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SIX CASES  
of  
ACUTE DILATATION OF THE HEART OCCURRING IN CHILDREN  
(5 of rheumatic origin, & 1 probably post-enteric)

With  
Remarks on the Diagnosis, Prognosis, Causes, and Treatment  
of the Affection, its consequences as an unsuspected  
cause of cardiac disease in adult life, and, a  
discussion of the relative value of super-  
ficial and deep cardiac dulness.

%%%%%%%%%

A THESIS  
For the degree of Doctor of Medicine in  
UNIVERSITY of GLASGOW.

%%%%%%%%%

By  
Agnes F. Blackadder, M.B.,Ch.B.

.....

September, 1901.

.....

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6.

ACUTE DILATATION OF THE HEART  
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CASE I

LEONARD B -----, AET 5½

A CASE OF ACUTE DILATATION OF THE HEART ,

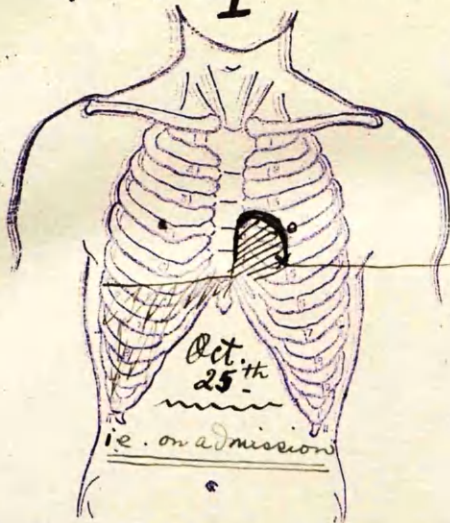
WITH PYREXIA, OF OBSCURE ORIGIN.

*Diagrams and Temperature Records.*

{ Narration of case notes.  
Summary of case, and Remarks.  
Diagnosis.  
Etiology, toxin, in operation.

Superficial or Absolute Dulness.

I



Measurements { R. border -> just to l. of mid-line  
L. border -> within nipple line

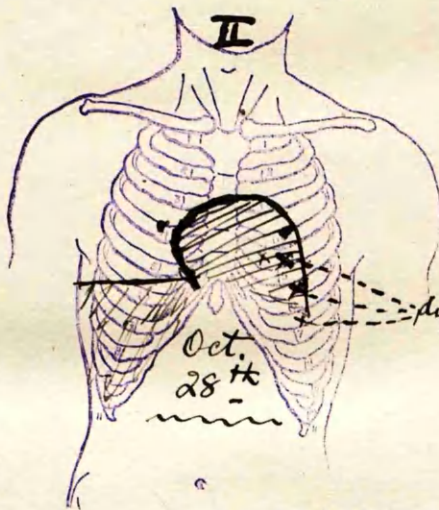
no murmur present.

apex beat in 4<sup>th</sup> space, within nipple line.

I

Oct. 25<sup>th</sup>  
min  
ie. on admission

II



Measurements { R. border -> 1 1/2" to r. of mid-line.  
L. border -> 3 1/2" to l. of mid-line.

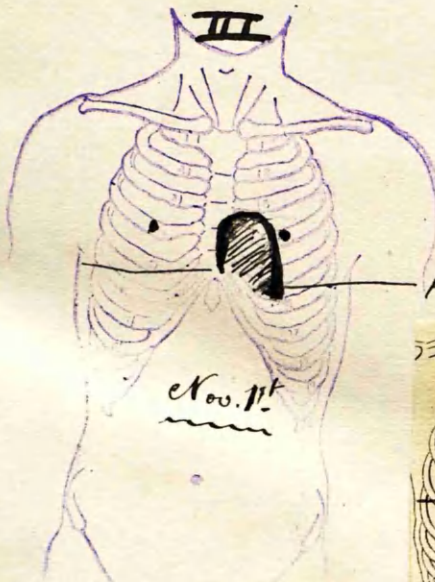
Soft systolic murmur at apex & aortic area.

diffuse wavy impulse 4<sup>th</sup>, 5<sup>th</sup> & 6<sup>th</sup> spaces.

II

Oct. 28<sup>th</sup>  
min

III



Measurements { R. border -> at the mid-line  
L. border -> within the nipple line

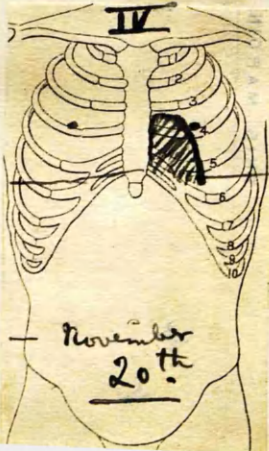
murmurs, systolic, inconstant.

apex beat in 5<sup>th</sup> space, in nipple line.

III

Nov. 11<sup>th</sup>  
min

IV



apex beat in 5<sup>th</sup> space, displaced slightly further to the left than on admission.

IV

November 20<sup>th</sup>



A CASE OF ACUTE DILATATION OF THE HEART, WITH PYREXIA,  
OF OBSCURE ORIGIN.

Leonard B--, aet 5 $\frac{1}{4}$ , was admitted to the Belgrave Hospital under the care of Dr Ewart, on October 25th 1900, complaining of shivering, nausea and general malaise.

HISTORY OF PRESENT ILLNESS. On the evening of October 23, (two days before admission) the child shivered and complained of a generalised headache. During the two following days he felt cold and retched several times, but did not vomit. At the same time a slight cough was noticed. The mother stated that some 16 days previously the boy had fallen on his head & that he had frequently since complained of pain over the occipital region.

PREVIOUS HISTORY. The child had always been healthy. He had whoopingcough at three years old, and measles. He had not suffered from scarlet fever, rheumatism, sore throats or "growing pains".

FAMILY HISTORY. Nothing of importance was elicited. There was no rheumatic fever in the family.

PRESENT CONDITION. (morning of October 25). The child was very pale, with dark rings under the eyes, but looked bright and cheerful, sat up in bed and said he felt "quite well". There were marks of recent herpes on the lower lip and chin on the right side. The body was well nourished, of square build, with short neck & broad chest, and without trace of Rickets. There were a few enlarged glands under the jaw, but none elsewhere. No erythematous rash either on the trunk or limbs was present. There were no swellings of the joints, nor were there any rheumatic nodules present. The temperature was 100.5° F.

Cardio-vascular System. Pulse--112; regular; of moderate tension. No pain or tenderness over the praecordium. Apex beat--4th space, just within the nipple line. No enlargement of cardiac dullness--the right border being at the middle line, the upper border at



the 3rd left space-- See diagram I. (Only the superficial dulness was recorded, as there was nothing to lead one to suspect any cardiac disease). The heart sounds were normal.

Respiratory system. R-- 44 per minute. The patient showed no distress, breathing quietly and easily. He had a short hard cough. The percussion note over the lungs was clear, and nothing was revealed on auscultation.

Abdomen.--Alimentary System--The tongue was covered with white fur, but was red tipped. The breath was foul. There were several partly decayed teeth; the four lower temporary molars were carious. The gums however were healthy.

The Liver and Spleen showed no signs of enlargement.

Urinary System.-- The urine was pale, clear, amber in colour, with a slight mucoid deposit; acid; of specific gravity 1028; contained neither albumen nor sugar.

Nervous System.-- There was no pain nor tenderness over the head. No paresis nor defective sensation was found. The reflexes were not exaggerated nor diminished.

The above notes are taken from the report made on the day of admission to hospital. The following is a summary of the reports made during the residence of the child in hospital,---

On Oct. 26 the cheeks were flushed, the eyes bright, and at noon the temperature rose to 102.6°. The pulse numbered 130 per minute. The apex beat had not altered in position, but there was a faint yet distinct thrill appreciable to the hand when laid flat over the praecordium. The superficial or absolute

dulness had increased in size-- the right border had moved from the midsternum to the right sternal margin, and the left border was in the nipple line. On auscultation there was heard at the mitral area a short soft systolic murmur almost indistinguishable from a prolonged first sound. The respirations numbered 50; a few fine crepitations were audible in the left axilla and at the left base. Otherwise the lungs were normal.

On October 27 the cheeks were brightly flushed, and the temperature reached  $104.8^{\circ}$  during the day. No pain was complained of. In the evening the child vomited soon after drinking a cup of milk. The condition of the heart was considerably altered. The rhythm had increased to 140 per minute. Pulsation was visible in the 4th and 5th spaces & could be felt as far as  $3\frac{1}{2}$  inches to the left of the midline (over  $\frac{1}{2}$  inch to the left of the nipple line). A marked systolic thrill was present, but there was no praecordial pain or tenderness. The apical systolic murmur was much louder, & was audible also at the pulmonic area.

On the morning of October 28th the cardiac dilatation reached its maximum size. The pulse numbered 120. A diffuse wavy pulsation was seen in the 3rd, 4th & 5th spaces, & on palpation an impulse was felt also in the 6th space. The area of superficial dulness is represented in Diagram II. At the level

Case I - Notes.

of the nipple the right border of cardiac dulness was  $1\frac{1}{2}$  inches from the midsternum (i.e. about  $\frac{1}{2}$  inch further out than on the previous day); the left border remained beyond the nipple line; so that the greatest transverse measurement was fully 5 inches. The systolic apical murmur was very soft, but at the aortic area a fairly loud systolic murmur was audible. A slight cough was still present, and the crepitations in the left axilla were more numerous.

On the morning of October 29 the temperature fell to  $99.2^{\circ}$ , and there was a great improvement in the patient's general condition. In the evening the heart was again carefully examined, and it was found that great changes had occurred in the 36 hours which had elapsed since the previous examination. The pulsation in the 4th & 5th spaces was scarcely visible. The superficial or absolute dulness had diminished very considerably. The right border was at the right sternal margin -- on the previous day it had been fully 1 inch further out. The left border had crept in a little and was found to be in the nipple line. The cardiac sounds were not quite so clear; the soft systolic apical murmur remained.

On October 30th the temperature remained normal & the general condition was satisfactory. The cardiac murmurs had disappeared.

On October 31st the area of cardiac dulness remained as on October 29th, but a faint thrill was felt on palpation, which had not been present on the previous day.

The cardiac murmurs reappeared; soft systolic murmurs were distinctly audible in the mitral and in the aortic region. Fine crepitations were heard at both bases.

On November 1st no thrill was felt, and the right border of cardiac dulness was found to have crept in from the right sternal margin to the midsternum. <sup>See diagram III</sup> At the mitral and at the aortic area a soft systolic murmur was heard with about two out of every three beats. These murmurs were very localised, and the question was raised whether they were exocardiac. However, they did not disappear entirely when the child stopped breathing, and it was impossible to decide their source of origin.

Little ~~change~~ alteration occurred from Nov. 1st to Nov. 13th. The child was allowed to sit up in his chair for one hour daily from Nov. 6th. During the week the murmurs came and went without any apparent cause.

On November 13th the cardiac rhythm was slightly irregular, a pause occurring after every fifth beat or so. The right border of cardiac dulness appeared to be at the left sternal margin; the left border remained in the nipple line. No murmurs were audible.

At the time the child left hospital, on November 21st, the apex beat was in the fifth space, just outside the nipple line, & the cardiac sounds were pure. At the time of admission, one month previously, the apex beat was in the fourth space, just within the nipple line.

Diagram IV was drawn on the evening before the child left hospital, On comparing it with the diagrams I & II, of October 25th & 28th respectively, the alterations of the area of cardiac dulness are readily seen.

TREATMENT. The treatment in this case consisted chiefly in absolute rest in the recumbent posture for ten days, After the tenth day, the patient was allowed to sit up quietly in bed, and in a few days, as the heart was not affected for the worse by sitting up, he was allowed to get up and to go about the ward. On admission he was put on a mixture of sod. and pot. iod., with sp. ammon. aromat.,<sup>salicyl.</sup> for two days. He then had a mixture containing arsenic, hyd.perchlor., and ferri perchlor. During the time of pyrexia he was given only milk; when the fever subsided he gradually returned to ordinary diet.

# Case I - Summary.

SUMMARY AND REMARKS. The above notes clearly show that this case presented very considerable enlargement of the praecordial area of dulness, coming on within a few days & subsiding almost as rapidly. When the child was first seen, attention was directed to the mother's description of a fall on the head some fourteen days previously, with subsequent occasional headache and tenderness over the occiput. The onset of a generalised headache, together with the presence of fever, led us to suspect an early stage of meningitis. Nothing was detected on careful examination of all the organs of the body; & it was fortunate that the heart was examined not only by myself but by Dr Cautley.

From the course of the temperature after admission it was evident that the child suffered from <sup>some</sup> general inflammatory condition. The nature of this condition was, and has always been obscure. Attention was concentrated upon the heart when, on the day after admission (4th day of illness), the area of praecordial dulness was found to be increased, & a systolic apical murmur was heard. On the 6th day of illness the dulness reached its maximum. Thirty-six hours later the temperature had declined, and the transverse measurement of the praecordial dulness had decreased by 1½ inches

Case I - Diagnosis.

Diagnosis. In this case, as in the other cases mentioned below, the diagnosis from pericarditis had to be carefully considered. In pericarditis there may be rapid enlargement of the area of praecordial dulness. A pericarditic murmur is not always the typical double murmur described in the text-books: it may be single, audible at any part of the praecordium and even beyond. Pericarditis of any extent is usually attended by cough and increase in the respiratory rate, and it is now recognised that there is a latent form of pericarditis, occurring especially in children, which runs its course without pain. As regards all these points, therefore, the case might have been one of pericarditis. On the other hand, there were signs incompatible with the presence of fluid between the heart & the chest wall. The chief differential point rests on the fact that the murmurs developed and increased while the area of praecordial dulness enlarged; whereas, in pericardial effusion, the murmurs disappear as the effusion increases. Again, the apex was lowered instead of being

raised, and a wavy impulse was visible in the 3rd to the 6th spaces on the day the praecordial dulness reached its maximum. There was never any bulging of the intercostal spaces. The dulness never showed the cone shape so characteristic of the dull area of pericardial effusion, nor was it increased upwards. This latter point is of some importance, as it has been said that pericardial effusion in children of a rheumatic diathesis starts and may remain around the great vessels at the base of the heart. The murmurs at first were indistinguishable from a prolonged first sound; as the disease progressed, they assumed the characters of murmurs of valvular origin: they were single, never harsh or superficial, nor altered by pressure with the stethoscope. They were heard over the aortic and mitral regions, regions where valvular murmurs (structural or functional in origin) are best audible, and they were propagated in the direction usual with murmurs arising at these orifices. The heart sounds were never distant or weak, on the contrary, they were unusually loud and clear when the area of dulness was greatest.



# Case I - Etiology.

## ETIOLOGY

The dilatation in this case was ~~therefore~~ in all probability due to the presence of a toxin in the blood. The nature of the toxin <sup>in</sup> operation remained a matter of conjecture. The toxins causing pyrexia of short duration and commonly known to affect the cardiac muscle were first considered-- Rheumatism, Scarlet Fever, Pneumonia, Influenza, and Enteric Fever.

Of these, Scarlet Fever and Enteric Fever may be dismissed from serious consideration. The course of the temperature resembled that of neither of these diseases, and there were no other symptoms present, such as sore throat, rash, or enlarged spleen.

As regards pneumonia, it is known that a deep-seated pneumonia may be present without other physical signs than an increased respiratory rate and fever. But in pneumonia the fever is continuous, declining by crisis, and the dilatation of the heart is of slight degree, and attended by graver symptoms of the patient's general condition.

In Influenza the toxin may affect the respiratory, cardiac or alimentary system. The heart may become dilated, with irregularity of rhythm, and even endocarditis may be present. The course of the temperature in Influenza is frequently very high, and is of short duration, as in this case. However, there are important points against the diagnosis of Influenza in this case,

The onset of the fever in Influenza is attended by severe pains in the head, back, and limbs, which are characteristic of the disease. In the case under consideration there were no such pains; the patient felt so well that he desired to get up. Again, the heart in Influenza is not so greatly dilated, and when dilatation of the heart does occur, it is a much more serious condition. The dilated heart of Influenza is long in recovering; the patient is left liable to severe and even fatal syncopal attacks.

The most probable toxin in operation was that of Rheumatic Fever. The chief symptoms in the case were fever, or a progressive pyrexia, concomitant with very considerable enlargement of the heart. Mentioning first the points which might be brought forward against a diagnosis of Rheumatic Fever, there was an absence of any joint lesions, of endocarditis, sore throat, and of any family history of Rheumatism. To these it can be replied that it is well known that a child may complain of "pain in the legs" for a few days, no notice being taken of him at the time, and later on, he may come under medical observation for cardiac valvular disease. Rheumatism is indeed a disease especially liable to attack

the heart, and commonly in children, without giving rise to any inconvenience beyond a trifling ailment which at the time is passed without exciting the parents' attention. When Rheumatism attacks the joints it is one of the most painful of diseases, but when it attacks a deeper structure it may run an entirely ~~course~~ / painless course.

Recently, attention has been particularly drawn to the fact that in Rheumatism the heart is frequently dilated, and that the dilatation in these cases may attain a very great size without any correspondingly remarkable alteration in the general circulation. Moreover, these cases may be unattended by any peri- or endocarditis. I shall refer to the pathology of such a condition later on, when I am comparing the various cases narrated below.

The fact that the systolic murmurs in this case varied from day to day, and that at the time of <sup>the patient's</sup> dismissal <sub>from hospital</sub> they had entirely disappeared, is no proof that they were not of organic origin. It is highly probable that, several months later, a systolic murmur would be audible. In his Lectures on Heart Inflammation in Children, (Brit. Med. Journ. Vol. I.) <sup>1894</sup> Sturges narrates ~~the~~ case of a child admitted to hospital for rheumatism. When she appeared to have recovered,

Footnote: ① When the patient attended the outpatient department in January, (i.e. two months after leaving hospital), the heart remained slightly enlarged towards the left side, and there was a soft systolic murmur at the aortic area. It was not possible to decide, however, that the murmur was organic in origin.

Case I. - Etiology.

examination of the heart revealed a lengthened first sound, a reduplicated second at the apex, and some irregularity of the cardiac rhythm. In a few days a soft systolic murmur developed, only to disappear again in a few days more. The heart was watched carefully for three months; for some considerable time the murmur was audible only when the child lay down, but after two months with perfect general health a systolic murmur appeared, and remained permanently. Sturges remarks that had the child been dismissed from hospital a little sooner the fact that endocarditis had accompanied the attack of rheumatism would have been overlooked.

It is indeed well to remember that although it is a common <sup>error</sup> to mistake functional murmurs for organic, the opposite error may also occur.

CASE II

JOHN F -----

A CASE OF ACUTE DILATATION OF THE HEART, ACCOMPANYING  
ENDOCARDITIS, OCCURRING IN A CHILD AGED SEVEN.

*Temperature chart*

{ Narration of case notes.  
Treatment.  
Summary of case, and Remarks.  
Diagnosis.  
Etiology.  
*Diagrams -*



15.

A CASE OF RAPID DILATATION OF THE HEART, ACCOMPANYING  
ENDOCARDITIS, OCCURRING IN A CHILD AGED FIVE.

John F--- aet 5, was admitted to the Belgrave Hospital under the care of Dr Ewart, on November 26th 1900, complaining of cough, languor and feverishness.

History of present illness. On November 15th the child attended the outpatient department for cough, and bronchitic rales were found on examining the chest. A cough mixture was given, & the mother was told to bring the boy back in a week's time. When the child returned on November 22nd, the mother said that two days previously he had taken a turn for the worse, he had seemed feverish at night & had complained of pain in the stomach". On examination the temperature was found to be  $101.5^{\circ}$ , & the tongue was thickly furred. A few bronchitic sounds were heard in the lungs; otherwise there was nothing else abnormal detected in the chest. The heart sounds were normal. The child said he had fairly constant pain, and pointed to the epigastrium as the position of the pain. Two days later the boy again attended the outpatient department. The temperature was  $99.8^{\circ}$ , the tongue was furred; he had epigastric pain and constipation. ~~Nothing was~~ found in the chest beyond a few bronchitic sounds. The heart sounds were normal; the cardiac dulness was not mapped out. Nothing was detected on examining the abdomen. As no bed was available the child was ordered to return in two days, and Hyd.  $\bar{c}$ . Cret. gr. i was given night and morning.

