

## THE CARDIOLOGY OF R. T. H. LAENNEC

by

JACALYN M. DUFFIN \*

René Théophile Hyacinthe Laennec (1781–1826) made an extensive study of the heart that has been examined by historians in different ways, all inadequate, ranging from open ridicule to bemused indulgence.<sup>1</sup> Words like “wrong” or “less excellent” have been applied to his conclusions and he has been accused of leaving the study of heart disease in a hopeless “snarl”.<sup>2</sup> Usually this aspect of Laennec’s research is neglected in favour of his more successful work on diseases of the lungs. Often, the fact that he ever listened to the heart at all has been considered pardon enough for his mistakes. But Laennec considered his work on auscultation of the heart to be every bit as important as that on the lungs. By his own account, it was for the express purpose of a cardiac examination that he created his first stethoscope.<sup>3</sup> Because he believed his technique offered a breakthrough in the diagnosis of heart disorders, he devoted long hours to the interpretation of its normal and abnormal sounds.

Examination of Laennec’s cardiology offers an insight into the concerns of a practitioner in the transitional period of anatomic-clinical medicine, when disease concepts moved from definition by associated subjective symptoms to definition by associated objective organic lesions. It also provides a unique opportunity to study the psychological and epistemological priorities governing an early nineteenth-century physician’s use of evidence. As Laennec explored the sounds generated by the heart, he accepted some and rejected others as signs of hidden organic lesions. The criteria he

\*Jacalyn M. Duffin, M.D., F.R.C.P.(C), D.3cC., J. A. Hannah Professor of the History of Medicine, Faculty of Medicine, Queen’s University, Kingston, Ont. K7L 3N6, Canada.

I am indebted to Mirko D. Grmek for his guidance in this research and to Goodman Cohen, Toby Gelfand, Frederic L. Holmes, Joel D. Howell, and John Harley Warner for their helpful comments on earlier versions of this paper. Support of the Jason A. Hannah Institute for part of this work is gratefully acknowledged.

<sup>1</sup> Standard histories of medicine recognize Laennec’s “lesser” achievement in cardiology as do several histories of cardiology: Terence East, *The story of heart disease*, London, Wm. Dawson, 1958, pp. 32–3; James B. Herrick, *A short history of cardiology*, Springfield MA and Baltimore, Charles C Thomas, 1942, p. 88; F. A. Willius and T. J. Dry, *A history of the heart and the circulation*, Philadelphia and London, W. B. Saunders, 1948, p. 317.

<sup>2</sup> Victor A. McKusick, ‘Rouanet of Paris and New Orleans: experiments on the valvular origin of the heart sounds’, *Bull. Hist. Med.*, 1958, 32: 137.

<sup>3</sup> R. Laennec, *De l’auscultation médiate ou traité de diagnostic des maladies des poumons et du coeur fondé principalement sur ce nouveau moyen d’exploration*, 2 vols., Paris, Brosson and Chaudé, 1819, vol. 1, pp. 7–8; also in the second edition, renamed, *Traité de l’auscultation médiate et des maladies des poumons et du coeur*, 2 vols., Paris, Chaudé, 1826, pp. 7–8. The historical vignette of Laennec’s discovery, including the role of the children playing in the courtyard of the Louvre, was presented by Laennec’s younger colleague J. A. Le Jumeau de Kergaradec, ‘Discours sur Laennec à l’inauguration de la statue de Laennec à Quimper’, *Bull. de l’Acad. de Méd.*, 33: 810. The authenticity of this account has been examined by M. D. Grmek, ‘L’invention de l’auscultation médiate: retouches à un cliché historique’, *Rev. du Palais de la Découverte*, 1981, no. spéciale 22: 107–16.

imposed on this ensemble of inductive evidence are interesting in themselves, because they were an expression of the probability-based decision making orientation of one whose existence straddled the sensualist philosophy of the late eighteenth century and the dawn of positivist thought. That Laennec's conclusions were inaccurate does not hamper our assessment of his priorities; in fact, the errors permit a certain enhanced clarity. In the case of the breath sounds, where the intended meanings of Laennec's signs appear to conform to those of the present, there is a great temptation to assume that they must indeed have been given the same significance. Laennec's heart signs and their evolution do not allow this type of simplistic assumption. They require a diligent effort on the part of a modern observer in order to be understood and this effort protects the comprehension from unwarranted assumption.

#### CARDIOLOGY IN THE EARLY NINETEENTH CENTURY

To discuss the cardiology of the early nineteenth century is to invite an accusation of anachronism. Diseases of the heart were considered to be plausible entities by 1800, but symptoms that could be regarded as diagnostic of cardiac problems were few. For centuries, the phenomena most commonly associated with disease of the heart were palpitation, chest pain, and sudden death. Other symptoms now commonly related to cardiac dysfunction, such as fainting spells, shortness of breath, and dependent oedema, were not so obviously linked to the heart and most appeared to be remote. As Saul Jarcho observed, it is necessary to unearth records from "unexpectedly diverse hiding places" in order to make a longitudinal study of a modern cardiac concept.<sup>4</sup> The purpose of this introduction is not to make a longitudinal study of a modern concept, but rather to determine the status of knowledge concerning diseases of the heart in the early nineteenth century and to relate this understanding to the cardiology of René Laennec.

It has been observed that there is a "dearth of recorded interest" in the subject of physical diagnosis of heart disease before Laennec;<sup>5</sup> this observation may be accurate, but it is also naïve. Late eighteenth-century physicians were not looking for signs of heart disease any more than they were looking for the signs of renal disease, liver disease, or any other organ-based illness. Given the spectrum of clinical diagnoses available to an early nineteenth-century practitioner, discovery and treatment of an organic lesion of the heart was not a concern. Diagnoses consisted of the accurate description and naming of the symptom complex accompanying illness. Laennec's invention of the stethoscope, in 1816, provided a practical means of bringing pathological anatomy to the bedside. Auscultation made possible the ante-mortem recognition of organic lesions in the lungs, the site of many common diseases of the time. The stethoscope permitted the identification of diseases in the living patient by detecting their associated physical alterations. It cleared the way for a shift in the conceptualization of all diseases from symptom groupings to organic alterations in the human organism and it made useful the accumulation of more than a century of

<sup>4</sup> Saul Jarcho, *The concept of heart failure from Avicenna to Albertini*, Cambridge MA, Harvard University Press, 1980, p. vi.

<sup>5</sup> Victor A. McKusick, 'The history of methods for the diagnosis of heart disease', *Bull. Hist. Med.*, 1960, 34: 16.

observation in pathology. There could be no interest in the physical diagnosis of heart disease until diseases were perceived to be due to *and identified by* physical alterations of the heart.

In 1800, the dominant method of defining disease in the clinical setting was the symptom-based classification of the nosologists, in which disease was perceived as the ensemble of symptoms suffered by and perceptible to the patient. Some of these symptoms were “pathognomonic” or diagnostic of a recognizable course or outcome. Such symptoms were considered to be signs as well, because the physician’s appreciation of this type of symptom gave additional information. A sign was generally thought to be a symptom that invoked a reasoned judgement, a judgement that led to the name of the disease or to an accurate prognosis.<sup>6</sup> Rarely would the sign implicate an organic lesion. Practitioners interested in pathological anatomy might suspect some structural alteration of the heart in certain patients, but this suspicion was insufficient to constitute a diagnosis.

While nosology dominated clinical medicine, pathological anatomy existed as an almost separate science.<sup>7</sup> Its perceived relevance to bedside medicine increased throughout the eighteenth century as reflected in the titles, which moved from the “graveyard”, *Sepulchretum*, of Théophile Bonet, to the *Seats and causes of disease* of Giambattista Morgagni, and the *Medical anatomy* of Antoine Portal.<sup>8</sup> But the utility of pathological anatomy was challenged by three principal objections. First, organic lesions were associated with the cadaver and might even be artefacts of death itself; symptoms were a feature of life. Second, although the internal changes might be truly associated with disease, they could not be detected before the death and autopsy of the patient. Finally, even if the organic changes could be detected by or inferred from the clinical presentation, nothing could be done to change them. The physician had to relieve the symptoms, the subjective suffering of the disease, and could never hope to alter internal defects of anatomy.

Pathological lesions of the heart had been recognized in all the great eighteenth-century compendia, from Bonet to Baillie, although there may have been a traditional prejudice against the notion of a diseased heart.<sup>9</sup> In 1715, Raymond Vieussens wrote an anatomical-physiological treatise on the structure of the heart and its movement in which he mentioned, but did not emphasize, structural changes as part of disease.<sup>10</sup> He

<sup>6</sup> See contemporary discussions of “*signe*”, “*symptome*” and “*pathognomonique*” in the following: François Double, *Sémiologie générale ou traité des signes*, Paris, 1811, vol. 1, pp. 149, 157–8; A. J. Landré-Beauvais, *Sémiotique ou traité des signes des maladies*, second ed., Paris, 1813, pp. ix–xx; *Dictionnaire de médecine*, eds. Adelon, Andral, Béclard, Biett, Breschet, *et al.*, Paris, Béchét jeune, 1826, vol. 16, p. 217; and *ibid.*, vol. 19, pp. 317–18. See also Lester S. King, *Medical thinking: a historical preface*, Princeton University Press, 1982, pp. 99–104.

<sup>7</sup> Stanley Joel Reiser, *Medicine and the reign of technology*, Cambridge University Press, 1978, pp. 1–22; Lester S. King, ‘Auscultation in England, 1821–1837’, *Bull. Hist. Med.*, 1959, 33: 452.

<sup>8</sup> Théophile Bonet, *Sepulchretum sive anatomia practica ex cadaveribus morbo denatis*, Geneva, Cramer et Perachon, 1700; Giovanni Battista Morgagni, *De sedibus et causis morborum per anatomen indagatis libri quinque*, Venice, Remondini, 1761; Antoine Portal, *Cours d’anatomie médicale*, 5 vols., Paris, Baudoïn, An xii (1804).

<sup>9</sup> James B. Herrick wrote that, according to Hippocrates “*cor aegrotari non potest*” (the heart could not be sick), ‘Certain textbooks on heart disease of the early nineteenth century’, *Bull. Hist. Med.*, 1941, 10: 137.

<sup>10</sup> Raymond Vieussens, *Traité nouveau du structure du coeur et des causes du mouvement naturel*, Toulouse, Guillumette, 1715.

suggested that alterations in the quality of the blood could favour organic change in the heart.<sup>11</sup> Jean-Baptiste Sénac also wrote a treatise on the structure and diseases of the heart and even chose to name his second edition *Diseases of the heart*, although it dealt mainly with the pathological lesions.<sup>12</sup> Sénac, like Vieussens, discussed changes in the pericardium and the heart muscle and, in addition, he described lesions of the valves, coronary arteries, and great vessels. He considered sadness to be a potential cause of heart disease.<sup>13</sup> In Britain, William Heberden's (1710–1801) classic description of the pain of angina pectoris<sup>14</sup> was linked to ossification in the coronary arteries in the work of John Fothergill (1712–80), John Hunter (1728–93), Caleb H. Parry (1755–1822), and Edward Jenner (1749–1823).<sup>15</sup> Here, then, the symptom of a specific type of pain became a sign of a specific internal organic change. As will be shown, however, acceptance of this notion was neither immediate nor universal, and objections were often justified. Sénac expressed the problem of accurate correlation of the variable symptoms with organic changes, when he lamented the sorry prospects of detecting such changes before the patient died: “But how amongst so many false appearances, so many complications and varieties can one disentangle the heart diseases?”<sup>16</sup>

In the early nineteenth century, prior to Laennec's discovery of auscultation, there was a certain *rapprochement* between nosology and pathological anatomy. For example, at the end of every chapter in his book, Matthew Baillie (1761–1823) discussed symptoms that accompanied alterations in each particular organ. Concerning the heart, he stated that the symptom of angina pectoris “would seem to be intimately connected to ossification of the coronaries”, but he readily admitted that for other organic changes, like valvular lesions, “no observations have yet been made by which practitioners may be led to conjecture what set of valves is diseased.”<sup>17</sup> Shortly after, several works appeared that sought to combine clinical presentation with pathological changes in the heart. These were the earliest clinico-pathological treatises on heart disease. The first of these, and the best known, was the *Essay on the organic diseases and lesions of the heart and great vessels* by Jean-Nicolas Corvisart des Marets, Napoleon I's personal physician and Laennec's teacher.<sup>18</sup>

<sup>11</sup> *Ibid.*, pp. 120–4.

