Thomas Lewis—the early years

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Apart from one year at Clifton College, Bristol, Thomas Lewis's schoolboy education was done entirely at home by his mother and by a tutor so it cannot have been the impress of a formal education that led to him being singled out from the rest of the first year pre-clinical class in Cardiff by the lecturer in physiology, T Swale Vincent. Lewis (Fig. 1) was to record this event later in life when he wrote,¹ "It was while working in his class of Physiological Chemistry that Vincent asked me if I would care to undertake a research with him on the proteins of unstriated muscle. The request came as a complete surprise to me. I readily agreed, and afterwards worked at his suggestion and under his supervision at the haemolymph glands. I have always been grateful to Vincent for giving me my first introduction to scientific work".

His first two scientific papers were both written in collaboration with Vincent and published in *The Journal of Physiology*, 1901; the first² was on the proteins of unstriated muscle and the second³ on the chemistry and heat rigor curves of muscle. He took a BSc pass degree in 1901 and proceeded to a double honours degree in 1902. In that year he was the sole author of two papers on the haemolymph glands, and published a further two papers on that subject in 1904. It was a group of seven papers, two of them running to over 11 pages in *The Journal of Anatomy and Physiology*, and all written by the age of 26 that won him the degree of DSc Wales in 1905.

He entered University College Hospital Medical School, London, as a clinical student in 1902, possibly choosing this school because Swale Vincent had been an associate professor at University College London. Very likely it was Vincent who introduced him as a member of the Physiological Society in 1904, which must have been a step of considerable importance for him because it was there that he started meeting the leading physiologists of the day,

such as Henry Dale, T R Elliot, E H Starling, and Leonard Hill. It was Hill who gave him the stimulus to work on the analysis of the pulse and during his six months as house physician to Sir Thomas Barlow, he made a detailed analysis of the clinical features of 20 cases of aortic regurgitation including on each a pulse tracing done with the Dudgeon sphygmograph and a measurement of the systolic blood pressure. These findings were written up in the *Lancet* of 15 September 1906, with the title "The pulse in aortic disease; the relation of pulse curves to blood pressure", and it was Lewis's first cardiological writing.⁴ He followed it with eight other articles on either the pulse or the blood pressure, both clinical and experimental observations. Using the Dudgeon really as a plethysmograph rather than a pulse recorder, and with a Riva Rocci cuff, he showed that it could signal the level of systolic blood pressure at a lower pressure than did palpation of the radial pulse. But he deliberately avoided any plethysmographic effect of the sphygmograph when he studied "The influence of the venae comites on the pulse tracing, with special reference to Valsalva's experiment, and diastolicity; a note on anacrotism." He did this by an ingenious modification whereby a weight was suspended underneath the instrument allowing the strap to be undone (Fig. 2). An unusual feature of this article for its day is the presence in it of two photographs. They show the arm veins with a venous tourniquet and during the Valsalva manoeuvre. He concluded that the rise of the sphygmographic curve in the Valsalva manoeuvre was caused by an increased blood pressure and not by venous congestion.

Analysis of the various waves in arterial tracings clearly fascinated him and his first paper on the subject in the *Journal of Anatomy and Physiology* in 1906⁵ discusses whether the first wave in the arteriogram is an artefact or a true pulse wave. His ability to devise conclusive experimental methods is

Fig. 1 *This is Lady Lewis's favourite photograph of her husband. It was taken at the time of their marriage in 1916.*

well shown in this paper where, by placing artificial waves into the tracing (by depressing the pad of the instrument), he shows that the primary wave cannot in fact be an artefact (Fig. 3). He goes on to state that the primary and the secondary waves are produced centrally, but that they may be enhanced peripherally. A paper on "The factors influencing the prominence of the dicrotic wave"⁶ contained good bedside and laboratory investigations. He showed that a dicrotic pulse could be produced when patients had venesection performed, while a reservoir system with an artificial "pulse" created by a silk tambour with a sphygmograph mounted above it led him to conclude that low blood pressure with short sharp heart systole and rapid output of blood from the periphery were the

chief factors involved in dicrotism.