Previous History. The child had suffered from Rickets in infancy, and had not been able to walk until he was three years old. He had never had Measles or Scarlet fever. He was frequently troubled with cough, but not with sore throat. For two months previous to admission there was an indefinite history of cough, with hot skin at night and weariness during the day.

Family History. No definite history of rheumatism in the family could be elicited, but the mother said that several members on her side had died "suddenly, with heart disease".

The following is copied from the notes made on admission:-

Present condition. The child is very pale, with dark rings under the eyes. The skin is clammy, the lips dry, the tongue furred, with a red tip. The body is thin; the bones show signs of Rickets: the chest has a slight degree of pigeon breast. There are a few enlarged glands under the jaw. The throat shows some enlargement of the tonsils, but no congestion. There are no rheumatic nodules, and no swelling of the joints. No pain or tenderness is present.

Cardio-vascular system. There is a systolic thrill over the praecordium. There is diffuse pulsation, the point of maximum impulse being in the 4th space, two inches from the midsternum. Owing to the prominence of the sternum in



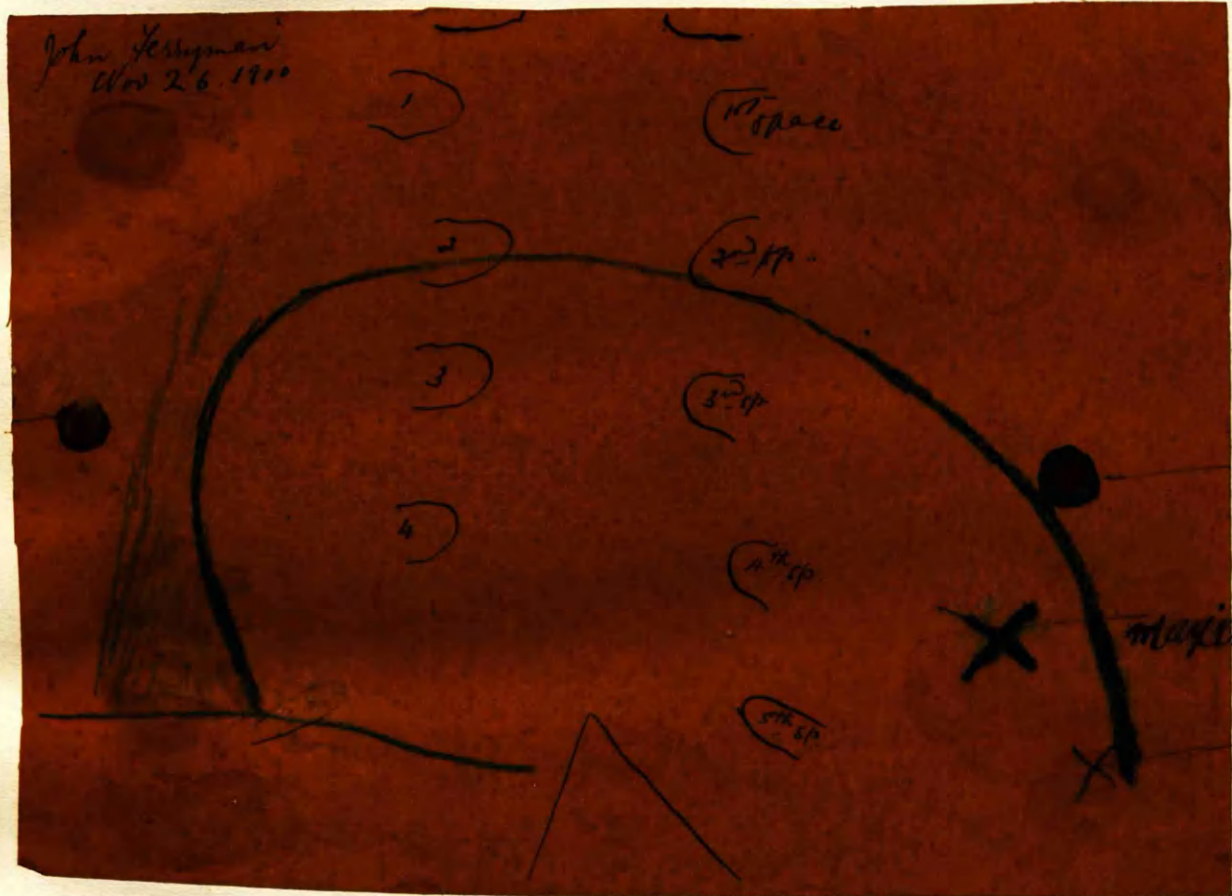
h.v.

Cape II

Diagram I

Taken on Nov. 26<sup>th</sup>

Dilatation of the Heart, accompanying Endocarditis  
Superficial Dulness Tracing.



nipple over  
4<sup>th</sup> rib;  
2<sup>3</sup>/<sub>4</sub>" from the  
midsternum

Maximum  
impulse.

fainter  
impulse.

Tracing shows extent of Superficial Dulness on  
Nov 26<sup>th</sup> (6<sup>th</sup> day of illness.) The measurements, as  
made out by percussion with the finger, are:  
 { R. border = 2" from the midsternum  
 { L. border = 2<sup>3</sup>/<sub>8</sub>" " " "

The shaded area outside the R. border was made out by  
percussion with the pleximeter.

the pigeon breast there is a slight resonance on percussion even over the centre of the praecordium. The "absolute" or superficial cardiac dulness is therefore judged to exist over the area where the percussion note is greatly impaired. This area is very considerably enlarged-- the right border extending to 2 inches to the right of the midsternum, and the left border to ~~2~~ inches to the left of the midsternum. The upper border is at the upper edge of the 3rd rib. With the pleximeter the impairment of the note due to the relative or deep dulness is found to extend still further on both sides. (No record was made of the deep dulness. See Diagram I). On auscultation a loud systolic murmur is heard at the mitral area; it is conducted upwards & inwards as far as the pulmonic area.

Respiratory system. Evidences of bronchitis are present. A few rhonchi and rales are heard over the lungs; at the bases, more especially at the left base, fine crepitations are present.

Abdomen. The liver is palpable below the costal margin, as is to be expected from the deformed shape of the chest wall. No evidences of enlargement of the liver or ~~spleen~~/ spleen are present.

Urinary system. The urine is clear & pale; acid; sp.gr. 1008; no albumen or sugar.

The following notes summarise the alterations in the condition of the heart from day to day during the week after admission to hospital.

On November 27 ( the morning after admission) the temperature had fallen to 99°. The child had perspired profusely during the night. The right border of dulness had moved in ¼ inch towards the middle line.

On November 28 no alteration was found in the right border of dulness, but the left border had moved in almost ½ inch--see

Diagram II.

On November 29 there was considerable diminution of the area of cardiac dulness, the right border having moved in 1 inch, and the left border almost ½ inch since admission on the 26th. <sup>see Diagram III</sup> The systolic murmur was louder and propagated further to the left than on admission, and there was accentuation of the pulmonary second sound.

On December 2nd the right border had moved in another ¼ inch. The systolic murmur had acquired a musical character. No rales were heard in the lungs.

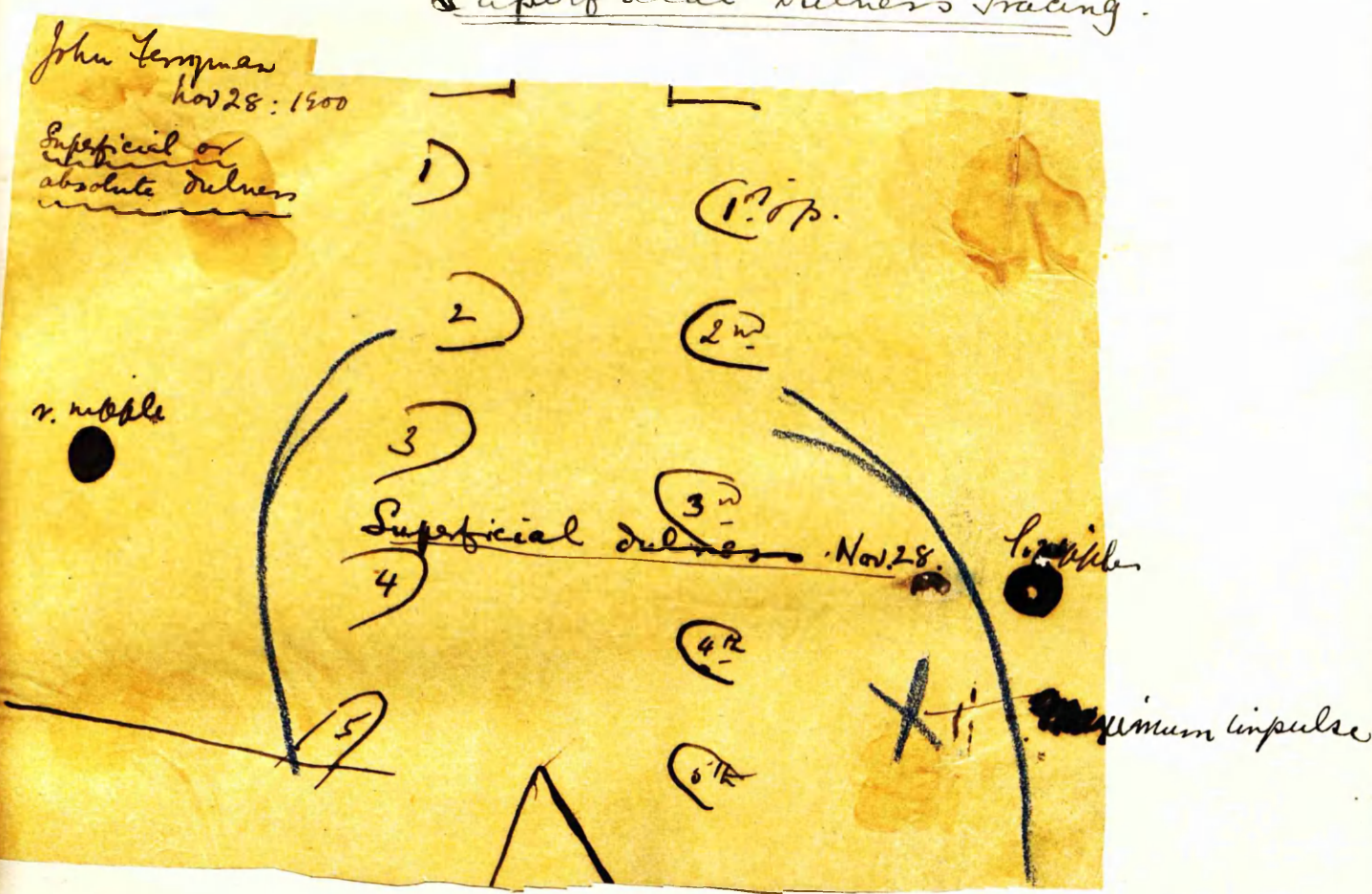
On December 6 the right border of the superficial dulness could

Case II

Diagram II

Taken on Nov. 28<sup>th</sup>.

Shows Dilatation of the heart, accompanying endocarditis.  
Superficial Dulness Tracing.



Tracing shows extent of superficial dulness on Nov. 28<sup>th</sup>, the 8<sup>th</sup> day of illness.

The measurements are : { R. border =  $1\frac{3}{8}$ " from midsternum.  
L. border =  $2\frac{1}{2}$ " " " "

The upper border could not be defined on account of a blister that had been applied.



II Diagram IV

Taken on Dec. 9<sup>th</sup>.

Deep or Relative Dulness Tracing.

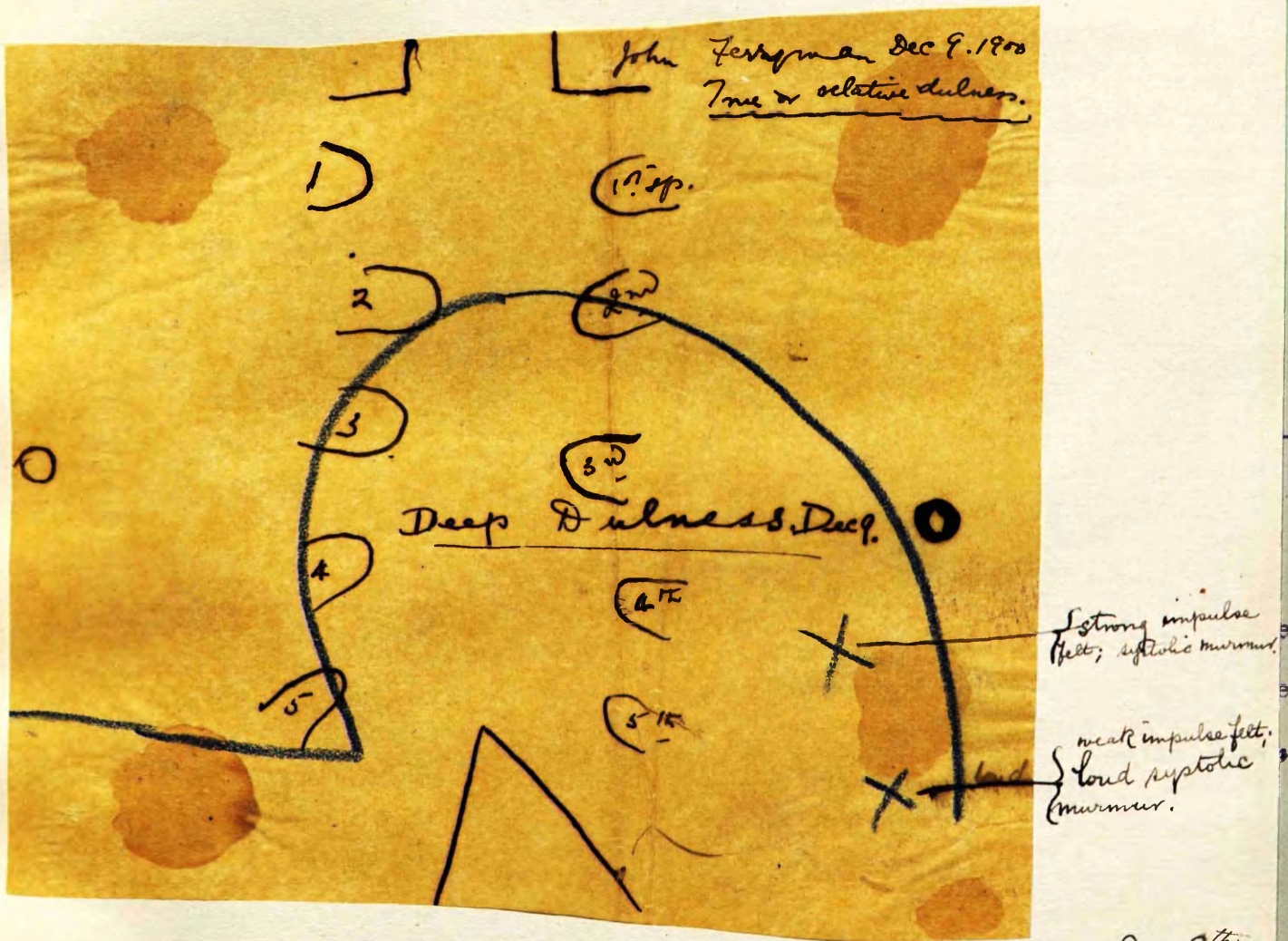


Diagram showing the deep or relative area of dulness on Dec. 9<sup>th</sup>

On Dec. 9<sup>th</sup> the resonance of the pigeon-breast was so great near the l. border of the sternum that it was impossible to define accurately the superficial dulness; while it was much simpler to define the deep or true dulness.

The above tracing probably represents the normal size of the heart <sup>in this case</sup> because it must be remembered that although the R. side of the tracing is 1" from the midline, the prominence of the sternum makes the surface measurement greater than the true transverse measurement of the heart.

could not be defined, owing to the great resonance of the sternum, and it was presumable therefore that it had moved in as far as its normal position, i.e., the mid or left sternal margin. The apical systolic murmur had become louder and more musical, and was conducted further into the left axilla.

On December 9 the superficial dulness could not be defined at all on account of the resonance over the prominent ribs and sternum. The deep or true dulness however was comparatively easy to percuss out, and a tracing was made--see Diagram IV. This diagram shows that the heart had regained its normal size.

On December 18 the cardiac dulness remained unaltered. The apex beat was faintly perceptible in the 5th space just within the nipple line. The systolic apical murmur was still loud and musical, the pulmonic second was highpitched and accentuated.

TREATMENT as usual consisted of absolute rest in bed for almost a fortnight. In addition, Caton's method of treatment by blisters over the praecordium was adopted. On admission, the child was put on a mixture containing Sod. Salicyl. gr. vi, with Pot. Iod. gr. ii t. i. d. On Dec. 3rd the pupils appeared somewhat dilated, & the boy talked nonsense excitedly. The mixture was stopped, and by the evening the child had regained his normal condition. The mixture was resumed at midday Dec. 5; on the following morning the boy again talked wildly. It was then found that the artificial preparation of Sod. Sal had been used. When the natural salt was substituted no further symptoms occurred.

# Case II - Summary

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## SUMMARY AND REMARKS.

The history of this patient, as so frequently happens with children, was somewhat indefinite. However, it was quite clear that he was suffering from some pyrexial condition certainly for four days before admission, & very probably for two or three days longer. During the four days before admission the symptoms consisted of "feverishness" & epigastric pain. He was twice seen in these four days, and was thoroughly examined. The temperature on these occasions was over 100°, and there was no cardiac murmur. The cardiac dulness was not mapped out; it is probable that some degree of dilatation would have been discovered if the dulness had been mapped out.



Case II - Summary.

He was admitted on Nov. 26th, with a definite systolic murmur, and marked dilatation of the heart. This dilatation affected chiefly the right heart, the right border being two inches beyond its normal position. At the same time there was a generalised bronchitis, rhonchi & rales being audible over the whole chest. Thus there was an abnormal strain thrown upon the right side of the heart, which, already weakened by the toxin circulating in the blood, yielded to the strain and became dilated.

After admission the temperature rapidly declined, and reached normal in four days. The dilatation gradually diminished, and in eight days the heart regained its normal dimensions. The systolic murmur at the mitral area, however, remained, and even two months later, when the child attended the outpatient department, it was still distinct. The persistence of this murmur confirmed the

Case II - Summary

22

opinion that it originated not from dilatation, but from endocardial inflammation.

It is only right to mention here that in this case the distortion of the chest added some difficulty to the physical examination. The percussion was on every occasion carefully performed; and the dilatation of the right heart was confirmed on the day of admission by two of the physicians. It must be remembered that the diagrams do not show an accurate representation of the size of the heart during the period of dilatation, because, owing to the prominence of the sternum, the transverse surface measurement of the chest shown in the diagrams is greater than the true transverse measurement of the heart.

Diagnosis. The diagnosis in this case was not so complicated as in Case I. The same arguments employed in Case I. would apply here for the exclusion of pericarditis with effusion. The condition undoubtedly was one of endocarditis with dilatation of the heart.

Etiology. As in Case I the pyrexia pointed to the presence of a toxin in the blood, & the cardiac dilatation was due in all probability to the action of this toxin upon the cardiac muscle. (see causes of dilatation, *page 81b*). As regards the nature of the toxin in operation, the fact that endocarditis accompanied the fever favoured the opinion that a rheumatic toxin was present. No inference can be drawn from the fact that the pyrexia subsided while the child was under salicylates. In relation to the cause the indefinite history of a hot skin at night for almost a month previous to the onset of the

Case II - Etiology.

24.

present illness, indicated a lowered state of health which probably rendered the child more susceptible to the infective organism. The arguments in favour of a rheumatic toxin, which were enumerated in Case I, hold good in this case also (see page 12).

CASE III Dorothy P-----

A CASE OF ACUTE DILATATION OF THE HEART,  
ACCOMPANYING ENDOCARDITIS, OCCURRING IN A CHILD AGED SEVEN.

*Temperature charts.*

Case notes.

Summary of the case and Remarks.