<sup>12</sup> Jean-Baptiste Sénac, *Traité de la structure du coeur, de son action et de ses maladies*, Paris, J. Vincent, 1749; and second edition, renamed, *Traité des maladies du coeur*, Paris, 1778.

<sup>13</sup> *Ibid.*, 1749, vol. 2, pp. 286–7.

<sup>14</sup> William Heberden's description was first delivered as a lecture in 1762 and published in 1768. It also appeared in his *Commentaries on the history and cure of diseases* (1801), repr., New York, Hafner and the New York Academy of Medicine, 1962, pp. 362–9.

<sup>15</sup> Caleb H. Parry, *An inquiry into the symptoms and the causes of the syncope anginosa*, London, Cadell and Davis, 1799. See also Joshua O. Leibowitz, *The history of coronary heart disease*, London, Wellcome Institute for the History of Medicine, 1970, pp. 73–103.

<sup>16</sup> Sénac, *op. cit.*, note 12 above, 1778, vol. 1, p. 34. Portal, making the same lament, suggested as an explanation that clinicians were not good pathologists and pathologists were not good clinicians: *op. cit.*, note 8 above, vol. 1, p. v.

<sup>17</sup> Matthew Baillie, *Morbid anatomy of some of the most important parts of the human body* (1793) reprinted in Alvin E. Rodin, *The influence of Matthew Baillie's Morbid Anatomy*, Springfield, Charles C Thomas, 1973, pp. 83–95, esp. p. 94.

<sup>18</sup> J.-N. Corvisart, *Essay on the organic diseases and lesions of the heart and great vessels* (1806), ed. C. E. Horeau, repr. of 1812 English edition trans. Jacob Gates, New York, Hafner and the New York Academy of Medicine, 1962.

Corvisart's *Essay* is divided into six parts. Five more or less concern the different sites of organic alteration, pericardium, heart muscle, tendinous parts (valves and chordae, etc.), varia (carditis, rupture, tumour and septal defect) and aorta. The last part contains a discussion of aetiology, diagnosis, prognosis, and treatment. Case histories illustrate the commentary on pathological lesions. Corvisart described clinical entities that resemble the conditions now associated with coronary artery disease, but perhaps because of his political milieu, he appears to have been unaware of, or unwilling to credit, the theories on this disorder expounded across the Channel. He associated stenosis of the mitral valve with a palpable "thrill", a vibration felt by the fingertips on the chest.<sup>19</sup> Corvisart was especially interested in enlargement of the heart, which he called "aneurysm", and he sub-divided it into "active" (thickened muscle) or "passive" (thin muscle). He tried to detect aneurysm by laying a palm on the patient's chest or by tapping on the precordium to determine the extent of cardiac dullness, and was quite confident in his ability to distinguish active from passive aneurysm in the clinical setting.<sup>20</sup> He expanded on the value of percussion two years later in his translation of Auenbrugger's *Inventum novum*.<sup>21</sup>

In defining the clinical signs of organic heart disease, Corvisart included the colour and appearance of the face, the state of the pulse, the presence of dependent oedema, dyspnoea, enlargement of the liver, palpable thrill, and the signs of lung engorgement, hydrothorax or enlarged heart as elicited by percussion. Here were many signs, but even Corvisart acknowledged that, at times, it was still necessary to distinguish disorders of the heart from those of the lung.<sup>22</sup>

Unlike Sénac,<sup>23</sup> Corvisart thought that heart diseases were common, perhaps even increasing in frequency, and that they could be caused not only by abnormalities in heart structure but also by emotional and psychological distress, such as that created by the travail of the recent French Revolution. He was a mechanist, who had referred to the human body as a machine.<sup>24</sup> For example, he thought that coarctation of the aorta led to changes in the heart muscle,<sup>25</sup> but he did not rule out the psychic causes of organic lesions in the heart. He wrote:

If anyone could candidly deny or only doubt of the fatal physical influence of the passions over the heart, it may be sufficient for him to be informed that it may be lacerated in a fit of anger and instant death ensue; and I am not the only physician who has thought that [the heart's] organic lesions were more frequent in the horrible times of the revolution than in the usual calm of social life . . . This organ is the point in which the effects of all the moral affections, gay or melancholy, seem to be concentrated. The unexpected news of pardon strikes a criminal dead who was going to be executed. A

<sup>19</sup> *Ibid.*, p. 185. Support for this statement is contained in a manuscript consultation published by P. Soulié, 'Corvisart et le diagnostic clinique du rétrécissement mitral', *Hist. de la Méd.*, 1955, no. 7, pp. 55–64.

<sup>20</sup> Corvisart, *op. cit.*, note 18 above, pp. 126–8.

<sup>21</sup> Léopold Auenbrugger, *Nouvelle méthode pour reconnaître les maladies de la poitrine*, trans. J. N. Corvisart, Paris, Migneret, 1806, pp. 420–32.

<sup>22</sup> Corvisart, *op. cit.*, note 18 above, pp. 315–30.

<sup>23</sup> Sénac, *op. cit.*, note 12 above, 1749, vol. 2, p. 318; Corvisart, *op. cit.*, note 18 above, p. 15.

<sup>24</sup> Corvisart, *op. cit.*, note 18 above, p. 18.

<sup>25</sup> *Ibid.*, pp. 77–8.

lover dies at the very moment the flame of his passion was to be satisfied; the one is destroyed by terror; the other apparently thunderstruck with a paroxysm of passion . . .<sup>26</sup>

As most of Corvisart's research on organic heart disease took place during the strife of the French Revolution, the Terror, and the wars of the First Empire, it is almost inconceivable that he autopsied *any* patient, cardiac or other, who had not suffered emotional hardship. Later observers contended, perhaps with some justification, that there was no true rise in frequency of the heart diseases and that Corvisart's sensitivity to the increased psychological stress among his contemporaries caused him to examine the heart more closely, seeking (and finding) anticipated organic changes.<sup>27</sup>

In 1809, Allan Burns (1781–1813) published his *Observations on some of the most frequent and important diseases of the heart*.<sup>28</sup> He was unaware of Corvisart's work, but he shared many of the same preoccupations, including an interest in enlargement of the heart. Burns divided enlargement into two categories, corresponding to Corvisart's "active" and "passive" aneurysm: enlargement with increase in solid muscular substance and simple dilatation. He associated the former with the palpable sensation now known as "precordial lift" and considered the presence or absence of lift to be a means of distinguishing between the two.<sup>29</sup> He was not familiar with percussion and was less able than Corvisart to detect enlargement in general. Burns endorsed the contributions made by his countrymen to the coronary artery theory of angina pectoris.<sup>30</sup> He avoided discussing the origins of the organic lesions and accused his contemporaries, including "the venerable Portal", of offering only vague conjectures.<sup>31</sup> Although he scorned the concept of "acrimonious humours" as a cause of organic heart disease, Burns was not prepared to find a purely mechanical cause for the dilatations of the heart. He developed a sophisticated theory of back pressure to explain dilatation and cardiac oedema, but he rejected Parry's accurate observation that dilatations occur in those parts immediately proximal to an obstruction.<sup>32</sup> "Suffice it to say", remarked Burns, "that, in general, the dilatation is not caused by any mechanical agent. We know that individuals are predisposed to certain diseases, and that these different affections are produced by similar exciting causes."<sup>33</sup>

Two other, slightly later, treatises on heart disease deserve mention here because Laennec cited them both; however, as his translator suggested, he may not have given them deep consideration.<sup>34</sup> The first was the *Delle malattie del cuore* (1810) of

<sup>26</sup> *Ibid.*, pp. 30, 275.

<sup>27</sup> John Forbes, Laennec's translator, claimed to hold this opinion with R. J. Bertin: see R. T. H. Laennec, *A treatise of diseases of the chest*, trans. John Forbes, London, Underwood, 1827, p. 584n.

<sup>28</sup> Allan Burns, *Observations on some of the most frequent and important diseases of the heart* (1809) repr., New York, Hafner and the New York Academy of Medicine, 1964.

<sup>29</sup> *Ibid.*, p. 40–2.

<sup>30</sup> *Ibid.*, pp. 136–52.

<sup>31</sup> *Ibid.*, p. 44.

<sup>32</sup> Parry, *op. cit.*, note 15 above, pp. 113–14; *idem*, *Elements of pathology and therapeutics*, London, Underwood, 1815, p. 162.

<sup>33</sup> Burns, *op. cit.*, note 28 above, pp. 44–6.

<sup>34</sup> John Forbes referred Laennec's readers to other works on heart disease because the "paramount importance of auscultatory diagnostics in his mind has rendered this epitome too brief . . . [it is excellent] as far as it goes." *Op. cit.*, note 27 above, p. 579n.

Antonio Giuseppe Testa, who also accepted the coronary artery theory of angina and who envisaged vitalistic causes of organic heart disease.<sup>35</sup> The second, *Die Krankheiten des Herzens* (1814) by Friedrich Ludwig Kreysig, a prolix three-volume work, was a compendium of anatomical abnormalities found in the heart. It has been suggested that Kreysig relied more heavily on the publications of others, including Corvisart and Burns, than he did on his own clinical experience.<sup>36</sup> Like them, he exhorted physicians to perform autopsies in order to improve the understanding of heart disease. He adopted the coronary theory of angina, the valvular significance of palpable thrill and once mentioned having “heard” a swishing sound, but he ignored percussion.

The cardiology of the early part of Laennec’s career can be summarized in three statements: first, there was general acknowledgement that illness could be related to organic changes in the heart; second, there was a fairly well-developed system of study of these organic lesions; and, finally, there was a less well-developed system of clinical signs indicative of the pathological change. A few of these signs, like the palpable thrill and percussion, were objective, independent of the patient’s perception or description. Many, like the pain of angina pectoris, were intimately related to the patient’s subjective experience of illness and most, like dependent oedema, dyspnoea and cyanosis, were not specific to heart ailments at all. Virtually all practitioners admitted the problems of detecting organic heart disease in the ante-mortem setting. Corvisart claimed a special ability, which he called “tact”,<sup>37</sup> for the experienced practitioner, an ability that helped him to choose the correct organic diagnosis between the various possibilities suggested by a complex and imprecise ensemble of observed phenomena.

#### LAENNEC’S CONCEPT OF HEART DISEASE

##### *Pathology*

Laennec retained Corvisart’s classification of organic heart disease, substituting only the word “hypertrophy” for Corvisart’s “active aneurysm” (see table 1). He coined the term “hypertrophy”, was the first to apply it to the myocardium, and pointed out that it could exist in the absence of dilatation. His emphasis on the independence of changes in each of the chambers was coupled with an almost total silence on the physiological circumstances that may have led to these alterations. He classified both hypertrophy and dilatation with diseases of “nutrition”: more heart was present than in the normal state and no other prior organic cause was implicated. He observed that atrial dilatation occurred most commonly in the presence of diseased atrio-ventricular valves, but he did not speculate on any causal relationship between the two conditions.<sup>38</sup>

<sup>35</sup> Antonio Giuseppi Testa, *Delle malattie del cuore*, Florence, 1810. See James B. Herrick, op. cit., note 1 above, pp. 33–4, 78, 139.

<sup>36</sup> Friedrich Ludwig Kreysig, *Die Krankheiten des Herzens*, 3 vols., Berlin, 1814–17. See James B. Herrick, op. cit., note 1 above, pp. 81–3.

<sup>37</sup> Corvisart, op. cit., note 18 above, p. 19.

<sup>38</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 281–4.

TABLE 1: LAENNEC'S CLASSIFICATION OF ORGANIC HEART DISEASE\*

---

*Diseases of the myocardium*  
Hypertrophy: left, right, both  
Dilatation: left, right, both  
Dilatation with hypertrophy  
Partial dilatation  
Hardening  
Softening: violet, yellow, white  
Atrophy  
Gangrene  
Displacement  
Prolapse  
Congenital abnormalities  
Carditis  
Communication between right and left heart  
Rupture  
Fatty Degeneration  
Ossification of the myocardium\*\*  
Tubercles  
Cancers  
Cysts: serous, acephalocysts  
Inflammation of the internal membrane  
Valves: induration and ossification  
Detached eustachian valve  
Mitral aneurysm  
Polyps

*Diseases of the pericardium*  
Pericarditis: acute, chronic  
Hydropericardium  
Pneumopericardium  
Accidental productions\*\*\*  
Ossifications

*Diseases of the vessels*  
Aorta: narrowing, incrustations, inflammation  
Pulmonary  
Coronary

*Neuralgias*  
Heart  
Vessels

---

\* derived from the *Traité de l'auscultation médiate*, second ed., 1826.