Appointments in 1907 to the Dreadnought Seamen's Hospital, Greenwich, and to the City of London Chest Hospital still left him enough time to work in E H Starling's laboratory at University College where he undertook technically demanding haemodynamic work on cats. In one series a brass tube was placed in the pericardial sac and the chest wall then closed around it. Thus, by opening or closing the tube the intrapericardial pressure could be varied. Using this model,⁷ Lewis was able to show that the rise in blood pressure, which occurred with the prolonged inspiration of vagotomised animals, "velocity curve", was a result of increased diastolic filling of the heart and not, as previously supposed, of

Fig. 2 Dudgeon sphygmograph modified so that a weight (not shown) would compress the artery thus avoiding the plethysmographic effect of the strap, which is undone. (*J Physiol* 1906; 34: 393.)

a change in the lung resistance. In another study⁸ he recorded the intrapericardial, arterial and intra-tracheal pressures simultaneously and showed that a rise in intrapericardial pressure of 1 mmHg lowers the systemic blood pressure by 8 to 9 mmHg and vice versa. The conclusions were drawn from experiments on no less than 44 animals, and is an early example of Lewis's capacity for sustained hard experimental work. This paper also contained human work and one conclusion was that the pulsus paradoxus is a misnomer because the blood pressure almost always fell when a patient took a deep breath. The same volume of the *Journal of Physiology* contains Lewis's first paper on cardiac arrhythmias, written with A Salusbury Macnalty. It is entitled "A note on the simultaneous occurrence of sinus and ventricular rhythm in man" and is illustrated chiefly with

polygraph⁹ curves, but there is one electrocardiogram recorded on 12 November 1908, the first he ever published. It has a simultaneous arterial trace and was taken by Professor A D Waller (Fig. 4).

There are two unusual features to this paper. Firstly it is difficult to read and one gets the impression that Lewis is fumbling with a subject that he does not fully understand. Secondly it is the one and only time that he expresses thanks to a technical assistant in this case "Gratitude to Mr Symes for the trouble he has taken in rendering me conversant with the nature of the apparatus". A unique feature is the first appearance of his famous "ladder" diagram for arrhythmia analysis (Fig. 5): unknown to him, it had, however, been described independently previously.¹⁰ Presaging the pattern of his future research work, he made at this time an abrupt transition from study of the pulse and blood pressure, about which he only once ever wrote again, to a study of arrhythmia.

His later clinical work on the jugular venous pressure, however, was heralded on 30 October 1908 when he gave a lecture on the normal venous pulse at The London Hospital Medical School.¹¹ Presumably the contact here was Leonard Hill and not James Mackenzie because he had only just been introduced to the latter. Another lecture later in the year at The London was on the occurrence of heart block in man and its causation,¹² when he analysed 27 cases from the published reports with necropsy control, noting that six had gummatous lesions of the atrioventricular bundle and concluded that atrioventricular bundle lesions were the usual cause of complete heart block.

Throughout his career Lewis did remarkably little investigative work in cardiac auscultation and his 1909 paper, "notes upon the cardio-respiratory murmur . . ."¹³ was one of the few. He points out that it is in fact an interrupted breath sound and noted in a few cases that it occurred in presystole because of the action of the atria. A paper in 1909 with the promising title "Irregular action of the heart in mitral stenosis . . ."¹⁴ turns out to be a disappointment. He had a patient with intermittent slow heart action in which atrial activity is shown in the phlebogram but in whom the presystolic murmur has disappeared,

Fig. 3 Sphygmograph pulse tracing, with artefacts (1) induced by finger pressure on the pad to show that they differ from the natural pulse wave (2). (*J Anat* 1906; 41: 138.)

Fig. 4 The first electrocardiogram published by Lewis. Taken by Professor A D Waller. Note tracing is inverted, recorded from right to left, and that the P waves are labelled a. Time marker in 1/5 s above, and radial pulse below. (J Physiol 1908; 37: 457.)

and he reaches the extraordinary conclusion that this must be because the right atrium is beating while the left one is paralysed.

