Title | Heart disease in childhood

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W. M. Reed
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Heart Disease in Childhood.

{ A Clinical Study. }

Being a Thesis submitted for M.D. graduation by

William Cash Reed

(M.B. & Ch.B. 1877)

2 Atheneum Street, Plymouth
Devonshire. April, 1880.
Preliminary Remarks.

The following paper on "Heart Disease in Childhood," purports to be a Clinical Study of cases which I had the opportunity to observe during the year 1879. They came under observation either at the Out or In-Patient Department of the North-Eastern Hospital for Children, London, during my term of Office as House Surgeon.

The Notes were taken by myself comprise a record more or less complete of about 50 cases. Either of disease of the heart itself, or some morbid condition bearing upon the study of heart affections in the young.

The tracings which are selected from a series of about 150 taken by myself are executed with a Lond's American Phymocardiograph.

The drawings were kindly done by a relation as I found it impossible to get time to do them.

The paper is a record of cases, with the deductions which appear warranted after a careful study of them, and such exhibits at once the clinical feature & the deduction, the latter being solely the outcome of the former, whether or no such conclusion have been before established.

William Casel Reed.
Cases of Congenital Origin.

Case I. Marked cyanosis & clubbing without cardiac or pulmonary lesion to account for them:

For seven months a little boy aged 1½ yrs. attended as an Out-patient. He suffered from a cough on & off during this time & was markedly cyanotic. His feet & hands were clubbed (see trace p.4). No murmur could be detected except on one occasion when there was a low pitched midsystolic.

The supposition here was that possibly an open foramen ovale might exist; though it was borne in mind that the cyanosis present might harmonize with any case of venous obstruction. The child died about the middle of October.

The following is a brief account of the post mortem which I regret to say had to be done with utmost haste.

The heart was normal, except that in the right auricle there was a large mass of adherent papula, which was probably formed towards the latter end of life. There was atrophy of the muscular tissue of the auricle so that the periendo-cardium were in contact.

It has been observed, I think by Dr. Tilbury Fox, that theoretically, a pre-capillary murmur loudest at the base of the heart ought to be present in cases of open foramen ovale. I have seen one case which would certainly support this view.
Both lungs were fully expanded. It appeared that the clubbing had somewhat diminished since death, the result perhaps of that general shrinking which occurs then. I have had no opportunity to verify this observation, though it would be interesting to know whether the lessening of the clubbing were, or were not, in excess of the general shrinking of tissues which follows dissolution.

Right Hand.  

Left Foot.
On May 14th 1878 a male child aged 2½ yrs. who had been attending for a couple of months on account of a rather persistent cough was found to have a "mitral systolic murmur". About a month afterwards he was treated for an attack of diarrhoea. The general health not improving a more careful inquiry was made into the history of the case, when it was elicited that he had been "dwindling away" though had his life purple. The child was noted as "very irritable" so that it was almost impossible to make a satisfactory examination. He was not now cyanotic, the hands and feet were pallid. The systolic murmur at the apex was distinctly audible to the ears of several auscultators, amongst them one whose name is well known and justly honoured in the profession. The child from this time forward (a period of 17 months) continued gradually to waste and died on December 7th 79.

**Post mortem:** On opening the left pleura some two or three ounces of fluid were found contained in that cavity. The pleura was partly thickened at its parietal surface united by strong bands of adhesions to the chest wall's upper surface of the diaphragm. The lung itself was shrivelled exceedingly from edema, (chronic interstitial pneumonia) studded with points of bronchopneumonia.
The right lung appeared healthy. Heart & great vessels perfectly normal. The glands in the median sternum were many of them greatly hypertrophied, some up to 1/4 to 1/4 inches in length, 1/2 in. in thickness. On cutting into them, they gave the sensation almost of making a section of a serous humour, but the naked eye the appearances resembled a hard adenomatous growth. Spleen very large & hard, having on its surface a white superficial patch about an inch in diameter. Lungs & kidneys appeared healthy.

Remarks:

The interest in this case centres in the fact that the physical signs so far as it was possible to detect them indicated cardiac mischief, presumably of congenital origin. The pallor, which this not usual in such cases, is well known to exist at times, was not regarded as counter-indicating the diagnosis. Short: the murmur excited there is no doubt, the its mode of production is very obscure. Bearing in mind the production of murmurs by pressure upon the lumen of a blood vessel not uncommon in children, it is not unreasonable to surmise that the murmur in the present case had a similar origin, though its maximum intensity was in the region of the apex. I would submit that it was due to one or more of the greatly enlarged glands pressing upon one of the large vessels at the base of the heart.
In a child of such tender years the valves lie so closely together that the localization of murmurs is neither so easy nor so certain as in the adult.

Base III.

Definite development associated with cyanosis.

On October 17th 79, a female child aged 3 months was brought to the out-patient department suffering from broncho-pneumonia. The cough had existed five days. It was noted that the heart sounds were conducted freely over the chest, that there was a systole murmur at the apex of the heart. Moreover, the child was cyanosed. Three days afterwards the child died.

The following is a description of the heart, by Dr. Fraenkel to whom I took the organ having its dissection somewhat completed. The aorta gave off first the arterios inominata, then the right carotid, then the left carotid, or left subclavian artery, then "termination in a small vessel continued into the descending aorta. The descending aorta seems at first to be chiefly derived from the pulmonary artery, but there is a decided constriction at the point of entry of the ductus arteriosus." The foramen ovale is patent.

Remarks: The case would (as Dr. Fraenkel pointed out) appear to prove in which sufficient time had not elapsed after birth before death followed the changes which ought to follow birth to have taken place. If life had been continued one may conceive..."
that the connection between the pulmonary artery & the ductus arteri
ovum might by obj. have become closed, it conincidently will
that process the terminal portion of the ascending aorta would
probably have expanded, the descending portion of the aorta
would have come to be continuous with the ascending part as
ought to be.**

Dr. Paccott was further of opinion that
the change in the ductus arteriosus & patent ovale may
have been caused by atelectasis in a young & delicate child.

The murmur at the apex seemed to have no such origin as tightness
of the left auriculo-ventricular opening, it may therefore have
been associated directly with the pyriform for it is well known
that in the acute inflammatory attacks of children a murmur
heard at the apex of the heart is not infrequent. (See p. 24)

In regard to the pyriform I must agree with those who do not see
sufficient cause in an open patent ovale alone to produce
that appearance. The difference in contractile power of the
two auricles in early life would scarcely be sufficient to
divert the blood from one to the other, nor would the action
of their forces contribute to that result either.

A picture of the heart is given overleaf.

**There is however a form of malformation described in
which the descending aorta is pinched off from the pulmonary artery.
Right ventricle giving off pulm. tr. artery & comm. communicating with the aorta by still patent ductus arteriosus.
case IV. Entire absence of Pulmonary Artery.

The subject of the following notes, a little boy, aged 8 mos. first attended as an out-patient May 12th, 1799. Typical cyanosis was present. Finger ends but slightly clubbed. What clubbing was present resembled rather in character that associated with phthisis.

no systole could be detected.

May 16. The heart sounds were masked by the breath sounds, but occasionally at the end of inspiration, during the interval before expiration a distinct blow. Murmur was to be heard. If the child happened to hold its breath for a moment whilst it struggled to rid itself of the annoyance of the asthmatic fit, the murmur was again most distinctly audible. "Loudest in the pulmonary region.

May 26. Child not so well. No murmur to be heard.


Aug 7. More cyanosis, breathless. No fever. Since the weather has been warm another attack. That it has been worse.

Sept. Some ulceration of fauces present, the ulcer contracting sharply with the cyanotic fauces-breacous membrane.

Died.

J. Pracoker who exhibited the heart before the Pathological Society, gave the following account of it:

"The heart is of normal size. The right ventricle is large, the walls thick. The left ventricle is natural in size. There is a large aperture at the top of the interventricular septum so that the aorta arises about equally from the two ventricles. The aorta is wide at its ascending portion. There is no apparent trace of the pulmonary artery. The aorta is unfortunately cut off short, but what appears to be the ductus arteriosus arises from the aorta somewhat anterior to its proper site, it divides into two large branches, which doubtless formed the channels by which the blood was conveyed to the lungs.

"The foramen ovale is entirely closed."

Remarks - Before availing myself of Dr. Pracoker's profound knowlege on this subject, I endeavoured to trace the intima from the common vessel into its branches, with the result
offending what appeared to be the innominate, left subclavian, and carotid arteries, I was thus sorely puzzled to know how the blood had been conveyed to the lungs. That the ductus arteriosus must have taken it thither I felt sure, though that perhaps its gradual closure & consequent cutting off the pulmonary supply had been the proximate cause of death. It appeared contrary to all precedent to assume that there had not existed any communication with the lungs except through the capillary system, for in such case the child must surely have perished at birth, as soon as ever the maternal circulation was cut off. And this child not only lived in fair health for 13 months, but was a well nourished baby. Had it been born a monstrosity one could have reconciled such a condition with a total abolition of the pulmonary circulation.

There was one peculiarity in this case which seemed at first sight rather anomalous, viz. that the child should be worse in warm weather than in cold. It would seem however that in warm weather the vital functions going on with more activity would evince their hunger for oxygenated blood to a greater degree, & this could not be supplied, the organs in no more obviously suffered, whereas in cold weather the child's functions approximated rather to a condition of hibernation. I am indebted to Dr. C. Turner under whose care the child was for this idea.
Case of absence of Pulmonary Artery.
The ventricles laid open showing the communication of the common vessel with them both: a deficiency of the interventricular septum.
A little girl aged 4½ yrs. first attended as an Out-patient May 29th with the following history. Has been ailing since birth, always the cough also especially at night.

"Marked rickets is present so that in walking which she can only do to a very slight extent, the feet are almost at a right angle with the legs. The fingers can be overextended to a most unnatural degree. Fingers thus much clubbed. Abnormal membrane of the tongue cyanosed. Temperature 97. Physical signs. The precordial dulness is increased. The chest is somewhat hyperresonant to light percussion over the anterior margins of the lungs.

On auscultation there is a loud superficial first sound murmur heard over the area indicated, but having a maximum intensity over the lower area. In the region of the apex breathing just below the manubrium sterni. The two sounds of the heart are heard with most distinctness, there also the murmur is heard. It is also audible over the back of the chest.

It is noticeable in this case that the ribs form a medium of conduction of the murmur which is heard to replace the first sound almost entirely when auscultation is made over them, but placing the stethoscope below the sternum, the normal sounds of the heart become distinct.
**Case VI**

**Congenital Pulmonary Aneursy.**

Richard S. aged 5 yrs. admitted October 22nd 1879.

An aneurysm which has existed since birth is very marked at the present time, especially about the cheeks and also nose. The mucous membrane of the lips is very blue. Tongue is too much clubbed. There is rapid breathing, constant thirst, continuous writhing of the lips with the buccal muscles.

**Physical Signs.**

The apical beat is at the normal site, but the carotid pulsation is felt an inch below it. The heart's dulness is somewhat increased. There is a localized, very superficial systolic murmur, of a flute-like character, having a maximum intensity at the third left costal cartilage. This peculiar character is conducted only in the direction marked thus in the scheme, though there is a systolic bruit audible over a larger area. This latter systolic bruit is heard at the apex distinctly, where the first sound is loud, booming. The breathing is loud. Throat is the midsternal region, this part of the chest is tympanitic on light percussion. The chest is projected forward in the cardiac region as shown in the outline over-leaf. There are prespi- tations + thorchi in the lungs, especially at the left back.