Remarks upon the progressive alteration of  
the heart sounds.

Diagnosis.

Etiology.

*Diagrams.*

Bacteriological Investigation of the blood.



A CASE OF ACUTE DILATATION OF THE HEART ACCOMPANYING ENDOCARDITIS  
OCCURRING IN A CHILD AGED SEVEN.

Dorothy P--- aet 6 $\frac{1}{2}$  was admitted to the Belgrave Hospital for Children, under the care of Dr Ewart, on Dec. 3rd 1900, complaining of headache, shivering and pains in the legs since Nov. 28th.

**HISTORY OF PRESENT ILLNESS.** ON the evening of November 28th the child said she "felt very ill" and has ever since complained of shivering, pains in the legs, languor and headache. On the evening of December 2nd she vomited. The mother stated also that for a month previously the child had been more or less ailing, complaining of being tired, wanting to lie down during the day.

**PREVIOUS HISTORY.** The child has always been "delicate". She had diphtheria at three years of age, measles at four years. She never had scarlet fever or whooping cough. No history of rheumatic fever could be elicited, but she has often suffered from sore throats & pains in the legs.

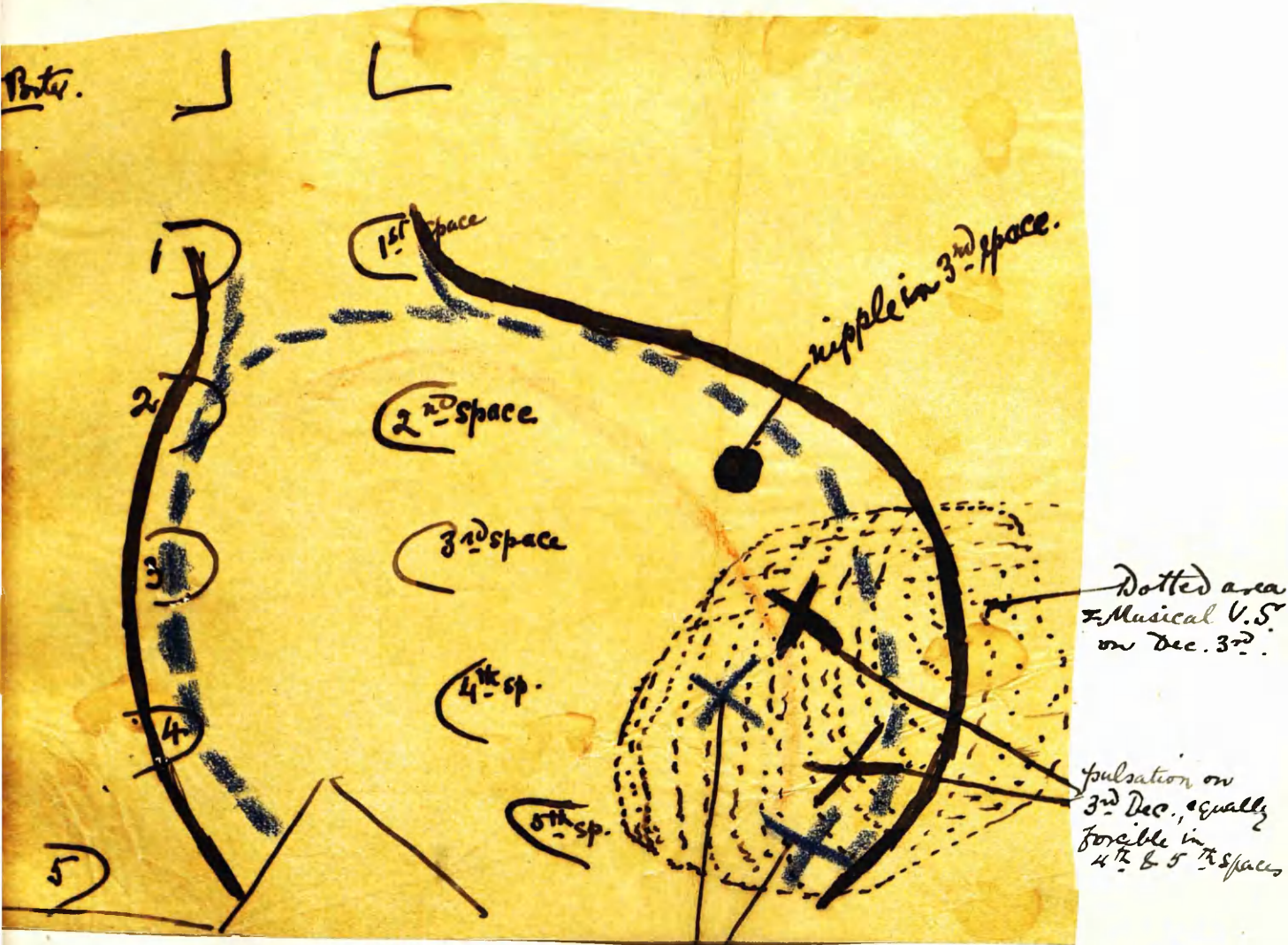
**FAMILY HISTORY.** Nothing of any value was elicited.

**PRESENT CONDITION.** The child is pale, with red hair and a fine delicate skin. The skin is moist and clammy with perspiration. There are no swellings of the joints, no rheumatic nodules, no erythematous or other rash. The glands under the jaw are slightly enlarged. She is of thin slender build and has coxa vara of the left

Taken on admission, Dec 3<sup>rd</sup>  
& on Dec. 9<sup>th</sup> & 11<sup>th</sup>

Case III Diagram I

Dilated left heart; showing subsidence to normal size.  
3 Tracings of Deep or Relative Dulness, taken on 3 separate occasions.



Explanation of above 3 Tracings.

1. Outer black outline = area of deep or relative dulness, made out with aid of pleaximeter, on Dec. 3<sup>rd</sup> 1900.  
Dotted black area indicates site over which the V.S. is audible on that date.
2. Interrupted blue line = area of deep or relative dulness, similarly mapped out, on Dec 9<sup>th</sup> 1900.
3. Red line = area of deep or relative dulness, similarly mapped out on Dec. 11<sup>th</sup> 1900.  
The right border could not be accurately defined.

Note that the deep dulness at l. side is to the l. of the apex beat on both days.



hip. The tongue is thickly furred, the breath foul, the tonsils enlarged but not congested. There are several decayed teeth, but the gums are healthy. The temperature is  $103^{\circ}$ . No pain or tenderness is complained of.

Cardio-vascular system. Pulse--140; regular; full; of low tension. A somewhat diffuse impulse is visible in the 4th & 5th spaces, most marked in the 4th space. No thrill or praecordial tenderness is present. Percussion shows enlargement of the praecordial area of dulness. A tracing of the deep or relative dulness shows the upper left border to be at the upper edge of the 2nd left rib. The right border is one inch to the right of the midsternum; the left border, at the level of the 4th space, is  $3\frac{1}{4}$  inches to the left of the midsternum--the normal limit of the left border, in the nipple line, being only  $2\frac{1}{4}$  inches to the left of the midsternum.

Diagram 1 shows the tracing made at this date. On auscultation at the apex both sounds are heard; the first is accompanied & followed by a soft short systolic murmur propagated only a little way into the axilla. The pulmonary second sound is accentuated.

Respiratory System. Nothing abnormal is present; there is no cough. Respirations number 34.

The liver is just palpable below the costal margin.

The urine is clear, pale, acid; contains no albumen or sugar.

It is important to mention here that in the Outpatient Department ~~11/9/11/11~~ the child's chest was thoroughly examined by the outdoor physician that morning at 9 a.m. He reported

that

that there was nothing in the heart beyond what might accompany any feverish condition; and the child was admitted on the supposition that some lung mischief was developing. At 11p.m., when the above notes of the PRESENT CONDITION were made, the apical murmur was so loud that it could not possibly have been overlooked had it been present in the morning.

The following summarises the notes made during the child's residence in hospital:--

On Dec. 4th, the day after admission, an oval patch of erythema, about 1½ inch long, was found, symmetrically situated on the extensor surface of both arms, just above the elbow. A larger patch was seen on either side of the neck. The temperature gradually fell till it reached normal on Dec. 6th. The child lay quietly on the **back**, was very pale, and vomited once each day, usually towards the evening. The area of cardiac dulness diminished very ~~little~~, if at all, during these days, but the systolic apical murmur day by day became louder & more musical.

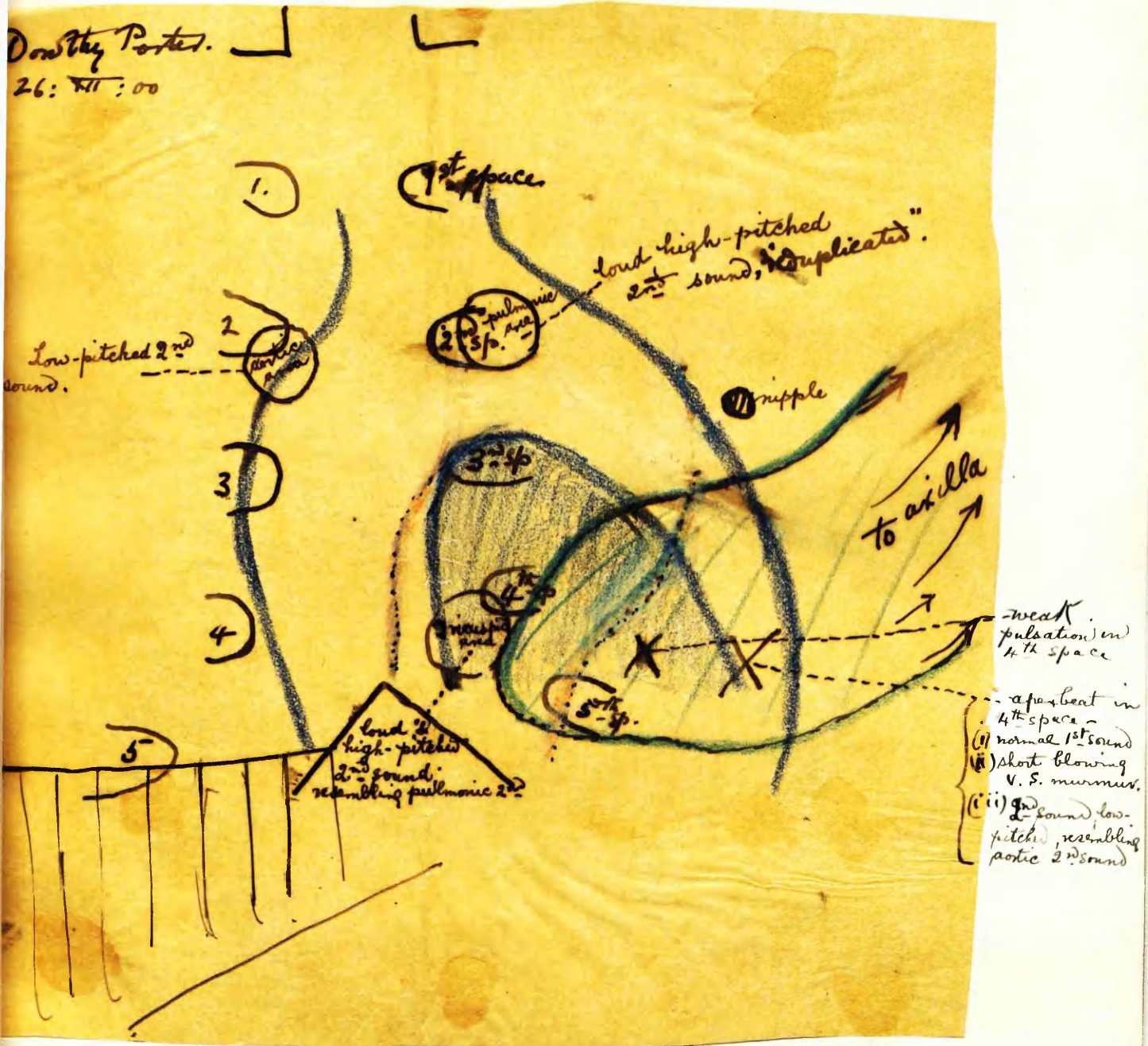
On the morning of Dec. 9th the right and left

borders of the cardiac dulness were found to have moved in almost  $\frac{1}{2}$  inch. A tracing was made, without reference to the tracing made on the day of admission. Both were subsequently

transferred to one sheet so that the two diagrams might be more readily compared, and the alteration in the area of dulness seen at a glance. (See Diagram I). On auscultation at the apex both sounds were heard, the second rather accentuated. A systolic murmur, short, harsh, with a high pitched musical squeak in the middle, accompanied & tailed off from the first sound; it was conducted into the left axilla & was audible at the angle of the left scapula.

On Dec. 11th the apex beat was felt in the 4th space, within the nipple line, having moved in fully  $\frac{3}{4}$  inch. The left border of the cardiac dulness had moved in quite  $\frac{1}{2}$  inch since Dec. 9th, & 1 inch since admission, so that it was now within the nipple line, & could be accounted normal. The right border was difficult to define, owing to the resonance of the sternum, but appeared also to be normal. (See Diagram I)

Shows superficial, & also deep, dulness, after recovery from dilatation.



Tracing made from chest on 26<sup>th</sup> Dec. 1900. (Percussion by ordinary method - without foldimeter)

{ Outer blue line = deep or relative dulness.  
 { Inner blue shaded area = superficial or absolute dulness.

Green outline = area of musical, short & blowing V. S. murmur.

Notes - The high-pitched "reduplicated" pulmonic 2<sup>nd</sup> sound is heard over area enclosed by red lines; it is not heard to the left of the red dotted line. Note that it is not heard over the apex, nor over that part of the chest where the left ventricle comes to the front. Over the apex the 2<sup>nd</sup> sound resembles that at the aortic area.

On Dec. 14th the child was allowed to sit up in bed, and on Dec. 15th and 16th she was allowed to get up for a few hours. On the evening of the 16th the dulness was found to be the same as that traced on the 11th. The apical systolic murmur had lost its musical squeak, & was uniformly harsh.

The child was kept in hospital for the Xmas treat, and on the morning of Dec. 26th, before dismissing her, another tracing was made, which records both the deep and the superficial dulness--- see Diagram II. The character of the cardiac sounds & murmurs was also of great interest, and their area of conductivity was very carefully marked out in the tracing. At the apex a loud second sound, of low pitch, was heard. The second sounds at the aortic & at the pulmonic regions were both accentuated; the aortic was of lower pitch than the pulmonic. At the tricuspid region the second sound was high pitched, resembling that heard at the pulmonic region. I shall refer to the significance of these observations later (see page 32).

Treatment consisted of absolute rest in the recumbent posture for eleven days, with a mixture containing sod. salicyl., pot. iod., and spt. ammon. aromat.

SUMMARY AND REMARKS. The observations made on this case from day to day point to the presence of endocarditis of the mitral valve, associated with a moderate degree of dilatation of the heart, more especially of the left side. The child walked up to hospital on the morning of admission, and though the chest was stripped in the outpatient department, nothing was reported as worthy of note with regard to the condition of the heart. When the child was examined late in the evening of the same day, a hasty percussion of the superficial dulness called for no remark. The standard by which the precordial area of dulness is measured in a child varies with the age, and with the size of the child, so that slight enlargements, especially of the superficial dulness, are difficult to detect. It was only after the diffuse impulse & murmur were noticed that attention was drawn to the state of the heart. Then on

careful percussion, the heart was found to be enlarged, & a tracing was at once taken of the deep or true dulness. During the first five days after admission the child vomited daily, & the respiratory rate varied from 28 to 36 per minute. The temperature fell gradually, from 103.5°, and reached normal in five days. Three days later, the measurements of the cardiac dulness were normal.

A soft systolic murmur was audible at the apex on admission. As the temperature fell, and the dilatation subsided, this murmur became louder & harsher, and developed the characters of a murmur due to mitral regurgitation--- being conducted into the left axilla, and audible at the angle of the left scapula behind.

REMARKS ON THE PROGRESSIVE ALTERATION OF THE HEART SOUNDS in this case.--The heart sounds were carefully noted daily, and it was observed that the pulmonary 2nd sound, at first only slightly accentuated, gradually

became louder, & finally acquired a reduplicated character, i.e. the 2nd sound resembled two sharp clicks without any appreciable interval between them. This reduplicated 2nd was also audible at the tricuspid area, & over that part of the chest wall which covers the right ventricle (see diagram II). At the aortic area & at the apex the second sound was not reduplicated, & was of a lower pitch. Now it is held by many that the reduplicated second in cases with backward pressure arising from mitral regurgitation or other causes, is due to asynchronous closure of the pulmonary & aortic valves. If that theory were true in all cases, the reduplicated second in the above case should have been heard at the aortic area and at the apex. But in this case it was clear that <sup>whereas</sup> the sounds at the aortic and mitral areas resembled each other, & ~~that~~ the reduplicated 2nd was audible only over the right ventricle. Thus the reduplication appeared



to originate solely at the pulmonary area, and was probably due to asynchronous closure of the pulmonary cusps of the pulmonary valve.

DIAGNOSIS. The diagnosis of endocarditis of the mitral valve was simple ; but the dilatation was more difficult to detect. Enlargement of the left side of the heart does not cause so great an alteration in the area of superficial dulness as does enlargement of the right side of the heart; & the diagnosis of dilatation depends upon the altered position of the apex, a diffuse apex beat, and enlargement of the deep or true dulness beyond the nipple line. Other points are mentioned under Case I, page 9.

ETIOLOGY. The presence of pyrexia and the symmetrical distribution of the erythematous patches pointed to the presence of some toxin circulating in the blood. In this case, as in Case II, there was an indefinite history of general malaise for about one month previous to the

onset of the acute illness for which the child was admitted to hospital. The system must therefore have been in a condition ill adapted to resist the invasion of any organism. The history of frequent "sore throats" and "pains in the legs" was strongly suggestive of the existence of a rheumatic diathesis. The presence of endocarditis was in itself a strong argument in favour of a rheumatic toxin in operation, and the absence of any other cause of endocarditis rendered the diagnosis practically certain.

BACTERIOLOGICAL INVESTIGATION. On December 3rd the finger was pricked & a drop of blood was drawn up with due aseptic precautions into two fine capillary pipettes. A Pasteur pipette was filled with equal parts of bouillon & milk, rendered slightly acid with lactic acid. The blood from the capillary pipettes was added to the Pasteur pipette, which was then sealed at both ends and incubated at blood heat. Seven days later (Dec. 10) the tube was broken at one end & a drop of its contents placed upon a slide, stained & examined. Nothing however was found. The tube was again sealed and incubated at blood heat. On Dec. 14th several films were made from the tube, and on examining these with the microscope minute cocci were seen, lying in chains, each coccus being much smaller than an ordinary streptococcus. This organism was partly decolorised by Gram's method. Subcultures from the pipette were made on Dec. 14 on gelatine, agar, acid agar and serum agar. These were incubated at blood heat-- some tubes aerobically, some anaerobically.--A.

A.1. On both the anaerobic & the aerobic, especially on the aerobic tubes of gelatine and agar, whitish grey colonies appeared in one day. On staining, they were found to consist of large cocci. Subcultures made on gelatine and potato grew luxuriantly within 48 hours, and were found to be sarcinae.

A.2. As regards the tubes of serum agar, nothing grew on the aerobic tubes. On two out of the five anaerobic tubes thin filmy colonies were visible by transmitted light on Dec. 18 (i.e. in 4 days). These, on staining, were found to consist of large cocci in chains. The individual cocci were much larger than those which had been found in the pipette contents. They stained with Gram and resembled ordinary cocci of the streptococcus variety. Subcultures were made from the two anaerobic serum agar tubes on Dec. 18.--A.2.i.

A.2.i. On Dec. 19 the subcultures made from the two anaerobic serum agar tubes showed no growths, except on one anaerobic agar tube. This growth was found to consist of sarcinae and ordinary streptococci. Nothing ever grew on the other subculture tubes.

A.2. Later on, on Dec. 24th, a tube of serum agar with a filmy colony (see A.2. above) was again examined. The colony was found to contain fine cocci in chains, smaller than the ordinary streptococci subcultures were made from this colony upon gelatine, agar, and serum agar, both anaerobic and aerobic, but nothing ever grew on any one of the subculture tubes.