\*\* Laennec admitted that he had never seen a case.

\*\*\* A general term used by Laennec to indicate non-inflammatory new tissue. The group included benign tumours, carcinoma and tubercles.



Laennec shared Corvisart's major interest in volume changes of the myocardium, but he was not oblivious to other aspects of heart pathology. He recognized two forms of congenital heart disease: patent ductus and septal defects. He also left an original description of one type of valvular change, which he called "globular excrescences", the origin of which still remains obscure.<sup>39</sup> In 1802, at the age of twenty-one, he published his first article, a clinico-pathological case report on a 22-year-old male whose pleural effusion and right ventricular dilatation had been predicted by Corvisart, using percussion on the cadaver just prior to autopsy. Corvisart had also hinted that the left side of the heart would contain "organic lesions and that it was even probable that the primary cause ("*cause première*") of the disease would be found there."<sup>40</sup> In addition to proving the precision of Corvisart's predictions about the lungs and right ventricle, the autopsy revealed ossification of the mitral valve.

In 1809, Laennec gave a lecture (in Latin) before the Société de l'École de Médecine on the subject of angina pectoris.<sup>41</sup> This was never published, but an incomplete manuscript essay in Latin, which is preserved with his scientific papers, probably contains some of the text.<sup>42</sup> He demonstrated his awareness of the coronary artery theory of angina and he cited "Jenner and others",<sup>43</sup> but he was not convinced by their arguments. He rejected this theory because of conflicting results from autopsies: ossified coronary arteries could be found in persons dead of conditions unrelated to angina; conversely, apparently normal coronaries were observed in others dead of what he considered to be unmistakable angina.<sup>44</sup> Consequently, Laennec doubted the causal relationship of coronary ossification to angina pectoris. Filed with the essay is a 1810 manuscript record entitled "*angina pectoris?*" of the patient Nicolas Millot, who was thought to have suffered, and died, from angina.<sup>45</sup> At autopsy Millot was found to have had normal valves and coronary arteries. Although such a situation is entirely possible, a close reading suggests that the severe pain Millot described, "as if

<sup>39</sup> Laennec, op. cit., note 3 above, 1826, vol. 2, pp. 630–51. See also Frank D. Mann and Ruth J. Mann, 'Laennec as a critical pathologist', *J. Hist. Med.*, 1981, 36: 446–54, esp. pp. 451–2. The valvular changes may be due to disseminated intravascular coagulation. Joel D. Howell has suggested that the entity is probably that of marantic endocarditis (personal communication).

<sup>40</sup> René Laennec, 'Observation d'une maladie du coeur', *J. de Médecine*, An x (1802), 4: 265–307.

<sup>41</sup> 'De angina pectoris commentarius', was read to the Société de l'École de Médecine, 31 October 1809. See *Bull. Soc. Éc. Méd.*, 1809, no. 10: 135.

<sup>42</sup> An undated manuscript *mémoire* on angina pectoris probably contains some of this lecture: Laennec, MS. Cl. 7, lot e–2. Other papers in Latin, including several consultations dated 1810, are kept with it. MS. Cl. 7, lot e–1, and e–3. Laennec's scientific manuscripts are preserved in the Musée Laennec of the Bibliothèque Universitaire de Nantes and in the Bibliothèque Interuniversitaire de Médecine de Paris. An indispensable guide to these papers is the catalogue edited by Lydie Boulle, Mirko Grmek, Catherine Lupovici, and Janine Samion-Contet, *Laennec: catalogue des manuscrits scientifiques*, Paris, Masson and Fondation Singer-Polignac, 1982. All manuscript references will be made to the classification codes in this catalogue.

<sup>43</sup> Laennec, MS. Cl. 7, lot e–2, f.9.

<sup>44</sup> E. H. Desportes, *Traité de l'angine de poitrine*, Paris, Méquignon, 1811, pp. 69–83; John Warren, 'Remarks on angina pectoris' [1812], *N. Eng. J. Med.*, 1962, 266: 3–7. A discussion of the controversy still surrounding the coronary theory in the 1920s was presented by Reidar Lie, 'The angina pectoris controversy during the 1920s. Why was the coronary theory accepted?', at the American Association for the History of Medicine Annual Meeting, Philadelphia, April 1987.

<sup>45</sup> Laennec, MS. Cl. 7 lot e–2, ff. 25r.–26v.

someone were trying to tear off his left breast . . . [from] a square area the size of two playing cards overlying his heart”, may not have been cardiac in origin. In other words, this case may have served Laennec as evidence against the coronary ossification theory, a justified opinion; however, in using the particular case of Millot, the evidence may not have been valid.

Laennec had these reservations about the coronary theory at least seven years before his discovery of auscultation, and his opinion remained unchanged until his death.<sup>46</sup> He described angina as a nervous “lesionless” disease, or “*névrose*” localized in the heart, that bore no constant relationship to the coronary arteries or the myocardium. For a while, he believed himself to be afflicted with angina, as well as other illnesses that he considered to be of a psychic nature, such as asthma, hypochondria, and gout.<sup>47</sup> Later, he may have perceived the lack of definite stethoscopic findings in this disease as more support for this opinion. For him, persistence of normal heart sounds implied the absence of organic change.

### *Ætiology*

With most of his contemporaries, Laennec did not think of mechanical derangements as the only cause of organic changes in the heart. Valvular stenosis, even if it could be diagnosed in the living, was poorly understood and rarely seen as a primary alteration. Valvular insufficiency, on the other hand, was not recognized until 1831.<sup>48</sup> The “mechanical” obstruction posed by persistent elevation of systemic blood pressure, perhaps one of the most common causes of circulatory strain, was undetectable at the bedside, invisible in the cadaver, and as yet had no place in medical patho-physiology.

A student steeped in the sensualist philosophy of the post-Revolutionary Paris school and familiar with ideas of Cabanis,<sup>49</sup> Laennec avoided discussing the causes of any disease. Nevertheless, he did provide a discussion of the *possible* causes of organic heart disease in both editions of his book. Corvisart had suggested that all valvular lesions were due to syphilis, but Laennec thought that other processes were probably involved.<sup>50</sup> He denied the essential role of any form of inflammation in the production of valvular change, a stance he adopted in many other areas of medicine.<sup>51</sup> This position may have stemmed from his opposition to François-Joseph-Victor Broussais (1772–1838), whose emphasis on irritation and inflammation in the

<sup>46</sup> Laennec, op. cit., note 3 above, 1826, vol. 2, pp. 487–8, 745–52.

<sup>47</sup> Alfred Rouxau, *Laennec after 1806*, Paris, Baillière, 1920, facsimile repr., Quimper, Cornouaille, 1978, vol. 2, pp. 82–4, 171–4, esp. p. 174.

<sup>48</sup> Credit for the first recognition of valvular insufficiency is usually given to James Hope, *Treatise of diseases of the heart*, London, Kidd, 1831.

<sup>49</sup> P. J. G. Cabanis, *Du degré de certitude de la médecine*, in *Oeuvres philosophiques*, ed. Claude Lehec and Jean Cazeneuve, Paris, Presses Universitaires de France, 1956, pp. 58–64. On Cabanis and his influence on early nineteenth-century French medical aetiology, see E. H. Ackerknecht, *Medicine at the Paris hospital 1794–1840*, Baltimore, Johns Hopkins University Press, 1967, pp. 3–12; and Martin Staum, *Cabanis, Enlightenment and medical philosophy in the French Revolution*, Princeton University Press, 1980, pp. 104–7.

<sup>50</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 255, 335; 1826, vol. 2, pp. 494, 619–20.

<sup>51</sup> This reservation led Laennec to keep pleurisy and pleural effusions out of his clinical description of tuberculosis: pleurisy was clearly an inflammatory condition; tubercles were not. He seems to have arrived at the concept of accidental production to avoid having to speculate on causes of organic lesions with no immediately obvious aetiology. In this category, he included cancer, benign tumours, and tubercles. See

production of virtually all diseases was itself a major irritation to Laennec.<sup>52</sup> He admitted only that valvular alterations might be secondary to long-standing chronic illness and could themselves cause other organic changes, such as dropsy in the legs, oedema in the lungs, and haemorrhages.<sup>53</sup>

Laennec also criticized Corvisart's emphasis on coarctation of the aorta as a frequent cause of heart disease, because the condition was rare.<sup>54</sup> Comparing a subject's heart size to that of his fist, Laennec endorsed Allan Burns's notion of a heart too small for the body and suggested it as a reason for an individual's susceptibility to heart disease.<sup>55</sup> He also offered the following blend of psychology and physiology as an explanation for organic change:

The energetic and frequently repeated action of all muscles causes them to increase in volume . . . as the arm of a soldier or the hands of labourers . . . As a result, one realizes that palpitations, even if they are only of nervous or emotional origin, could lead to a true augmentation in the substance ("nutrition") of the heart, if they occur too often.<sup>56</sup>

John Forbes, Laennec's outspoken translator, criticized him for not placing enough emphasis on the emotional and nervous causes of heart disease.<sup>57</sup>

### Treatment

In the early nineteenth century, organic heart diseases were considered to be incurable, but this did not preclude their treatment and Laennec was far from being a therapeutic nihilist. In 1819, he recommended blood-letting by leeches or venesection for the complications of swelling and dyspnoea. He found that the heart sounds and murmurs were altered by bleeding and concluded that the stethoscope would be a useful means of controlling the beneficial effects.<sup>58</sup> In 1826, he modified his therapeutic recommendations to include dietary measures and digitalis, but of the latter he was uncertain. "Its effect [as a diuretic] has never been obvious to me . . . even when the dose was increased to the point of causing vomiting and vertigo . . . in short I can only consider it to be an heroic measure."<sup>59</sup> In using these high doses, a practice reminiscent

---

Laennec's article, 'Anatomie pathologique', in C. Panckoucke, (editor), *Dictionnaire des sciences médicales*, Paris, 1812, 2: 46–61. Also Laennec, op. cit., note 3 above, 1819, vol. 1, pp. 18–40 and *idem*, op. cit., note 3 above, 1826, vol. 1, pp. 577–80. For more on the derivation of Laennec's classification and his concepts of disease see Jacalyn M. Duffin, 'The medical philosophy of R. T. H. Laennec (1781–1826)', *Hist. Phil. Life Sci.*, 1986, 8: 195–219.

<sup>52</sup> Laennec often attacked Broussais for his aetiological theory of disease by inflammation. See op. cit., note 3 above, 1826, vol. 1, pp. xx–xxxii, 538–9, 598–603 and Collège de France lecture 10, 1822–23 MS. 2186(IV), f. 81v. and lecture 35, 1823–24, MS. Cl. 2 lot a (B), f. 280v.

<sup>53</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 251–4; 308–24, 335–53; 1826, vol. 2, 490–4, 572–87.

<sup>54</sup> *Ibid.*, 1819, vol. 2, p. 256; 1826, vol. 2, pp. 495–6.

<sup>55</sup> *Ibid.*, 1819, vol. 2, pp. 256–7; 1826, vol. 2, pp. 496–7.

<sup>56</sup> *Ibid.*

<sup>57</sup> John Forbes, in Laennec, op. cit., note 27 above, pp. 583–4n. The link between emotion and cardiac muscle in the patho-physiology of heart disease was still firmly entrenched in the later medical thought: see Joel D. Howell, "'Soldier's heart': the redefinition of heart disease and speciality formation in early twentieth-century Britain", in W. F. Bynum, C. Lawrence, and Vivian Nutton, (editors), *The emergence of modern cardiology*, *Medical History* Supplement No. 5, London, Wellcome Institute of the History of Medicine, 1985, pp. 34–52.

<sup>58</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 240–1.