Nevertheless it is quite obvious that at this stage of his career Lewis in getting into his stride regarding the analysis of cardiac arrhythmias. A paper on "Single and successive extrasystoles" shows electrocardiograph tracings which he identifies as ventricular extrasystoles, one figure showing three in a row. As

seen previously in Fig. 4, the tracings, again made in Professor Waller's laboratory, are recorded from right to left and labelled with the polygraph notation a, c, and v! A polygraph recording in the same patient which must represent five successive ventricular extrasystoles is unhappily identified by him as "nodal rhythm".

It was not long, however, before he unravelled the nature of the irregular irregularity of the heart which

Fig. 5 His first use of the "ladder diagram" to illustrate the electrophysiology of arrhythmias. In this instance, it is an analysis of a polygraph tracing. The original legend read as follows. "A diagram illustrating certain changes of the ratio of auricular to ventricular rhythm, which may be supposed to occur as a result of altered auricular rate. A_s = auricular and V_s = ventricular contraction. R = the refractory period of bundle function as defined in the text. The oblique lines joining A_s and V_s represent the transmission of auricular impulses to ventricle. The lines x, y represent recovery of bundle functions. SP represents the building up and periodic destruction of stimulus matter in the ventricle. The complete development of stimulus matter is interfered with in cycles 1-8 by ventricular beats, and by auricular impulses c^2 , c^4 , and c^6 . In the last cycles there is no interference and spontaneous ventricular beats occur" (J Physiol 1908; 37: 453).

Mackenzie had described as nodal rhythm and a paper in the *British Medical Journal* on 27 November, 1909 entitled “Auricular fibrillation; a common clinical condition” gives a brief and masterly summary of the evidence conclusively pointing to fibrillation of the atria as the underlying condition.

A few months earlier he had, with Mackenzie's support and encouragement, founded the journal *Heart*, the first issue of which came out on 1 July 1909. It contained papers by Mackenzie on nodal bradycardia and by Lewis on paroxysmal tachycardia, which are interesting to compare because the former describes patients as having nodal rhythm when the tracings clearly show that they do not, while Lewis published a fine example of rapid “cannon” waves caused by nodal tachycardia and failed to realise at that time what he was seeing. It is important, however, in containing a first brief reference to his work on the experimental production of paroxysmal tachycardia—usually ventricular but sometimes atrial—by the ligation of the coronary arteries in cats and dogs. The full account covering 40 pages appeared in the second issue. Another paper on paroxysmal tachycardia appeared in the third issue¹⁵ and is notable for showing wide QRS complexes of two types with atrial extrasystoles in addition to the normal complexes (Fig. 6). Lewis could not account for this phenomenon at the time, but he later attributed it “to defects in conduction through some of the chief Purkinje strands (aberration)”. The patient under study had attended “the out-patient department at Welbeck Street”—No 58a was in fact his private consulting room. The fourth number of volume one was issued on 30 March 1910, and we can note at this point Lewis's refusal to publish the *Journal* at regular intervals. When he started *Heart* he said that the numbers would come out when there was

sufficient and good enough material to justify it. The final 66 pages of volume I are taken up with “Auricular fibrillation and its relationship to clinical irregularity of the heart”¹⁵ and it is illustrated by no less than 31 figures most of which are electrocardiograms taken with Engelmann's modification of the Einthoven galvanometer. The long paper was probably the most comprehensive and best account of atrial fibrillation available at that time. The fifth volume of *Heart* was completed on 16 July 1914, and in these five volumes Lewis had written 22 papers (only five jointly) with a range of four to 66 pages, average 25 pages each. It is interesting that he chose the physiologist W H Gaskell to write the prefatory note and he lauds him in the editorial preface as being largely responsible for the advances of recent years in circulatory physiology. Lewis, however, carefully avoided the use of the word “physiology” in defining the scope of the new journal which he states to be “new facts and new conclusions relating to haemodynamic problems” . . . and “the progress of knowledge of the mechanism by which the blood circulates in health and disease”. He refers—and how many editors of new journals have done since—to, “the difficulty of keeping in touch with the Papers that appear, so widely scattered in the various publications of more general scope”.