The conclusion was therefore that ordinary streptococci were present in the Pasteur tube, & that they had become stunted by being incubated anaerobically for so long a time.

B. On Dec. 15th the clots remaining in the Pasteur pipette were placed in (1) a tube containing ordinary bouillon and (2) a tube containing slightly acid bouillon-- the bouillon having been rendered acid by the addition of lactic acid. On Dec. 17th films wire stained from both tubes, and showed large cocci resembling sarcinae, and only a few of those minute cocci in chains which had been found in the Pasteur pipette on Dec. 14th. Subcultures from the acid broth were made on glycerine agar, aerobic and anaerobic. Luxuriant growths developed in a day or two, but on examination these growths were found to contain only sarcinae.

REMARKS ON THE BACTERIOLOGICAL INVESTIGATION OF THE BLOOD OF CASE III.

The films made from the acid milk & bouillon mixture were promising. (a) The Rheumatic diplococcus described by Poynton and Paine grows best in an acid medium consisting of milk and bouillon rendered faintly acid by the addition of lactic acid.

(b) It usually takes about ten days to grow-- the films in this case were not found until the Pasteur pipette had been incubated ten days.

(c) The rheumatic diplococcus grows in chains when in a fluid medium.

(d) The individual cocci of the rheumatic germ are much smaller than ordinary streptococci.

In all these points, therefore, the organism found resembled that described by Poynton & Paine. But, on the other hand, it differed in not being readily decolorised by Gram. The subcultures were in every case disappointing. The rheumatic germ grows like a staphylococcus on a solid medium and grows best on slightly acid media, anaerobically. The colonies are very delicate & appear as tiny grey discrete colonies best visible by transmitted light. This organism grew only on two anaerobic serum agar tubes; it formed grey

and delicate colonies, but these were filmy and confluent instead of discrete and circular. In addition, on staining they were found to resemble ordinary streptococci. Moreover, the subcultures from this tube were disappointing in that only one showed any growth, and that one contained ordinary streptococci.

The conclusion drawn from the research was that the cocci found in the Pasteur pipette were ordinary streptococci exhausted and stunted from the absence of air. The anaerobic subcultures scarcely grew at all; whereas the aerobic subcultures grew luxuriantly, and contained abundant sarcinae and ordinary streptococci.

The failure to find the rheumatic germ was, however, to be expected when so small a quantity of blood was taken from the patient. Dr Poynton informed me (after I had completed the examination) that the germ was exceedingly hard to cultivate, that it was to be obtained only with special precautions, chiefly from the exudations into the joints or pericardium, and that in order to find it in the blood a large amount (several ounces) would be required.

CASE IV      Ambrose D- ----

A CASE OF ACUTE DILATATION OF THE HEART? ACCOMPANYING  
ENDOCARDITIS, OCCURRING IN A CHILD AGED TEN.

*Temperature charts.*

Case notes.

Summary of the case, and Remarks.

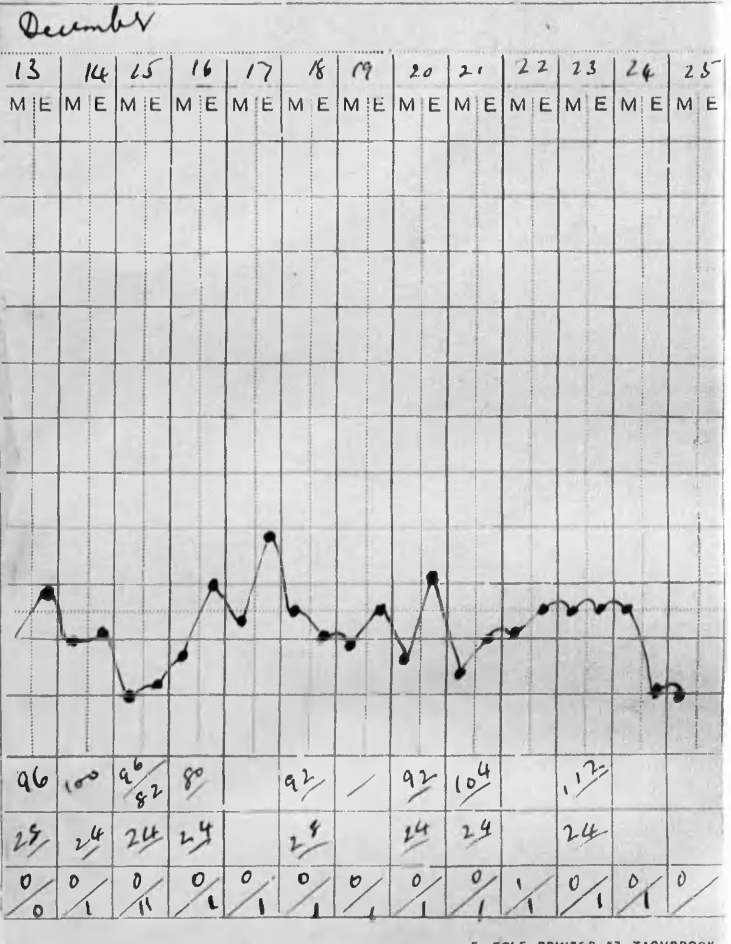
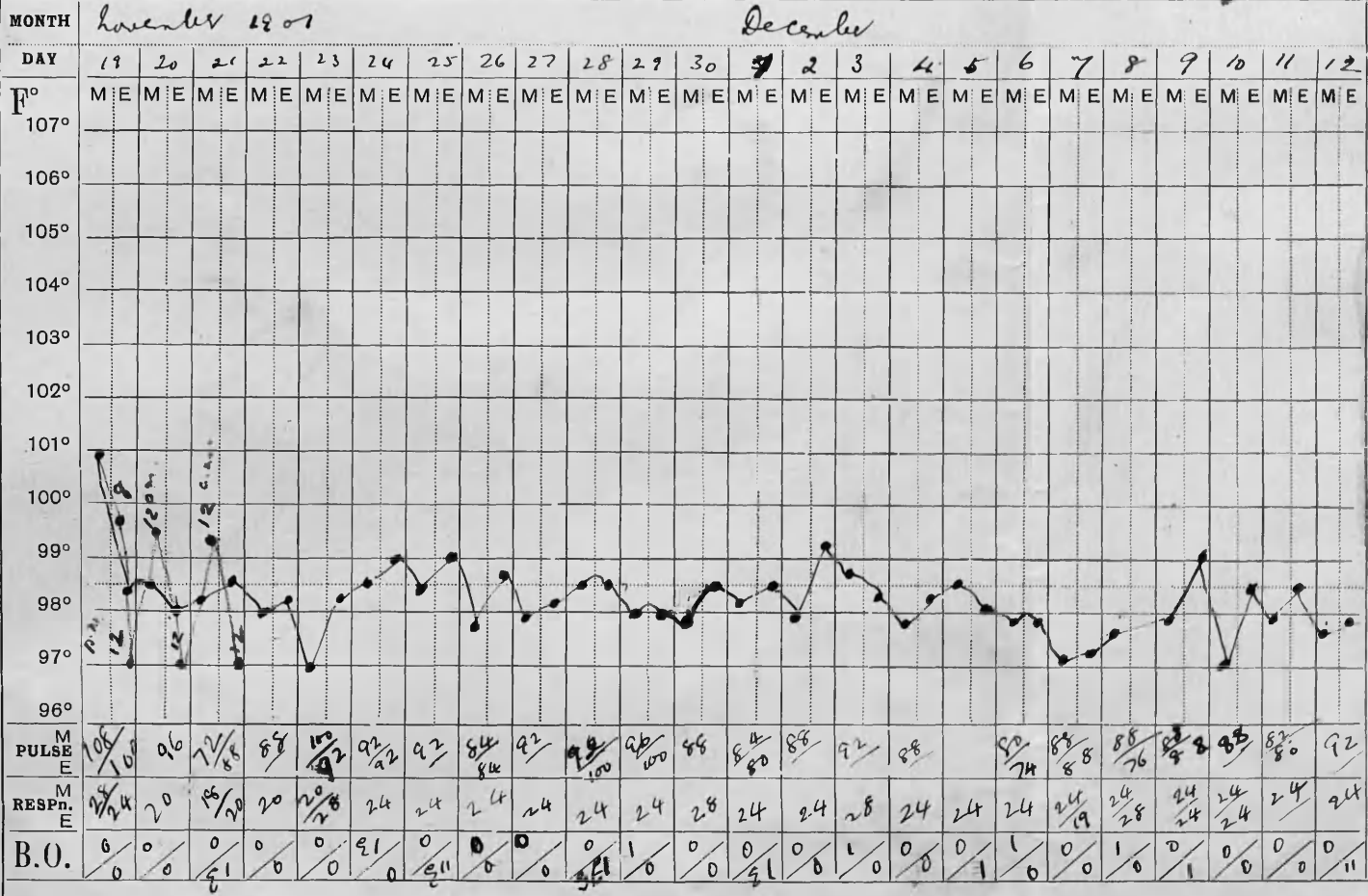
Diagnosis of the condition of the heart.

Etiology.

Bacteriological Investigation of the Blood.

# Case IV Temperature Chart

Ambrose Danell. Case IV - Blue lines show four hourly records during first 3 days.



A CASE OF ACUTE DILATATION ACCOMPANIED BY ENDOCARDITIS,  
OCCURRING IN A CHILD AGED TEN.

The following is copied from the notes made on admitting the case :--

Ambrose Daniell, aet. 10, was admitted on November 19th 1900, to the Belgrave Hospital, under the care of Dr. Ewart, complaining of faintness & feverishness.

History of Present Illness. A week ago the patient complained of "sore throat" which pained him for two days. During the week he had joint pains which shifted from day to day. He stayed at home from school, but did not remain in bed. Several times during the past few days he has had attacks of faintness, & the mother states that at these times he became very pale.

Previous History. The child has been subject to "sore throats". Three years ago, according to the mother's statement, he suffered from an illness similar to the present symptoms. At that time he was feverish and faint, but did not remain in bed although kept at home. No



definite history as to the duration of that illness could be elicited .

Family History. Of seven brothers and sisters, one sister had "rheumatism". There was no other point of importance in the family history.

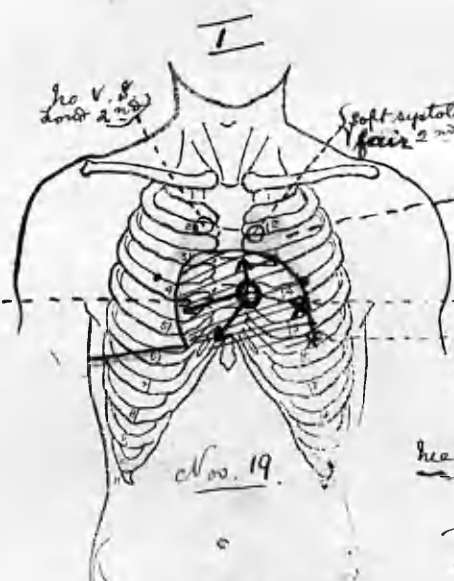
Present Condition. The patient is very pale, with dark rings under the eyes, & is disinclined to speak or move. The skin is clammy. There are no swellings of the joints, no rheumatic nodules, no erythematous or other rash. He is a well nourished child, with short neck & broad chest. The breath is foul, the tongue thickly furred, the tonsils large, but not inflamed. No pain or tenderness is present in the joints or elsewhere . The temperature is 101°.

Cardio-vascular system. The pulse is 112, regular and full. A wavy impulse is visible in the 4th and 5th spaces. On palpation the impulse is felt more strongly in the 4th space in the nipple line, & there is a

Andrew Daniel.

[These are exact copies of the original diagrams, & from these the facing Diagram IV was made up later.]

Supercial or Absolute Dulness.



Long harsh systolic murmur, conducted up the left side of sternum to 3rd rib, across to right of sternum, & to epigastrium

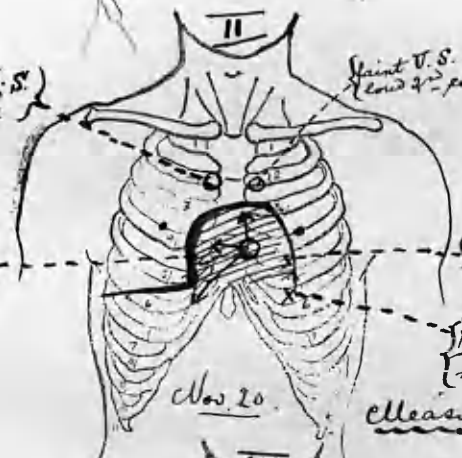
(note impaired over blue area upto roughly 3rd rib. ? deep dulness)

1st sound accompanied & followed by a loud musical systolic murmur & early diastolic murmur, short after a fair 2nd sound  
1st sound & soft systolic murmur, not so much to the left.

Measurements { upper border = upper edge of 3rd rib.  
R. border = 2" to r. of mid-sternum  
L. border = near nipple = 2 3/4" to l. of mid-sternum

Pulse regular, 96, full, fair tension.

Diagram II



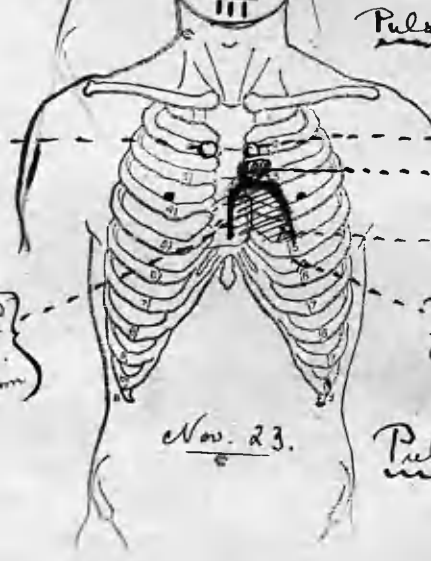
murmur as per above Diagram

Musical systolic murmur, loud 2nd sound, & early diastolic murmur. After smaller, felt as well in 4th as in 5th space. Systolic murmur, softer than in 4th space, & with only a slight musical sound in it; 1st sound is marked, but not very loud; 2nd sound is accentuated

Measurements { R. border = 1 1/2" to r. of mid-sternum  
L. border = 2 1/2" to l. of mid-sternum

Pulse "2, regular, small, good tension; arterial wall readily felt.

Diagram III



soft V.S. heard over sternum, & low 2nd sound. ? V.S. conducted from apex.

soft V.S., low 2nd V.S. lower here - ? conducted from apex.

low 1st sound, soft V.S. with a squeaky sound in it, low 2nd sound followed by a short whiff. After scarcely felt in 5th space; punctate in 4th 1st sound not very loud, soft V.S., low 2nd sound, conducted into axilla.

Pulse 88, regular, small, high tension, arterial wall felt for some way up arm.

Ambrose D

# Case IV Diagram IV

Tracing showing Dilatation of the Heart. { Superficial or Absolute Dulness

Ambrose Daniel

Nov 19<sup>th</sup> 823<sup>rd</sup> - 1900.

1<sup>st</sup> space

2<sup>nd</sup> sp.

3<sup>rd</sup> sp.

4<sup>th</sup> sp.

5<sup>th</sup> sp.

6<sup>th</sup> sp.

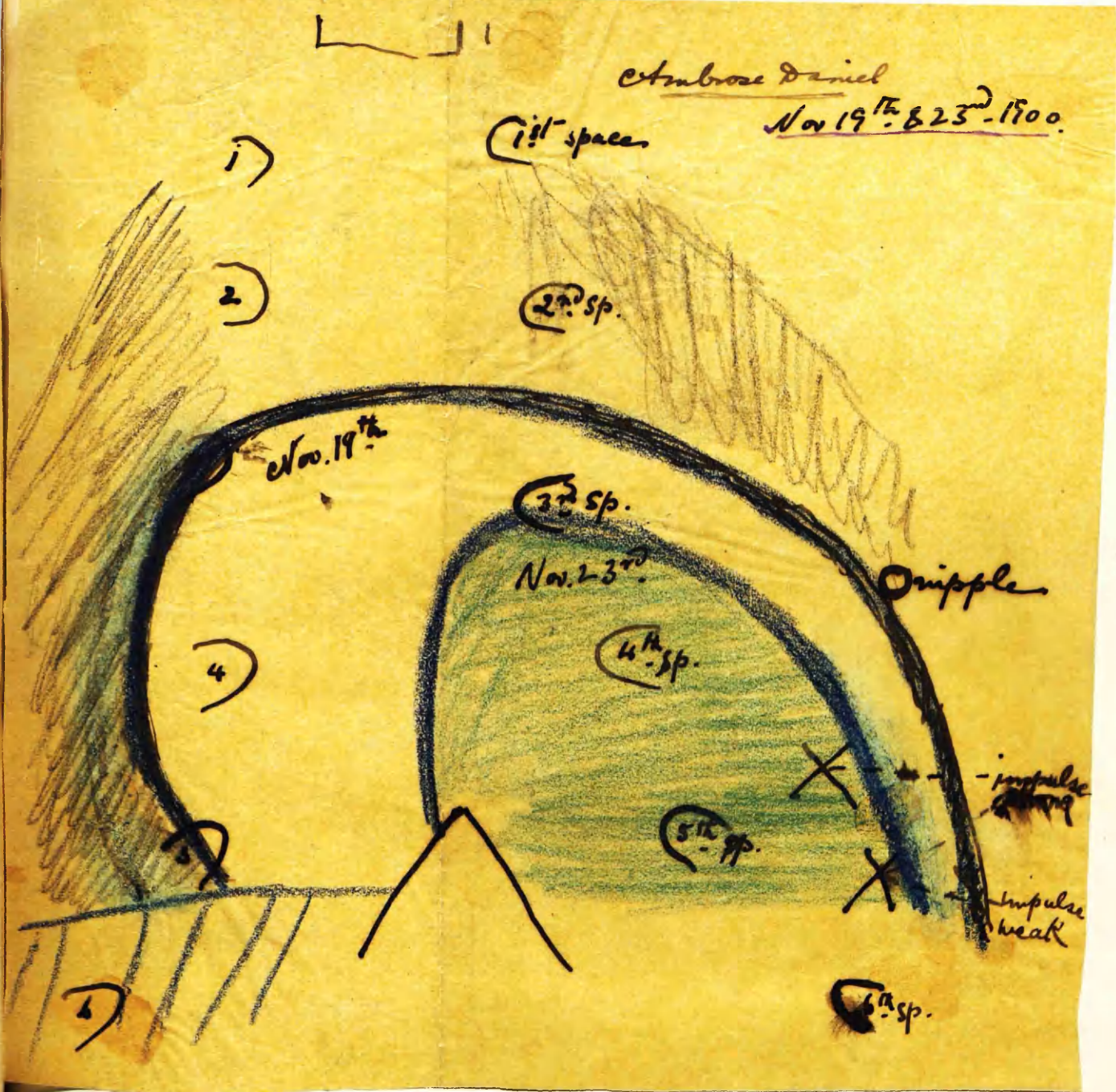
Nov. 19<sup>th</sup>

Nov. 23<sup>rd</sup>

Ripple

impulse

impulse weak



Compared with diagrams I & III, the rubber-stamp diagrams made at the time. The above diagram was made by tracing the chest landmarks, & then drawing upon the tracing-paper according to the recorded measurements, & the shape of the outline in the rubber-stamp diagrams, which were made on Nov. 19. 823. By this means the great diminution of dulness that took place in 4 days is seen at a glance. Thick black outline = absolute or superficial dulness, made out by percussion with the fingers in the ordinary way, on Nov. 19.

Green Area = absolute or superficial dulness made out by same method, on Nov. 23.