<sup>59</sup> *Ibid.*, 1826, vol. 2, p. 735.

of his controversial experiments with tartar emetic,<sup>60</sup> Laennec disregarded the original cautions of William Withering (1741–99)<sup>61</sup> and seemed to accept the unpleasant side effects as an inevitable part of treatment. A testament to the effects of this vigorous therapy is the sad account of Marianne Viccia, a 24-year-old woman admitted to hospital 22 March 1822. She suffered epigastric pains, nausea and vomiting, which vanished “when one stopped the digitalis and reappeared when it was restarted”.<sup>62</sup>

#### *Diagnosis*

Laennec recognized the general symptoms of heart disease: dyspnoea on exertion, orthopnoea, dependent oedema, anasarca, and palpitations. Unlike his British colleagues, he rarely timed the pulse, but he did notice quality and rhythm and startling variations in rate. He was able to say he had seen a pulse less than fifty beats per minute without symptoms, but he set less store by these examinations than had Corvisart.<sup>63</sup> Attributing the original description of swollen jugular vein to Giovanni Maria Lancisi (1654–1720), Laennec resurrected this sign as a valuable indicator of an enlarged (but hypertrophic rather than dilated) right heart.<sup>64</sup> He claimed that Corvisart had thought this was an unreliable sign of passive aneurysm of the right heart; yet, while he was still Corvisart’s student, Laennec’s attention had been drawn to pronounced beating in the jugular vein of a patient with precisely that pathological finding.<sup>65</sup> Laennec thought that percussion was less useful in the examination of the heart than it was in examination of the chest, but he relied heavily on the value of pre-cordial palpation: exaggerated impulse as a sign of enlargement; thrill as a sign of valvular disease.

#### *Stethoscopic signs*

Laennec described the diagnostic physical signs as a combination of alterations in all modalities of assessing the patient’s condition, including observation and palpation. He maintained that auscultation had merely heightened the utility of other methods, especially percussion,<sup>66</sup> but there is no doubt that he was very impressed with his stethoscope to the point of deserving the epithet “cylindromaniac”.<sup>67</sup> In the following presentation of Laennec’s auscultatory signs of the normal and diseased heart, frequent reference will be made to present-day cardiac concepts. Although these references may be criticized as “presentist”, they are not meant to exclude or intimidate the non-physician and can easily be skipped without losing the train of the argument. Their inclusion is intended as an attempt at critical judgement of Laennec’s

<sup>60</sup> In the second edition, Laennec dwelt at length on his experience with high doses of tartar emetic: *ibid.*, vol. 1, pp. 492–516. It may have been his experience with this hardy therapy that led him to remove the first edition’s reference to bleeding as potentially “the most harmful” of treatments: *ibid.*, 1819, vol. 2, p. 241.

<sup>61</sup> William Withering, ‘An account of the foxglove’ (1785), in *Classics of cardiology*, 2 vols., ed. Fredrick A. Willius and Thomas E. Keys, New York, Henry Schuman and Dover, 1941, vol. 1, pp. 238–9.

<sup>62</sup> Laennec, *MS. Cl. II*, f. 161 r.–v.

<sup>63</sup> Laennec, *op. cit.*, note 3 above, 1819, vol. 2, pp. 220, 237–40; 1826, vol. 2, pp. 476, 492.

<sup>64</sup> *Ibid.*, 1819, vol. 2, p. 268; 1826, vol. 2, p. 504.

<sup>65</sup> Laennec, *op. cit.*, note 40 above, p. 302.

<sup>66</sup> R. Laennec, *Notice des faits nouveaux obtenus par suite des recherches de M. Laennec*, Paris, Feugueray, 1826, p. 2.

<sup>67</sup> Evan Bedford, ‘Cardiology in the days of Laennec’, *Brit. Heart J.*, 1972, **34**: 1195.

observations and as acknowledgement of the need, too often overlooked in contemporary medical history, “to recognize qualitatively better or worse science”.<sup>68</sup>

*Stethoscopic signs: normal heart sounds*

The normal heart beat consists of two sounds: the first is synchronous with closure of the atrio-ventricular valves (i.e., mitral and tricuspid); the second, with the closure of the ventriculo-arterial valves, which Laennec called “sigmoid” (i.e., aortic and pulmonic). The exact physical cause of the sounds can be debated, but the synchrony with valve closure is well established.<sup>69</sup> Laennec heard the first sound at the same time as he felt the rise in the carotid pulsation and the palpable apex beat, and he noticed that the point of maximal loudness was over the apex of the heart. From these two observations, he concluded that this first sound was due to the contraction of the ventricles. He then assumed that the second sound must be due to the contraction of the atria; and supported this assumption with the accurate observation that it was loudest high on the sternum, and the erroneous conclusion that the ventricular contraction was audible.<sup>70</sup>

Using this interpretation of the heart sounds, his extensive knowledge of anatomy, and repeated physical examinations, Laennec then tried to determine the best site on the chest to hear the “contraction” of each chamber and to delineate the boundaries of the normal audible beat. He then established a list of sites to which augmented sounds would progress and from which diminishing sounds would recede: left side of chest to axilla and the stomach; right side of chest to axilla; left back; right back.<sup>71</sup>

Further support for his erroneous conclusion that myocardial contraction was audible came from Laennec’s extrapolation from his research on auscultation of contracting muscle. He believed that the contraction of skeletal muscle was an audible phenomenon and concluded that the heart, being a similar muscle, would also produce audible contractions. There is some controversy over whether or not auscultation of contracting skeletal muscle produces any sound. Whatever sound may be heard is attributed not to the muscular action, but to secondary circulatory changes or friction between tissue planes. After the publication of his first edition, Laennec performed experiments on the auscultation of muscle contraction during his retirement in Brittany, from 1819 to 1822. Taking advantage of any clinical opportunity, he listened to the muscle action of patients with tetanus and of a woman with “*catalépsie*” and he tried to distinguish on an acoustic basis between contractions resulting in movement and what he called the “*force de situation fixe de Barthez*” (isotonic contraction).<sup>72</sup> In

<sup>68</sup> Frederic Lawrence Holmes, *Lavoisier and the chemistry of life: an exploration of scientific creativity*, Madison, University of Wisconsin Press, 1985, p. xvii.

<sup>69</sup> The statements in this essay concerning cardiac auscultation have been confirmed by one or all of the following: Aldo A. Luisada, *The sounds of the diseased heart*, St. Louis, Warren H. Green, 1973; Aldo A. Luisada and Francesco Portaluppi, *The heart sounds: new facts and their clinical interpretation*, New York, Praeger, 1982; Abe Ravin, *Auscultation of the heart*, second ed., Chicago, Year Book Medical Publishers, 1967; Robert S. Winwood, *Essentials of clinical diagnosis*, London, Edward Arnold, 1981.

<sup>70</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 216–17; 1826, vol. 2, pp. 432–9.

<sup>71</sup> *Ibid.*, 1819, vol. 2, pp. 199–200; 1826, vol. 2, p. 387.

<sup>72</sup> Laennec, 1826, vol. 2, pp. 429–48; Laennec read a paper on these experiments at the Académie Royale de Médecine on 19 April 1825. Alfred Rouxeau, op. cit., note 47 above, vol. 2, pp. 307–8. Laennec referred to W. H. Wollaston’s lecture, ‘On the duration of muscular action’, *Phil. Trans.*, 1810, 100: 2–5.

this presentation, he cited the work of “Blaud of Beaucaire”, “Erman of Berlin”, and W. H. Wollaston of London, but there is no evidence in the scientific manuscripts that Laennec was aware of the work of his contemporaries Félix Savart (1791–1841) or Jean-Louis-Marie Poiseuille (1779–1869) on turbulence and sound.<sup>73</sup>

Laennec corresponded with Erman on the possibility of timing the variable contractions of muscle fibres in order to develop an auscultatory assessment of the strength of muscle action.<sup>74</sup> According to Laennec, this project failed because he was unable to count more than seven or eight discrete noises per second, even though his ear could distinguish many more. Nevertheless, he made detailed analyses of the work of the heart based on an attempt to time the duration of the sounds. Although the heart seemed to be in perpetual motion, he concluded that in a twenty-four-hour period the ventricles were actually at rest for twelve hours and the atria for eighteen.<sup>75</sup> These observations, made with stethoscope and watch, without the help of the direct vision or precise timing which would later be provided with electrical information or kymographic display, are remarkable for their relative accuracy. He applied the same blend of observation and analysis to sounds heard in a carotid artery to produce the most disarming pages in his book: a musical description of carotid bruits complete with staff, notes, slurs, and clef.<sup>76</sup> Laennec was an enthusiastic witness to several experiments performed by his English friend, David Barry (1780–1836), on the effects of atmospheric pressure on the cardiovascular physiology of the horse and the dog.<sup>77</sup>

Having defined rest periods of the heart chambers, Laennec suggested that Albrecht von Haller (1708–77) had overlooked the rest period after the contraction of the atria.<sup>78</sup> This has been cited as a misinterpretation of Haller, who had adopted William Harvey’s idea that the atria beat first and the ventricles, second.<sup>79</sup> In fact, Laennec’s so-called “reversal” of the order of the ventricular and atrial contraction has led to the assumption that he was ignorant of Harvey’s *De motu cordis* (1628), in which contraction of the atrium was clearly stated to precede that of the ventricle. Laennec was well aware of Harvey’s work and from it adopted the notion that the apex beat represented ventricular contraction.<sup>80</sup> Sénac had cited Harvey frequently, but placed

<sup>73</sup> On Savart and Poiseuille, see Victor A. McKusick and H. Kenneth Wiskind, ‘Félix Savart (1791–1841), physician-physicist: early studies pertinent to the understanding of murmurs’, *J. Hist. Med.*, 1959, 14: 411–23; *ibidem*, ‘Osborne Reynolds of Manchester: contributions of an engineer to the understanding of cardiovascular sound’, *Bull. Hist. Med.*, 1959, 33: 124.

<sup>74</sup> *Ibid.*, p. 430–9. The reference Laennec gave to Erman’s work was Gilbert’s *Annalen für Physick*, 1812, 1: 19. This article later attracted the attention of C. J. B. Williams and R. B. Todd. Laennec’s correspondence with Erman does not appear to have been kept with his scientific manuscripts.

<sup>75</sup> Laennec, *op. cit.*, note 3 above, 1826, vol. 2, pp. 405–8.

<sup>76</sup> *Ibid.*, 1826, vol. 2, pp. 424, 426, 433.

<sup>77</sup> *Ibid.*, pp. 415–20; Laennec, ‘Rapport sur les expériences de M. Barry’, *Archs gén. Méd.*, third year, 1825, 9: 605–8; David Barry, *Recherches expérimentales sur la cause du mouvement du sang*, Paris, 1825, pp. 56–9. Also see David Barry, *Discours pour le passage du sang à travers le coeur*, thèse méd., Paris, Didot, 1827, no. 117, p. 5.

<sup>78</sup> Laennec, *op. cit.*, note 3 above, 1819, vol. 2, pp. 217–18; 1826, vol. 2, p. 406.

<sup>79</sup> Roger Rullière, ‘Laennec, cardiologue: le bon grain et l’ivraie’, *Rev. du Palais de la Découverte*, no. spéciale 22, 1981, p. 136; Bedford, *op. cit.*, note 67 above, p. 1194. The passage Laennec is said to have misunderstood can be found in Albrecht von Haller, *First lines of physiology* (1786), trans. William Cullen, repr. New York, London, Johnson Reprint Corp., 1966, pp. 67–8.

<sup>80</sup> William Harvey, *De motu cordis* (1628), and English trans. Kenneth J. Franklin, Springfield, Charles C Thomas, 1957, pp. 26, 135. Laennec cited this book: *op. cit.*, note 3 above, 1826, vol. 2, p. 420.

no special emphasis on the sequence of chamber contraction. Perhaps as a result of his reading of Sénac, Laennec may not have thought the order important. Yet, both his “reversal” of the sequence of contraction and his ignorance of Harvey may be more apparent than real as the following discussion should demonstrate.

If every ventricular beat is preceded by an atrial beat, it must also be said that every ventricular beat is also *followed* by an atrial beat, albeit displaced considerably in time. Laennec might not have thought his description of the heart sounds to be inconsistent with the work of Harvey. It could have been the entire period of ventricular diastole that he meant by “the rest period after atrial contraction overlooked by Haller”. Support for this possibility can be found in the work of William Stokes (1804–78), who published a small treatise on auscultation in 1825.<sup>81</sup> Stokes adopted Laennec’s interpretation of the heart sounds and was the first to use the terminology of “first” and “second” sounds; however, his ordinal nomenclature was the direct opposite of the conventional. Stokes’s first heart sound was the one now called “second”, and vice versa. Why did Stokes create this curious grouping of the sounds giving a longer pause between the two components of each heartbeat (first atrial contraction, then ventricular contraction) than between the end of one beat and the start of the next? This may have been an attempt to reconcile the plausible observations of Laennec, including the rest period “overlooked by Haller”, with the undisputed work of Harvey. Because the heart sounds are not muscular in origin, Laennec’s “reversal” of the order of contraction is neither corrected nor improved by simply reversing his nomenclature, i.e. naming the first sound “atrial” and the second, “ventricular”. In fact, such terminology corresponds less well to the events observed.