*This is frequently be noticed in cases of broncho-pneumonia, sometimes in empyema in children.*
December 5th. At 5:30 this morning a sudden attack of an asphyptic character occurred. He screamed out first, then the eyes became fixed staring, the could not speak. The colour of his skin was bluish-black especially about the nose & cheeks. His head fell back the nurse supposed him to be dead, but she tried artificial respiration, she returned to consciousness. He has never had such a bad attack as this, but slighter once previously of a similar character. They have occurred usually when he has been sitting up, sometimes when taking food. These attacks have generally been relieved by laying him down.

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Temperature chart. Richard S.
Congenital Pulmonary Stenosis.

Frederick L., aged 10, was admitted December 20, 1796, when the following notes were taken.

“Born under care many months, suffering from a very loud apical pulmonary murmur. A doubtful aortic insufficiency also exists. There is very little hyper trophy and dilatation.

“Effects = great dyspnea, lividity of surface.”

Notes taken when the boy again came under observation Oct. 23, 1799. He is small and pale, looks at least two years younger than he really is. His complexion is fair, his skin particularly translucent, which with the presence of cyanosis present gives him a most peculiar look which is nearly to be expressed by the word “lividity.” Though I know not what else to call it. From birth he has been observed to be blue after any ordinary exertion, his fingers then are clubbed though only to a very moderate extent.

It is very subject to “chilblains” tone which he now has on one of the toes of left foot has existed for years. History of fits.”

Physical Signs:—The chest is a little rickety. The precordium bulges somewhat. There is some hyper-resonance to light percussion.

Oclusion commonest in the feet, but clubbing in the hands. (Bracon) In this connection it is necessary to bear in mind that the chest wall is frequently deformed when there has been inflammation of the lungs in early life, or atelectasis pulmonum.
cussion over the precordial region. The apex beat is felt diffused about the normal position, there, as also over the whole of the precordia, a very distinct apicalic thrill is felt. The cardiac dulness is somewhat increased, it is perhaps more globular in form than natural (see scheme).

There is a very loud superficial, rolling straining apicalic murmur at the head over the whole of precordia, having a maximum intensity in the pulmonary region. It is conducted as marked in the diagram, over the sternum it has rather a different character from that elsewhere. Here, (i.e., over the sternum) the bruit commences as broad elsewhere but ends in a higher pitched strain, alone not, this latter character resembles that of the localized murmur.

* It is difficult to estimate the size of a heart in cases where deformity of the chest exists, as the apparent dulness may be really increased, so that there is a larger portion of the heart in contact with the pericardium than is normally the case. (Pacock)

* The murmur associated with pulmonary stenosis may be loudest in the aortic area, brevity, in consequence of patency of the interventricular septum, the two currents of blood forced by the ventricles meeting each other in the aorta give rise to a murmur. (Pacock)
heard in the case of Richard S. (Case VI). It is difficult to describe but reminds one of a note produced by as kind of flapnote. The murmur is also heard over the back of the chest and in axilla.

Sphygmo grams from Congenital Pneum. V. VI.VII.

The irregularity at the beginning of this tracing is because the paper did not travel smoothly.

All these tracings agree in showing well filled arteries:
No. 1. Differs nothing from the normal corres two. to the good health enjoyed by the patient.
No. 2. is further, t. agrees with the asy named of the child.
No. 3. Has a peculiar systole at the apex.

The evidence obtained from the cardograms of which I made several is so decidedly negative that it did not seem worth while to insert them. A rickety chest & Empyematous lump made it difficult to obtain any information of a reliable character.
Anemic Murmurs.

When I read in Dr. West's work on "Diseases of Infancy and Childhood", from which I have learnt so much, that anemic murmurs are scarcely to be observed in children until the approach of puberty, it seems unlikely that I should have anything to say about them. Probably however the truth not infrequently found applicable in infancy, that nothing when looked for will be discovered, finds here an example. In the poor children of London I have heard nothing more commonly than murmurs over the base of the heart, great vessels which have precisely those peculiarities which characterize so-called "anemic" or anemic murmurs in the adult.

The maximum intensity is usually stated to be in the aortic area, in children however I have heard it more frequently located in the pulmonary region.

Such murmurs are of varying pitch, character; musical, coarse, purring, soft, blowing, harsh, etc., usually modified partly according to the amount of pressure brought to bear upon the vessel by pressure of the stethoscope, supposing for example auscultation be made in the neck.

A slight amount of pressure will not infrequently produce musical murmurs, whilst a greater amount will produce those of a very stridulous character, high pitch.

Anemic murmurs are not to be heard over an aortic area, except in the line of the great vessels, unless probably there...
be present some special object for conduction of the sound.
Such e.g. as enlarged glands, in which deep auscultation
may reveal the brut sound over the back of the chest.
These eminent sounds are systolic in rhythm, & at first, it might,
appear diastolic as well. On listening more carefully however,
it will be found that what appears to be the diastolic portion, is
in reality a systolic reinforcement of the continuous ominous
sound produced in the large venous channel adjacent.

In regard to the causation of these murmurs, one observer (S. Laws)
is of opinion that they are due to vibrations in a solid substance
viz. the walls of the vessels which do not contract fast enough
on their contents sufficiently. The walls are thus thrown into
vibrations, or a sort of "subtle trans" in the same way he thinks
that the heart walls may become tremulous to an extent capable
of producing a sound.

Whether it be so or not, the following, which attempts to explain
a condition of vaso-motor paroxysm, not of inhibition, but of an
opposite condition, one of contraction, has seemed to me a
possible explanation.

The hypersensitiveness of the nervous system in the child's
nature to think lends colour to the view, from the young par-
chial insinuation have such marvellous powers to produce
nervous phenomena, whether referable to the organic or to
an organic system of nerves.

Depressing influences are in operation either mental or phical.
We know that such produce an inhibitory influence on the vis
nervosa. Suddenly bad intelligence may make our feeling
unable to drag one leg after the other. Spasm of the ultimate arteries, vividness of surface are the expression of nervous shock, have the same effect as cold externally applied as seen in the case of contraction of the arteries of the extremity.

Now it may not be unreasonable to suppose that under any depressing influence, hypnotic or otherwise, there is vasomotor paralysis of this nature.

If then we have to do with an arterial system in a state of underrision rather than of relaxation, which latter appears to be the generally adopted opinion, then murmurs find an analogy in those heard in the arteries sinuose, or in the carotids on making pressure externally. Murmurs of this character may be we all know be appreciated not infrequently in our own carotids by pressure of the pillow.

Treatment: The morbid sounds will usually gradually disappear pari passu with the improvement in the general health. The Digitalis & Arsenic are helpful, somewhat more so I think, than Iow.
With regard to the murmurs associated with acute pyrexial attacks, with certain cases of Shorra (with Typhoid Fever), a few words are necessary. Those associated with the first named condition are very common in children, and may occur so early on in the course of the disease whatever it may be, (frequently broncho-pneumonia), as almost of necessity to exclude the theory that they are of "dynamic" origin, that sufficient time has elapsed for structural change in the valvular apparatus to have taken place. Murmurs associated with Typhoid I have rarely heard in children, though we have had a fair sprinkling of the disease in the Hospital during the year. The following case forms the basis of the remarks made in this connection.

James K., art. yrs. admitted August 18th, 79. There was no history of any importance, the boy had simply been observed to be out of sorts for about a fortnight. There is a murmur having a maximum intensity at the apex, it follows on the commencement of the first sound, becomes appreciable, turns up to the second sound. It may be described.

Those in which rheumatic endocarditis is not present, leading to structural valvular change, nor yet vegetations forming the valves.
Dec. 14. Boy has been improving steadily in every respect, and the murmur is scarcely to be distinguished.

The history, except for symptoms which will be observed the absolutely negative with the exception of the auscultatory phenomena. The murmur had none of the "anemic" character, but essentially those of one dependent upon regurgitation. It disappeared very rapidly. There was not the slightest edema, dyspnea, not one of those accompaniments which in some degree are almost always attached to anemic cardiac disease. The murmur therefore could scarcely have such a grave connotation, for what then was idiopathic of the cardiac musculature might permit a passive regurgitant flow through the mitral valve in all probability at first I thought of this as the only explanation, more especially as the "site" of the bruit, so to speak, in the cardiac region would favor this view. What more likely than that a weakened ventricle, as it contracted, should allow of an overflow into the auricle from want of tone in the muscular tissue surrounding the auriculoventricular opening. And yet if such were the case, one would look for some secondary manifestation of such misdirection of the blood current. There was none. The boy however was kept in bed constantly, which would lessen the likelihood of such occurring.

After reading Dr. Sanoomi's views on the origin of so-called "anemic" murmurs, a speck light was thrown upon this case.

Echocardiography of the Heart, 2nd Ed. p. 121. re.
but yet I cannot accept a "subclinical" of the muscular structure of the heart giving rise to vibrations, as the explanation in this case, but must conclude either that the brand was of some totally unexplained extracardiac origin (which seems scarcely possible) or else that it had a causation similar to that named at first viz, a passive distension of the lobes through the mitral opening owing to dilatation of the cardiac muscle.

James C. Physiopramy, Oct. 14, 79. (Democt elect.)

Tilb. after 15 days rest in bed. Murmur only just distinguishable, every faint.

These tracings are negative as far as the argument is concerned. They agree in the main characteristics, which are modified according to the circumstances under which the tracing was taken.
Exocordial Murmurs.

These murmurs which are exo-cardial, which are independent of pericarditis, though they may be soft and due to having limited pericardial inflammation, appear to have the following characters which differentiate them from murmurs having an endocardial origin.
1. They are very superficial.
2. Circumscribed area of audibility.
3. At some point not far remote the sounds of the heart may be completely inaudible and perfectly normal. The following notes of a case may be taken as typical of what I now refer to.

Ely, Iowa, Dec. 7. 1879.

Physical Signs: There is a distinct, very superficial, systolic murmur having a maximum intensity just internal to a level with the left nipple. The area of murmur is covered by the end of the stethoscope. The sound shades off in all directions from this point.

Dec. 17. Removed on account of Reckoia. Murmur entirely disappear. It is frequently observed in cases of cleft mitral valves. Changes that at some point or other over the cardiac area the heart sounds are to be heard tolerably normally. Thus appears to establish the fact that absence of the mitral and tricuspid valves is not the only elements in the production of the first sound of the heart.
The following case which occurred at the London Hospital in an adult is too interesting in this connexion to omit.

The physical signs present were a presystolic murmur having a maximum intensity in the region of the aortic arch, & another murmur with a max. int. at the apex. There was of course a difficulty in diagnosing Tricuspid Stenosis on account of its Extreme rarity but it was stated as the probable explanation whilst Mitral Stenosis was diagnosed.

Post Mortem. On the exterior of the right auricle there was abundant rough lymph, the roughness whatever of the tricuspid valve curtain.

On the left side there was some roughness on the auricular aspect of the mitral valve though doubtfully according to Dr. Sutro the Pathologist sufficient to pass me for a murmur. On the exterior, here again, of the auricle was lymph though in less abundance than on the right side.

The murmur heard at the aortic arch was therefore truly presystolic in rhythm, though of extracardial origin; that on the left side may have been either extracardial or endocardial in origin, probably the latter.

The case is important as showing the appreciable difference in rhythms of the contraction of auricles ventricles so that a murmur may be diagnosed as corresponding in point of time to the Systole of one or the other.
whilst it appears necessary to acknowledge in connection with the cases that with the present methods of diagnosis it is sometimes impossible to say whether a murmur is of pericardial or endocardial origin.
Pericarditis.