In March 1910, he gained the first of the newly established Beit Memorial Fellowships, and in July of that year in *Heart*¹⁶ he published a very fine paper delineating the pacemaker of the heart. Stimulating electrodes were attached to various points on the atria or the venae cavae, and very remarkably to the internal surface of the right atrium with the electrodes protruding beyond a glass tube which had been introduced via the internal jugular vein. The electrodes terminated in minute trout hooks—shades

Fig. 6 Electrocardiogram showing coupled atrial extrasystoles with two types of wide QRS complexes—later attributed by him to aberration. The original legend read as follows. “Electrocardiographic and radial curves. A ‘bigeminy’, consisting of normal beats and auricular extrasystoles, is shown. The abnormal beats are of Types II and III: they are arranged alternatively between the normal beats” (*Heart* 1909; 1: 273).

of his happy boyhood days fishing in Wales. He returned again to paroxysmal tachycardia "accompanied by the ventricular form of venous pulse"¹⁷ and it is curious to find him persisting with this designation because he remarks in the text that the pulsation is readily appreciated by the finger awakening the suspicion that it is not of ventricular origin. Only tentatively does he reach the conclusion that these rapid giant "a" waves are the result of impulse formation in the junctional tissues (Fig. 7).

Looking back to 1911, one can imagine the sensation which must have been created in that year by his publication of "*The Mechanism of the Heart Beat*". It was a 296 page account, divided into 23 chapters and with 227 figures, which covered all aspects of the heart beat in man and in animals, physiological and experimental (Fig. 8). All methods used were carefully described and it must have been a godsend to those starting to work in the still relatively new field of electrocardiography, of which in fact it was quickly described as the bible.

It contained, however, more than electrocardiograms. Quite a number of the photographic recordings have two channels of mechanical activity in addition to the electrocardiogram. The work was dedicated to James Mackenzie and Willem Einthoven as "a token of the appreciation of the services which they have rendered to the study of the clinical pathology of heart affections". Clinical pathology was a phrase which he was fond of using to describe what was really his own special contribution of applying experimental methods to the study of clinical problems and it was a disappointment to him 20 years later when the phrase had been adopted by clinical biochemists and others, causing him to adopt an alternative description, namely clinical science.

1912 saw the first of his books for the general medical profession which were written from a sense of duty in time he could hardly spare from his intense research activity. It was "*Clinical Disorders of the Heart Beat*" and was soon translated into French (Fig. 9).

Having visited Einthoven in Leiden in 1908, it naturally gave Lewis great pleasure to welcome him to a special meeting of the clinical section of The Royal Society of Medicine, which was held at University College Hospital Medical School on 20 March 1912. Lewis was at his most fluent. "The School", he said, "was proud to entertain such a man, a worker whose name was forever stamped in the annals of clinical medicine. (He was) an incalculable benefactor of the human race". Bearing in mind that his own research on arrhythmias went back only four years, his assertion at the meeting that "the chapter of the analysis of cardiac irregularities was fast closing" was breathtaking. Nevertheless even with hindsight one

may judge the essential truth of his statement by reading the first edition published in 1913 of his small handbook entitled "*Clinical Electrocardiography*". It could be used today without alteration as a first class manual on the main cardiac irregularities and it has excellent illustrations.

Fig. 7 Four possible explanations, in the form of ladder diagrams, for paroxysms of tachycardia which later were definitely identified by him as of nodal origin. The original legend read as follows. "Four diagrams illustrating the possible interpretations of mechanism in two portions of the preceding curves. Fig. 7, 1 and 2 are alternative explanations of the first short paroxysm of Fig. 3. Fig. 7, 3 and 4 are alternative and parallel explanations of the first five beats of Fig. 6. The arrows drawn above each diagram represent impulses which descend from the pacemaker and give rise to auricular contractions. A represents the auricle, B represents the junctional tissues, and V represents ventricle. The black dots are intended to represent the point at which the impulses may be supposed to arise" (Heart 1910; 2: 138).

THE MECHANISM OF THE HEART BEAT

WITH ESPECIAL REFERENCE TO ITS
CLINICAL PATHOLOGY

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1911

Fig. 8 Title page of "The Mechanism of the Heart Beat". London: Shaw and Sons, 1911.