Laennec’s failure to interpret the heart sounds correctly has astounded some historians of medicine; yet only rarely have they tried to explain his conclusions. It has been suggested that his errors were due to an over-emphasis of pathological anatomy and a relative indifference to physiology.<sup>82</sup> There is abundant evidence here to refute such a contention and Laennec would have refuted it too. He considered himself to be an active participant in the study of the living subject, since most of the sounds he described, such as breath sounds, rales, pectoriloquy, the heart beat and murmurs, were absolutely dependent on the co-operation of the living patient, and non-existent in the cadaver.<sup>83</sup>

#### *Stethoscopic signs: Abnormal sounds*

Corvisart had recognized the difficulty of separating pulmonary from cardiac diseases using palpation and percussion alone.<sup>84</sup> With the invention of the stethoscope, Laennec had partially solved this problem simply by improving the distinction of respiratory diseases from heart disease, and of individual lung diseases from one another. In turning his attention to the clinical delineation of the different

<sup>81</sup> W. Stokes, *An introduction to the use of the stethoscope*, Edinburgh, MacLaglan and Stewart, 1825, pp. 137–9.

<sup>82</sup> Rullière, *op. cit.*, note 79 above, p. 133; McKusick, *op. cit.*, note 2 above, p. 137.

<sup>83</sup> In presenting his classification of disease at the Collège de France in 1822, Laennec said, “Put aside metaphysics, we follow the physiologists exclusively.” MS. 2186(IV), f. 13v.

<sup>84</sup> Corvisart, *op. cit.*, note 18 above, pp. 315–24.

cardiac diseases, Laennec was convinced he was on to something extremely important and useful. With auscultation, he hoped to reduce the subjective aspects of physical signs: no longer would the physician be dependent solely on his own interpretation of a patient's account of subjective feelings; no longer would the signs of organic change be polyvalent symptoms that could occur in many different illnesses. The sounds provided by the stethoscope were produced directly by the organic change itself. Once their significance had been clarified by careful autopsy correlation, they would stand as universally valid, objective indicators of internal anatomical change. As his word *stethoscope* implied, Laennec intended the observer to *see* within the thorax by *hearing*. Auscultatory sounds were a capital endorsement of the desiderata of the sensualist Ideologue physicians: they were based on acute sensory observation; they minimized intervening theorizing.

Laennec's cardiology chapters and the thesis of his student, Adolphe Toulmouche, are full of enthusiasm for the advantages of auscultation. Phrases like "the only sure sign" and "the only constant and truly pathognomonic sign" were all, of course, applied to stethoscopic findings which were to replace the "equivocal symptoms".<sup>85</sup> In fact, where Laennec used these superlative modifiers for the lucidity of his own technique, he indulged in some of the muddiest prose in his book. For example, he wrote that "the signs [of right ventricular hypertrophy] are exactly the same as those for that of the left except that the ventricular beat is less soft;"<sup>86</sup> and then, "the signs [of biventricular hypertrophy] consist in a unification of the signs of hypertrophy of each ventricle, but with an almost constant predominance of those of the right".<sup>87</sup> The only explanation of "those [signs] of the right" in the second citation was the unrevealing statement in the first citation—a meaningless circle of words! Such confused phrases made excellent fodder for his detractors who, led by Broussais, complained, not inappropriately, of "the over-abundance of detail, and excessive nuances of perception".<sup>88</sup>

#### *Stethoscopic signs: Murmurs*

Diseased heart valves are often noisy because of turbulence in blood flow across their irregular tissues. Their pathological changes are usually quite distinct. Laennec did hear and describe murmurs, but he was not certain what they signified. At first, he stated that murmurs represented diseased valves, but since he thought ossifications and excrescences on the valves were rare, he devoted very little space to the subject.<sup>89</sup> In the second edition, he wrote that murmurs also indicated spasm, or prolonged contraction of a cardiac chamber, and he added a long discussion concerning abnormal sounds in a definite modification, if not a retraction, of his original stance.<sup>90</sup> In 1819, he tried to base distinctions between the potentially associated organic lesions on the quality of

<sup>85</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 268–70; 1826, vol. 2, p. 435. Adolphe Toulmouche, *Considérations sur les signes diagnostiques des maladies du coeur*, thèse méd. Paris, 1820, p. 30.

<sup>86</sup> Laennec, op. cit., note 3 above, 1826, vol. 2, p. 506.

<sup>87</sup> *Ibid.*, p. 507.

<sup>88</sup> F. J. V. Broussais, *Examen des doctrines médicales et des systèmes de nosologie*, second ed., 2 vols., Paris, Mequignon-Marvis, 1821, vol. 2, p. 751.

<sup>89</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 214–5, 308–24.

<sup>90</sup> *Ibid.*, 1826, vol. 2, pp. 421–57, 572–87, 755–6.



the murmur. For example, he associated the file sound (“*bruit du lime*”) with valvular ossification,<sup>91</sup> but he also described a “muffled noise” synchronous with the second sound that, for him, represented cardiac enlargement.<sup>92</sup> This latter noise, which occurs in stenosis of the mitral valve, is due to turbulence in blood flow at the altered mitral orifice, but Laennec ascribed it to hypertrophy of the atria because it seemed to represent a prolongation of the atrial contraction (the second sound). Indeed, left atrial hypertrophy does occur in mitral stenosis as a result of the altered haemodynamics, but only as a secondary change.

It has been said that Laennec’s thinking on the subject of murmurs “deteriorated” in the years between his two editions.<sup>93</sup> In 1819, he denied that any murmur was associated with ossification of the aortic valve, but he did accept murmur as a sign of mitral valve disease: “ossification of the mitral valve can be recognized with the cylinder by the following signs: the auricular sound becomes much longer and more muffled and is slightly choked and brusque, reminiscent of file sound on wood; sometimes this sound is similar to that of a bellows closed abruptly.”<sup>94</sup> However, four years later at the Collège de France, he retracted this statement “[there are] many varieties [of murmurs], file, grate, bellows, saw . . . [they] indicate spasm only. I used to believe they indicated a blockage, but [it is] obvious from exper[ience or experiment?] that [this is] not so”.<sup>95</sup> Nevertheless, in the same lecture, he spoke of Corvisart’s old sign of pre-cordial thrill, which he labelled “*fremissement cataire*” or cat-like purring, as a reliable, but questionably sensitive, sign of ossification of the valves that “never” appeared without a murmur.

In 1824, Victor Collin, Laennec’s junior colleague at the Necker Hospital, provided the details of some of the experiments that had caused Laennec to revise his opinion:

[The bellows sound or murmur] occurs in nervous individuals, hysterics, hypochondriacs, in the presence of haemorrhage often without any change in the structure or function of the heart. . . . At autopsy no consistent organic changes are found . . . Monsieur Laennec sees it as the sign of a simple spasm in the circulatory system. Several observations support this opinion:

1. its analogy with forced muscular contraction . . . [as heard] if one places the elbow on a table, the hand on the ear and repeatedly contracts the jaw.
2. the ease with which it appears upon compression of the arteries of healthy people.
3. its existence over arteries delivering blood to a haemorrhage.
4. its existence in the palpitations produced by anaemias.<sup>96</sup>

Collin also reaffirmed the reliability of the grating or file murmur as a sign of valvular narrowing:

M. Laennec regards this as a reliable sign of valvular narrowing by ossification, vegetation or any other cause. The site and timing of the contractions in which it is

<sup>91</sup> Ibid., 1819, vol. 2, pp. 215, 316.

<sup>92</sup> Ibid., 1819, vol. 2, p. 213.

<sup>93</sup> Rullière, op. cit., note 79 above, p. 134.

<sup>94</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, p. 136.

<sup>95</sup> Laennec, MS. Cl. 2 lot a(B), f. 308v. Many of the lecture manuscripts were written in point form with abbreviations. In translating these notes, I have completed certain words, but have made no attempt to restore the fragmentary phrases to prose.

<sup>96</sup> Victor Collin, *Les diverses méthodes d'exploration de la poitrine*, Paris, Baillière, 1824, p. 61.

heard indicates which orifice is affected. The possibility of detecting a very similar sound in an individual with the bellows sound by compressing an artery . . . seems to suggest it is only a modification of the latter due to a more pronounced spasm, maintained and caused by an additional and equally resistant obstacle.<sup>97</sup>

In short, the file sound may have been a reliable indicator of valvular change, but it was produced by accompanying *spasm* in the muscular chamber upstream to a lesion.

Two years later, Laennec did not cite Collin, but he included the example of the contracting jaw muscles. He even went so far as to cast suspicion on the value of pre-cordial thrill and its associated murmur as a sign of organic change:

These sounds are remarkable . . . they are the only auscultatory sounds that are not related to any organic lesion in which one can find their cause . . . The bellows sound is the result of spasm and does not imply any organic lesion in the heart or its arteries . . . It seems extremely likely that the thrill is due to a specific modification of innervation . . . [These sounds] occur in young hypochondriacs, . . . in those with fever and . . . above all in those with palpitations of purely nervous origin.<sup>98</sup>

Having originally considered murmurs to be signs of valvular disease, Laennec abandoned them all as signs of organic change. This stance was far from being unjustified. Non-organic or “functional” murmurs are extremely common, and occur without valve lesions in such hyperdynamic conditions as anaemia, fever, pregnancy, and thyrotoxicosis. Arterial bruits can also occur with anomalies in physical architecture that are of no pathological significance, as Collin’s experiments had demonstrated. Given the high incidence of tuberculosis and its common manifestations of fever and anaemia, it is probable that Laennec did hear murmurs in these seemingly “lesionless” states. Certainly the list of observations provided by Collin supports this contention. Laennec’s familiarity with the findings of his friend Jean Le Jumeau de Kergaradec (1788–1877) concerning the murmur overlying the normal human placenta probably further clouded the apparent significance of all murmurs.<sup>99</sup>

#### *Stethoscopic signs: Friction rub*

Pericarditis produces a characteristic sound called a “rub” (“*frottement*”), like creaking leather, which is probably due to friction between the inflamed surfaces of the membrane surrounding the heart. Laennec’s associate Victor Collin first described this sound in a case-history of pericarditis, in 1824.<sup>100</sup> Laennec may never have heard the sound, or if he did, it occurred so rarely prior to the more common autopsy finding of pericarditis that he completely denied its value. He did not name Collin and mentioned the sign only to reject it. “I thought for a while that this sound could be a sign of pericarditis”, he wrote, “but I have been convinced since that it is not.”<sup>101</sup> Collin’s hesitant description was confined to two patients, only one of whom

<sup>97</sup> *Ibid.*, p. 63.

<sup>98</sup> Laennec, *op. cit.*, note 3 above, 1826, vol. 2, pp. 421, 443, 453, 763–4.

<sup>99</sup> *Ibid.*, pp. 457–66.

<sup>100</sup> Collin, *op. cit.*, note 96 above, pp. 64–5.

<sup>101</sup> Laennec, *op. cit.*, note 3 above, 1826, vol. 2, p. 446.

had he personally examined during life without an autopsy to confirm his clinical impression. The second case, provided by an intern at the Hôpital St Antoine who had also examined the first, displayed the identical rubbing sound and was found to have pericarditis on post-mortem. It is possible that Laennec found the evidence of only one confirmed case, which he had not seen personally, too tenuous for inclusion in his treatise.<sup>102</sup>

Pericarditis is a complication of tuberculosis. It is impossible to imagine that Laennec did not encounter this change quite frequently at the bedside and in the morgue. It is possible, however, that he discovered the lesion at autopsy far more frequently than he heard the friction rub in the clinic. Absence of the friction rub is usual in chronic calcific pericarditis, which may complicate tuberculosis, and in advanced states of pericarditis, called tamponade, when effusion separates the membranes and prevents the physical production of the sound.