Clara M., aged 6 yrs. was admitted June 27, 79, with only slight symptoms of cough, chilliness. The relatives were not aware that the child was seriously ill. She had had no acute disease save scarlet fever three years ago; had been slightly ill for three months, was supposed to be suffering from "worms." There was no history of phthisis. Physical examination showed an increased area of precordial dulness; distinct precordial friction in the neighborhood of the mammary stieli; as well as an area of deficient breath sound, with some deficiency of pulmonary resonance over the apex of the left lung.
The diagnosis was pericarditis with effusion, bronchopneumonia. The child appeared by no means ill-nourished; the subcutaneous fat was in fair amount. The treatment consisted of injection of an ointment of bismuthum hydrargyri two parts, extract of belladonna one part, into the precordium; a mixture of iodide of potassium, bicarbonate of potass.
The physical signs of pericarditis remained marked for 21 days; the friction was of triple rhythm.
There was no evidence of endocarditis. The sphygmograph showed the pulse to be ofuble elevation, irregular to normal type. The cardiograph registered vibrations throughout the cardiac cycle. On July 18th scarcely any signs appeared.

carditis remained, and in a few days all had passed away. There were also no noticeable symptoms, except a peculiar toxop. On August 27th she was not so well; had a small, quick pulse; short hacking cough; superficial respirations (respirations 56). In two days there were almost complete dulness of the right chest; oesophageal breath sounds, mingled with fine crepitations; imperfect expansion. Much of this dulness soon passed off. The pleuritic effusion was absorbed leaving patches of broncho-pneumonia. On September 15th the breathing was laboured; there were evidences of more fluid in the chest. The abdomen was enlarged. The decubitus suggested peritonitis, but there was no evidence of ascites. The topos continued. She died suddenly on Sept. 17th, death being preceded by convulsions. The fundus of each eye had been previously examined with negative results, except dulness of the iris. The temperature for the first eight days was subnormal. Afterwards there was marked evening rise to nearly 100° F., till July 22nd, when a fall to about normal. From Sept. 17th to 19th, there was nocturnal rise to 100° or 101°. Necropsy:—The lungs were studded with small tubercles. There was a caseous nodule of the size of a small orange at the right apex. Several patches of broncho-pneumonic consolidation were found about a pint and a half of serous fluid was found in the cavity of the right pleura.
The pericardium was greatly thickened; the parietal layer was profusely studded with miliary tubercles; the visceral layer was smooth; the cavity contained about half an ounce of yellow fluid. The heart's surface was covered with a layer of lymph; on section, the muscle was pale and degenerated. There was no endocarditis. The valves were all competent. The liver & spleen contained tubercles. There was no fluid in the peritoneal cavity. The kidneys were healthy. Between the convolutions on the upper surface of the brain, here & there were small patches of pale extravasation. There was a granular (tubercular) deposit on each side of the longitudinal fissure. A nodule of the size of a halfpenny was found on the surface of the right corpus striatum, involving the superficial part of the substance, extending upwards into the lateral ventricle. This nodule was easily enucleated, that, on section, the usual caseous characters of a tubercular growth. The ventricle contained much fluid. Grey miliary tubercles were found extensively about the Sylvian fissure.

Comments on the case:

I. The triple rhythm of the friction. This characteristic of the pericardial rub was not heard at first, but after about a week it became manifest, villi very interesting as showing the participation of auricle in the phenomenon.

II. Though there was no evidence of rheumatism, the rheumatic origin of the disease was suspected. For it to be...
borne in mind that pro- or endocarditis or both, may be the first expression of the rheumatic diathesis, in many cases the more evident sequel, notably the articular. Moreover pericarditis may itself be the only evidence of rheumatism.

III. There was no wasting throughout; towards the close there was no evidence of lesion of nervous centres leading to the suspicion of tubercle, unless indeed the remarkable torpor of the child may be regarded as suspicious. During the violent convulsions terminating in death, i.e., examined the eyes with the ophthalmoscope, the pupils were widely dilated with negative result.

In regard to convulsions in children I may remark that when they are unilateral, they last longer than when bilateral. This hint among many I learnt from an "old nurse." I have verified the truth of it.
Trace up from the case of Clara St.

Sphygmoman June 2, 79.

Small, feeble but irregular pulse, corresponding to the superficial heat of a weak heart.

Apex cardiopacks, July 1st & 9th, respectively.

The latter was taken as the only point where cardiac action was felt.

There is no evidence whatever of cardiac action here. There is simply the record of vibrations throughout. This favours the opinion either that the pericardium contained fluid which differentiated the heart's impulse, or else that the apex is pressed against a rib. Of course the cardiac action must be very feeble to produce a negative result according to either hypothesis, but it was so in this case.

The post-mortem proved the former supposition to be correct.

The above tracing of the right wrist, Sep. 14th, shows how in inflammation of a serous membrane modifies the type of a pulse.
Leftwrist. Sep. 16. 79.

There was at the time, this was taken, a short cough, of frequent paroxysms of hurried breathing. There were not sufficient grounds for the reasons already given to regard this case as initially tubercular, hence in thinking over the diagnostic importance of the peculiar breathing, its cardiac origin was uppermost in my mind, if I remembered the description of how a person under certain circumstances of cardiac lesion (the palsy) appears as if were to "forget to breathe," I have seen such a case in the London Hospital which this expression admirably describes. & our little patient seemed to "forget to breathe."
Myocarditis.

Case of Myocarditis without Either peri-or endocarditis.

Ages 14, articulation was admitted Feb. 17, 1879, with the following history: Child has had a cough for three weeks. Shortness of breath has been observed a week. She has been kept from school that long; of time. There is loss of appetite, no vomiting, the bowels are regular.

Station of Admission:—Heart found to be enlarged but there is no murmur. Aperistalsis greatly displaced downwards. On auscultation a double first sound is heard, a sharp 2nd.

Child kept in bed in mixture of iron & quinine ordered.

Feb. 27th. Has lost power in right arm & leg, indicates that she suffers pain in the right side of the trunk. She cannot speak towards turning the mouth was noticed to be drawn somewhat to the left side.

March 1st. Did not talk at all to her friends when they came to see her. Appetite poor.

Feb. 2nd. Symptoms as before. There is hyperesthesia of the right side of trunk & limbs. The tongue is protruded towards the right side. Appears to have difficulty in swallowing at times. Appetite still indifferent.

Feb. 6th. Sphygmogram shows pulse of low tension, fully diastolic undialed.

Mixture of Diphteric, Aperineum & camphor water ordered.

Feb. 7th. Slept better took more nourishment. Committed this morning. Some blood in the vomited matter.
Mel 9th 10 pm. Vomiting has continued throughout the day, it occurs immediately after food.
Pulse = 120. Very feeble. Resp. = 60 per min.
Do have nutritive Parmata.
Mel 10th Died.

 Necropsy: Extensive endocarditis; heart's Substance appears mollassed on Section. There is Thrombosis of auricle, filling of the arteries at base of brain on the left side.

The diagram over leaf illustrates the condition. The formation of an enormous amount of fibrillated tissue, at the expenses of the muscular structure, which is pushed aside, thus apparently losing transparency vibration.

Note: The temperature during her stay in Hospital at no time rose above 101.3.
Section of heart substance from case of myocarditis.
comments on the case:

(1) In looking up the history of myocarditis, without either of its usual accompaniments peri- or endo-carditis or both, I have been able to find but scanty information, especially concerning the disease as it affects the child. Dr. West in his interesting chapter devoted to diseases of the heart in childhood says nothing about the disease from which fact alone one might postulate its extreme rarity. Some authors speak of suppurative carditis occurring independently of pyemia. I give instances of three cases, two of which were children, (it is not stated in the third case whether it was a child or not). They then go on to describe a sort of "rapid cisthosis" of the heart, but state that it never occurs without endocarditis usually peri-carditis also.

Further, in speaking of chronic myocarditis, they state that it "probably never occurs without inflammation of the external or internal membrane of the heart, except as a consequence of pyemia.

If this last statement be correct, our case must be regarded as unique, for there was no evidence whatever of accompanying inflammation of the external or internal covering. The heart was carefully examined subsequently by Dr. Sharwood Dyer, microscopical Pathologist to the London Hospital, under whose care the child was.

Pathological Anatomy 2nd Ed. p326 et seq.
The ease then would appear to be one of Acute fibroid de-
generation.

11. A reduplicated 1st sound: — Derived in relation to the post-
mortem revelations, this is a very interesting phenomenon
suggests an instance of want of synchronous systole of
the two ventricles, the result of the disease.

It is easy to conceive that the presence of bundles of now-
contradicted fibrous tissue diffused amongst the muscular
fibre of the musculi papillares, might give rise to an
earlier closing of the left auriculo-ventricular opening
than of the right, or vice versa; or else, that the presence
of the same foreign substance in the ventricular walls
might occasion a delay in the contraction of those of our
ventricle. In this view the more the fibrous tissue in
the one preponderated over that in the other, the longer the
delay in the systole of the one containing the greater
amount. But it appears the left walls are more
subject to the disease than the right, hence, the first
part of the double first sound would be due to the contrac-
tion of the right ventricle.

But there is another view which suggests itself to me, viz., that hanging upon the normal production of normal
first sound.

If the first sound be due to the three elements, (1) closure
of the auriculo-ventricular valves, (2) Impulse of heart
up against the thoracic parietes, (3) Muscular
torsor, e.g., vibrations incident to the vital 
-
fracture of muscular tissue, then a want of synchronism between the three acts, might theoretically give rise to a triple first sound, or a want of synchronism between any two, the third, might in like manner give rise to a double oscillated first sound.

Now it would appear that here we have just such a condition as would be likely to produce reduplication from this latter cause.

But there is yet another view. Those mentioned imply a mechanical causation of want of harmony, but it may have a nervous origin (incident possibly to the disease) whereby an inhibitory influence is exercised through certain nerve filaments upon the heart, so part of that organ.

III. Hypoesthesia occurred on the paralyzed side.

IV. From the characters of the ophthalmoscopic tracing taken on the 9th of March, an exceedingly bad prognosis was given, it may add without exaggeration that the indications from this source were almost the only grounds upon which the deduction was made.

V. The thrombosis of auricle is very interesting as causal of the cerebral embolus, this latter of the signs of paralysis.

(Unfortunately at the time we did not carefully make out which arteries were plugged, for, as so often happens in such cases, the investigation being deferred was never made.)

I may mention that this was narrated by Dr. G. Turner at the Nervous Society in London.
Endocardial Affections.
Mitral Stenosis.

Fanny P. admitted April 19th, 1879; act. 9.

History: Unfortunately, I have omitted to make a record of this, but am quite under the impression that there was an absence of satisfactory evidence of rheumatism. If so, the case would be in accordance with the observations of Dr. Hilton Fogg, that mitral stenosis is most frequently met with in persons who have not betrayed obvious signs of rheumatism. (See comments at the end.)

Physical signs: Apex beats between 6th & 7th ribs, two inches below nipple, & 3/4 inch to the left of nipple line. Fremitus is almost imperceptible in the area. There is a loud presystolic murmur audible in the mitral area. After the second sound there is a very brief interval when a sound resembling somewhat the normal first sound commences, but is immediately followed, & supplanted by a very rough murmur which seems right up to, & terminates in a short sharp first sound. Thus: —

Pulmonary second accentuated. The murmur is audible in the left axilla, not in the back.
There is no abnormal pulsation or thrill in either the suprasternal notch, or in the epigastrium.

