by a distinct systolic apex bruit & the second sound is accentuated" He further says "The urine quickly presents characteristic appearances" I suppose by this he means us to understand that the cardiac symptoms may precede the urinary. If so I shall feel more confident in the accuracy of my observations.

I have now detailed & discussed my cases ~~but have not yet referred to~~ & it is evident I regard the murmurs in a slightly different light from that in which "functional" murmurs are usually ~~looked upon~~ viewed. In the pleurisy Pneumonia's & Kidney cases, it is not possible this cardiac change may have been brought about with salutary intent[A] may the incompetence not represent an effort on the part of nature to spare & save a damaged or diseased organ by allowing an escape of blood back into the

circulation, instead of pumping it on with increased rapidity into a disabled organ already gorged & so aggravating matters there? The early appearance of the murmur would seem to justify this. In the first 8 cases (if we exclude no. 2) we can hardly set the heart down as debilitated. Actual disease had not begun. If we look on the murmur then as representing a sclerotic incompetence I must explain my case of Epilepsy by supposing the attacks to be occasioned by cerebral congestion. A few more observations on the behaviour of the heart in Epilepsy might help to clear up the very vague pathology of this disease. I would not push my explanation of this functional murmur in the Syphus & Erysipelas cases for there are other causes to account for cardiac weakening; but I think it will bear investigation in the nephritis cases. In similar cases concurrent observations on the bowels, skin & lungs would be of great service.

On looking over the literature of this subject I find that Latham (Collected works page 40) has recorded a murmur over the 2 & 3 Left costal cartilages & 2" Left interspace "either in

A. I think I have hardly made this statement clear enough. Cardiac Irregularity, & increase in pulse tension are recorded by all writers as frequent indications of commencing kidney disease; but Goodhart (if I understand his language correctly) is the only writer who indicates that a distinct murmur may occasionally be the first circulatory symptom of kidney affection & that it may occur before urinary changes are observed.

those who were undoubtedly consumptive or in those who were too justly suspected of being so."
I have already quoted Goodhart's observation pg 57. A few writers recognise the cardiac changes in nephritis but he is the only writer I am acquainted with who indicates that cardiac changes may be detected before the urinary.
The Practical Value then of these observations is: That a murmur, functional in nature is frequently met with — probably more frequently in children than in adults —, in patients complaining, but in whom the most careful physical examination reveals nothing: That this murmur generally precedes the disease by a short time but may even be detected for considrably longer periods: That it disappears soon after, sometimes even before he is actually recovered & does not return: & That it is probably most frequently met with in acute diseases of the lungs and kidneys.
Certainly in the cases I have recorded, the results of a careful physical examination were entirely negative. One dare not diagnose a pleurisy because a pain is felt in the side nor a tubercular meningitis because a child

appears to be a little out of sorts but taking its food. The outpatients room is a good field for observation, but I expect the private physician will come across more cases. I conclude my paper by quoting from Dr. Russell's Observations on diseases of the Heart "The constancy with which the heart participates in and indeed is the leading factor in the production of ~~debility~~ the subjective phenomena in debility from whatever cause it may originate seems to be but imperfectly realized & does not obtain that careful consideration which apart from its intrinsic interest is demanded by it, seeing the organ holds the foremost place in what has been aptly termed the 'Lispod of life'. In the debility accompanying even brief pyrexia the organ presents very manifest evidence of its sympathetic response to even slight disturbance of the normal health. The suddenness with which it occurs & the like suddenness with which it disappears on the subsidence of the condition producing it show very strikingly the readiness with which the heart is affected by conditions which may in themselves be regarded as practically unimportant. (pg 31)."

I have referred to the following works but found no record of similar experience save in Latham

Bramwell Diseases of the Heart Edin. 1884.
Foster M. Physiology. Lond. 1879.
Smith Eustace Diseases of Childrn Lond. 1886
Goodhart C.J. Diseases of Childrn Lond. 1885
Bartholow R. Practices of Medicine Lond. 1881.
Watson T. The principles & Practice of Physic Lond 1857
Latham. Collected Works Sydenham Society

and Goodhart.

I. Trousseau's Lectures on Clinical Medicine

II Swan (Joseph) An essay on the connection between the action of the heart & arteries & the functions of the nervous system. Lond 1829.

III Williams, (James Calthrop) Practical Observations on Nervous & sympathetic Palpitation of the Heart. Lond 1836.

IV Shapter (Thos) Notes & observations on diseases of the heart & of the Lungs in connection therewith. Lond 1874

V Williams (John Calthrop) Practical remarks on palpitation & other functional diseases of the heart 2 Ed. Lond 1832.

VI Moodie (John) Sounds of the heart. Edin. 1830.

VII Morison (Alex). Observations on some points in Dental valvular disease of the heart. Ed. 1880

XI Balfour (G.W). A Lecture on the diagnosis of disease of the heart. Ed. 1874.

XII Balfour (G.W) Diseases of the Heart. Lond. 1876

XIII Russell. Diseases of the heart. Edin. 1886

XIV Flint (Austin) A practical treatise on the diagnosis & pathology & the treatment of diseases of the heart. Phil. 870

XV Walshe (WH). A practical treatise on diseases of the Lungs & Heart Lond. 1854.

XVI Corvisart (Hobbs' translation) Treatise on Disease of the heart & great vessels 1813. Lond.

XVII Stokes (W) The diseases of the heart & Aorta. Dublin 1854