*Stethoscopic signs: Hypertrophy and dilatation*

Laennec, like Corvisart and Burns, was far more interested in the conditions of hypertrophy and dilatation than he was in the state of the valves or arteries. Since these changes were the most obvious and seemingly the most common anatomopathological changes, they dominated his anatomico-clinical research on the heart. Useful diagnostic signs would be those that could reliably and precisely indicate the status of cardiac muscle. Signs of valvular disease, coronary disease, and pericarditis were less important. From a modern perspective, hypertrophy and dilatation are only very rarely the primary alterations even if they are the most obvious findings at autopsy. Usually these alterations are secondary to some other prior pathological change, be it anatomical (such as valvular lesions, lung disease, and infarct) or metabolic (such as uraemia, toxæmia, and hypertension). Laennec heard sounds that could be considered diagnostic of these primary conditions, but, as with the file murmur, he always tried to associate them with the secondary changes in the myocardium.

The auscultatory signs of altered myocardium were described in the same terms in both editions of his book (see table 2). In fact the only changes in this section were the careful correction of the word “*cylindre*” to “*stethoscope*” and the sometimes hostile, sometimes complimentary references to R. J. Bertin’s book of 1824, on diseases of the heart.<sup>103</sup> If Laennec’s interpretation is divorced from his description, these signs can be summarized in the following manner: distinct heart sounds implied dilatation of the ventricles; silent or absent sounds implied hypertrophy. In laying down the stethoscopic signs of hypertrophy and dilatation, Laennec relied on the site of the

<sup>102</sup> There is evidence that Laennec and Collin did not always see eye-to-eye on the matter of teaching or popularizing auscultation. In a not-so-subtle criticism of Laennec’s lengthy verbal descriptions, Collin advised that a stethoscopist should “avoid tedious detail and infinite subdivisions, convinced that the many nuances belonging to the major stethoscopic sounds can be appreciated only by attentive and repeated observation and that even a very long description gives only an incomplete idea of their nature.” Op. cit., note 96 above, p. 2.

<sup>103</sup> As Laennec himself declared, his hostility was directed not so much against Bertin as against his editor, Bouillaud, for his Broussais-like theories concerning inflammation. Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 258–85; 1826, vol. 2, pp. 497–531, esp. 538–9n. See also MS. Cl. 2 lot a(B), f. 307r. R. J. Bertin, *Traité des maladies et les gros vaisseaux*, ed. Jean-Baptiste Bouillaud, Paris, Baillière, 1824.

TABLE 2: SUMMARY OF LAENNEC'S CRITERIA FOR THE DIAGNOSIS OF MYOCARDIAL HYPERTROPHY AND DILATATION

<i>Lesion</i>	<i>Impulse</i>	<i>Percussion</i>	<i>First sound</i>	<i>Second sound</i>	<i>Other</i>
LVH*	great	dullness	soft, prolonged in proportion to degree of hypertrophy	soft, short	pulse increased
RVH*	great, lower sternum		soft, less than in LVH	soft	jugular distention
LVDIL	decreased		clear, loud	loud	soft, weak
RVDIL		unreliable	loud, lower sternum	loud	jugular distention

\* The signs of biventricular hypertrophy were a combination of LVH and RVH.

KEY: R = right; L = left; V = ventricular; H = hypertrophy; DIL = dilatation

audible sounds and its extension to unusual locations. The palpable impulse of the heart on the chest wall was also an important consideration. Sometimes he seems to have included not only the sound, but also the impulse carried to the side of the examiner's head by the stethoscope.<sup>104</sup> He assumed that variations in the quality and intensity of the heart sounds reflected organic changes in the myocardium, rather than in the other components of the circulatory system. This assumption may have been fostered by his musical literacy, which encouraged him to explore the relationship of loudness to strength. Its justification came from his work on skeletal muscle. The final criteria established by Laennec imply an inverse acoustic translation of increased muscle bulk. The origin and potential accuracy of these signs will be assessed in the three detailed case-histories presented below.

#### CASES

Despite the superlative adjectives he used, Laennec seems to have had some uncertainty about his signs. He cautioned his reader not to place too much confidence in the stethoscope and uncharacteristically dwelt on the value of other signs.<sup>105</sup> In his four years at the Collège de France, he devoted only ten out of a total of 161 lectures to the subject of "*les maladies cardiaques*".<sup>106</sup> The drafts of all but one of these ten have been lost or destroyed for an unknown reason, at an undetermined date, by an

<sup>104</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, pp. 206–9; 1826, vol. 2, pp. 394–7.

<sup>105</sup> Laennec, op. cit., note 3 above, 1819, vol. 2, p. 274–5; 1826, vol. 2, pp. 517–8.

<sup>106</sup> Collège de France lectures 44 and 45, 1823–24, MS. Cl. 2, lot a(B), f. 306–308v. Laennec appears to have repeated a two-year cycle of lectures first given in 1822–4; therefore, the ten lessons were probably repeated at some time between 1824 and 1826.

unidentified hand. This is an exceptional omission in an otherwise complete collection, and it may hint at an attempt by Laennec's friends to suppress his less successful ideas.<sup>107</sup> Perhaps more astonishing than the paucity and disappearance of the Collège de France lectures are the poverty of case examples in the published chapters on the heart, and the fact that these cases scarcely change between the two editions. In several instances they even fail to support Laennec's contentions.

In the 1819 edition of Laennec's book, out of a total of fifty case observations only six concern the heart and great vessels: Ponsard, Potel, Villeneuve, Lefebvre, an unidentified 35-year-old labourer and Millet.<sup>108</sup> Of these, four had been examined with the stethoscope, another four had been examined by autopsy, but in only two was any correlation made between the stethoscopic signs and the post-mortem findings. Lefebvre, who had died in 1803 prior to the invention of the stethoscope, was selected because of the spectacular post-mortem findings on the valves. No mention was made of auscultation in the case of Millet, who died in May 1817 of a dissecting aortic aneurysm with left ventricular hypertrophy. Villeneuve died in 1819, but auscultation had done little to elucidate his pericarditis. His case history too was selected for the autopsy results. Potel, a 39-year-old woman who died in early December 1817, was found to have loud heart sounds which were correlated with the autopsy findings of pulmonary tuberculosis, myocardial dilatation, and an enlarged liver. The 35-year-old labourer, who had died in April 1819, had a murmur corresponding to mitral valve insufficiency, which had not yet been recognized as a patho-physiological entity. His case was used to illustrate the diagnosis of ventricular hypertrophy by variation in the intensity of the heart sounds. Ponsard was still alive in 1819. Obviously, his case could offer no pathological proof of the presumed meaning of his very loud murmurs, which were attributed to diseased valves.

In the 1826 edition, out of fifty-one observations there were again six cardiac cases: Ponsard, Potel, Lefebvre, the unidentified 35-year-old labourer, Millet and Dirichard.<sup>109</sup> In fact, the apparently new case of Dirichard had already been published in the 1819 edition, in the section on lung diseases.<sup>110</sup> In other words, Laennec made no new contributions to his chapter on heart disease, although he revised some of his conclusions. He removed the cross-references made in 1819 to five cases elsewhere in the text where he had unabashedly admitted that the findings seemed to contradict his conclusions.<sup>111</sup> There was an update on the still-living Ponsard who had returned to visit "*le docteur Laennec*" in 1822. Despite the justifications offered earlier in this essay for Laennec's eventual rejection of the murmurs as signs of valvular disease, it must be said that both the 35-year-old labourer and the young Ponsard, still without autopsy and still with his loud heart murmurs, were left as case examples of mitral valve disease!

<sup>107</sup> For a discussion of Laennec's conflict with organicism, the view that every disease could be associated with organic change, see Jacalyn M. Duffin, 'Vitalism and organicism in the philosophy of R. T. H. Laennec' *Bull. Hist. Med.*, Dec. 1988, 62.

<sup>108</sup> Laennec, observations XLV-L, *op. cit.*, note 3 above, 1819, vol. 2, pp. 321-4, 337-44, 346-53, 382-91, 398-403, 411-8.

<sup>109</sup> Observations XLVI-LI, *ibid.*, 1826, vol. 2, pp. 582-5, 623-30, 636-42, 642-50, 675-80, 696-703.

<sup>110</sup> Laennec, *op. cit.*, note 3 above, 1819, case XXXVI, vol. 2, pp. 54-62.

<sup>111</sup> *Ibid.*, pp. 265, 278.

It seems that shortly after the publication of his first edition, Laennec had given up trying to make some coherent order out of the stethoscopic findings in the heart. Although he had changed his mind about the meaning of the murmurs, he did not see fit to correct the case examples in his publication accordingly. A survey of the several hundred manuscript case records kept in the Musée Laennec at Nantes tends to confirm this impression. Shortly after the discovery of mediate auscultation, Laennec made a diligent effort, in most cases, to describe the heart sounds and to make an ante-mortem diagnosis of the organic state of the heart. Sometimes this was accomplished by verbal “stretching” of the pathological diagnosis to fit the predictions of the clinical findings.<sup>112</sup> By 1824, this practice seems to have been abandoned: the heart sounds were rarely mentioned and any attempt at an ante-mortem cardiac diagnosis, if it appeared at all, was based on information other than that provided by the stethoscope.

Two cases, one from May 1820, the other from November 1823, illustrate the change in Laennec’s interpretation of murmurs. In the first, of a 35-year-old woman, a palpable thrill and a murmur “softer than a file sound” led the examiners to predict mitral valve ossification, which was confirmed by autopsy.<sup>113</sup> In the second, a 25-year-old *menuisier* named Jean Juelle, a left precordial lift and a file sound murmur synchronous with the first heart sound caused the first observer, probably a student, to predict ventricular hypertrophy and ossification of the aortic valves. When Laennec examined Juelle the following week, he heard the same murmur and a crepitant rale and had the following words added to the diagnosis: “pulmonary oedema—spasm of the arteries”. Autopsy demonstrated the presence of left ventricular hypertrophy beyond question, but it is not clear whether or not the student intended to soften the significance of the valvular findings out of respect for Laennec when he wrote his report. He wrote: “one of the sigmoid valves of the aorta was entirely ossified at its base and open (“béante”): nevertheless it reduced the calibre of the artery only a little at this point . . . The internal membrane of the arteries, the entire length of the aorta, and into the internal iliacs, the subclavians and the carotids . . . was perfectly healthy.”<sup>114</sup> The valvular change was confirmed but spasm, a phenomenon of life, could not be demonstrated or denied by autopsy.

Unfortunately, there is no trace of the manuscript versions of the cardiology cases published in Laennec’s treatise. Many of the published pulmonary cases do exist in manuscript, making the absence of the cardiac observations look suspiciously similar, if not related, to the disappearance of the Collège de France lectures on the heart. Nevertheless, the examination of one case selected from the treatise, and two chosen from the collection of hospital records, can shed some light on Laennec’s method and

<sup>112</sup> One example of this is the case of Moissonet, a 67-year-old male seen in December 1821, who had very clear heart sounds suggestive of dilatation. The pathology report reads “heart not very large . . . [but] cavities were perhaps a little larger than the walls were thick”: MS. Cl. I, lot b., f. 5–7. Other examples include Redon, Cl. I lot b, f. 75–77; and anon., Cl. I lot b, f. 69–72. Most of the cases displaying an attempt at ante-mortem diagnosis of organic heart changes are in Classeur I, lot b (1816–1819); those without are in Classeur III (1823–1826).

<sup>113</sup> Laennec, MS. Cl. Id, 6r.–12v. This observation, like Case 2 above, was most likely collected by René Laennec’s cousin and student, Mériadeuc Laennec.

<sup>114</sup> Laennec, MS. Cl. II 80r.–82v.

on how he came to his conclusions. In the footnotes, the medical reader can find a representation of each case in twentieth-century terms. The primary objective in this analysis is to demonstrate that Laennec's diagnostic signs could successfully predict hypertrophy and dilatation.