Further history: Augt. 9th: there is evidence of dilatation, the transverse dulness 4 inches & there is considerable pulsation below the sternum. Child is in poor spirits, complains of precordial pain. The pulse is very feeble. There is generally much cardiac excitement after food & she gets cyanosed. She had an attack of pneumonia early in November & died suddenly on the 9th. The treatment consisted mainly in the administration of digitalis & iron.

Necropsy: In my absence for a holiday this was performed by my friend Dr. T. H. Watson who reported that both lungs were congested & kindly preserved the heart drawings of which are given overleaf. The left auricle was hypertrophied & the mitral curtain & cord tendineae were matted together into a mass which was traversed by a hollow narrow that it only admitted the point of the little finger. The mitral opening viewed from the auricle had the appearance of a buttonhole as shown in drawing No. 2.
Drawing of J. Pi's heart.
showing condition of mitral valve from ventricular aspect.
Left auricle of same, laid open and expanded, showing the "button hole-like" initial opening. To the left are seen the fossa and annulus ovales.
Remarks: (1) On the general history of mitral stenosis. Dr. G. W. Balfour regards such cases as "by no means infrequent congenital." (a) As a comment upon this remark I would call to mind, first, the extreme rarity of congenital affections occurring on the left side of the heart, & secondly that in a clinical review of 136 cases of disease of the heart in children, as the basis of a course of lectures on that subject by Dr. A. E. Laneam (b) there is no evidence of the congenital origin of one of the cases of atresia of the mitral orifice. Such evidence, I take it, would be found in the hypoplasia of the arterial system. No such hypoplasia is recorded in connection with the cases referred to. I think Dr. Hilton Fagge's observation spoken of before suggests a ready explanation of this form of heart disease. Cases of a non-rheumatic origin are yet (as Dr. West points out (c)) dependent upon an altered state of the circulating fluids. By this, I suppose is meant an overloading with excrementitious materials.

2) Medical Times & Gazette for 1879, see several numbers.
of which, indeed, rheumatism is but the expression. In it we have the type of a comparatively acute disease which has a tendency to give rise to retraction of the free edges of the valves of the heart, & hence incompetence of those structures. Whereas in that condition of the system which may occur after scarlet fever, or measles for example, there is likewise a storing up in the blood of excrementitious products, from defective elimination by means of the great emunctories. The mobil condition thus induced having a tendency to run a more chronic course, has also a tendency to induce a more chronic kind of pathological change, which in the case before us finds its expression in that slow, fibrous proliferation which results in a making together of the valve curtains, & the cordae tendineae. On account of the direction & force of the blood current from auricle to ventricle during the gradual production of this change, the mass has a tendency to project frondell-like into the ventricular cavity. (a)

(a) See "Medical Times & Gazette" 1879 p. 713. Dr. Sansom's lectures before referred to.
II The character of the murmur: it is double syllabled, the first portion as noted resembles somewhat in character the commencement of a normal first sound, tailing off into a rough prolonged thrill. In the only other typical case of pre-systolic murmur which has come under treatment during the year at the Ho. East. Hospital there was the same double character of the murmur. The explanation would seem to be as follows.

The moment the ventricular diastole commences the left auricle, constantly in a state of tension from over distention relieves itself of some of its contents by, as it were, a slight elastic recoil. The blood thus passing through a narrowed, sometimes eroded orifice before it has received the impulse of the active auricular contraction gives rise to the first comparatively soft syllable, & the reinforcement of a hypertrophied left auricle following, produces the second & longer syllable. The limit of first sound may be represented phonetically thus: m-rr-m. But seeing that a so-called "reduplicated second sound" is so common an occurrence in cases of mitral stenosis, I have heard it urged that the sound attributed to the
premature closure of the pulmonary semi-

lunar valves was in truth due to a mode

of production very similar to that given

as explaining the first part of a double

syllabled murmur. I will not go into this

point, but merely remark that it

seems scarcely conceivable that the short,

sharp click indicative of the semi-
lunar closure should be simulated by the earliest

portion of an essentially presystolic event,

which would presumably be of a soft

character & relatively more prolonged.

III The cardiac perturbations, & the cyanosis

after food are interesting as showing the ease

with which an enfeebled organ is reflexly

affected. Being then stimulated to overwork

there is an expenditure of energy without

any corresponding good to the economy, the

walls contracting upon only a partially filled

ventricle, & an indifferent supply of blood

being evidenced by the cyanosis supplied to

the organism.

IV The sudden death. The congestion of

the venous system would appear to have

been as extreme as was consistent with

life, when a super-induced pneumonia

increasing the venous hypostasis caused the fatal result.
Tracings from the case of Fanny P.

Inductions.

1. Peculiar as a consequence of imperfect filling of aorta.
   The initial arterial expansion was so weak that it was
   with difficulty that a tracing could at any time be ob-
   tained, other (as above) no differentiation of the various
   events is observable.
   There was a very typical tracing except that it is more regular
   than is frequently the case. *

From region of left auricle, Sep. 5th 79.

Showing the respiratory wave to which are superadded
deviations due to cardiac action, though it is impossible
 unlawful with precision as to the origin of each.
A marked tendency to truncation is observable which
would accord with the laboured auricular contraction.

† Another, Guy’s Hospital Reports 3rd series, vol. 71, p. 335.
At apex when the murmur was most audible to the touch.

Apex cardiophasm. Aug. 9. 79.

Unusual protracted diastolic phase, followed by a sudden, sharp peak of ventricular contraction.

There is evidence: (1) of auricular contraction marked here, then in the tracing, then (2) of ventricular systole, followed by (3) the evidence of the blood wave (if that view which appears the most generally accepted be the correct one.) This elevation is very clearly marked, corresponding to the imperfect filling of the aorta, finds its expression in the typical pulsation of mitral stenosis. The suddenness of ventricular contraction is out of proportion to the work done.

Apex cardiophasm. Sep. 6. 79.

The peaks above the horizontal line (which is a little too high) were recorded when the chest was more or less in a state of collapse. I. The fact is here manifest (or repeatedly evidenced throughout the tracing) that the fuller the chest the lower does the ventricles in contracting manifest itself either to the finger or to the tampon of the instrument, probably because the anterior superior of the lungs being distended overlap the heart. The converse proposition also holds good.

II. In the interval between inspiration i.e. when the chest wall is at rest in the collapsed state, the second peak in the cardiac cycle (marked thus) is most clearly demonstrated.
Mitrail Regurgitation and doubly, Obstruction also.

Rosie M. act. 9, a delicate-looking blonde, was admitted May 19th, 1879. The following is a short account of the physical signs. Apex beats between 6th and 7th ribs 2 inches below 3/4 inch to the inner side of the nipple, there is a slight systiotic thrill & doubtfully also a presystolic. The horizontal absolute dullest = 2 1/2 inches in the inter-mamillary line. There is pulsation over the aorticulus cordis & in the epigastricum also. A soft blowing murmur is to be heard all over the heart's area, especially marked at the apex; heard all over the chest. The murmur sounds as if composed of two syllables.

The intercostal depressions coincident with the heart's beat were very marked in this case & they afforded some interesting information. From their extent it might have been inferred that auricular contraction contributed in part to the result, & if, as it would have afforded important presumptive evidence that mitral stenosis existed, it that we had to do with the contraction of a hypertrophied auricle. This doubt was however set at rest by means of chronometry of the pulsation, in a way subsequently
The depressions by the beating outwards were found by this means to be perfectly synchronous, & it was thus established that they were due simply to ventricular contraction causing a beating outwards in the region of the apex beat & a drawing inwards beyond that area, the latter probably being due to atmospheric pressure tending to fill up the vacuum produced behind the costal parietes by the change of shape & position of the left ventricle. Moreover the murmurs were now found to be synchronous with these depressions.

Afed barthogram on admission 21st. June 30. 79.

The long diastolic interval is suggestive of a condition of the left auriculo-ventricular opening which is capable of giving rise to a pre-systolic bruit totally independent of that which is associated with stenosis of the mitral orifice. The drawing of E. D.'s heart (p. 69) shows well the condition to which I refer, & which so far as I can find out seems to be unnoticed in the text-books. If the mitral curtains are puckered at their free edges & yet permanently widely separated from one another, there
seems to be a condition capable of giving rise to an auricular systolic murmur without stenosis. In this case such a murmur being apprehended on the study of the cardiogram, it was carefully listened for, as is stated probably heard. Such a long diastole indicates a laboured auricular contraction in a marked manner, we know that free regurgitation was taking place through the mitral valve. The 2nd tracing does not show this but it was taken when the child's condition had immensely improved.
Abstral Regurgitation: Murmur of unusual character in unusual position.

Emily Dact. II was admitted Sept. 16th, 1879.

History: had scarlet fever four years ago, never had rheumatism. Two years ago she was under the care of Dr. Bathurst Woodman as an outpatient at this hospital, & was said by him to be suffering from disease of the heart. Was in Victoria Park Hospital for diseases of the chest 18 months ago.

Physical signs: (see diagram). There is a very distinct systolic thrill & considerable pulsation over the 2nd left interspace. There is a loud, rough, harsh, systolic bruit, having a maximum intensity to the left of the manubrium sterni; it is exceedingly loud here, also very distinct in the line of the sternum & aortic area. Hard all over the chest.

The point to determine in this case was whether we might not have to do, with the unusual lesion of tricuspid regurgitation, but though the site of the bruit pointed to that, yet its character, & the absence of jugular pulsation, seemed to negate the assumption. The view taken by one of the Physicians (Dr. Sansom)
who saw the case was that, that segment of
the mitral valve which lies nearest to the
septum ventriculorum was the subject of
dense vegetation, or great roughening, & thus
the sounds were transmitted in the direction
indicated. Whether this be correct or no
it is a pretty explanation & corresponds with
one by the same author equally ingenious to
be found in histoticles on diseases of the
heart,” p. 169.

Sphygmogram. Aug 18/79.

Sitho. Dec. 19/79. (Pulse sustained)

Apex cardiogram. Aug 18/79.


Left Beat of Heart. Aug 18/79


At about one cardiac area, where the 2nd sound (probably pulmonary
very distinctly to be felt. (So to speak.)
On comparing the two sphygmograms it is observable that during a month's rest and treatment, the pulse had gained in volume, and this furnishes a suggestion of importance towards the interpretation of the second tracing taken over the seat of the lesion. Assuming that the vibrations communicated to the fingers are registered by the instrument, it is observable that the first of the two tracings taken over the lesion at the time of admission, there is no evidence of the heart's impaction; whilst in the next tracing there is very clear, rhythmic evidence of such impaction, the heart having in the meantime increased in force. This fact is brought out in the most evident manner possible in the two apex cardiograms.

The tracing taken where the second event was appreciable to the sense of touch has exquisite symmetry though the various events registered are difficult of interpretation in the present state of our knowledge. The only apparent want of perfect harmony is at one point where the paper travelled rather more slowly than during the rest of the time. But the most striking feature in this tracing is the effect of respiration upon the impulse. The peaked
elevation corresponds to the collapse of the thoracic parietes in the interval between expiration & inspiration.
Myocardial Incompetence, very great hypertrophy of left ventricle.

Jane S., aged 61/2, admitted April 1st, 1879.

History: Rheumatic.

Physical signs: There is some bulging in the cardiac region, flattening of the intercostal spaces. The apex is most distinctly felt to beat under the sixth rib, two inches below and one inch to the outer side of the nipple. The impulse is heaving and diffused; no marked thrill is to be felt. The movements of the heart can be felt as far out as midaxillary line, to which point the dulness extends. There is a loud systolic murmur to be heard at the apex; the pulmonary second is accentuated.