LES DÉSORDRES CLINIQUES
DU
BATTEMENT DU CŒUR

PAR

TH. LEWIS

Professeur à l'École de Médecine de Londres.

TRADUIT DE L'ANGLAIS

PAR LE D^r C. CHAUVET

Médecin consultant à Royat.

PRÉFACE DE M. LE PROF. TEISSIER, DE LYON

AVEC 47 GRAPHIQUES DANS LE TEXTE

PARIS

LIBRAIRIE FÉLIX ALCAN

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Fig. 9 Title page of the French translation of "Clinical Disorders of the Heart Beat" 1913.

At the meeting Lewis referred briefly to the form of the electrocardiogram that occurred with right ventricular hypertrophy—"in lead 1 a small peak R and deep depression S, and in lead 3 a large and exaggerated peak R". He showed tracings from a case of congenital pulmonary stenosis. He also mentioned the fact that together with the obstetric registrar at UCH, Dr Clifford White, he had been examining newly born children and had found the same type of curve, "the right ventricle in the new born child was strong". In the book previously referred to he has a beautiful pair of illustrations showing the regression of physiological right ventricular hypertrophy between two hours and six weeks after birth (Fig. 10).

He returned to the subject of ventricular hypertrophy later in a paper in which he describes a new method of assessing ventricular hypertrophy at necropsy by weighing separately each ventricle and the septum, and summarising the results as the left/right ratio, showing this normally to be 1.8. Though he recognises that "high blood pressure appears to be especially potent in creating a

preponderance of the left ventricle"—the average left/right ratio being 2.31, he is keen to emphasise that uniform hypertrophy is the commonest type, "In renal disease this is the rule"—yet his figure for this group at 1.97 is well above the normal and is identical with all aortic cases. He conveniently avoids the question as to which ventricle contributes to the septal hypertrophy. In 80 patients he compares his clinical notes on cardiac pulsations with the post-mortem heart weights and finds no correlation with an epigastric thrust and right ventricular weight, and though a forceful thrusting and displaced apex was found usually in those with enlarged left ventricles he makes too much of a single case with a huge right ventricle and a heaving impulse citing it as "a most notable instance of the unreliability of those physical signs", and was apparently unaware of Mackenzie's account of the right ventricle forming the apex and of his clear description of the difference between outward movement and retraction at the apex and epigastrium in right and left hypertrophy. In 96 patients he shows that the electrocardiogram fairly

Fig. 17.

Fig. 18.

Fig. 10 Electrocardiograms showing resolution of the physiological right ventricular hypertrophy of the newborn. Originally this figure appeared as two illustrations (Fig. 17 and 18) from "Clinical electrocardiography". London: Shaw and Sons, 1913: p. 26. The original legends read as follows. "Fig. 17. Curves from a child two hours after birth. The relative heights and depths of the peaks is such as is expected where there is relative preponderance of the right ventricular muscle. Fig 18. Curves from the same child, but six weeks later. The right-sided preponderance is not evidenced by these curves to nearly the same extent."

often exhibits a QRS angle of 120 degrees in mitral stenosis but it worries him that quite a few do not. He is also concerned because in aortic disease many did not show a deep S in III. He overlooks that his figures show tall R waves in leads I and II in those cases, and also that left axis deviation (though he does not use that term) is something different from left ventricular hypertrophy.

I mention all this at some length because it leads him to conclude that “mechanical factors are by no means the only important causes of hypertrophy of the heart” and this must have coloured his later views which played down the importance of valve lesions and emphasised the pre-eminence of the myocardium. The normals in the series were from a paper by Gilder and himself¹⁸ where he included a case with a QRS axis of +120 “with a history of bronchitic trouble”, and where he took an $R3 > R1$ as abnormal—concluding erroneously that normal subjects may have “right or left-sided ventricular preponderance”.