*Case 1*

Dirichard, a 45-year-old man, was first admitted to hospital in August 1818 for dyspnoea and swelling. On physical examination he was found to have a prominent pre-cordial impulse, a loud first heart sound and an absent second heart sound. Because of these findings, ventricular hypertrophy was suspected. He improved and was discharged a month later, but had to be readmitted for the same symptoms in November and again in January 1819. He felt heart beats in this throat, his dyspnoea increased and he developed rales. He died on 8th February 1819 after a pulmonary "haemorrhage". Autopsy confirmed the finding of ventricular hypertrophy, as well as engorgement of the lungs and a small pleural effusion. In addition, the aortic ring and the ascending aorta were dilated, although the valve cusps appeared to be normal.<sup>115</sup>

Laennec used this case in the first edition as an example of pulmonary hemorrhage. The loudness of the first (ventricular) sound and absence of the second sound were associated with ventricular hypertrophy as an incidental finding. In the second edition, Laennec moved the case to the chapter on heart disease, emphasizing the absence of the second heart sound as an indicator of hypertrophy. In making his initial clinical diagnosis, Laennec seems to have ignored his own statement that the ventricular sound should be soft in ventricular hypertrophy. This contradiction may have been the reason why he did not place the case in the cardiology section of the first edition. Instead, he heard what he might have expected to hear: a loud "ventricular" sound to correspond to a large ventricle. Later, Laennec focused not so much on the loud first sound, but on the soft second sound, finding that the case was more compatible with his pronouncements than he had thought. He judged the absence of the second sound to be the result of poorly heard atrial contraction, mechanically distanced from the chest wall by the enlarged ventricle. Here, evidently, hypertrophy was associated with a soft second heart sound.<sup>116</sup>

*Case 2*

This case illustrates the difficulty in applying the diagnostic sign of soft heart sound to indicate hypertrophied myocardium. It is kept in the manuscript collection at Nantes and the writer, who wrote in the first person, did not identify himself. It is most likely the work of Laennec's young cousin and student, Mériadec Laennec, who

<sup>115</sup> Laennec, observation XLIX, *op. cit.*, note 3 above, 1826, vol. 2, pp. 642–50. See also note 110, above. The pulmonary "haemorrhage" may have been acute pulmonary oedema. Since the outflow valves were normal, but the aortic ring and artery were dilated, it seems that this man had probably suffered from aortic insufficiency secondary to aneurysmal dilatation, perhaps of hypertensive or syphilitic origin. The first sound was loud due, not to ventricular contraction, but to the crisp closure of the partly cartilaginous and stiff mitral valve. The second sound was absent because the aortic cusps closed inefficiently, or not at all in the presence of a dilated aorta and aortic ring. The hypertrophy and dilatation correspond to patho-physiological changes in the heart, secondary to long-standing elevation of end-diastolic pressure.

<sup>116</sup> Some clinicians continue to cite ventricular hypertrophy as a reason for a soft first heart sound. See Luisada and Portaluppi, *op. cit.*, note 69 above, p. 32.

two weeks earlier, had been assigned to the service of Laennec's friend Joseph-Claude-Anthelme Récamier (1774–1852) at the Hôtel Dieu, when Laennec had to leave Paris for Brittany because of his poor health.<sup>117</sup>

On 23 October 1819, Monsieur Récamier asked me to consult on a 65-year-old patient, who was tall, thin and still very muscular. This man had a high fever and a slight disturbance of his thoughts. The respiratory sound on both sides was replaced by fairly loud mucous rales which seemed to be due to an uninterrupted series of bursting bubbles. On the lower right, the respiratory sound was a little clearer. The voice resonated well throughout the chest, but posteriorly, there was obvious egophony, especially on the left. The heart beats were muffled and their impulse scarcely discernible; they were masked by the mucous rattle of respiration. Monsieur De Lens maintained that this sound was synchronous with the beats of the heart; for my part, I thought it was recognizable as a mucous rale and that it seemed to me to be quite separate.—In this patient I announced the presence of a double pleurisy and a *weak* (“*faible*”) *heart* [writer's emphasis]. The autopsy thoroughly disproved the second part of my diagnosis.

Opening of the cadaver 24 hours after death . . .

*Chest:* The lungs did not adhere to the chest wall . . . When they were removed it could be seen that there was no effusion in the pleural cavities, but the posterior portions of both lungs, especially the right, were covered with a soft, yellow, opaque pseudo-membranous exudate that came off easily when the pleura was scraped. The right lung was more voluminous and heavier than the left and was like liver tissue . . . a true *hepatization* . . . the left lung appeared to be healthy and only a little engorged with blood in the posterior portions.

The heart, quite large when compared to the size of the subject, adhered to the pericardium posteriorly and inferiorly by very short, firm, cellular bands. The left ventricle alone took up almost the entire volume of the organ; its cavity, however, was a little smaller than normal, capable of holding at most an almond in its shell, but the walls were up to 15 *lines* [one *line* = 1/12 inch] in thickness and the septum up to 10. This considerable thickness was generalized and scarcely varied from the apex to the base. The fleshy columns were not in proportion to the rest of the ventricle, appearing as they should do in the normal state. The right ventricle . . . was small and its walls of moderate thickness (3 *lines*). The atria were unremarkable except for a few small, soft, yellowish, non-adherent, polypoid concretions, which extended into the veins.—The valves and the internal lining of the arteries were unremarkable.—The flesh of the heart was quite red and fairly firm.

Thus, in this patient the double pleurisy was confirmed, but the pneumonia and heart disease were not. After a brief examination the latter ought to have been recognized; yet I found the heartbeats weak! This double error in diagnosis was all the more humiliating for me as I made it in the presence of Messieurs De Lens and Kergaradec.<sup>118</sup>

This case is particularly interesting because of the error. The writer readily admitted not only his mistake and embarrassment, but also his surprise at the paradoxical

<sup>117</sup> Rouxau, *op. cit.*, note 47 above, vol. 2, pp. 222–5.

<sup>118</sup> Laennec, MS. Cl. I, lot d, f. 1r.–3v. Few details were given about this patient's history, but it does seem clear that he died of pneumonia and that his enormous left ventricular hypertrophy was of long standing and independent of valvular changes. Why then were the heart sounds “*faible*”? It is possible that the noisy rales of the acute process in the airways drowned the cardiac sounds. It is also possible, given the adhesions in the pericardium, that a pericardial effusion had reduced the intensity of the heart sounds. On the other hand, if Laennec's observations, both in the physical examination and at autopsy, were correct, the case could have been representative of either of two rare diagnoses: restrictive cardiomyopathy or idiopathic hypertrophic subaortic stenosis (IHSS). In Laennec's lifetime, restrictive cardiomyopathy may have occurred more often than it does now, as a result of amyloidosis secondary to tuberculosis. IHSS is an uncommon congenital condition without valvular abnormalities, which leads, among other things, to



observation of a tremendously thickened heart muscle producing such a feeble sound. He did not appear to have entertained the possibility of a muscle being weak in spite of its increased bulk. This case was attended only a few months after the publication of Laennec's first edition. Although the writer had failed to apply the book's recommendations at the bedside, the autopsy served as a resounding confirmation of the statements Laennec had made therein about the stethoscopic signs of hypertrophy.

### Case 3

The third case, also with the Nantes manuscripts, was entitled "*pleurésie ancienne à droite—pleurésie récente à gauche—gastro-entérite?*". It is clear, however, that the patient succumbed to heart disease. Unfortunately, because the pre-mortem diagnostic predictions were not included in the final report, it is impossible to know whether or not the cardiac pathology had been anticipated. The detailed auscultatory description has been preserved and would have been available to Laennec for post-mortem correlation even if he had not recorded his clinical diagnostic impression.

Necker Hospital, St Joseph's ward, no. 7, January 18, 1822.

Brasard (Jean Nicolas) 75-year-old worker in tobacco admitted 5 January 1822. For about seven or eight years, this strong, muscular and slightly stout man, had been subject to recurrent oppressions accompanied by a cough productive of quite abundant sputum and vague chest pains. From time to time, these symptoms would worsen and he would feel episodes of suffocation followed by dizziness and sometimes fainting. Two years prior to admission, he suffered fluxion of the chest and was treated at Hôpital Cochin with phlebotomy, leeches, and revulsives to the legs and feet. During the last three months, the attacks have been worse and the cough and palpitations more frequent. The patient lost his appetite, felt his strength dwindling, and suffered pains in all his limbs. All these symptoms increased in the last ten days.

On admission his cheeks were red, respiration short, rapid and accompanied by wheezing and a loud mucous rale in the trachea. The pulse was weak, but the heart, on the other hand, was strong ("*fort*"). The impulse of the heartbeat seemed to be greater on the right than on the left . . . the 15th, same condition; the 16th dead at 1 a.m.

Opening of the cadaver on January 18 . . .

*Chest:* The right lung adhered all over to the parietal pleura so firmly that one could not remove it without taking the membrane along with it. Transverse indentations corresponding to the ribs were noted on its surface . . . The lung was of normal volume; its blackish-red tissue was still crepitant although it was well infiltrated with a frothy sanguinous serosity. No tubercles were seen . . .

The left lung was bathed in a half-quart of sanguinous serosity . . . Its tissue, more crepitous than that of the right, was similarly infiltrated.

The heart was much larger than the fist of the subject. The left ventricle was huge and contained a few friable, black clots; the walls of the ventricle were scarcely thicker than they would be without the dilatation of this cavity. The right ventricle also offered a cavity much larger than normal proportional to the enlargement of the left; its walls were barely thickened.—The muscular flesh was firm and the membrane was dark red, almost violet, a redness that one also noticed along the internal lining of the pulmonary

---

reduced ventricular out-flow and sometimes to lowered arterial blood pressure. Both diseases are associated with "distant heart sounds". The first sound is soft because of the delay in the rise of intraventricular pressure or because of incomplete closure of the mitral valve. The second sound is soft because of the low arterial pressure and the slow closure of the aortic valve.

arteries and into the abdominal aorta. The aortic “sigmoid” valve bore several cartilaginous points, which grated under the scalpel when scraped.

*Abdomen:* . . . the interior of the stomach was very red and its veins were engorged with black blood. The liver was large and very heavy.

Conclusion: old pleurisy on the right, recent pleurisy on the left and possibly gastro-enteritis.<sup>119</sup>

This case confirms another of Laennec’s cardiac signs: loud heart sounds in the presence of dilatation, without hypertrophy.

## CONCLUSIONS

### *Heart sounds*

Laennec’s interpretation of the heart sounds and his rejection of the significance of heart murmurs were incorrect, but his observations were sound and some of the signs he described were actually useful in predicting the pathological changes that interested him most. Some historians have suggested that since Laennec’s judgements about the meaning of the heart sounds were not right, then all his cardiac semiology must have been without value.<sup>120</sup> This is not true of either Laennec’s cardiology, or that of many of his successors. The significance of certain murmurs was appreciated and implemented in diagnosis long before the interpretation of the heart sounds was definitively resolved.<sup>121</sup>

The 1832 dissertation of J. Rouanet (1797–1865) contained the first interpretation of the heart sounds as products of valve closure, but it would take many years of controversy before this thesis was accepted.<sup>122</sup> Confusing the issue was the apparent inaccuracy of Harvey’s observation that the palpable apex beat corresponded to ventricular systole. The apex beat does occur when the contraction of the ventricle

<sup>119</sup> Laennec, MS. Cl. I, lot c, 16r.–19v. Despite Laennec’s conclusion to the contrary, it seems that this patient died of florid left- and right-sided failure. The man was on the heavy side and had a long history of “oppressions”, a word used by Laennec to describe not only dyspnoeic conditions, but also the pain of angina pectoris. The origin of his heart failure may well have been hypertensive and/or ischaemic as repeated small infarctions led to cardiomyopathy. What was Laennec hearing? The second sound might have been due to the loud snapping closure of the sclerosed aortic valve, perhaps even further accentuated by elevated blood pressure. The weakness of the pulse, however, is against the presence of hypertension. The loud first sound is more difficult to explain with the clinical information given. We are told that the pulse was weak, but its rate was not mentioned. Almost certainly, there was tachycardia to correspond to the tachypnoea, and tachycardia is a cause of accentuated first heart sound by the mechanism of abrupt closure of the atrio-ventricular valves. Laennec attributed this loudness to the fact that an enlarged, dilated heart struck the chest wall over a wider area. In fact, dilatation is a feature of heart failure and in heart failure, unless there is a rhythm disturbance, tachycardia is almost always present. Enhanced heart sounds, therefore, can occur with dilatation and in a convoluted way can be an indicator of that pathological condition.

<sup>120</sup> See McKusick, op. cit., note 2 above, pp. 137–45.

<sup>121</sup> For example, James Hope and Thomas Hodgkin made successful interpretations of murmurs of aortic and pulmonic origin prior to the clarification of the meaning of the heart sounds. See East, op. cit., note 1 above, p. 34–6.