Upper limit of dulness = border of 3rd rib.
Right "" "" = 1/2 right of sternum.
Left "" "" = midaxillary line.

Defective breathing at left apex. Both bases resonant, air entering fairly well; some crepitations however. Edge of the liver somewhat firm, felt 1/2 inches below costal border. No oedema, lips rather blue.

Readmitted Oct 18th. The physical signs are about the same as formerly noted. The
Maximum is extremely strobulus & is loudest with deep inspiration. The apex beat is distinctly felt 4 inches below the nipple. There is no epigastric pulsation. Heart's movements are felt 1/2 an inch beyond the median line of axilla.

October 30th: Frequently complains of pain over precordia. To have emb. belladonnae.

(Originally taken on micro & transferred to paper.)

These tracings were taken at the several points marked 1, 2, 3, 4 on the accompanying diagram (p. 61) i.e. at points over an extremely hypertrophied & to some extent dilated ventricle, successively further removed from the centre of contraction, if one may so say. Dr. Sanderson

(2) "Handbook of the Physiological Laboratory" p. 234.
has pointed out a fact, the bearing of which on the present case may be best appreciated by quoting the words of Dr. galabin, who thus refers to Dr. Sanderson's observations. "If needles be introduced from without so as to penetrate the living heart of an animal at different points, the one introduced at the centre of impulse has no transverse motion, whilst the external end of the others move outwards during systole in proportion to their distance from it, this denoting a drawing inwards of their points. Hence the spot at which the so-called apex beat is felt is the centre towards which the muscular mass of the ventricles draws itself together, & the same "hardening of the ventricles, produces a heating outward over a limited area, & at the same moment a retraction beyond that area. Hence it follows that tracing taken at different points over the heating area vary according to their distance from the centre of impulse, or, as it would appear better to designate it, centre of contraction; for the motion of effect is beside the mark in the present enquiry.

(a) "Guy's Hospital Reports for 1875, 3rd Series, Vol. XX, p. 268."
No. I Differs but little from a normal tracing.

II Evidences a more laboured systole.

III Fails to properly differentiate the events occurring in the cardiac cycle.

IV Taken at the furthest limit to the left where cardiac action felt, looks chaotic, & yet appears to have registered the impulse pretty faithfully.

The sphygmogram shows the systemic circulation to be in a very satisfactory condition, as indeed it was.
Mitrval Regurgitation & dilated Left Ventricle.

Emma D., aged 41 admitted Nov. 7th, 1879, with a loud rough systolic murmur of low pitch heard as indicated, & evidence of considerable dilation of the left ventricle.

(Was in the hospital 12 months.)

Treated with digitalis & iron & kept constantly at rest.

Temperature sub-normal throughout.

Died very suddenly indeed Nov. 28th preceded for a day or two by frequent vomiting.

Necropsy. Lags oedematous. Some fluid in pleura & pericardium. Pneumonic consolidation of right lung (lower lobe). The heart (see drawing p. 69.) weighs 10 1/2 oz. every vestige of clot having been removed. The right side is dilated. The left auricle is greatly dilated, it is expanded into a thin, elastic, membranous reservoir. Left ventricle hypertrophied & dilated, the latter preponderating. The transverse diameter is greater proportionately than the longitudinal. The left auriculo-ventricular orifice admits three fingers easily, & is greatly thickened & puckered all
round the free edges thus rendering the opening permanently patent.

No. 1. Left wrist. Nov 8, 79.

No. 2. After cardiogram. Time date.

No. 1. Evidences great irregularity, when compared with No. 2 shows that many of the ventricular contractions were powerless to produce any corresponding systolic diastolic distension of the radial artery. It is the pulse of very imperfectly filled arteries, & shows a very great loss to the systemic circulation by free regurgitation through the mitral valve. The initial expansion in the artery when the ventricle does succeed in contracting upon a good column of blood in the aorta, is fairly satisfactory but simultaneously, the blood is diverted from its preferred course through a permanently widely open mitral orifice, hence the "systolic distension" of Flenderson or blood wave is poorly represented. Moreover, the pulse is fully diastolic.

No. 2. There is evidence of hypertrophy (truncation) and an exceedingly short, practically absent in fact, diastolic interval, suggestive
of an almost constant passive flow from auricle to ventricle.

Remarks. I. The entire absence of embolus (or the arteries of the brain were most carefully examined, especially about the medulla) associated with the conditions known to be present would suggest a typical example of death from asystole. A gradual failure of arterial pressure, had been slowly but steadily occurring, coupled with a proportionate venous congestion, general throughout the system, until at last the left ventricle fails in its function & life is extinct. The cardiogram conveys I think a most vivid picture of the heart, enfeebled by disease, & the state of the circulation within it. The tracings viewed together are fairly typical of one of the most appalling forms of heart disease.

II. Weight of heart. I am able to state of the weights of the human heart at different ages (1) the following facts, amongst others are stated, viz. that from the ages of 10 to 14 the

(1) Dr. T. B. Peacock. "On some of the causes & effects of valvular disease of the heart." Being the Londonian Lecture, p. 57, 88.
mean weight of the female heart is 150g. This little girl was 11 years old. The greatest weight attained by the human heart in health is between the ages of 60-70 in the male when the mean weight is according to the same table 10g. 13.33dr. Thus it seems that the heart of this child increased in weight to such an extent by hypertrophy as nearly to equal in weight the healthy adult heart when developed to its greatest extent.
Drawing of B. D.'s heart. (see description)
A portion of the left auricle is drawn over by the side of the aorta, in order to show its characters.
Frances R., aged 11 yrs. 4 mos. admitted July 8th, 1879.

History: — Quite well till two years ago, when for 6 months she was troubled with pain referred to the precordia, she then complained of aching of the feet, which subsequently swelled, & have continued to do so on & off for 12 months. She has never had rheumatism or scarlet fever.

Physical signs: — Apex beats 1½ inches below, ¾ inch to inner side of the left nipple. There is pulsation over the acrobrachial cord but no thrill to finger.

Transverse deep delness = 2¾ inches.

In mitral area there is a soft low-pitched systolic murmur running right up to the 2nd sound.

In aortic. The systolic murmur is also heard, but much less distinctly, but here there is a long diastolic whiff occurring directly after the 2nd sound.

Both murmurs heard in left axilla July 23rd. Has complained of left hemiplegia for about a fortnight & yesterday said she had pain in the left hypochondrium. Temperature 103.4 yesterday, & the day previously today it has been high.
August 3rd. A distinct systolic bruit is now heard in aortic area.

September 6th. Frequently, (sometimes two or three times a day) she has attacks, intermediate in character between angina pectoris, & cardiac asthma. Pain she refers to the precordia extending over left hypochondrium to the left back, & sometimes over the left shoulder to the scapular region, but not down the arm; sometimes the pain last almost all day. She desires to breathe, but the deepest respirations afford her no relief ("air hunger" of the Germans.)

The attacks are relieved by Nitrite of Amyl inhalations.

Temperature, normal throughout, except as noted above, under date July 23rd.

Treatment consisted mainly of the exhibition of iron.

The child was sent to the Convalescent Home at Lorrigan on the 21st September.
Remarks: I. The rise of temperature noted on July 23rd, accompanied by the pain in left hypochondrium might be taken as strong presumptive evidence that some embolic infarction had occurred in the spleen. I think that in the case of the child more caution must be exercised than in the case of the adult, in ascribing in such circumstances embolus as causal of the symptoms, for it is to be borne in mind the extremely slight causes which give rise to hyperpyrexia in children whose nervous systems are so exquisitely sensitive. Apropos of this, I may mention how I soon learned as house surgeon at the Children's Hospital, that an elevation of temperature occurring on a Sunday evening was a less cause for apprehension than on other evenings, because on Sundays the children's friends visited them, and the additional excitement, if possibly an occasional clandestine present in the way of unsuitable food, were the causes of the hyperpyrexia.

II. The development of a systolic bruit in the aortic area on August 3rd, is to be noted in connection with the way in which endocarditis is frequently known to successively involve, first the mitral curtains, then
extending upwards, ultimately to reach and include the aortic valves. Dr. Samson (a) in speaking of this says "The disease spreads from the valve (the mitral) to the endocardium lining auricle & ventricle, its effects may often be traced by the appearance of a milky patch of thickened endocardium stretching in a direct path across the ventricle from the mitral to the aortic valves."

As much as there was a regurgitant murmur before the destructive became audible, it is obvious that the aortic valves were involved early on, & the systolic murmur must be taken as evidence of some superinduced morbid change, or else & what is more likely, that as the heart gained in strength under rest & treatment, it acquired sufficient power to develop a lesion for which the necessary conditions were present but the force was wanting.

Professor Sanders has drawn special attention to this subject in his "Variation & vanishing of Cardiac Organic valvular murmurs," Edinburgh Medical Journal January 1869. Dr. P. Balfour has also an interesting chapter on this subject.

(a) "Diseases of the Heart" 2nd edn. p. 148.
II. Anginiform attacks. I do not find that Dr. West makes any mention of angina pectoris occurring in children, but from the clinical history of the attacks in this case (the description was spontaneous, not merely assent to enquiries) they appear to have been, if not actual angina, yet anginiform, if one may so say in character. Though angina is doubtless frequently associated with some lesion of the heart proper, notably fatty degeneration, & calcification of the coronary arteries (2), yet some authors would limit the affection to cases in which no ascertainable structural lesion of the heart exists. It would appear (3) that Dr. Heberden was the first to describe the affection in his Commentaries on the history & cure of diseases" (1806, & he points out that of 100 cases he had seen 50 were women, 1 a boy, & the rest men, near, or past 50 years of age. These statistics bear out, I believe, the views held now.

Dr. R. H. Temple in his interesting work on the heart (3)

(3) "Manual of Diseases of the Heart" and "Angina Pectoris" by R. Hunter Temple M.D. 1875.
(2) "Lectures on practice of physic. Dr. T. Grainger Stewart. Session 1875/6."
a book I think, but little read, though full of matter the result of great experience, insists upon the dissociation of true angina from cases in which lesion of the heart exists. He says, “True angina thus characterized, is not a common disease & although, as I have stated, it is essentially a nervous affection, yet it may accompany organic lesions of the heart. But I entirely agree with Dr. Flint that such connection is rare. That Physician found that out of more than 150 cases of organic disease of the heart, observed by him before the year 1859, angina pectoris existed only in 7, 8 in 188 cases of a collection analysed by him in preparing the 2nd edition of his work in 1867, the disease in question existed only in 8. The proportion being 15 in 338. Again, the same authority states that during ten years he noted only 14 cases of true angina pectoris, that is to say, where the disease was unconnected with cardiac or aortic lesions.

In regard to the pathology of the disease, most writers seem to regard it as probably a neuralgia of the cardiac plexus, though other explanations have not been wanting such as II. Result of spasmodic contraction of the heart.

III. Overdistension of the organ giving rise to (2) Lectures by Dr. Grainger Stentor before referred to.
a pain of the character of that which occurs in flatulent colic.

II. Vascular spasm in the substance of the heart. Dr. R. H. Semple speaks of it as "neurosis" of the pneumogastric, in which in all probability the phrenic is also involved.

Dr. J. W. Balfour would condemn an important element in the pathology, whilst Dr. Sleson appears to hold the same view, but regards "degeneration" as playing a part.

In regard to children, it would appear that angina is not an affection quite unknown in them, yet any rate one which is extremely rare. Dr. West as Shaw said makes mention of the disease in his chapter devoted to the affections of the heart, although his teaching therein detailed, is founded on the clinical study of 122 cases. Again, Dr. Sleson in his lectures before referred to, embracing as they do the study of 136 cases makes no mention whatever of the affection, as an affection sui generis.