From 1908 onwards the Cambridge Scientific Company had been collaborating with Einthoven on a marketable form of his galvanometer, and W D Duddell FRS designed a superb and small modification of the original instrument (which occupied two rooms and took five people to operate) and it was equally well produced by the company. The first complete Cambridge electrocardiograph, which was finished in 1911, was supplied to Lewis but it was not until the technician B A Robinson, who was personal assistant to Sir Horace Darwin at Cambridge, arrived at UCH in July 1912 that the apparatus became operational. One of the first clinical

applications was a phonocardiographic study and Lewis extended L Bull’s method of simultaneously recording the sound and electrical records¹⁹ by using two string galvanometers side by side projecting onto the same photographic plate, and with this he obtained exquisite heart sound records. In normal subjects he measured several physiological intervals such as the beginning of Q wave to S1; and from analysing the records of several patients with mitral stenosis (Fig. 11) he states clearly that the murmur is the result of the high velocity of blood flow which in its turn depends on the difference in pressures between the left auricle and the left ventricle. “The murmurs” he says “first appear at those portions of diastole at which the differential pressure is greatest”.²⁰ He returned to the subject a year later with a paper on “Illustrations of heart-sound records”.²¹

The phonocardiograms were recorded with a carbon granule microphone constructed by the Cambridge company on lines suggested by Einthoven, and they produced a double string carrier for twin channel recording.²² In fact he later had two double string carriers, one left and one right handed, so that he could project the images of four strings and obtain a four channel simultaneous tracing from different sources. It was the twin string instrument that enabled him to proceed with what is probably his finest piece of investigatory work with the electrocardiogram, namely “The excitatory process in the dog’s heart”.

Two papers on this subject were communicated to the Royal Society and published in their *Philosophical*

Fig 11 Simultaneous electrocardiograms and phonocardiogram in a patient with mitral stenosis. The murmur is marked M and is preceded by an opening snap—which Lewis invariably called a reduplicated second sound. (Heart 1913; 4: 256.)

Transactions. Part I was on the atria and his co-authors were John Meakins of Montreal and Paul D White of Boston,²³ while Part II was on the ventricles and the co-author was M A Rothschild.²⁴ The twin string enabled him to use a lead from the right shoulder to the left groin as the standard tracing, with the second channel employed for direct electrograms from either the epicardium or the endocardium—in the latter case the electrodes being introduced via the pulmonary veins or superior vena cava. It was shown that the region of the sinoatrial node became electro-negative relative to all surrounding points as the atrium contracted and that therefore it seemed to be the point of origin of the excitation wave. The receipt of the excitatory wave by the muscle immediately beneath the electrode contact, signalled by a prominent spike, was labelled the *intrinsic deflection* (Fig. 12). In studying the spread of the excitation wave the positions of the electrodes were measured in relation to fixed points, and at the end of the experiment the heart was distended with formalin and then embedded in paraffin from which small blocks of tissues were cut and studied. Therefore every electrogram could be accurately placed in relation to muscle bands and the sinoatrial node.

Fig. 12 Twin channel electrocardiograms from a dog's heart. 1 is from the sinoatrial node; 2 is lead II. (Phil Trans R Soc Lond [Biol] 1914; 205: plate 40.)

In the second paper the distribution of the excitation wave over the surface of the ventricles is immaculately plotted, and after the experiment the heart is stained in bulk with Best's carmine in order to show up the chief branches of the bundle and whole Purkinje network. The importance of the Purkinje system is emphasised by a fine experiment, in which a knife introduced through the right auricular appendage is used to sever the right branch of the bundle. The result showed the impulse to arrive at contacts on the left ventricle at an unaltered time, while contacts on the right ventricle or on the endocardium of the conus showed a considerable delay in excitation.

In a later communication, this time to the Physiological Society,²⁵ Lewis gives convincing evidence that the Q wave results from activation of the septum from the left bundle division. He derives this conclusion from dog work showing that the earliest reading of an intrinsic deflection is obtained from the septum beneath the aortic valve and also perceptively points out that Q is absent in tracings from the toad and tortoise, in which animals no septum exists. This was only one of five papers in the *Physiological Proceedings* for 1915 and it marks the end for him of an era of electrophysiological investigation.

I am deeply indebted to Lady Lewis for permission to reproduce her own copy of Sir Thomas's photograph, and grateful for the photographic help extended by Mr Stephen Paratian.

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