<sup>122</sup> J. Rouanet, *Analyse des bruits du coeur*, thèse médecine, Paris, Didot, 1832, no. 252. See East, op. cit., note 1 above, p. 35. Discussions of the various theories were provided by the following: G. Andral in his edition of R. Laennec, *Traité de l’auscultation et des maladies des poumons et du coeur*, fourth ed., Paris, Chaudé, 1837, vol. 3, pp. 34–42; J. Bouillaud, *Traité clinique des maladies du coeur*, Paris, Baillière, 1835, pp. 25–6, 102–37; L. L. Rostan, *Cours de médecine clinique ou sont exposés les principes de la médecine organique, etc.*, 3 vols., Paris, Bechet, 1830, vol. 1, pp. 315–18; C. J. B. Williams, *Pathology and diagnosis of diseases of the chest*, third ed., London, Churchill, 1835, pp. 163–79.

causes it to rise up and hit the chest wall, but it *seemed*, “logically”, that it should be due to distension of the ventricle during diastolic filling. Harvey’s was seen as the “old” opinion, and the “new” was actually a partial reversion to Cartesian physiology, in which the apex beat had been interpreted as the dilatation of the heart caused by the expansion of blood heated there.<sup>123</sup> This backward-looking stance had many respectable adherents including Dominic Corrigan (1802–80)<sup>124</sup> and Joseph Beau (1806–65).<sup>125</sup> Their objections represented more than reactionary resistance to new ideas. They were the tenable conclusions resulting from careful physiological experiment, observation, and reasoning.

Pierre Huard analysed this dilemma of the “cardiac controversy” as a problem that defied resolution through the application of such techniques as then existed. The rupture of this long standing “epistemologic obstacle” was accomplished “only in 1864, when Chauveau and Marey, with their closed-thorax cardiac catheterization, succeeded in having the heart record its own movements on a rotating drum.”<sup>126</sup> This work did not imply that other interpretations of the heart sounds had been irrational, but it did guarantee their obscurity. In other words, resolution of the interpretation of the heart sounds was impossible with the technology of early nineteenth-century anatomico-clinical medicine. Substituting first and second heart sounds for Laennec’s confusing nomenclature of “*contraction ventriculaire et auriculaire*” leaves a collection of easily identifiable cardiac cases whose semiology is exact.

#### *Stethoscopic signs*

Laennec set rigid standards in establishing his new elements of diagnosis, the stethoscopic signs. This type of inductive evidence was relatively new and there were no guidelines to the acceptable limits of accuracy, or the tolerable margin of error.<sup>127</sup> Laennec moved through this uncharted domain, apparently oblivious to the new methods in probability and statistics that could have helped him to decide on the utility, or not, of a sign. He also seemed to ignore, perhaps deliberately, any consideration of the *degree* of patho-physiological disruption required to generate (or obliterate) the abnormal sound; and he seems to have been unaware of at least two French contemporaries’ interest in fluid dynamics and/or sound. By recognizing the position from which Laennec made his judgements, it is possible to reconstruct his priorities, when evaluating stethoscopic signs.

Laennec was inclined to ascribe one, and only one, lesion to each stethoscopic sign. Initially, he considered the lesion as a necessary cause for each abnormal sound, so that

<sup>123</sup> For a brief explanation of the Cartesian explanation of cardiac physiology, see T. S. Hall, *Ideas of life and matter*, Chicago and London, University of Chicago Press, 1969, vol. 1, pp. 257–9.

<sup>124</sup> D. Corrigan, ‘On permanent patency of the mouth of the aorta’, *Edinb. med. surg. J.*, 1832, 10: 225. Throughout this discussion, Corrigan referred to the rise in arterial pulse as “arterial diastole”.

<sup>125</sup> Beau published frequently and at great length on this subject. See for example, J. Beau, *Traité expérimentale et clinique d’auscultation appliqué à l’étude des maladies du poumon et du coeur*, Paris, Baillière, 1856, pp. 234–314, especially p. 275.

<sup>126</sup> Pierre Huard, ‘L’auscultation cardio-pulmonaire depuis Laennec’, *Rev. du Palais de la Découverte*, no. spéciale 22, 1981, pp. 170, 174.

<sup>127</sup> Ian Hacking, *The emergence of probability*, London, Cambridge University Press, 1975, pp. 31–48; Lester S. King, ‘Evidence and its evaluation in eighteenth-century medicine’, *Bull. Hist. Med.*, 1976, 50: 174–90.

in every instance of the sound's occurrence Laennec could be certain that the lesion was present. Gradually, for some sounds, he came to accept the possibility that one or more lesion(s) could be (a) sufficient cause(s) of the abnormal sound, so that in each instance of hearing a particular sound he would have a limited range of possible pathological lesions. When this happened he usually rejected the value of the sound as a sign of pathological change. These priorities can be restated more clearly with twentieth-century decision making vocabulary.<sup>128</sup>

In order to be useful, Laennec insisted, a sign must be specific, certain, and rigidly infallible in all cases. "Specificity" means that a sign is peculiar to one condition only. If a sign is not specific, then it can occur in conditions other than the one for which it is intended to be an indicator, resulting in a false positive. Specificity was not Laennec's word for this priority; he preferred the words "*pathognomonique*" and "*constans*". Nevertheless, this concept of specificity held absolute control over his formulation of auscultatory semiology. He abhorred the false positive.

Laennec also insisted on a certain level of sensitivity in his signs. If a sign is "sensitive", then it will appear in every example of a condition regardless of how minimal the change may be. If a sign is not sensitive, there may be cases which escape detection: false negatives. Sensitivity is independent of specificity. For example a sign may be very sensitive to a certain condition and also non-specific, resulting in many false positives, but no false negatives. In Laennec's formulation of auscultatory semiology, the importance he gave to sensitivity was secondary to that of specificity. These priorities will be illustrated with a few examples from the material already presented.

For the coronary theory of angina pectoris, Laennec rejected the value of the typical chest pain, described by William Heberden as a sign of coronary ossification. Ossification of the coronaries could occur as an incidental finding in patients who died of diseases other than angina pectoris. Moreover, it was not always found in patients like Nicolas Millot, who seemed to suffer from the pain of angina pectoris. In the former instance, absence of the sign, i.e. pain, was a false negative; in the latter, presence of the sign was a false positive. It is interesting that in selecting the case of

<sup>128</sup> Although economists, psychologists and game theorists were long aware of these concepts, their conscious recognition in medical decision making, and incorporation into medical vocabulary, appeared in the first third of the twentieth century, with an appreciation of potential inaccuracies of lab testing for venereal disease and tuberculosis. On the history of these concepts see Stanley Joel Reiser, 'The emergence of the concept of screening for disease', *Milbank Quarterly*, 1978, **56**: 403–25; Ward Edwards, 'The theory of decision making', *Psychol. Bull.*, 1954, **51**: 380–417. For more detailed medical definitions of the terms "sensitivity", "specificity", "false positive", and "false negative", and explanations of their use in medical decision making, see Robert S. Galen and S. Raymond Gambino, *Beyond normality: the predictive value and efficiency of medical diagnosis*, New York, etc., John Wilkey and Sons, 1975, pp. 10–14; Lee B. Lusted, *Introduction to medical decision making*, Springfield, Charles C Thomas, 1968, pp. 107–11; Harvey N. Mandell, 'Sensitivity, specificity and predictive value vs. instinct', *Postgrad. Med.*, 1984, **75**: 24–8; Barbara J. McNeil, Emmett Keeler, and S. James Adelstein, 'Primer on certain elements of medical decision making', *N. Eng. J. Med.*, 1975, **293**: 211–21; Robert W. Sappenfield, Myrton F. Beeler, Paul G. Catrou, and Donald Boudreau, "Nine-cell diagnostic matrix: a model of the diagnostic process; a framework for evaluating diagnostic protocols", *Am. J. clin. Path.*, 1981; **75**: 769–72.

Millot, Laennec indicated his belief that refutation of the theory was best supported by the latter circumstance and this suggests a philosophical distinction between him and his late eighteenth-century precursors.<sup>129</sup>

Laennec did continue to acknowledge the frequent association of heart murmurs with valvular change, as in the case of Ponsard. Valvular alteration may have been a sufficient cause for murmurs; in other words, murmurs may have been *sensitive*, but they were not specific. Analogous sounds with no valvular pathology could be heard in anaemia, tachycardia, and pregnancy. They could be produced experimentally by the compression of healthy vessels anywhere in the body. Laennec rejected these newly-discovered signs because false positives occurred too frequently to fulfil his standards of specificity. When he proposed that certain murmurs were signs of spasm, he moved beyond the realm of autopsy proof. No confirmation or rejection of these statements could be found, or would be expected in the cadaver. He had exceeded the limits of his method. His interpretations of heart murmurs could not be relied upon as signs, since they were only uncontrolled speculations at best.

The sound of friction rub provided an instance of a false negative. The sign had not been present ante-mortem in every case diagnosed at post-mortem. Laennec was more tolerant of false negatives than of false positives, but such a situation made him reluctant to proclaim the reliability of the sign. In other words, the friction rub may have been specific, but it did not appear to be sensitive enough to be practical.

Only the variations of loud and soft heart sounds seemed to correlate well with the pathological changes of hypertrophy and dilation. These are the signs in which Laennec was most confident and, as cases 1, 2, and 3 show, they did appear to be both specific and sensitive.

Examples of Laennec's criteria of acceptability of stethoscopic signs are not confined to auscultation of the heart. His description of pulmonary catarrh or bronchitis illustrates the one sign – one lesion principle. He named many different types of rales, each corresponding to what we would now describe as non-specific clinical variations of bronchitis, based on sputum colour, quantity, and texture; variations that may in fact reflect differing bacterial or viral pathogens.<sup>130</sup> These subdivisions, like his qualitative distinctions between murmurs, were too subtle to be workable and since they did not correspond to any reliable clinical or therapeutic distinction, they fell into disuse. His distrust of the false positive is apparent in his consideration of the causes of pectoriloquy and egophony. Pectoriloquy is an increase in volume of the voice heard through the chest. Laennec tried to establish degrees of this sign to correspond to different lesions. When he found that a single degree of the sign seemed to appear in two different conditions, bronchiectasis and cavity, he was obliged to justify his decision at length, relying heavily on the anatomical similarities of the two lesions.<sup>131</sup> Egophony is the

<sup>129</sup> Karl Popper's doctrine of falsifiability as a criterion of demarcation of science is an explanatory model of change in scientific thinking. It is interesting to note that Laennec seemed to have adhered to this principle, whereas a generation earlier, Lavoisier did not. See Karl Popper, *Conjectures and refutations: the growth of scientific knowledge*, fourth ed., London and Henley, Routledge and Kegan Paul, 1972, pp. 256–7; Holmes, *op. cit.*, note 68 above, pp. 499–500.

<sup>130</sup> Laennec, *op. cit.*, note 3 above, 1826, vol. 1, pp. 145–9, 158–9, 163–6, 173–9, 188, 195, 201–6.

<sup>131</sup> Laennec, *op. cit.*, note 3 above, 1819, vol. 1, p. 127; 1826, vol. 1, pp. 212–15; Collège de France lecture notes, 1823–24, MS. Cl. 2, lot a(B), f. 261r.

characteristic bleating sound of the voice auscultated through the chest: Laennec thought egophony always represented pleural effusion. Gabriel Andral (1797–1876) was unable to convince him that it could exist under certain false positive circumstances, i.e. when there was no effusion.<sup>132</sup> Laennec argued against this possibility with a vengeance.<sup>133</sup> If Andral's observations were correct, then, according to Laennec's strict priorities, the continuing value of his sign would be cast in doubt. In his auscultatory work, there were no exceptions to the priority of specificity.

Laennec's diagnostic criteria for heart disease were rapidly eclipsed, and his modest contribution duly forgotten. Yet within his limited conceptual framework, his auscultatory heart signs did have some diagnostic utility, independent of the accuracy of his interpretations of their physiological correlatives. It is hoped that this analysis, of Laennec's attempt to construct a workable diagnostic system of cardiac pathology with new conceptual and methodological tools, serves as an epistemological justification for his conclusions, and as a model for the decision making criteria applied by one early nineteenth-century physician to the resolution of scientific problems.

<sup>132</sup> G. Andral, *Clinique médicale ou Choix d'observations recueillies à la clinique de M. Lerminier*, Paris, Gabon, vol. 2, 1824, pp. 571–2.

<sup>133</sup> Laennec, *op. cit.*, note 3 above, 1826, vol. 2, pp. 83–4.