Pencil shading = indefinite area of relative or deep dulness made on Nov. 19, by pleximeter; it extended up to the 1<sup>st</sup> space? was it all due to the heart.

systolic thrill. Percussion reveals enlargement of the area of praecordial dulness. The right border measured 2 inches from the midsternum; the left border is in the nipple line-- i.e. 3 inches from the midsternum. The upper border is at the upper edge of the left 3rd rib. ( See Diagrams ~~IV~~ Only the superficial or absolute dulness was recorded in the notes). On auscultation over the impulse in the 5th space there is heard a loud first sound, a fainter second sound, and a soft systolic murmur, which is not conducted very far to the left. Over the impulse in the 4th space the first sound is louder, there is a musical squeak in the middle of the systolic murmur, & the "reduplicated second" so characteristic of mitral stenosis, is distinctly audible. Over the tricuspid area

left

(at the junction of the 4th<sup>^</sup> rib with the sternum) there is a loud harsh systolic murmur, conducted along the sternum

as far up as the 3rd rib, and across the sternum for quite  $1\frac{1}{2}$  inch to the right of the midsternum. At the aortic region no murmur ~~is~~ heard, and the second sound is accentuated. At the pulmonic region there is audible a soft systolic murmur, and a loud second sound.

Respiratory System. Nothing abnormal is found.

Abdomen. The liver is felt about ~~one~~  $\frac{1}{2}$  inch below the costal margin . There is no evidence of enlargement of the spleen, and the other organs appear to be normal.

The Urine is clear, pale, acid, without albumen or sugar.

Case IV - Notes.

The following summarises the notes made on the case during the child's residence in hospital.

On November 20, the day after admission, the child was very pale, and perspired profusely. At noon the temperature was 99.5°. The cardiac impulse remained diffuse. On percussing the superficial dulness, the right border was found to have moved in quite  $\frac{1}{2}$  inch, and the left border had moved in  $\frac{1}{4}$  inch. (See Diagram II). On auscultation near the apex the "reduplicated second" was clearly distinguished on careful analysis to consist of a second sound, immediately followed by a short, soft, early diastolic murmur. The tricuspid systolic murmur was not so loud as on the previous day.

From the day of admission the child steadily progressed towards recovery. The temperature reached normal

Case IV - Notes

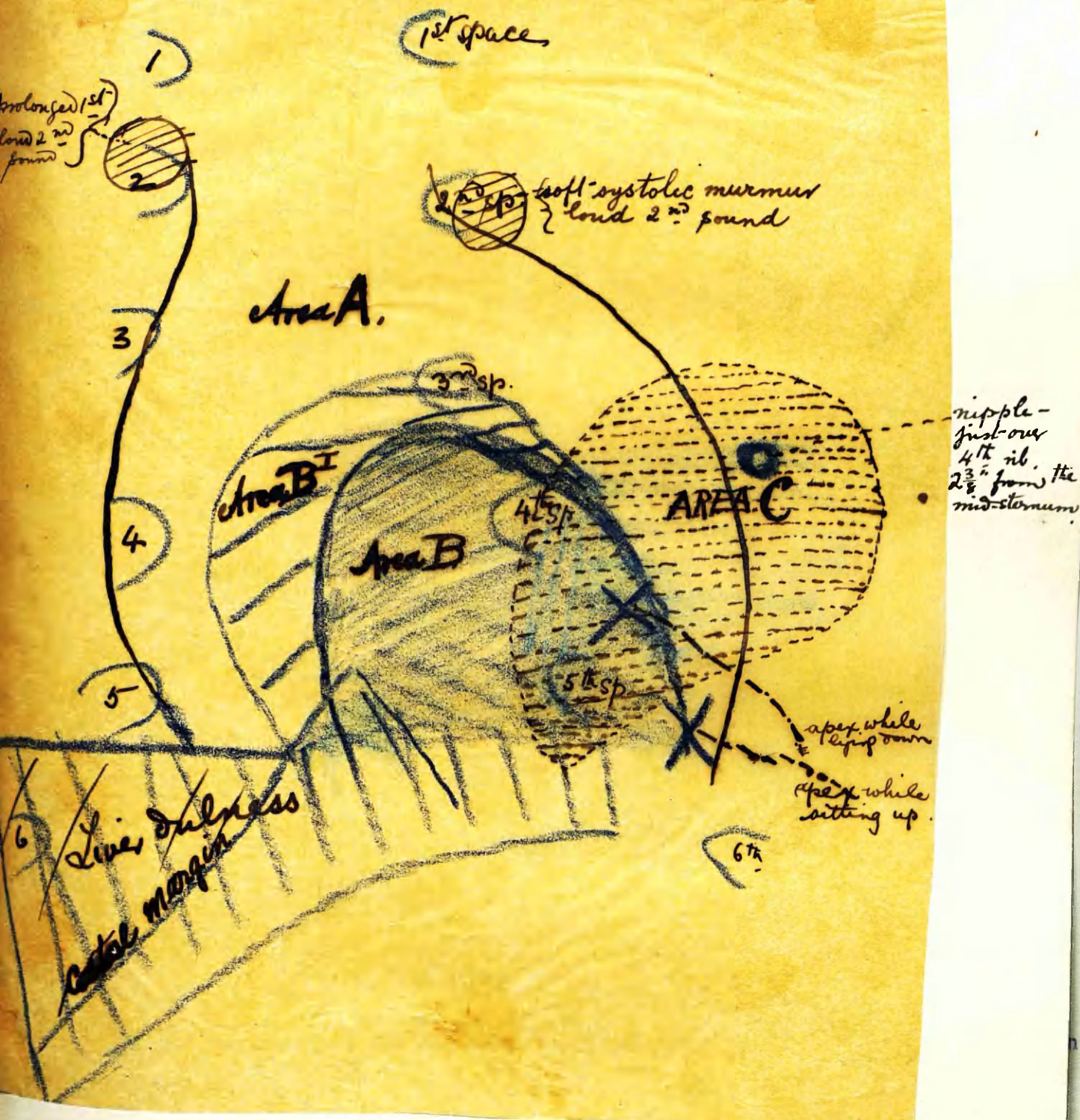
in four days. The pallor and languor remained for several days longer. The area of Superficial cardiac dulness rapidly diminished, until on the evening of November 23, four days after admission, it reached its usual size. The right border was at the midsternum, the left border was almost 1/2 inch within the nipple line. Scarcely any impulse was to be felt in the 5th space, and the impulse in the 4th space was small and punctate. Percussion therefore revealed some dilatation of the right side of the heart. (Diagrams III and IV illustrate these points.) Auscultation revealed but little alteration in the cardiac sounds at the apex; the systolic murmur had acquired a musical squeak, and the second element of the so-called "reduplicated second" audible just within and above the apex, was unmistakably a whiffing, soft early diastolic murmur. The tricuspid murmur had disappeared.

On November 25 the general condition was so much improved that the patient was allowed to sit up in bed.

# Case IV Diagram V

Explanation on fly-leaf below

Ambrose Daniel  
13: FTI: 00





## Case IV.

Area A in Black Outline = area of deep or relative dulness - made out chiefly by aid of pleximeter.

Area B includes { **B**, in thick blue shading = superficial or Absolute dulness made out by percussion with fingers { Upper body = 3<sup>rd</sup> ribs  
Right " =  $\frac{1}{8}$  to r. of mid-sternum  
Left " = 2  $\frac{1}{2}$  l. " " "

{ **B<sup>I</sup>**, in thin blue shading = area where dulness not so absolute as over B, yet more dull than over A.  
This condition remained for several days.

Area C, in pink with dotted black outline, indicates the area over which the early diastolic murmur was audible. Over this area the sounds heard were:-

- (i) 1<sup>st</sup> sound, heavy, but not unduly loud.
- (ii) Systolic murmur, short, blowing, high-pitched, accompanying & following the 1<sup>st</sup> sound.
- (iii) 2<sup>nd</sup> sound, loud, resembling that heard at pulmonic area.
- (iv) Early diastolic murmur immediately after 2<sup>nd</sup> sound, softer, longer, & of lower pitch than the V.S. murmur.

Over the apex, in the 5<sup>th</sup> space, the sounds heard were:-

- (i) 1<sup>st</sup> sound, not very loud except when child sitting up, when it became loud & thumping, with slight roughness immediately preceding it.
- (ii) Systolic murmur, rough, high-pitched, accompanying & following 1<sup>st</sup> sound.
- (iii) 2<sup>nd</sup> sound, not quite so loud or high-pitched as that heard at pulmonic area and over Area C.
- (iv) No diastolic murmur.

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Case IV - Notes

good tension, and the wall of the artery was easily felt for several inches up the forearm. Percussion with the pleximeter revealed no alteration in the area of cardiac dulness since November 28th. On auscultation the first sound at the aortic, pulmonic and tricuspid areas was so soft as to suggest the presence of a systolic murmur. Otherwise the cardiac sounds were unaltered.

On December 13th the heart was again very carefully examined. While the patient was in the recumbent posture the apex appeared to be in the 4th space; when he sat up an impulse was palpable, though not visible, in the 5th space. Percussion of both the deep and the superficial dulness showed no alteration from the notes made on November 28th. Tracings were therefore made so that a permanent record might be kept of the size of the heart while the child was in health. These are shown in Diagram V -- it is seen at a glance that a degree of dilatation of the right ventricle remained. The heart sounds & their several areas of conductivity were then noted and recorded in the Diagram. The alterations in the heart sounds are worthy of note,

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Case IV - Notes

and may be briefly mentioned here. When the child was in the recumbent posture the heart sounds were somewhat distant. When the child sat up, however, the first sound at the apex (5th space) was loud, approaching in character the "slapping" first sound associated most frequently <sup>with</sup> as to <sup>be</sup> almost pathognomonic of mitral stenosis. When the arms were raised above the head a roughness was audible, preceding the first sound. This roughness did not amount to a murmur; (it gave the impression rather that the vibrations were too few in number to produce a musical note). Just within the apex the "reduplicated" second sound became audible. The area over which this was heard was carefully mapped out, and is best understood by referring to Diagram V--Area C.

On the afternoon of December 17th the child appeared rather languid, and the temperature rose to 100°. Percussion revealed slight enlargement in the area of cardiac dulness; (and unfortunately the records of the alteration in size were mislaid. The cardiac action was excited and the heart sounds were louder than usual. At the apex the first sound was

Case IV - Notes

loud and heavy, accompanied and followed by a harsh systolic murmur with a musical ~~spe~~ak in the middle; the second sound was very weak. Just within and above the apex the second sound was clear, and was followed by a long murmur resembling a distant rumbling, which lasted all through the diastole.

At both the aortic and the pulmonary regions the first sound was muffled: at the aortic area the second sound was weak; at the pulmonary area the second sound was loud. At the tricuspid area there was no murmur.

On Dec. 18 the notes made confirmed the report of the previous day. Pulsation was visible from the 3rd to the 5th space, and epigastric pulsation could be felt. When the child sat up for a few minutes the lips became livid; after he had lain down for a few minutes they regained their rosy colour. For the first time it was

observed that the fingers had a slight but distinct tendency to clubbing. The nurses said that they had noticed that after the child had been up for any time his hands were usually somewhat blue.

On the next day, Dec. 19th, pulsation was seen only in the 4th & 5th spaces. Auscultation revealed changes in the heart sounds corresponding to the improvement in the child's general condition. At the apex the first sound was preceded by a roughness similar to that found on Dec. 13th, and the second sound was no longer weak. The early diastolic murmur had disappeared, but on listening carefully a soft mid-diastolic murmur could be heard. So soft indeed was this murmur that it was suspected only because the diastole was not a silent pause, as it normally is.

Three days later the child was quite recovered,

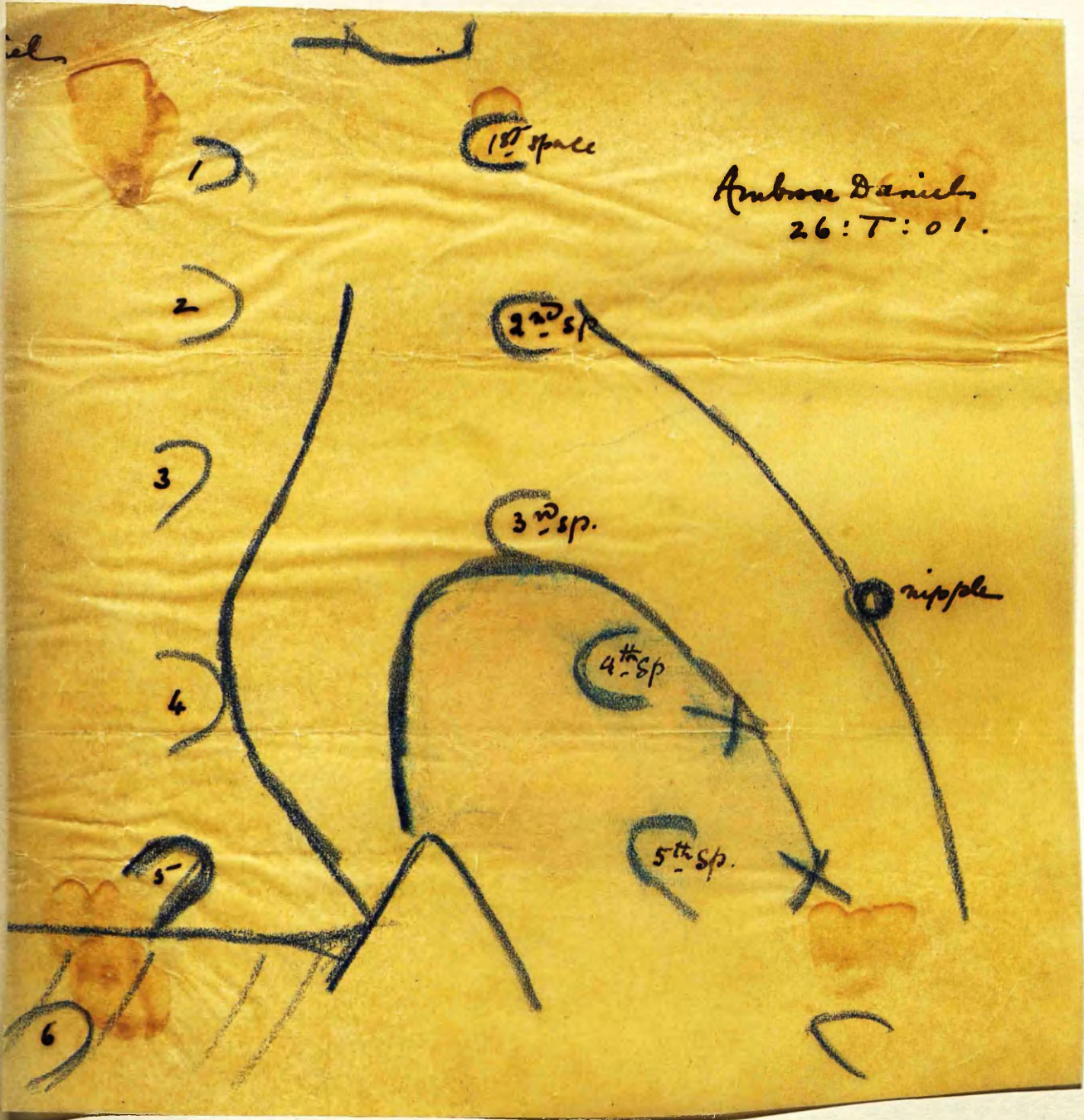
and ran about without any trace of lividity in the lips. The cardiac pulsation was as on Dec. 13th, namely, a small impulse in the 5th space, within the nipple line, and a stronger impulse in the 4th space. The cardiac dullness was as on Dec. 13th. On auscultation just internal to the apex, and in the 4th space, there was heard what at first resembled a double murmur, but on careful analysis the apparent second murmur was found to consist of two elements-- a weak second sound, immediately followed by a short, whiffing, early diastolic murmur. At the other areas the sounds were unaltered.

The child was dismissed from hospital on Dec. 26th with the cardiac dullness & sounds practically the same as on Dec. 13th ( See Diagram V ).

The child attended the outpatient department in January and February. During that time the condition of the heart remained unchanged.

The TREATMENT in this case was as in Case II.

Case IV      Diagram VI  
Tracing Taken one month after leaving hospital.



Ambrose Daniels  
26:T:01.

Outlines made by ordinary percussion with fingers; and the above is the original tracing.

The outer outline shows the area of deep or relative dulness.

The red area is the area of superficial or absolute dulness.

The tracing shows a degree of enlargement of the right heart.

DIAGNOSIS of the heart condition. The

various physical signs pointed to the presence of a double mitral lesion. Moreover, certain facts in the history of the case, and in the physical signs, suggested that the valvular disease was of some standing. (i) As regards the history, it is highly probable that the valves became damaged during the "feverish attack" which the mother remembered so distinctly to have occurred three years previously. The mother was "quite certain" that on that occasion the boy had at least one severe attack of faintness, that he had never before been so affected, & that he had not again had any similar attacks up to the time of the ~~present~~ illness for which he was taken into hospital.

(ii) The physical signs pointed to mitral stenosis of some standing---(a) the pulse was small, firm, of unusually high tension for a child. (b) Even after the acute dilat-



ation had entirely subsided, the right heart remained somewhat enlarged. The right border of superficial dulness, when the child was quite recovered, remained a little to the right of the midsternum, instead of being at the left sternal margin, as in the case of a normal heart.

(c) The slight degree of clubbing of the fingers pointed to a long-standing obstruction in some part of the circulation. In most cases this sign is noted in association with pulmonary obstruction and enlarged right heart.

(d) The diastolic murmurs were of a character which was not compatible with a mitral stenosis of only one week's duration. Much difference of opinion exists as to the time of appearance of the presystolic, early and mid-diastolic murmurs in cases of mitral stenosis; but the majority hold that the presystolic murmur appears in the early

stages of the disease, the early and mid-diastolic murmurs being of later development. However, much stress cannot be laid upon the diastolic murmurs as a proof of old valvular constriction when we remember that there are certainly <sup>some</sup> cases with presystolic murmurs in advanced stages, and others with early or mid-diastolic murmurs in recent stages, of mitral stenosis. When the right auricle is dilated, as it was in this case, it is not strong enough to produce a presystolic murmur by its contraction. When the right ventricle also is dilated it may be so weak that its suction force is unable to produce any early diastolic murmur. Thus it is readily understood why in some advanced cases of mitral stenosis there is no murmur present at all. In this case there were not, except on the first day or two after admission, any signs of dilated right ventricle --- such as tricuspid regurgitation, epigastric pulsation, or venous pulsation in the neck.

Mitral regurgitation was present in this case, but regurgitation was not the predominant condition at the mitral valve. This <sup>was</sup> opinion ~~was~~ arrived at by noting that there

Case IV - Diagnosis of Heart Condition. 55

was little enlargement of the left ventricle in comparison with that of the right ventricle, and that the apical systolic murmur was not conveyed any distance into the axilla.

The sharp differentiation of the sounds heard at the apex--i.e., over the left ventricle<sup>l</sup>, and over the right ventricle, was well marked. These are shown in Diagram VI, and were verified on several occasions, during subsequent days.

Case IV - Etiology

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Etiology. The pyrexia, endocarditis and dilatation in this case, were considered to be produced by the presence of the rheumatic toxin in the blood. The reasons which pointed to the rheumatic rather than any other toxin were :- (i) the pyrexia at its onset was accompanied by sore throat and joint pains, two of <sup>the</sup> most common accompaniments of rheumatism; (ii) the child frequently suffered from sore throats, a complaint which is common in those of a rheumatic diathesis; (iii) there were cardiac signs leading one to believe that the child had in all probability suffered from endocarditis three years previously, the symptoms at that time being so slight as to call for little notice. It is a commonly recognised fact <sup>that</sup> endocarditis of rheumatic origin often occurs in children, accompanied by few or no general symptoms; so that medical

advice is not sought until, perhaps years later, the damaged valve gives rise to circulatory troubles.

(iv) There were no symptoms of other diseases which are known to cause endocarditis or dilatation of the heart, such as Influenza, pneumonia, scarlatina, etc.

A drop of the blood was taken with due aseptic precautions, and examined for the rheumatic germ; but the results proved negative. The details of this examination are given below (p. 58). Poynton & Paine, who have recently succeeded in isolating a diplococcus which apparently is the specific germ of Rheumatic fever, state that the germ is exceedingly difficult to cultivate, & that it is useless to examine the blood unless a large quantity (several ounces) be available.