And finally I may mention that out of the 40 to 50 cases of heart disease in children, which it has been my privilege to observe during 1879, one case of true angina has come under notice. These latter facts lend colour to the view that degeneration
plays an important part in the pathology of the affection.

III. In regard to the cardiac dyspnea in this case a word will not be out of place. It would appear to be a reflex act due to the pulmonary congestion, probably reflex through the pneumogastric nerves. It seems moreover to bear a somewhat similar relation to the lungs, to that borne by angina to the heart. It is known to occur in cases of acute pericarditis from the suddenness of the onset of the symptoms, by which the mechanics of the circulatory system are interfered with and to be not an uncommon concomitant of aortic disease. In the other cases however of this latter disease observed during 1879 there was no such cardiac asthma present.
Spasms of heart attack of cardiac breathing. Aug 11/79.

† Left wrist. July 9/79.


2. S. No. At right angles to interface.


* Shows a properly regular, full, normal type of pulse; its wideness more of that small, true character, described by some as associated with a contracted state of the arterioles occurring in angina pectoris. If the arterial system were very thin, one might reasonably expect some lowering of the temperature during the attacks. I could find none however, on applying the thermometer to the subcutaneous vessels in this.

† Fibro elevation, but pulse well sustained. An unusually long diastolic interval, compare with cardiograms 1 & 2, in which a similar long diastole is apparent.
Cardiogram No. 1. Shows a powerful ventricular systole followed by the evidence (marked o) of the "recoil towards the heart apex of the blood which has been forcibly impelled against the auriculo-ventricular values at the moment of their closure. (a) The next slight elevation evidence, according to the same writer "contraction of the heart as a whole."

Finally the slight elevation at the commencement of diastole may be due to closure of the aortic values, or else to the "reflex of blood which closes those valves." Dr. Galabin would differentiate between the two, by using the stethoscope & noting what was occurring in the trace at the moment of the closure of the aortic values.

The flatness of the diastolic portion is puzzling, but must I think be accepted as positive, though perhaps unexplainable evidence, because it occurs in two tracings taken with the tampon placed in a different manner as stated. This was of course done to eliminate the possibility of too great pressure being exercised, & the second tracing proves the pressure to have been so slight that the greatest altitude attained in systole is not recorded & the same diastolic flatness apparent.

It is rather singular that in cardiogram No. 3 there is about as much evidence of auricular tension as there is absence of it in Nos. 1, 2. There is a circumstance however which may throw some light upon the subject viz., that when Nos. 1 & 2 were taken the young girl was at rest in bed; No. 3 after she had been dismissed from the hospital some 10 or 11 weeks during which time she probably moved about as usual. Anything that tends to increase the strain upon the heart must also tend to exaggerate the tension within the auricles. It may be noted also that No. 3 shows the heart beating more than half as quickly again than it was when Nos. 1 & 2 were taken. But accepting the usual explanation that diastolic flatness indicates low auricular tension & surveying the tracings once more, one can scarcely fail to note that when the auricular tension is low, the elevation (marked 0) is apparent, & as the converse proposition when auricular tension is high as evidenced by a short oblique diastolic line, this elevation(0) is absent. Can it be that one may conclude from this that when the patient was in a state of rest the auriculo-ventricular valves did close enough to impress their closure on the tracing.
but that when the heart was beating more rapidly, faster regurgitation resulted, and we have no evidence of closure of these valves. Could this be established, the evidence would be of no slight value diagnostically.

Arterial tracing from Frances R.

Right wrist. Dec 4, 1797

Femoral artery, in the groin, same date.

Dorsalis pedis artery, same date.

From the corresponding arteries of a perfectly healthy boy of the same age.

Left wrist. Dec 4, 1797.

Femoral artery in groin, same date.

Dorsalis pedis artery, same date.

The "waler hammer" character cannot be said to be marked at all in the case of Frances R. though there was free regurgitation.
Concerning the arterial tracings.

The object for which these were made was to determine if possible whether or not in the child the observations of Dr. Galabin (a) concerning the causation of the "water hammer" pulse would find support. To determine this point, it will be necessary to glance at the paper referred to, to give a brief review of it. Dr. Galabin agreed with Sir Dominic Corrigan that the "aortic," i.e., the "water hammer" pulse is due to the blood being propelled into comparatively empty arteries; but thinks this explanation incomplete.

The writer contends that the element of suddenness finds here no explanation, because, as he points out, hypertrophied muscle contracts in a slower and more deliberate manner than healthy. It is not necessary to glance at many of the tracings given in this paper only to prove this, notably those e.g., of Jane S. (see p. 61). They evidence by the marked truncation a hypertrophied, hence slowly and deliberately contracting left ventricle. We must next concede in agreement with Dr. Galabin that the suddenness is not explained by the

(See Medico-chirurgical Society's transactions Vol. 59. 1876.)
fact that dilatation is present without hypertrophy, in cases of aortic insufficiency. So pass on, let me give in the words of the writer a proposition he lays down. The water hammering quality of the aortic pulse does not exist in the pulse wave as it is thrown into the aorta, but is a character which is gradually developed as the wave recedes from the heart, & reaches its maximum at a point which, in some cases, is as distant as the dorsalis pedis artery.

The carotid pulsation in these cases, which is often so well marked, is, he says, not due to the suddenness of the wave but to its amplitude which is consequent upon the hypertrophy & dilatation of the left ventricle. The proposition next laid down is as follows: 'The transformation which the aortic portion of the pulse of aortic regurgitation undergoes by which the quality of suddenness is developed, is only an exaggeration to an extreme degree of a change which takes place to some extent in the normal pulse.' I will not attempt to summarize the explanation given of this change, which certainly is very abstruse, if not particularly lucid. I would merely point out a probable verification in the case of
Annice C. (p. 88) of at any rate the main point contended viz. the gradual development of suddenness in the arterial system, which quality may attain its maximum at the waist, or before that point, or may not do so before the dorsalis pedis artery is reached.
Aortic Regurgitation probably Mitral also.

Annie C., aged 9, admitted Sept. 15th, 1879.

History: has been ailing 6 months, first noticed swelling of the feet, accompanied by "pains all over." Mother states that the clubbing of the hands & feet which at present exists, has only occurred lately, not since birth.

Physical signs: Apex beats ½ inch below & the same distance to inner side of nipple. There is visible pulsation over the scrobiculus cordis, carotids, & in episternal notch. A thrill is felt in the two latter situations. In the mitral area is heard a faint systolic murmur. In the pulmonary there is both a systolic & diastolic murmur, but the latter is heard most distinctly in the aortic area where the systolic is not heard. The basic murmur is conducted upwards along the sternum & the apical one a little above & outside the left nipple. Neither murmur is heard over the child's back.

"Stills" show what appears to be a synchronous systole of apex & aorta.

Ring finger left hand. Middle finger left hand.
Remarks. I. The clubbing if it be, as stated, of only recent occurrence, is interesting as showing that a peripheral evidence of venous obstruction may date from the onset of the conditions giving rise to it. I remember hearing of a case in which marked cyanosis & dyspnea dated from the receipt of a sudden fall down some steps in a young girl. In such case one would assume, that a sudden jerk had caused a rupture of a previously falselty closed septum of the heart. I do not know whether in this case clubbing also followed as time elapsed, but it is most probable that it would occur.

II. Chronometry of pulsations.

Dr. Balfour describes a method of determining the rhythm of pulsations over the cardiac area, when there is an universal undulatory motion observed over the precordia, & when, in each case the pulse is over 90 per minute. In such cases he uses a pellet of beeswax to which is attached a briquette surmounted by a small flag. Thus any variation in time of one or more sections of the pulsating area may be an important guide to diagnosis.

I have however followed a modified plan suggested by Dr. Saneom who uses it especially.
to demonstrate the want of synchronism between the pulsations of left auricle & ventricle in cases of an auricular systolic murmur and the former chamber is hypertrophied. The plan he adopts is as follows. Cut a small circle of diachylon plaster, & transfer the centre with a pin, place the plaster over the area to be compared, & elongate the pin, by placing over it a small rolled strip of paper. Such I did in the present instance & found the pulsation in the episternal matrix synchronous with that of the ventricle. Of course I did not expect it to be otherwise, for one may exclude aneurism altogether from the diseases of children, but the evidence was interesting as establishing the fact that though the systolic expansion of the ventricle & that of the aorta are not exactly synchronous, yet so far as the eye can determine they are so.
Apex cardiogram Sept. 15th 1879.

The peculiarity about this tracing is that the deeper the inspiration, the more marked the heart's impulse, which is the converse of what usually occurs.

Taken in Episternal notch. (aorta) Sept. 15th 1879.

Left wrist. Sept. 1879.

Dorsalis pedis. Sept. 1879.

Dito. Dito.

Sphygmographic tracings show a pulse getting progressively "sordid" in character, the maximum being reached as far as the dorsalis pedis. The tracing taken from the aorta itself in no way differs from the normal.
It was urged by the Physician under whose care the child was (himself, rather sceptical as to the value of evidence obtained by the sphygmocardiograph) that the flat interval recorded in the last two tracings might have a different explanation. That it might be due to the slipping off of the tampon from the artery, or else that the artery moved for beneath the surface that its recoil was scarcely registerable by the instrument. I must say that neither of these objections appear to me to have any weight whatever; for in the first place, if the tampon slipped from the artery home is it that the upstroke is registered distinctly throughout? Moreover I may say that the manipulation of the instrument can scarcely be so faulty as to allow of this without one’s being aware of it. In regard to the 2nd objection I can only say, the artery is not at all far from the surface but is close below it, & may be distinctly seen to pulsate with the naked eye. And even if it were far from the surface the one condition viz. interposition of structures would affect all parts of the tracing alike, & would not make one faulty & another correct. Nay further, the aorta e.g. is far from the surface
in reference to the supratemporal notch, & yet the tracing taken in that region shows no modification at all referable to this circumstance. There is but one objection that I can see, to be admitted as having any weight in calling in question the reliability of the conclusions drawn from these tracings. The quasi-objection then is, that when, 18 days after taking the first series of tracings, I took others of the dorsalis pedis arteries, I did not find the same character of the diastolic period, the down stroke of which was more very much more gradual. But there is another, I venture to think, a much more reasonable way of accounting for this, than admitting the inaccuracy of the 1st tracings; viz. that during the interval of 18 days, the child had lain in bed, & had gained so much flesh, & was altogether so improved in regard to his general health as to be, I wont say scarcely recognizable as the same child, but so as to give one the most vivid appreciation of the great value of rest in such cases.

Inasmuch therefore as the heart itself had become compensated, & there was now an accurate balance of the circulating apparatus, I think we may fairly conclude that the
arterial supply farthest from the heart had proportionally increased, giving evidence of a fuller and better sustained blood-wave. I decidedly think moreover that now the radial pulse communicates to the finger a more distinctly "water hammer" character, than it did before, & the dorsalis pedis less so. (a)

(a) The notes independent of course of the present arguments lean this way.
E. P. act. 10, a thin delicate looking boy, was admitted Dec. 16th, 1879.

History:—Had rheumatism last winter just after Christmas, but recovered & was apparently quite well till three weeks ago, when he had a return of the complaint which necessitated his being kept in bed. He has always been perfectly healthy with the exception of these two attacks of rheumatism.

State on Admission:—Physical signs:—There is pulsation at the root of the neck & a thrill is to be felt over the carotids. Dullness is about normal, but the apex beat is displaced downwards 1½ inches below the nipple and in a line with it.

Soft systolic & diastolic murmurs heard all over cardiac area, having a maximum intensity about mid-sternum.
Apex Cardiogram, Dec. 1879.

Strong systolic, irregular action. Evidence of hypertrophy.

Right Carotid.

Left Radial.

Right Femoral artery in pain.

Dorsalis pedis artery.

The "water-hammer" pulse is not developed anywhere in the arterial system, showing that the influence on nutrition exercised or marked by in arterial peristalsis may be apparent (for this has as noted was very thin vascularity) without the evidence of a viable arterial supply being manifested. The evidence of irregular action is distinct in the sphygmograms. It is probable that the vibrations registered in the dorsalis pedis tracing were not endothelial, but were communicated by the instrument, possibly from the hand.
basis of probable Aortic Stenosis of Rheumatic origin.

Adm. March 21st, 1879. (age 2½)

History: Rheumatic fever 14 months ago.

Physical signs: Apex beat 12 inch below 
8 to inner side of nipple. There is a very slight 
thrill in this region. A systolic murmur 
is to be heard all over the cardiac area, but 
especially distinctly over the mitral & aortic areas. 
In the former area it is somewhat booming, whilst 
in the latter it is rather rasping as well as louder. 
2nd sound not perceptibly altered in either area.
The bruit is distinctly heard in both axilla, 
posteriorly in the interscapular region & over the cervical spine.

There is pulsation in the region of both carotids 
& the bruit is distinctly audible over them. 
A venous hum is also heard in the same region.
There is a perceptible thrill over carotids 
also in suprasternal notch. No evidence 
of dilatation. Heart action is somewhat intermittent.

May 2nd: No thrill except at apex; thrill systolic. 
Murmur very distinct at suprasternal notch. The 2nd sound to be heard all over cardiac area except in this region.

May 7th: Child's general health & appetite are excellent. Later on it is noted that the child appears in rude health.

* = Apex beat, ... line of conduction of bruit. * Distant pulsation.
This was a very curious as well as interesting case, and gave rise to a good deal of speculation at the time as to what really was the nature of the lesion. I have only given an outline of the notes of the case, but will refer to one or two points not given there in endeavouring to argue out which valve (or valves) was diseased. I am inclined to think that all things considered the lesion must be said to have had a point of maximum intensity at the episternal notch. Three explanations present themselves for consideration:

1. Aortic stenosis, either congenital or rheumatic.
2. Pulmonary stenosis.
3. Mitral insufficiency.

I will examine all three. (1) Congenital aortic stenosis is not very uncommon, nor is it necessarily accompanied either by any hypoplasia of the general system, nor other sign of its existence. Conrieart, as mentioned by Dr. Peacock, records the case of a female, aged 76, always ailing, & with symptoms of disease of her heart after her 67th year, whose aortic orifice was reduced by disease of the valves to a mere chink, which could only be opened to the extent of 1 or 2 lines, the valves too mere thickened & rigid.

But still more remarkable cases are recorded...
by other writers. One of these was that of a woman aged 76 who died after an operation for strangulated hernia at the Royal Free Hospital. Dr. Peacock says she had enjoyed good health throughout her life, until she was seized with the strangulation, could go without difficulty to the top of the house where she lived. The aortic valves were found the subjects of extensive disease, two of the cushions blended into one, & the aperture reduced to a mere slit 10 lines in length. Dr. Stokes amongst many most interesting & no less truly remarkable cases mentions one in which a gentleman past middle age had enjoyed "uninterruptedly good health" & yet "the aortic opening exhibited the most extreme degree of obstruction from osific deposit that he had ever seen. At first it appeared as if there was no opening, but when examined on the ventricular side, a very small slit was discernible, about 4 lines in length & 1 in breadth, through which it was just possible to pass a fine probe". We learn from such cases that obstruction may be well carried on in the subjects of aortic stenosis, & that dyspnoea on exertion does not necessarily occur.
The latter was present in one little patient, but as the notes state, at times he had the appearance of being in rude health, & was exceedingly well nourished throughout.

(2.) Pulmonary stenosis may exist even to an extreme degree without cyanosis, but the murmur was quite different in character, & its path of conduction was quite different from that which usually obtains in this affection of congenital origin. And its occurrence with cyanosis may be practically excluded, so exceedingly rare is it.

(3.) Mitral Insufficiency; probably present for the reasons mentioned immediately.

The child was frequently under observation after his stay in the country in Sept. The child remained just about the same, but he was continually having exacerbations of rheumatism, giving rise to stiffness, swelling of the joints & frequently to stiff neck. He always appeared well nourished, particularly sturdy. It would appear then correct to surmise the rheumatic origin of the lesion, which would seem most likely to be stenosis of the aortic orifice. It is

probable that thickening of the mitral cunctains
was also present partly because rheumatic
endocarditis usually as we have seen, primarily
involves this valve partly because the auscu-
tatory signs would support the view.

over pulmonary area July 9 79

mitral area (breath being held) same date

ditto (during respiration)

Apex cardiopano Sep 25 79

taken after a stay at the convalescent home.

region of aorta.
These tracings were taken with a view to determine whether it were possible to gain evidence by such means as to whether in the present case independent aortic disease existed. It will be observed from the notes that the ordinary means of diagnosis left this a question vexata. That no indisputable evidence was obtained is perfectly clear; yet there are one or two considerations suggested by the tracings which may be held tentatively. And it is by arguing upon such evidence en masse that the sphygmocardiograph is likely ultimately to come into regular routine use in clinical research.

1. A tracing taken in the aortic area, (of which there is only a rough sketch accompanying, as I have not the original) shows vibrations throughout the respiratory cycle.

2. The pulmonary tracing evidences them also, but to a less extent, though on another occasion the reverse obtained.

3. There was no evidence of these vibrations being produced in the instrument itself, or by the operator.
Mitrval Obstruction & Regurgitation & Aortic Sten.


History: The rheumatic history is somewhat obscure, but the disease appears to have been present in a subacute form about four weeks ago. She has reacted rapidly. She is now very thin, has a hollow, unhealthy appearance.

Physical signs: Transverse cardiac dullness = 2½ inches. Pulse over sinotubular line.

Apex beats 2 inches below & ½ inch to outer side of nipple. At the point marked X there is a soft systolic murmur, somewhat stridulous in character. On auscultating at a point corresponding to the normal apex beat, the murmur is as loud as at the other point, but here it is more decidedly stridulous. Pulmonary 2nd accentuated & somewhat changing.

The systolic murmur is so very extensively conducted over the thorax, both back & front, that it would be probably impossible to point out any spot where it may not be distinctly heard.

Oct. 14th: Over aortic area systolic & diastolic murmurs are to be heard, and in the region of the apex there is a short diastolic murmur.
Oct. 16th. For two days the child has complained of pain in the region of the left kidney. Today the pain is acute, & on examining the urine a trace of albumen is found to be present.


Oct. 23rd. Breathing abnormally harsh on left side of chest posteriorly, but there are no accompaniments.

Nov. 2nd. Torpid lately, asleep much.

It is here noted that the basic diastolic murmur has a maximum intensity decided over the pulmonary area. There is one point just below & internal to the nipple where both sounds are heard pretty natural, but another point there is a suspicion of a pre-systolic roughening.

Dec. 1st. At the apex the systolic murmur is heard to run right up to the 2nd sound, leaving no short interval appreciable. The basic diastolic murmur has so similar a pitch to the apical systolic, as to have led the former to be overlooked when the patient was admitted.

At apex there is still the suspicion of a slight diastolic murmur.

Dec. 29th. Complained of pain last night in right side of the face & neck. Today
Hemiplegia of the left side of face, mouth is drawn to the right, articulation is indistinct. No power in the left hand, but she can move the left arm & leg when told to do so. Sleeps a great deal. For several days four distinct bruits have been audible, obstructive & regurgitant, corresponding to the two valves, the mitral & aortic. The pulsation of the left carotid artery is very distinct, & its external division, together with the posterior branch of the temporal artery are seen as a continuous pulsating cord.

Albumen 1/3 in urine.

Dec 31st. Child rather better. The hemiplegia is less marked today.

Temperature has been throughout, with scarcely any variation, precisely of the type shown by the accompanying chart. Treatment has consisted of Dig.: Digitalis & Ipec.

The further history was that of occasional rallying, with nothing special to note. She died on the 8th February 1830. The following is an account of the autopsy sent by my friend & successor J. W. Hobson. Mr. B.
Heart. Hypertrophied. Aortic valves thickened & somewhat stiffened; mitral thickened but admitted three finger tips. Very friable vegetations in many places; a piece on one aortic segment, two large ones on ventricular wall below valve, very numerous on auricular aspect of mitral, on auricular wall also, in appendix. An uneven surface was left under the vegetations.

Brain. A hemorrhagic focus of softening in white substance of right frontal lobe, corresponding to about the junction of the first frontal with the ascending frontal convolution. The spot was not so large as a walnut.

Lungs & pleuræ. Fluid in both pleuræ, more in right. Lungs congested, lower lobes hardly crepitant, edematous.

Kidneys. Not obviously inflamed. No inlets.

Spleen. Large, several small inlets, none recent.
Remarks.

I. The apical diastolic murmur.

I have failed to find much reference to this phenomenon in books, & still less to the mode of production of such a sound. I refer, in cases of mitral regurgitation to a soft, short murmur, occurring immediately after the 2nd sound, usually indistinguishable at the back but having a maximum intensity at the apex.

Thus:

The murmur is represented by the red.

It appears to be dependent upon a passive outflow from left auricle to left ventricle due to the overdistension of the former cavity on account of regurgitation through the mitral valve, so that the auricle is put on the stretch & thus relieves itself the moment the ventricular walls begin to relax themselves. And the blood thus flowing over roughened surface gives rise to this short, low-pitched sound, which is then followed by the auricular systole which may or may not, according to circumstances, generate the true presystolic murmur.

II. Renal Infarction.

The notes of Octr. 16th make it probable that
such had occurred, & the appearance of albumen in the urine, gives still further colour to that view. The frequency however of albumenuria in cardiac disease must be borne in mind dependent as it is upon intravenous pressure, but inasmuch as in this case there was no deficiency in the amount of urine excreted, the failure of the urine to excrete of producing this result.*

III. The point of maximum intensity of the aortic murmur.

In speaking of anemic murmurs (as called) in children, I have mentioned that according to my experience, they more frequently have a maximum intensity in the pulmonary than in the aortic area, but that they are generated in the aorta, is I believe generally accepted. A diastolic aortic murmur follows the ordinary laws of conduction, viz., in the direction of the blood current; & this doubtless explains how it is that such a murmur is frequently heard to the left of its point of generation with far greater distinctness than actually at that point. A change in the relative position of parts induced by the primary lesion may still further tend to deflect the sound. And though in this the heart makes it doubtful however whether the supposition of injection were correct.
point of view it may be contended that no such change has been observed to occur in the adult heart, yet it must be remembered that in the case of a child we have to do with a growing viscus, which tends to adapt itself to any superinduced condition of disease, or, in other words, that there is an inherent adaptiveness in a developing organ.

IV. The character of the sound at the apex. It is noted it runs right up to the second sound leaving no short interval appreciable. This fact taken in connection with another, viz., that the basic murmur was of almost exactly the same pitch as the other one, the former was at first overlooked, so that the two running into one another failed to be differentiated. The following represents the condition diagrammatically.
Apeo cardogram, Oct 21/79.

Dorapoint where murmur was most stridulous in character.