BACTERIOLOGICAL INVESTIGATION. On the evening of November 21st the fore finger was pricked with due aseptic precautions, and the drop of blood was drawn up into two fine capillary pipettes. These were immediately sealed at both ends. On the following morning the ends of the tubes were broken, and the blood was blown with due precautions into two tubes of slightly acid bouillon, and also into two tubes of blood serum. All were incubated at blood heat in a jar containing pyrogallic acid.

BOUILLON. I. No cloudiness developed in the bouillon tubes. On November 28th <sup>subcultures</sup> were made from the clear bouillon, upon serum and glycerine agar tubes. These were incubated both aerobically and anaerobically. By Dec. 4th no cultures were visible on any one of the tubes.

II. On Nov. 29th similar subcultures were made from the second tube of bouillon. The results were negative.

SERUM. I - On November 26th, ( i.e. four days after the blood was placed on the surface of the blood serum), a cream coloured flat circular colony appeared upon one of the serum tubes. It was found to consist of cocci and diplococci which stained by Gram's method. Subcultures both aerobic and anaerobic, were made upon agar, glycerine agar, gelatine, and glucose agar. Abundant growths appeared upon all, gelatine was liquefied, and it was evident that the microbe was a staphylococcus.

II. Nothing grew upon the second serum tube, although it was incubated both anaerobically and aerobically, and was watched daily till Dec. 6th.

The above examination showed that the specimens of blood were obtained almost free from any contamination. Nothing at all resembling the germ described by Poynton & Paine was found.

CASE V Edith L -----.

A CASE OF ENDOCARDITIS ASSOCIATED WITH MODERATE  
DILATATION OF THE HEART AND PAIN IN ONE JOINT.

- Temperature chart.*
- Case Notes.
- Diagram*
- Summary and Remarks, including Diagnosis.

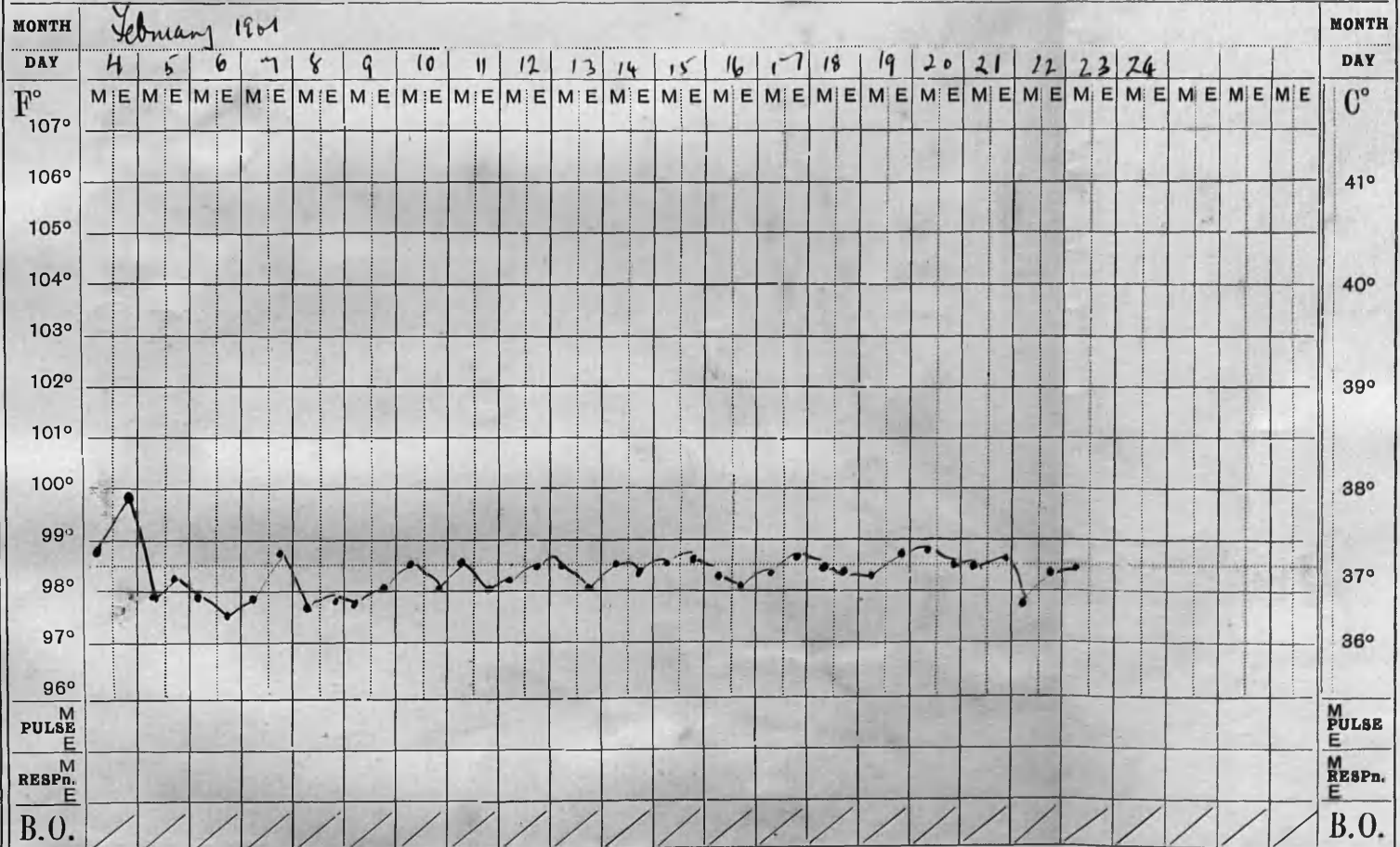


# Case V Temperature Chart.

Edith Lewis act. 6. **Temperature Chart. Case V.**

Day of illness 7 8 9 10 11

*morning & evening chart.*



A CASE OF ENDOCARDITIS ASSOCIATED WITH MODERATE DILATATION  
OF THE HEART AND PAIN IN ONE JOINT.

Edith L-- , aet. 6, was admitted to the Belgrave Hospital for children under the care of Dr Ewart, on February 4th, 1901, complaining of pain in the left leg, of three days' duration.

History of present illness. On January 28th the child came home from school feeling very tired. On the afternoon and evening of the 29th she shivered a great deal, and said her throat was sore. She did not vomit. On the morning of February 1st, she complained of stiffness & pain in the left leg, & would not allow it to be moved. She remained in bed after the pain in the leg set in.

Previous history. The child had been healthy, though not robust, and had not suffered from sore throats, and had only rarely complained of any "growing pains" in the legs.

Family History. Nothing of any importance was elicited. She was the eldest of a family of four, all being strong children.

The following summarises the notes taken after admission to hospital.---

Present Condition.--- The child was pale, of dark complexion and slender build. There were no rheumatic nodules, no rash, and no glandular enlargements. The left leg was held rigidly flexed at the hip-joint. She lay on the back, refusing to turn, or to sit up, complaining that any movement caused great pain in the left leg. All attempts to move the leg on the part of the physician were strongly resisted. There was no swelling apparent about the hip-joint. The tongue was furred, with a red tip; the temperature was 99.8 at the time of examination.

Cardio-vascular system. Pulse:--100; regular. The cardiac pulsation was visible in the 4th & 5th spaces, & on palpation was felt more strongly in the 4th space. There was no thrill. The area of cardiac dulness was found to be moderately enlarged on percussion. The measurements were:--

SUPERFICIAL DULNESS. { L. border--- $\frac{1}{4}$  inch outside the nipple line.  
R. border---midsternum.

DEEP DULNESS.---- { L. border---almost  $\frac{1}{2}$  inch beyond the nipple line.  
R. border--- $\frac{1}{2}$  inch to the right of the midline.

On auscultation a soft systolic murmur was heard at the apex, with a highpitched squeaking in the middle. The pulmonary second was accentuated.

Case V

Diagram to illustrate

- (1) slight degree of dilatation of heart.
- (2) subsidence of dilatation in 2 days.

Edith Lewis

5:11:01

X 11:01



This diagram shows the tracings of both the superficial and the deep cardiac dulness, on two occasions.

The blue outlines = superficial & deep dulness on Feb. 5<sup>th</sup>

The red dotted outlines = superficial & deep dulness on Feb. 7<sup>th</sup>

The tracing shows at a glance that in 2 days the right & the left borders crept in  $\frac{1}{2}$  an inch.

Nothing was found in the lungs or abdomen.

On the next day (FEB.5), the temperature was normal, but the pain in the left hip continued. The cardiac dulness was found to have moved in about  $\frac{1}{4}$  inch on the right side. A tracing of the cardiac dulness was therefore taken--see Diagram 1.

Two days later (FEB.7) the left hip was still painful, but the child permitted it to be moved a little. The cardiac dulness was found to be normal in size, and a tracing was made--see Diagram 1. This tracing shows that the transverse measurement of the dulness had decreased by  $1\frac{1}{4}$  inch since the tracing taken 36 hours previously. The apical systolic murmur was more musical.

On the following day (FEB.8) the child said she had wakened up to find the pain entirely gone from the left leg, & that she now felt quite well. The cardiac condition was unaltered.

On FEB.16 the child was allowed to get up, & on the 26th it was noted **that** the cardiac sounds were heard at the apex, the 1st sound being accompanied & followed by a musical murmur with a

squeak in the middle. The pulmonary second sound was accentuated. Near the apex there was occasionally heard a short whiff, closely following the second sound, and suggesting the presence of a "reduplicated second".

During the next week the area of **praecordial** dulness remained unaltered. The "reduplicated second", however, was only rarely audible. Sometimes one would conclude that one must have been mistaken in thinking one had heard any whiff after the second sound at the apex; and then one would hear it again on another day, a soft short blowing sound, present after every second or third heart beat, not affected with respiration. Sometimes the cardiac rhythm was a little irregular, hurrying with slight exertion, & as readily slowing down again. The apical systolic murmur became louder as the days passed, and was conducted a short distance into the axilla by FEB.25, the date of the patient's dismissal from hospital.

The treatment in this case was as in Case II.

SUMMARY AND REMARKS.

The illness in this case was very definitely stated by the mother to have commenced with sore throat and shivering seven days before admission to hospital. The pain in the left hip joint came on suddenly, remained for a week, and then as suddenly disappeared. It is highly probable that at the onset of the illness there was a considerable degree of pyrexia, and that the pyrexia had almost run its course before the mother sought medical advice for the child.

Attention was drawn to the heart on hearing a soft apical systolic murmur, evidently, from its character, a murmur of recent origin. The dilatation was of so moderate a degree that unless the heart had been carefully watched and accurate records and tracings taken, the dilatation would have passed unobserved. As the worst features of the illness had disappeared by the time of admission to hospital, so it is probable that there had been a greater degree of dilatation, which had practically subsided before the child was seen by a physician.

The pyrexia was considered to have been of rheumatic origin, because of the onset with sore throat ( a common rheumatic symptom), the sudden appearance and equally sudden disappearance of acute pain in a joint, and the presence of a murmur of mitral regurgitation. This murmur was considered to be of recent origin because (i) on admission it was very soft, and was not conducted any distance from the apex, and (ii) as convalescence progressed the murmur became more audible, & was conducted a short distance into the axilla.



CASE VI                      Lily F -----.

A CASE OF PYREXIA, OF OBSCURE ORIGIN,  
ASSOCIATED WITH A MODERATE DEGREE OF  
ACUTE                      DILATATION OF THE HEART.

*Temperature charts.*  
{ Case Notes.  
  Treatment.  
  Diagnosis and Remarks.  
*Diagram.*



Case VI.

## CASE VI.--A CASE OF PYREXIA OF OBSCURE ORIGIN, ASSOCIATED WITH A MODERATE DEGREE OF CARDIAC DILATATION.

Lily F---- aet 6½, was admitted to the Belgrave Hospital for Children under the care of Dr Ewart, on February 6th, 1901, complaining of headache and malaise of about a fortnight's duration.

HISTORY OF PRESENT ILLNESS. For about a fortnight the child has complained of feeling tired, and has lain down on returning from school, being fretful and drowsy at the same time. During the past week she has been too ill to go to school. On Feb. 2nd she shivered and vomited several times. Since that day she has stayed in bed "feeling too ill to move". She has had incontinence of the urine for the past week and the mother says it has an unusually disagreeable odour.

PREVIOUS HISTORY. The child has always been delicate, suffering every winter from attacks of "Weakness", during which she has more or less incontinence of the urine. Two years ago she had pneumonia.

FAMILY HISTORY. Nothing of any importance was elicited at the time of admission to hospital.---but see REMARKS p 75.

The following summarises the notes taken during the child's residence in hospital.

PRESENT CONDITION. The child is very pale, & looks very ill. The breath is foul, the tongue furred, with red tip. The teeth are decayed; the lower gums red & somewhat swollen. There are nervous blinkings of the eyes and twitchings of the mouth, & there is a slight dry cough, resembling a "nervous throat cough". The skin is not clammy, there is no rash, no joint swellings or rheumatic nodules. The tonsils are large, but not congested. The temperature is 103°F.

Cardio-vascular system. Pulse--120, regular, of low tension. The apex beat is in the 5th space, just within the nipple line (2½ inches from the mid-line); there is no thrill

SUPERFICIAL CARDIAC DULNESS--

}	L. border--2¼ inches from the mid-line, within the nipple line.
	R. border---½ inch to L. of the middle line.
}	upper border---4th rib.

DEEP CARDIAC DULNESS--

}	L. border-- 2¼ inches to L. of mid-line.
	R. border--Left sternal margin.
}	Upper border--upper edge 3rd rib.

On auscultation at the apex both sounds are heard; the 1st is accompanied and followed by a soft blowing murmur,

scarcely to be distinguished ~~at first~~ from a prolonged 1st sound. A systolic murmur is faintly audible at the pulmonary area.

Nothing worthy of note ~~is~~ found in the respiratory system or in the abdomen. The respirations numbered 40 per minute.

The urine is pale, acid, 1020, without albumen or sugar. It has a disagreeable sourish smell, and the child has constant desire to pass water.

Feb. 7. Child perspired profusely during night, & the temperature fell to normal at 8 a.m. At 2 p.m. the temperature again rose <sup>to 102.5</sup> <sub>^</sub>, & the patient vomited. There was diffuse pulsation visible in the 3rd, 4th, & 5th spaces. The cardiac dullness was slightly increased, and a tracing was made (see Diagram). The apical systolic murmur was more distinct.

Feb. 8. Herpes appeared on the upper & lower lip, on both sides. The cardiac pulsation remained visible. The

Case VI

Diagram I

Tracing showing slight dilatation of the heart, subsiding to normal in one day; this tracing shows the size of the heart on three occasions.

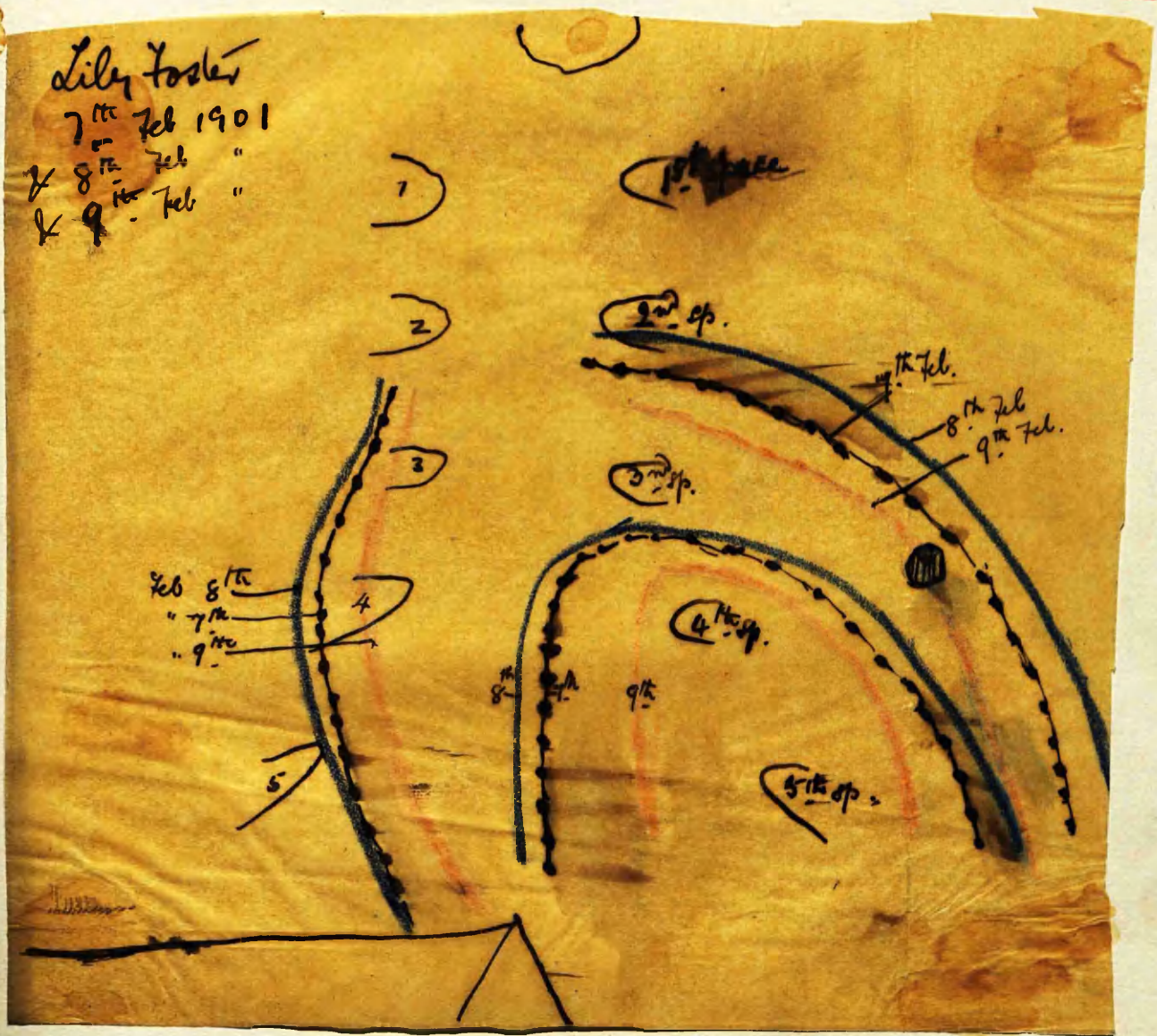


Diagram shows three tracings, of the superficial & of the deep cardiac dulness, taken on three consecutive days.

Dotted black outlines = deep & superficial dulness on Feb 7<sup>th</sup>.

Blue outlines = slight increase; deep & superficial dulness on Feb 8<sup>th</sup>.

Red outlines = subsidence to normal; deep & superficial dulness on Feb 9<sup>th</sup>.

## Case VI - Notes.

cardiac dulness was still further enlarged. The left border of the superficial dulness was  $\frac{1}{2}$  inch further to the left than on admission; the right border was  $\frac{1}{4}$  inch further to the right than on admission. The deep or true dulness showed the enlargement to be still greater ( see Diagram ). The murmurs were fainter than on the previous day.

February 9. The general condition of the patient was greatly improved. The temperature varied from  $97^{\circ}$  to  $99.8^{\circ}$  during the day. There was no pulsation over the cardiac region, and the apex beat in the evening had regained its normal site. The cardiac dulness, both superficial and deep, was almost the same as on admission. A tracing was made ( see Diagram ). The heart sounds were loud, and a soft systolic murmur was audible at the apex.

From February 10 to 16 the systolic apical murmur remained very soft, sometimes scarcely audible, and so variable that it was considered sometimes to be functional, sometimes cardio-pulmonary. The temperature fell by lysis, and remained normal after Feb. 11.

On Feb. 16th, there was slight pulsation felt in the **5th** space. The apical systolic murmur was scarcely audible except when the child was made to lie down. The cardiac rhythm was slightly irregular, a pause occurring after every 4 or 5 beats

On Feb. 17 the systolic murmur was heard at the apex & at the pulmonary area. The cardiac rhythm remained irregular; the heart beats hurried when the child sat up, with every now & then a somewhat prolonged diastole. Occasionally a sound was heard near the apex which greatly resembled a "reduplicated second".

During the following few days the "reduplicated" sound was heard at times. The cardiac rhythm became more regular, & on Feb. 21 the child was allowed to get up.

She appeared so well when up that she was dismissed on

Feb. 25. At the time of discharge from hospital the cardiac

dulness was normal and ~~there~~ was a soft systolic murmur

at the apex & <sup>at the</sup> pulmonary area, not conducted into the axilla.



During the last week of the child's residence in hospital the temperature had shown slight nocturnal rises, from half to one degree above normal, for which no cause was detected until on Feb. 21<sup>st</sup> there was a discharge from the R. ear

A week after dismissal from hospital the child attended the outpatient department. She had been taking a mixture containing quinine & iron, & appeared to be much improved in general health. On examining the heart, the sounds were found to be normal; there was no murmur audible at any region.

TREATMENT. The treatment in this case consisted of absolute rest in the recumbent posture for quite ten days after admission. She was put on milk diet, with a mixture containing sod. salicyl., pot. iod., and spt. ammon. aromat. After a week she was given quinine and iron.

Case VI - Diagnosis & Remarks.

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DIAGNOSIS AND REMARKS. In this case the presence of dilatation of the heart was undoubted, but it was of so transient a nature that unless frequent examinations had been made, & tracings recorded, the condition would certainly have been overlooked. On admission the heart was carefully examined & found to have a normal praecordial area of dulness. The dilatation reached its maximum on the second day after admission, the transverse increase then being only about  $1\frac{1}{4}$  inch greater than normal; and disappeared thirty-six hours later. A soft systolic murmur (& pulmonary region, was heard at the apex) on admission, & became more marked on the following day. After the pyrexia had subsided, the murmur became somewhat variable, sometimes being very loud, audible over the whole praecordium, sometimes being very soft, audible only at the apex & the pulmonary region.

It was never conducted into the left axilla, nor was it ever heard behind at the angle of the left scapula. While the child remained in hospital no definite conclusion was arrived at as to whether the murmur was of functional or of organic origin. When, however, the murmur disappeared entirely after a short course of treatment with iron and quinine, with open-air exercise, it was considered to have been of functional origin.

ETIOLOGY. The cause of the dilatation of the heart in this case was obscure, and was not determined during the time the child resided in hospital. The cause of pyrexia in any given case is best approached by considering (a) the local inflammatory conditions which give rise to pyrexia; and (b) the possible fevers and general inflammatory conditions which give rise to elevations of temperature.

(a) Local inflammatory conditions. The occurrence of a large crop of herpes about the lips is a clue of some value in a case so remarkable for its absence of positive

symptoms or physical signs as the case under consideration.

Herpes about the lips is usually associated with catarrhal inflammations of the respiratory passages, and it has been seen with catarrhal inflammations of other mucous membranes. The respirations in this case were somewhat hurried (36 to 40), though not laboured, and there was a small hacking cough. The possibility of a deep seated patch of pneumonia being present in this case had to be considered, although careful examination of the lungs on several occasions revealed nothing abnormal. The existence of a lobar pneumonia as described in the text-books was of course negatived by the character of the temperature chart and the absence of physical signs. There is no doubt, however, that in clinical work one meets with many aberrant forms and degrees (both in severity and extent) of pneumonic inflammation. Moreover, in children a considerable rise of temperature is commonly seen to accompany a slight bronchial catarrh which presents few or no signs to the stethoscope.

Some physicians use the term "Herpetic" fever in connection with cases which present a rise of temperature extending over a few

days, with no physical signs except a crop of herpes about the mouth or nostrils. But it appears to me undesirable to call a pyrexial condition "herpetic" when the herpes is after all a consequence, not a cause, of the pyrexia.

(b) Fevers and general inflammatory conditions. So strongly did the mother state that the child's condition had altered for the worse on FEB. 2nd, when she shivered & vomited, that the illness was considered to be one of sudden onset as long as she remained in hospital. The pyrexial conditions of sudden onset were therefore considered, and the symptoms weighed in that light. Sudden vomiting in a child previously healthy is always suggestive of Scarlet Fever. The absence of any history of rash does not disprove the suggestion: for the rash is of so fugitive a character that it may readily escape the observation of the laity, especially of the lower classes. On the other hand, the absence of desquamation and the fact that no other cases occurred in the ward, negatives the suggestion. Influenza was for a time considered a probable diagnosis. Influenza is of sudden onset, with considerable prostration, & the course of the pyrexia may resemble that in this case. The toxin of influenza is known to affect the heart, <sup>and some cases</sup> causing dilatation,

Case VI - Diagnosis and Etiology

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irregularity, and sometimes even endocarditis. The twitchings of the face and the incontinence of the urine in this case might have been due to the effect of the Influenza toxin upon the nervous system. But, on the other hand, the characteristic severe pains of Influenza were not present, and the rapid recovery from the cardiac weakness was unlike that usually met with after Influenza. After Influenza, if the heart was affected during the attack, the patient is liable to palpitation, syncope & other signs of cardiac trouble for many months or even years after the illness.

Rheumatic fever is frequently accompanied by dilatation of the heart, and may be attended by great prostration. Sometimes no other symptoms may be present. In this case, however, the dilatation increased while the child was under observation, i.e., at the latter end of the illness for which she was brought to hospital, and the dilatation was of only slight degree, and subsided in two days. In these points the case differed from all the above cases, <sup>(Cases I to V)</sup> which were almost certainly of rheumatic origin.

Case VI - Diagnosis & Etiology . 77

Enteric Fever was a diagnosis at first not entertained, because of the supposed sudden onset of the illness. However, just after the child left hospital on Feb. 25, a sister was brought up to the out-patient department, looking extremely ill, with furred tongue, a temperature over 100°, an enlarged spleen, ~~and~~ a few rose spots on the abdomen, and general bronchitis. The mother stated that this child had been ill for a fortnight with headaches, prostration, the illness resembling in every respect that of Lily, the child who had been taken into hospital. Careful inquiry then elicited the fact that Lily had been quite well up to about 14 or 16 days before admission, that she had then become languid, complaining constantly of feeling tired and ~~h~~badachy. The mother had not paid much attention to her, however, because "I was used to Lily having bad turns every winter". On FEB. 2nd the vomiting attracted the mother's attention seriously, but the child had been too ill to bring up to hospital before Feb. 6th. By that time the mother considered Lily to be improving.

Case VI — *Diagnosis & Etiology*

Nothing could shake the mother's opinion that the two children had suffered from identical symptoms both in point of duration and of severity. The sister was kept under observation, and treated as an Enteric patient. The spots, enlarged spleen and bronchitis all disappeared: and in two weeks the child looked well and healthy, as if nothing had been wrong with her.

A new and unexpected aspect was thus thrown upon the case of Lily, who had been in hospital with pyrexia and cardiac dilatation of obscure etiology. The temperature chart was examined with this new light, and it was seen that it had declined by lysis. Had the chart been continued with four-hourly records for some days longer this would have perhaps been more apparent. The wide variations of the evening and morning temperature on admission resemble the ordinary course of an Enteric fever at the beginning of the third week. Thus the pyrexia in this case probably lasted quite three weeks, and declined by lysis. Enteric fever varies so greatly in different individuals, and may be so mild, especially in children, that the rapid recovery of health in this case is no valid argument against the presence of the disease. Moreover, the



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Case VI - Etiology.

sister recovered even more rapidly, having a bright colour in the cheeks only a fortnight after she came to the Outpatient Department with ~~the~~ enlarged spleen and spots, feeling almost too languid to sit up during <sup>the</sup> examination of her chest.

The most probable diagnosis therefore in this case appears to be Enteric fever. But it does not follow that the dilatation of the heart was due to the effect of the enteric toxin upon the cardiac muscle. When Enteric affects the heart I have usually found that although the degree of dilatation may be no greater than was present in this case, yet it is accompanied by grave constitutional symptoms. In addition, the heart sounds in such cases are distant and feeble, & the pulse is of very low tension. After considering the physical signs of the heart in this case (especially the clear sounds and moderate pulse tension) I have ~~come~~ to the conclusion that the dilatation was due to the anaemic <sup>state of the blood after Enteric</sup> and that the murmurs were similar to the haemic murmurs so frequently associated with anaemia. It is no cause for surprise that at the end of an untreated attack of Enteric Fever a delicate child should show signs of anaemia, accompanied by cardiac dilatation. This opinion is confirmed by the fact that the patient rapidly recovered under treatment with iron.

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REMARKS.

CLINICAL SIGNS AND SYMPTOMS IN WHICH THESE CASES DIFFER FROM  
OF TOXIC ORIGIN,  
THE USUAL CASES OF CARDIAC DILATATION. -- Certain toxins, such  
as enteric, diphtheria, and influenza, exert a special deleterious  
action upon the heart, which leads to dilatation of the  
organ. In these cases, however, according to my experience amongst  
children, the dilatation has characteristic features--(i) it is  
of slight or moderate degree; (ii) it is accompanied by a weak and  
irregular pulse; (iii) the heart sounds are either (a) distant &  
enfeebled, or (b) sharp, clear, and rapid, resembling the "gallop  
rhythm" met with so often in the last stages of valvular disease;  
(iv) the constitutional symptoms, even where the dilatation is  
very slight, are extremely serious, necessitating stimulating  
treatment.

(Cases I to V)

In the cases under consideration, on the other hand, the  
following points were observed:--(i) the dilatation, except in  
Case ~~V~~ <sup>①</sup>, was of considerable degree; (ii) the pulse was never  
rapid, nor was the tension ever very low; (iii) the heart sounds  
were little if at all enfeebled, & the aortic second sound was  
accentuated; (iv) the constitutional symptoms, even where the dil-  
atation was extreme, were never very grave.

The obvious conclusion is that in the above cases the  
propelling power of the ventricles was almost intact, & that the  
cardiac muscle was but little impaired. This important point leads  
us to a consideration of the various causes & pathology of card-  
iac dilatation.

① Case VI is omitted from comparison here as the dilatation  
there was not of toxic origin, & it did not differ  
from the usual cases of anaemic dilatation.

CAUSES.

The CAUSES of Acute Dilatation may be broadly divided into two groups. In the first group the heart wall itself is at fault: the vascular system may be normal, but the heart is unable to perform its share of the work, and so yields or dilates. This occurs chiefly in toxic or in anaemic states. In the second group the heart wall is unimpaired. The heart is suddenly called upon to perform an undue amount of work, and being unprepared for so unaccustomed an output of energy, its cavities yield & dilate. This form of Acute Dilatation is met with after violent exertion, or in cases of Acute Bright's disease, where the peripheral resistance is suddenly greatly increased.

The Acute Dilatation in the cases under consideration undoubtedly belonged to the first of these groups. The cardiac muscle had no undue strain from extra work, and the vascular system presented no increased peripheral resistance. The toxin in operation was not in all cases determined. In Cases I to V the toxin was almost certainly of Rheumatic origin. In Cases II and III it was associated with recent endocarditis; and in Case V, with a rheumatic joint lesion. In Case IV the patient had evidences also of a slight degree of chronic dilatation, especially of the right heart, consequent on mitral disease of some standing. In Case VI, after reviewing the various possible causes, it appeared most probable that the patient was suffering from anaemic dilatation after an attack of Enteric Fever. I have described it at length so that it may be compared with Cases I to V, which were almost certainly of Rheumatic <sup>i.e., toxic</sup> origin.

Before discussing the pathology of acute dilatation in rheumatism, it would be well to review the history of this affection.

HISTORY OF ACUTE DILATATION OF THE HEART OF RHEUMATIC ORIGIN.

Whereas chronic dilatation of the heart has been long recognised by the medical profession, it is only recently that attention has been directed to acute, or, as some prefer to call it, rapid or temporary dilatation. The recent text-books make no mention of the fact that there may be enlargement of the heart dulness with an attack of acute endocarditis. It appeared to me impossible that this fact could have entirely escaped observation until Lees in 1898 drew attention to its occurrence in children. I therefore looked up several of the older writers, and found that my suspicion was correct. In 1839, in "Diseases of the Heart" Hope writes that an enlarged praecordial dulness is a common sign in acute endocarditis. He states-"Percussion is dull over an area of 4,9 and even 16 square inches. I cannot easily comprehend how the walls of the heart can simply swell to such an extent.... Dulness from this cause may be discriminated from that produced by fluid in the pericardium by the impulse sensibly striking the walls of the chest!" The phenomenon must have been noticed even before his day, for he adds-"M. Bouillaud, if I understand him, ascribes this to "turgescence of the heart, from the inflammatory fluxion."

In 1873 Walshe points out that with acute endocarditis the heart dulness, both superficial & deep, may undergo a "trifling increase" because the walls of the organ are turgid..The area of

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of dulness is never seriously increased, unless there be considerable distension of the heart by accumulated blood; then, mainly to the right".

In 1879 Goodhart, in Guy's Hospital Reports, describes several cases with acute dilatation of the heart associated with Bright's Disease which had followed Scarlet Fever. In The remarks at the end of his paper he says that Sir Willnam Gull used to teach that pericarditis in Rheumatic Fever is often accompanied by acute dilatation of the heart, which is most often mistaken for effusion of fluid in the pericardial sac. Goodhart observes that this teaching bears out his statements as to the possibility of the occurrence of acute dilatation. He adds the significant words that the condition "is hitherto unrecorded"!

No mention of cardiac enlargement accompanying acute endocarditis is made by Watson or Fagge.

In 1889 Henoeh, in his Diseases of Children, remarks--"Of the acute form of dilatation of the heart described by Steffen and others, the diagnosis of which rests only on percussion, and which is said to come on in endocarditis ( as the

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result of engorgement & overwork of the heart), as well as in infectious diseases, I think I have myself observed a few cases..... I must admit, however, that in regard to the majority of these cases I feel a little doubtful, owing to the rapid recovery from the dilatation, within a few days".

These last words are worthy of particular notice; they have been confirmed by subsequent writers very fully.

The next reference of value was made by Sansom. In the International Clinics, (vol. I. page 7, 1894), he describes a typical case of acute rheumatism, and states that there is often increase of the praecordial dulness in such cases. He adds--"I am convinced that the rapid increase of dulness over the heart in Rheumatism ~~are~~<sup>is</sup> not all due to pericardial inflammation.... the whole heart may become swollen and dilated-- swollen with the products of inflammatory exudation, dilated because of the enfeeblement of the muscle of its right and left chambers..... These variations in the bulk of the heart may be observed in some cases to be considerable from day to day, and there may be ~~the~~<sup>repeated,</sup> enlargement at intervals of a few days, just as there may be repeated swelling in the joints". He gives diagrams of a case, ~~in~~<sup>in</sup> which the right border was considerably beyond the right sternal margin, and

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the left border beyond the nipple. A diagram of the same heart, taken twenty-four hours later, showed the dilatation almost completely gone--the right border was at the mid-sternum, the left border within the nipple. In CASE I. (ante) the dilatation was almost as rapid.

Of late years, however, much attention has been drawn to the subject. Dr. D.B. Lees read a paper at the Medico Chirurgical Society of London in 1898, on Acute dilatation of the Heart in Rheumatic Fever. He first observed that the dilatation which occurred with rheumatic pericarditis was greater than that due to any other other cause. Subsequent observations gave him reason to state---"In Rheumatic Fever there is almost always more or less dilatation of the heart". He showed diagrams at the meeting, of great dilatation, occurring without any murmur being present. From this fact he pointed out the extreme importance of percussion in diagnosis with regard to the condition of the heart. Unless percussion was always carefully performed, dilatation would in some cases be overlooked, and the heart even might be considered to be normal.

History of Acute Dilatation in Rheumatism.

In the same year, and at the same Society, Lees and F.J. Poynton read a paper, in which a number of cases were recorded, and diagrams shown, fully illustrating the fact that great dilatation of the heart may set in acutely and as rapidly subside, in rheumatic fever. In the following year Poynton described a remarkable case, where there was very slight pericarditis, but enormous dilatation of the heart, the right border extending to the right of the right nipple. The patient died, and the post-mortem examination confirmed the diagnosis made during life. The microscopical examination of the heart wall will be referred to later, when dealing with the pathology of the condition.

In the chapter on Acute Endocarditis in Clifford Allbutt's System of Medicine ( Vol. V ) reference is made to this condition thus-- "On percussio n it is only in exceptional cases that we notice the increase of the area of cardiac dulness, due to the dilatation of the left ventricle, the right, or of both".

On the other hand, in the 1900 edition of his work on diseases of the heart, Sir William Broadbent writes thus, when



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describing Acute Endocarditis---"the **area** of cardiac dulness may become much enlarged when the inflammatory process involves the muscular substance of the heart as well."

Sansom also, in his Diseases of the Heart and Aorta, writes --"I am convinced that, exceptionally, the whole heart may become enlarged, without any of the ordinary evidences of pericarditis or endocarditis, in association with rheumatism".

Enough has been said to show that there is ample evidence from many observers as to the **occurrence** of acute dilatation of the heart in rheumatism.

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PATHOLOGY OF ACUTE DILATATION OF THE HEART IN RHEUMATISM.

The early writers above mentioned ascribed the acute enlargement of the praecordial dulness to "swelling of the heart", both from accumulation of inflammatory products and by abnormal distension of the cavities of the heart with blood. The true pathology of the condition remained for long a matter of conjecture, owing chiefly to the fact that the patients usually recover, and no postmortem examination is therefore available. It has been long recognised that the muscle of the heart wall is liable to become affected in certain of the specific fevers, and the fact that cardiac dilatation was frequently found to accompany Rheumatism lent confirmatory evidence to the growing suspicion that Rheumatism also was due to the action of a specific germ. The clinical symptoms however differed widely-- in the specific fevers the dilatation was of slight degree, accompanied by serious general symptoms; in Rheumatism the dilatation might be enormous, recover rapidly and be unaccompanied by any serious symptoms. It was therefore impossible to consider that the effect of the rheumatic toxin upon the heart resembled that found in the specific fevers.

IN 1882 West showed at the Pathological Society of London a specimen of a greatly dilated heart, with fatty degeneration visible to the naked eye. Great interest was excited by the history of the case, which was that of a young man who had suffered from Rheumatic fever, remained in bed only one week, and then developed signs of cardiac failure. This had become worse, & death had occurred about two months after the first onset of the illness. Thus it was proven that Rheumatism might lead to a rapid fatty degeneration

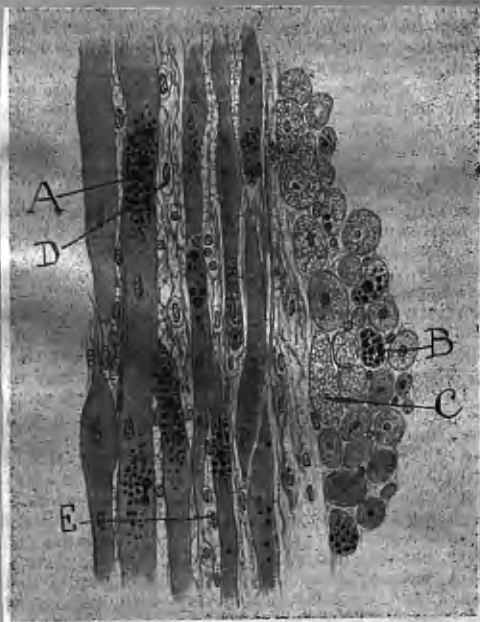
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of the cardiac muscle. No light however was thrown upon the path-  
ology of acute dilatation as described by Lees & Poynton and the  
above ~~six~~<sup>five</sup> cases. Sequeira ( Med. Chi. Soc., 1899 ) argued ably  
that great dilatation must be preceded by pericarditis, because a  
healthy pericardium would prevent the occurrence of dilatation  
beyond a certain point. He considered that in Rheumatism there was  
no myocarditis except that due to extension from endo- or pericard-  
itis, & that a postmortem examination of cases presenting great  
dilatation would reveal pericarditis together with a slight degree  
of myocarditis immediately beneath the pericardium.