1. Leftwoed, Oct 21/79

Ditto.

But less pressure applied than in no. 1. lid 210 with the least possible pressure.
Both show posterior expansion, feeble pulse wave vs marked diastolic. The "water hammer" was very evident to the finger, it is well brought out in the trace on.
Laura W. aged 8. First attended April 17th, 1879.
The mother complained that the child would not go to bed. This symptom abated, but on June 5th it was reported to have returned.
She was admitted into the hospital a week afterwards when the following notes, given here in brief, were taken:
She was laid up with rheumatic fever about Christmas.

Physical signs:—The apex beat diffused 1½ inches below, & slightly to the inner side of the left nipple. The parasternal transverse horizontal dulness measures 5½ inches, the absolute 2¾ inches.
June 15th. Dulness has decidedly diminished, showing the beneficial influence of rest. There is now at the apex a loud blowing systolic murmur heard also extensively over the back, & along the great vessels. Pulmonary 2nd accentuated & metallic.

Discharged relieved July 12th.
On July 17th the mother brought the child with the idea that she would be sent to the Eynsford Convalescent Home. It was found, however, that there was swelling of the limbs, free perspiration, a coated tongue, & vomiting...
a temperature of 104°. The heart's dulness much extended & a prominence observed over the precordia. Readmitted.

July 18th. Pulse 140. On the 21st the heart's dulness extended up to the first interface. A loud friction could be felt all over the heart's area.

July 27th. Friction as before. Has lately complained of pain in the right hypochondrium. Otherwise she has varied but little, except that now & then she has attacks of considerable dyspnea. The signs of chronic pneumonia now became apparent.

August 2nd. Pneumonia continues. Edema of feet considerable.

August 3rd. The endocardial bruit audible all over the chest. Much epigastric pulsation. Edema of feet & lower limbs continues.

August 17th. Pulsation of the liver marked. Lower border extends to within an inch of umbilicus. Over the epigastrium where the pulsation is exceedingly marked the 2nd sound is of an extremely metallic character, it is scarcely at all as over the rest of the cardiac area. Much blood mixed with sputum, lately.

Died August 19th.
Necropsy. The heart greatly enlarged. It consists of the left ventricle. The surface is covered by a thick layer of lymph. No adhesions & no fluid in pericardium. Slight vegetations on the aortic valves & those of a lead-like character on the borders of the mitral, with thickening & contractions of the edges of the latter. Walls of left ventricle thick, pale & mottled. Systole much dilated & the columns carneae stretched & standing out prominently from the wall. Right ventricle dilated. Both auricles dilated.

Lungs. Base consolidated; section granular.
Liver. Somewhat nutmeg, & fatty.
Kidneys. Firm, & congested.

Remarks.

1. The pulsation in the liver due to triphed incompetence & consequent "setting back" of the venous wave along the inferior vena cava & hepatic vein, is interesting from its comparative rarity in childhood.

2. A peculiar feature of auscultation in the epigastrium was as noted Sept. 17, the limited area over which the metallic character of the 2nd sound sustained. It suggests that a cartilaginous structure is a present one to conduct.

* The pulmonary artery is apparently dilated, & the aorta contracted.
the rapid vibrations of a high note for the metallic 2nd expresses great tension in the pulmonary circuit.

III. The apparent dilatation of the pulmonary artery, the apparent contraction of the aorta is of interest considered in relation to the great & progressive venous congestion & the proportional arterial failure.

IV. The temperature chart shows the type of pyrexia associated with passive & secondary pulmonary congestion i.e., such as independent upon cardiac failure for its initiation. The temperature attains none of that great height found in the acute primary pneumonia to which children are so subject. In the case before us on only one occasion did it rise (as that only to the extent of 103°).
Cardiogram taken at heart's apex, July 1st, 79.

There is nothing strikingly abnormal about this, but it is suggestive of a rather laboured systole (hypertrophy) and a short diastole.

1. July 1st, 79

2. July 27, 79

3. July 30, 79

4. Aug. 9, 79

The Pulse tracings 1. 2. 3. 4.

Show very significantly the progressive failure of the systemic arterial circulation.

1. A fairly well sustained pulse throughout.

2. Less sustained with a tendency to flattening at the end of the diastole.

3. A still more horizontal diastolic interval.

4. About the same as 3, but there is probably a slightly greater pressure of the instrument on the artery in taking No. 3.

Sphygmographic tracing from left wrist during a paroxysm of cardiac breathlessness, to which the child was very subject.
L. E's heart, showing greatly hypertrophied muscles papillares of left ventricle (the mitral valve has been cutaway) to the right the lymph covered external surface of same ventricle.
Cardiograms from healthy hearts.

The above were taken from the region of the heart's apex in children who were healthy, and maybe taken as normal tracings allowing for the atypicalities of London children in a crowded locality. The last tracing evidences slight irregularity in rhythm; they vary considerably.
bass of this b. showing how rhonchi snapping positive
Evidence to a tracing.

1. Apex cardiospasm July 24/79

2. Ditto Aug 17/79

3. Spiggamspasm July 24/79

4. Ditto Aug 17/79

Three tracings were taken from their b. at 3 yrs. who
was suffering from Bronchitis, with at first remarkably
loud thud of rhonchi. There was also a slight
anemic beat at the base of the heart.

No. 1. Shows the rhonchi registered throughout.
No. 2. Their total disappearance. The note on
this date say, "No rhonchi to be heard now, child's
cough is quite well."

No. 3. Shows pulse somewhat deficient in tone,
which has vastly improved in No. 4.
Nos. 1 & 2. Show the variation which the pulse may undergo in different conditions in the same individuals.

No. 1 was taken during an acute pyrexial attack occurring at the same time as the pyrexia of scarlet fever in a child in the same ward.

The pulse is over 130 in the minute, extremely strong.

No. 2 was taken when the child was convalescent, and is of full volume.
Organic Disease of the 1st Degree.

Helen B. apex cardigrams July 24/79.

Ditts. Physical signs. Same date.
This is a good example of what may be called, "Organic cardiac disease in the child of the 1st degree." There is mitral regurgitation with very no secondary manifestations. Traceup evidence a laboured cardiac cycle, short-fillyed arteries, owing to the backward diversion of the blood current through the more or less patent mitral orifice.

Helen B. apex cardigrams. Oct 21/79.

Ditts. Left ventricle.
This was a case of mitral regurgitation well compensated. The initial arterial expansion is rather feeble, the pulse wave not altogether satisfactorily sustained.
The case was one of mitral regurgitation. No 2. Shows rather too rapid descent; satisfactory compensation scarcely taken place. Build ultimately became so well, it was able to take so much exercise that it seemed almost impossible to believe so case a serious one.

Corresponding tracings, dec. 19/79. Showing differentiation of apex tracing as improvement took place, coincidently a better sustained pulsæ.

1. Apex card. -

2. Ditto.

3. Right cor. -

4. Ditto.

Explanation: - No. 1 (a) Elevation at commencement of diastole in a "a" point (b) a 2nd elevation (c) Ventricular systole, inefficacious, with a sudden collapse. Then follows (d) a feeble tidal wave, and finally (e) the diastolic wave.

No. 2. Gave precisely the same indications only here, the influence of respiration at first sight appears to produce a chaotic tracing. Precisely the same elements contribute to its production as those which produce No. 1. Only that here, in No. 2, the respiratory curve is evident. Nos. 3 & 4 are from the right cor. The former at a greater pressure than the latter. They show pulse of fair elevation, but not sustained; there is a very feeble tidal wave, indeed, other than a small diastolic wave. Pulse is regular but is that of deficient mitral function. Evidence of fair forces of left ventricular contraction. Probably there is pretty fair compensation.

* * *

The echo may possibly be due to the blood passing over a roughened auricular surface of the mitral valve. The physical signs would support this supposition.
Ada S. Initial regurgitation & doubtfully aortic ditto.

Sphygmomen: July 30/79.

Sxex cardiospasm (wilio respiration) same date.

Taken over region of pulmonary artexy where the closure of semi-

lunar valves was very distinctly felt.

The second noise which appears sometimes at the base of

the long down stroke, corresponding with the point of time

to the closure of the semi-lunar valves, may be due to that event.

If there, it is the only record that I have succeeded in

getting in traenpi from children, though Dr. Galabin gives

many examples of it from adult hearts.

The 2nd noise would appear due to the systolic expansion of

the pulmonary artery.

This case immensely improved, twas reported by herself on

our occasion “Lucky well.”
Summary of Deductions.

I. The signs (cyanosis, edema) usually considered as being strong presumptive evidence of the existence of congenital cardiac malformation, may exist independently of such causation; a systolic murmur may be superadded to these signs, or yet no defect of the heart be present.

II. Cyanosis may coexist with an open foramen ovale, but probably has a closer causal relationship with a certain form of deferred development (case 3).

III. There may be an entire absence of the pulmonary artery, in which case the blood is conveyed to the lungs through a persistently patent ductus arteriosus. A blowing murmur may be present in such case, perhaps dependent upon an incomplete interventricular septum which may coexist.

IV. Congenital pulmonary atresia is commonly associated with Ricketts' syndrome. (a) The murmur is very superficial, is accompanied by thrill, has a maximum intensity in the pulmonary region, but beyond that it has an extensive area of audibility over which its character is partly modified.
(c) Asphyxic attacks threatening desolation may rear.
(d) Illerations dependent upon malnutrition are common.

V By anemia dyspnea may occur.
By anemia or mental defect, dyspnea may occur, the former disappearing as the latter improves with development.

(The note warranted 310 5. I have not thought it worth while to encumber the text with.)

VI Anemic murmurs are common in children even of tender age.

VII Given a case presenting the physical signs of broncho-pneumonia, or also of pericarditis, the possible tubercular origin of the latter lesion as well as of the former, should not be overlooked.

VIII Myocarditis may exist without either of its usual accompaniments peri or endocarditis.

Further the subnormal temperature and the "compensatory pulse" described by writers as found associated with congenital defect were present in the cases here referred to, nor was there any peculiarity in the position assumed by the patient which is sometimes observed.
IX. Attacks of an angina-like character may occur associated with organic valvular changes.

X. The prognosis in cases of Rheumatic fever in the child cannot be too guarded for an apparently slight attack may give rise to disease of the heart progressing rapidly from bad to worse, in spite of all means used to arrest it.

XI. Rheumatism is the commonest proximate cause of heart disease in childhood, though other conditions of pyrexia associated with the retention of excretaitious products in the blood may give rise to organic changes.

XII. Whilst it is necessary to plan a guarded progress in cases of heart affection yet bearing in mind that the young heart has a special power of self-adjustment and adaptability, such progress may, on the whole, be more cheetful than in the case of the adult.

XIII. The occurrence of dilatation is to be looked forward to with apprehension not only as an Event, reality perhaps especially prone to occur in children in whom valvular disease exists, but also as determining the issue of such disease in an especial manner.
XIV. The sphygmograph is of the same clinical use in the child as it is in the adult, with the former as well as the latter frequently giving peculiar indications of interest & importance, especially in relation to prostration.

XV. The cardiosphygmograph whose use hitherto would appear to have been rather confirmatory than diagnostic, seems likely to be especially useful when the evidences obtained by it are viewed in the light of those taken by the sphygmograph in the same individual. In case the evidences obtained by the one confirm those of the other, frequently a joint consideration of the two furnishes a reliable deduction.

XVI. Murmurs have been associated with, probably dependent upon, (a) the presence of a distended abdomen, (b) collections of pus in the pleura, & such murmurs have disappeared when the pressure was removed.