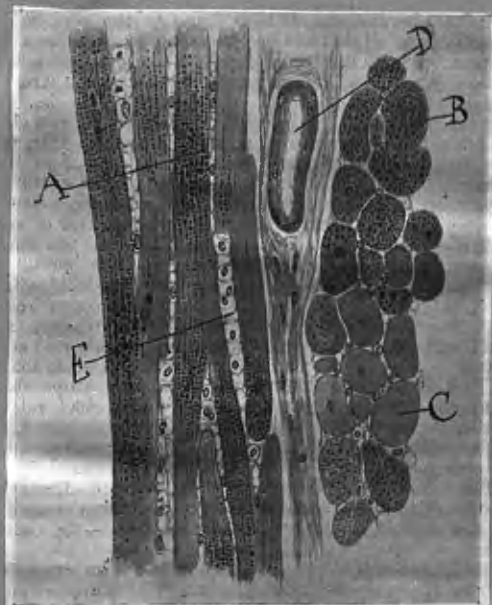
In the same year Poynton recorded a case of acute dilat-  
ation accompanied by pericarditis. The patient died; the post-  
mortem examination confirmed the observations made during life.  
There was dilatation of the right side of the heart so great that  
the right border extended beyond the right nipple; very little  
pericardial fluid was present. The microscopic examination of the  
heart wall revealed certain changes in the muscular fibres & the  
interstitial tissue which were not localised, as had been expected,  
to that part of the heart wall immediately below the pericardium,  
but were generalised throughout the myocardium. The chief hist-  
ological change consisted in the presence of foci of inflammatory  
cells between the muscular striae; the muscular fibres themselves  
showed slight granular & fatty changes, but these were not so  
great as are found in alcoholic hearts with only slight dilatation.  
These results were confirmed in the same year by the postmortem  
examination of the heart of a young adult who had suffered from  
Rheumatic fever in childhood, and whose death had occurred during

a second attack of the disease. The heart wall in this case showed (i) foci of recent inflammation and (ii) a degree of fibrosis. The latter was probably due to the fibrotic change which takes place in old inflammatory foci, and was the result of the inflammation which had accompanied the first attack of Rheumatism.

Further investigations confirmed these points. The changes in Rheumatic dilatation are (i) generalised throughout the myocardium and (ii) interstitial rather than parenchymatous. The latter fact explains the clinical observation that the propelling power of the ventricles in Rheumatic dilatation is but little impaired (see p 80). I add here two illustrations from the Lancet (May 12 1900) which enable one to comprehend at a glance the different points in the pathology of Rheumatic and Diphtheritic dilatation of the heart.



part of the left ventricle from the case of diphtheria fixed in Herrmann's fluid. A, Muscle fibre showing much localised fatty change. B, Muscle fibre in transverse section showing fatty change. C, Muscle fibre in transverse section showing destruction of the protoplasm. D, Muscle fibre entirely destroyed. E, Cellular elements between the muscle fibres. The more gross changes are alone shown in this figure. The alterations in contour and size of the fibres are distinctly seen in this section.



part of the left ventricle from the case of acute rheumatic carditis fixed in Herrmann's fluid. A, Muscle fibre showing much fatty change. B, Muscle fibre in transverse section showing fatty change. C, Muscle fibre in transverse section, not fatty. D, Bloodvessel in a connective tissue space. E, A capillary between two muscle fibres. The finer details described in the paper would necessitate several more plates for their representation. The gross changes only are delineated in this figure. The muscle fibres are seen to have preserved their contour.

The Diphtheritic heart shows (i) much fatty degeneration of the muscle; (ii) much destruction of the muscle; & (iii) little interstitial change.

The Rheumatic heart shows (i) Slight fatty & granular change in the muscle striae; (ii) Little destruction of the muscle; & (iii) inflammatory foci scattered throughout the myocardium, between the muscular striae.

Recent reserches by Poynton and Paine have resulted practically in the discovery of the germ of Rheumatic fever. This germ is a minute diplococcus, which grows in chains in liquid media, and in a staphylococcal arrangement on solid media. It grows best anaerobically in a slightly acid medium, & is easily decolourised by Gram. This germ was obtained from eight successive cases of Rheumatism, from the joint exudations, blood of the heart, pericardial fluid and urine from the bladder. Cultures injected intravenously into rabbits produced all the symptoms of acute rheumatism. The germ was then isolated from the blood of the animals and cultivated with success. Koch's postulates were thus fulfilled. Microscopic sections of the various organs revealed the presence of the diplococcus. In the heart the germ was found under the endocardium & the pericardium.

The first question that arises to the mind is -- Is it possible for a toxin circulating in the blood to produce the above described changes in the cardiac wall? The second question is -- What is the cause of the inflammatory foci between the muscular striae? These questions are best answered by considering Diphtheria & Enteric, & by reasoning from known to unknown conditions. In these diseases the specific germ has a special seat -- The throat in one, the intestinal canal in the other. At these sites leucocytes appear in abundance to endeavour to combat the toxin or destroy the germs before the toxin can circulate in the blood and produce its poisonous effect upon all the tissues & organs of the body. In our present state of knowledge therefore, which has as data the presence of

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(1) inflammatory foci in the heart, and (2) of a diplococcus which in animals causes all the symptoms of acute rheumatism, I think we are justified in ~~assuming~~ <sup>concluding</sup> that in Rheumatism the heart is the special habitat of the specific germ, just as the throat in Diphtheria, the intestinal canal in Enteric, are the habitats of their respective germs.

DIAGNOSIS.

CLINICAL DIFFICULTIES IN DETERMINING THE PRESENCE OF  
CARDIAC DILATATION.

Owing to the small size of the measurements in children special difficulties are experienced in determining the presence and extent of cardiac dilatation in them, and much care and patience is required to avoid error. Apart from this source of fallacy there are certain other difficulties which may occur at all ages. Thus, as regards Inspection and Palpation, reliable evidence of dilatation is afforded by the presence of a displaced apex beat or a diffuse and wavy pulsation over the praecordium. But these signs may be obscured by a fatty or a muscular chest wall. Moreover, with a slight or moderate degree of dilatation such signs are entirely absent. As regards Auscultation, no reliance can be placed upon this method of physical examination as a means of detecting dilatation unless it be of considerable extent, when the cardiac sounds may be clear and "clicking", or accompanied by various murmurs.

It is therefore evident that the detection of cardiac dilatation depends chiefly upon Percussion. The accurate performance of percussion is thus of the utmost importance, and recent methods of physical examination have greatly improved in this respect. At first the obstacles to the performance of accurate percussion appear almost insuperable. The heart in health



Definition of Deep & Superficial Dulness.

is overlapped by lung above and on both sides, and the more central portion lies immediately beneath the chest wall. The orthodox teaching, supported by scientific reasoning, points out that in order to map out the uncovered area of the heart it is necessary to employ a light or gentle stroke in percussion. By this means an absolutely dull note is obtained, due to the short or high pitched vibrations set up on striking a solid organ. If the percussion were not extremely gentle, the pulmonary resonance from the adjacent lung tissue would be elicited, thus rendering obscure the accurate limit of uncovered heart. This area of uncovered heart is described as the SUPERFICIAL or ABSOLUTE DULNESS. On the other hand, a heavy or strong stroke in percussion is necessary to detect the boundary of the entire cardiac area. A gentle stroke over the area of overlapping lung tissue would excite vibration over so limited a distance that it would elicit only the note of pulmonary resonance, and fail to detect the solid cardiac tissue beneath. The heavy stroke however sets up vibrations over a greater distance & thus ~~the~~ the dull note due to the presence of a solid organ is heard together with the pulmonary resonance. The area made out by heavy percussion is known as the area of DEEP, TRUE or RELATIVE DULNESS.

ORDINARY METHODS OF PERCUSSION.

(a) Direct method. The finger is laid flat on the chest parallel to the middle line and first the right and then the left boundary of the heart should be determined. Next, the upper boundary is made out by laying the finger in a sloping direction from the left nipple to the second costal cartilage, and moving it downwards in the same plane until the change of note is detected. It is to be remembered that the left border of TRUE DULNESS extends somewhat to the left of the apex beat, and that owing to the presence of the great vessels the upper boundary cannot be determined over and near the sternum. In attempting to mark out the area of Superficial dulness, it is best to find first a part which is absolutely dull and thence to proceed gently outwards and upwards, to decide where resonance begins-- at that point the lung overlaps the heart. On the other hand, in attempting to mark out the area of Relative dulness, it is best to start from a part of the chest known to be covering lung tissue. Having thus obtained the note of pulmonary resonance it is advisable to work gradually nearer the cardiac area, listening for a change of note. At the extreme edge of the heart this is so faint that it is readily missed, the pulmonary resonance being so predominant.

(b) Mediate or Pleximeter method. It is held by some that a suitable pleximeter diminishes the loudness of the pulmonary vibrations and so detects the cardiac dulness at its extreme edge. Sansom's pleximeter, Dr Ewart writes, "acts as a mute for the sonorous vibrations". This effect is obtained on account of the fact that the hard vulcanite of which the instrument is composed damps the large or deep-toned sound waves, conducting preferably the short high-pitched vibrations. The pleximeter in question is easily



portable (see fig.) and consists of vulcanite, a short stem joining two flanges. One flange is held flat on the chest wall by two fingers, one on either side of the stem, while the other flange is percussed by the middle finger of the right hand.

Difficulties in percussion due to abnormalities:—Apart from

the difficulties met with in appreciating the percussion notes in the normal chest— difficulties which can be overcome by training and experience— there are often puzzling conditions arising from (a) abnormal lung states, e.g., emphysema, pleurisy, contracted & fibrotic lung, etc.; (b) abnormal states of the abdominal organs, e.g., distended stomach or intestines, etc., and (c) individual variations of the chest wall, such as deformity or the ribs or ster-

num or increase of muscular or adipose tissue. Any one or all of these difficulties may be present and cause trouble in mapping out the dulness even of a normal heart.

Difficulties in percussion due to age.-- There is special difficulty in deciding whether a heart is or is not dilated when the patient is of tender years. There are in such cases two points of difficulty--(i) the area of dulness as mentioned above is in childhood extremely small, and (ii) the normal area of dulness varies with the age and size of the child. Normally at six years of age Dr D.B. Lees gives as the limits of DEEP Dulness--  
LEFT--3rd c.c. to 4th space, just inside the nipple line  
RIGHT-  $\frac{1}{2}$  inch to right of middle line, inclining slightly inwards to the liver dulness.  
From six to twelve years the area of DEEP dulness remains practically the same as these measurements. Under six years the left border of the deep dulness is in the nipple line. On the other hand the SUPERFICIAL Dulness varies between the ages of seven and twelve-- its upper border being at first at the level of the upper edge of the fourth rib, and later on at its lower edge (Sturck).

Relative Value of the Deep and the Superficial Dulness---

The question whether deep or superficial dulness gives the more accurate information as to the size of the heart is one of prime

importance. Superficial dulness is employed & taught in many schools, especially in Scotland. Some schools, however, teach that accurate records are obtained only by the percussion of the deep dulness. Arguments can be brought forward in support of both methods.

Ewart, Lees & others consider that percussion of the superficial dulness gives information more as to the condition of the lungs than as to the size of the heart. The superficial dulness indicates only the part of the heart which is immediately beneath the chest wall, and its size therefore must depend largely upon individual variations both of the chest wall and of the lungs. Thus, in emphysema, this area is diminished & may be altogether absent. In children especially the chest wall is so elastic, the lungs are so near the surface, that the pulmonary resonance makes it almost impossible to define with any precision the superficial dulness.

On the other hand, it is certain that in the majority of cases, especially adult cases, the <sup>percussion of the</sup> superficial dulness is more readily & easily performed, & can be mapped out with fair accuracy even by those who have had but little experience or training. Thus it has been argued that the results obtained by percussion of the superficial dulness are on the whole more likely to be correct. Even, so careful an observer as Lees states that the limits of the

deep cardiac dulness cannot be defined with mathematical precision and that "only an approximation can be obtained". To avoid pitfalls in this direction Dr Lees speaks of "fingerbreadth" measurements rather than inches. Other teachers speak of the importance of estimating so slight a degree of dilatation as  $\frac{1}{8}$  or  $\frac{1}{4}$  inch, and insist on this as an illustration of the greater value of deep percussion, which measures the true size of the heart.

Where authorities differ so widely it is evident that each observer who follows his own methods must after careful experience be enabled to set up a table of comparison for himself by which he can estimate the presence and degree of dilatation. Thus, although it may be true that no two independent observers would mark out precisely the same limits of the same heart, yet each man would be able to state that, according to his method, the heart was dilated, say, to the extent of about  $\frac{1}{4}$  inch. The practical conclusion is, that in making hourly or daily records of a case, the percussion and tracings must be performed by the same observer.

Method adopted in the above cases. ~~During the percussion~~ of the heart in the above cases the child was always lying on the back with the arms close to the side. Both superficial and deep dulness was mapped out at least once daily, and on many occasions twice or thrice during the same day. The limits were then marked on the skin with an aniline pencil. Next, as landmarks, the nipples, the suprasternal notch, and the epigastric notch were pencilled. Then tracing paper was laid on the chest and held in position by a nurse, whilst with a pencil the skin marks were readily drawn upon the almost transparent paper. To prevent fallacy due to preconceived

notions, the diagrams in almost every case were made without reference to any previous tracings. In most cases I employed direct percussion; on a few occasions I found the pleximeter of great aid. When arranging the diagrams it occurred to me that by superimposing the records taken on separate occasions, and tracing them on the same sheet, the great variation in the size of the heart would be seen at a glance. In order that as complete a picture as possible might be presented I at the same time added to the diagrams marginal notes describing the characters of the cardiac sounds.

Practical Points illustrated by the diagrams.--

1-- Slight Dilatation. Where the dilatation was slight, as in Cases III, V and VI, I found that it was only by means of deep percussion that I could tell that dilatation was present. The diagrams show both the deep and the superficial dulness on several occasions in these cases, and it can be seen at a glance that, although the superficial dulness underwent diminution as the cases recovered, it would have been difficult to state definitely on the first day of examination that there was abnormal enlargement of the superficial dulness. Without careful daily percussion such cases of dilatation would pass unobserved, unless the deep dulness was mapped out at the beginning.

2-- Considerable Dilatation.--When a considerable degree of dilatation was present I found that the superficial area of dulness was relatively much more increased than was the ~~deep~~ area of deep dulness. Thus, in Cases I, II and IV, the superficial dulness alone was recorded at first; the deep dulness was very little larger, and it was so hard to accurately define its limits that I thought it wiser

not to record it in a diagram. As the patients recovered, the superficial dulness diminished, and became more and more difficult to define. In Case IV, where the sternum was somewhat prominent, it became at last impossible to elicit any "absolute" dulness.

My observations with regard to the percussion of the heart in children may be summarised under three headings:--

(1) The ordinary rules of percussion, i.e., a light stroke for the superficial, a heavy stroke for the deep dulness, hold good in many cases; but there are other cases in which the reverse holds equally true. In most cases I found it also a simple matter to map out the deep dulness with a light stroke. <sup>①</sup>

(2) In children with a normal heart, the deep dulness is more easily mapped out than the superficial dulness, and therefore the results of deep percussion are more likely to be correct. The younger the child the more necessary is it to employ deep percussion in estimating the size of the heart.

(3) Where cardiac dilatation is present it is obvious that both the superficial and the deep dulness must be increased; but whereas a slight increase in the superficial dulness may in a child escape observation, or may be considered to be normal, a slight increase in the size of the deep dulness will readily attract attention as abnormal. Especially is this true when the right ventricle is enlarged, because the sternal resonance prevents the detection of the increase (unless very great) of the superficial dulness to the right.

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① I have since heard that Lees employs a light stroke in percussing the deep dulness. He maintains that in children the change of note is more readily made out by gentle percussion.

PROGNOSIS  
OF ACUTE DILATATION OF THE HEART

When Acute Dilatation is associated with severe symptoms of cardiac failure, such as breathlessness, pain, vomiting and syncope, the prognosis is necessarily most grave. But in the more moderate cases, cases where the dilatation per se gives rise to few or no symptoms, the physician may have difficulty in forming a prognosis. Acute Dilatation is not a primary disease, and therefore, except in the severe cases, the prognosis depends more on the cause than on the extent of the dilatation. For this reason, a prognosis cannot be ventured upon without first making an exhaustive inquiry into the cause of the condition.

As regards the cause in operation the recognition may be simple in cases of Enteric, Influenza, Diphtheria, Pyaemia, or Anaemia. The prognosis in Anaemia is good; in Diphtheria it is most grave, fatal syncope being liable to ensue on the slightest exertion. In Influenza the immediate prognosis is not so serious, but the patient is left with an "irritable" heart, palpitation and irregularity of the cardiac rhythm coming on with little provocation. In Enteric and in Septic conditions of the blood the prognosis will depend upon the course of the disease and the presence of complications. In all these diseases subjective symptoms are severe if the dilatation become of any considerable extent.

Sometimes, however, the recognition of the cause of the dilatation may be so obscure that no decision can be arrived



at. It sometimes happens that in such cases the form of the dilatation itself is an aid to the diagnosis of the cause of the dilatation. Thus, an examination of Lees' and Poynton's cases of rheumatism accompanied by dilatation of the heart shows that the dilatation was often of marked extent, though with few subjective symptoms. In our present state of knowledge we are justified therefore in assuming that dilatation presenting these features is of rheumatic origin, even in the absence of recognised rheumatic lesions. So true is this that the presence of such dilatation may enable us to diagnose the cause of pain in a joint not easily examined--e.g., in Case V, the presence of cardiac dilatation confirmed the suspicion that the painful hip-joint was affected by rheumatic synovitis.

As regards the immediate future, the prognosis of Acute Dilatation due to rheumatism is more satisfactory than is that due to any other cause. A slight degree of dilatation of the heart does not add any gravity to the usual prognosis of rheumatic endocarditis, because, as Lees points out, "Acute dilatation of the heart is a frequent, almost a constant occurrence, in a Rheumatic attack". The prognosis is however much less favourable when there is a history of previous endocarditis, myocarditis or pericarditis, because the cardiac wall is then weakened by previous disease. The prognosis is also graver when pericarditis sets in as a complication, because a pericardial sac softened by inflammation will permit the

Prognosis.

heart to dilate to a dangerous degree.

As regards the prognosis of the distant future it is as yet impossible to speak with certain voice. It is highly probable, when one considers the pathology ( see p<sup>88</sup><sup>top</sup>), that after each attack of rheumatism, even those attacks which occasion so little disturbance at the time that the patient does not seek medical advice, a certain degree of muscular degeneration and interstitial fibrosis occurs. This must lead to more or less impairment of the heart power. This impairment may perhaps be in some cases so slight that it in no way affects the prognosis of a long and active life in healthy circumstances, but may greatly hasten the ordinary causes of cardiac degeneration in middle life & old age, and may thus be a fatal source of danger in diseases (especially diseases of the lungs, kidneys or arteries) which would have been readily enough resisted by a healthy heart.

The point of all others which is illustrated by the above cases is the importance of promptly putting children to bed when suffering from slight ailments attended by any disturbance of the temperature or of the cardio-vascular system. It cannot be doubted that many of the cardiac lesions of unknown origin revealed in adult life have arisen in this way.

TREATMENT. The treatment of acute dilatation in rheumatism does not differ from that of endocarditis. Absolute rest is indicated; and the patient must be kept in the recumbent posture for some time after the dilatation has subsided. Opinions vary widely as to the length of time it is necessary to keep the patient in bed. In the above cases the children were not kept in the recumbent posture longer than a week after all signs of dilatation had subsided; but it is impossible to form any definite opinion as to the proper line of treatment in cardiac cases without having statistics and reliable records of the condition of the heart throughout life, the manner in which the patient resists future diseases, especially diseases which affect the cardio-vascular system, etc.

As regards the use of drugs, in the above cases sod. salicyl was employed, together with pot. iod. and sp. ammon. aromat. The dilatation in these cases subsided as satisfactorily and as rapidly as in any of the cases recorded by Lees or Poynton. Sod. Salicyl. in these cases did not appear to exert any depressing effect upon the heart; but this good effect may have been ~~gained~~ by the combination of pot. iod. and ammonia in the mixture.

The point of practical importance in the treatment is that when a child has a temperature without any discoverable cause he should be put to bed lest he be developing acute dilatation of the heart.

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