

# Emergence of Rheumatic Fever in the Nineteenth Century

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**T**HE CHILD SUFFERING CONGESTIVE HEART FAILURE from rheumatic heart disease, so common a century ago, is fortunately now quite rare. To the medical students I teach, rheumatic fever is a disease of history. They still memorize T. Duckett Jones's 1944 criteria for diagnosis (as they do Robert Koch's postulates), but nearly all await the opportunity to apply them—that is, until the last several months when the unexpected and unpredicted return of rheumatic fever in several isolated epidemics surprised pediatricians and epidemiologists alike.

So complete seemed the demise of rheumatic fever that pediatrician-epidemiologist Leon Gordis's 1985 T. Duckett Jones Lecture before the American Heart Association was entitled, "The Virtual Disappearance of Rheumatic Fever in the United States: Lessons on the Rise and Fall of Disease." That same year Milton Markowitz (1985) delivered the Lewis W Wannamaker Memorial Lecture that he titled, "The Decline of Rheumatic Fever: Role of Medical Intervention." In 1986, the 86th Ross Conference was dedicated to the clinical problem of "Management of Pharyngitis in an Era of Declining Rheumatic Fever." Clinicians over the age of fifty will recall that as late as the 1950s and 1960s rheumatic fever was a major health problem that touched in a central way many of the basic sciences (bacteriology, immunology, pathology, epidemiology, genetics, endocrinology, pharmacology), most of the clinical services (internal medicine, pe-

diagnostics, surgery, psychiatry), as well as many of the medical technologies (culturing, immunological assays, erythrocyte sedimentation rate, electrocardiogram, and the X-ray). The clinical entity that Walter Butler Cheadle refined in London in the 1880s and that Jones clarified 60 years later in Boston now is virtually gone, despite its recent recrudescence.

What is equally striking as the decline and reappearance of rheumatic fever is that rheumatic fever, as Cheadle understood it in 1890, may very well not have existed a century earlier. How, then, did rheumatic fever emerge? I argue that biological, technological, clinical, institutional, possibly even geographical and climatic elements of rheumatic fever's "ecology" changed in the nineteenth century in a way that focused attention on damage done to the heart as the clinically most important facet of the disease, and it was this cardiac emphasis that precipitated and shaped the clinical recognition of rheumatic fever.

In the late eighteenth century, rheumatic fever was imbedded in the diagnostic category of "rheumatism," a broadly defined group of illnesses characterized by fevers, aches and pains of the limbs, and debility (Swan 1749, 245–46; Cullen 1781; van Swieten 1776, vol. 13). "Rheumatism" was a routine medical diagnosis in the eighteenth century. I believe that the biological nature of a portion of rheumatism may well have changed in the late eighteenth century so that the heart, especially two tissues of the heart (the pericardium and the endocardium) became inflamed, an injury that had not commonly occurred previously.

This biological alteration must remain to a degree speculative. What is without question, however, was that the late eighteenth century witnessed the beginning of a clinical appreciation of cardiac involvement in ills also characterized by "rheumatism." William Charles Wells, a native of Charleston, South Carolina, who trained in Edinburgh and later remained loyal to Great Britain after the Revolution and practiced medicine in London, definitively linked heart disease with rheumatism in 1812 (Wells 1812; Keil 1936, 1939). Nevertheless, it is clear from reading late eighteenth and early nineteenth century accounts of patients suffering from rheumatism that a few practitioners were becoming aware of the cardiac connection before Wells. For example, Gerhard van Swieten (1776, vol. 13, 32) in mid-eighteenth century reported that "sometimes, when the pain in

the limbs ceases, there arises an anxiety in the breast, a palpitation of the heart, and intermitting pulse." While it is certain that van Swieten understood the occasional involvement of the heart, he did not believe it was a common element of rheumatism. William Cullen (1781, 156) in the 1760s called attention to the "frequent, full and heard pulse" that accompanied some rheumatic patients. Corvisart ([1806] 1984), in *Organic Diseases of the Heart and Great Vessels* (case 37) described one patient with rheumatic pains who on autopsy had vegetations on the mitral valve. In the first extensive statistical analysis of rheumatism, John Haygarth noted ([1805] 1977) that 55 of 93 patients with acute rheumatism who had their pulse recorded had a heart rate greater than 96 beats per minute. Haygarth did not make the connection between heart damage and rheumatism, and an elevated pulse does not always mean heart disease (fever in itself alone can raise the pulse). But Haygarth's observations indicated that physicians were beginning to look at the cardiovascular system. Twelve of Haygarth's patients died. The detailed case histories indicate that three died either with severe chest pain or with shortness of breath. While it is not possible to be certain of the exact pathological cause of death, it is certainly possible to speculate that pericarditis and/or congestive heart failure contributed to these deaths. Giovanni Morgagni ([1761] 1960, letter 57) in his *On the Seats and Causes of Diseases* supports the paucity of cardiac involvement in rheumatism. In reading the case histories and discussions that accompanied mitral valve disease, none clearly suffered from rheumatism. And Morgagni, when analyzing rheumatism, could recall only two people dying with this diagnosis.

Wells did not claim priority for his observation linking heart disease with rheumatism. Rather, he credited David Pitcairn, a prominent British physician, with the initial association in 1788. Pitcairn failed to publish his remarks on the subject, so Wells considered his paper serving the purpose of recording Pitcairn's idea, to which he added seven cases of his own. Wells also remarked that Matthew Baillie, a Scottish pathologist, had made the initial pathological investigation of a patient with rheumatism dying from heart disease:

The muscular parietes of the heart being generally very thin in proportion to the enlarged size of its cavities, the heart has little power to propel an increased quantity of blood into the more distant branches of the arterial system. At times there is much difficulty

of breathing; and there is a purplish hue of the cheeks and lips. . . . The causes which produce a morbid growth of the heart are but little known; one of them would seem to be rheumatism attacking this organ (Baillie 1797, 44–46).

In a footnote, Baillie confirmed that “Dr. Pitcairn has observed this in several cases.” Baillie’s description contained no prior reference to rheumatism, so it is not possible to understand how he arrived at his conclusion that rheumatism was responsible.

Wells also called attention to the experience of David Dundas, sergeant-surgeon to the king, who reported in 1809 nine patients with heart disease and rheumatism that Dundas had seen in thirty-six years of practice. Most had suffered chest pains, anxiety, an increased pulse, ascites, pleural fluid, or peripheral edema following one or more attacks of rheumatic fever. Seven of the 9 were under 22 years of age; and 7 died, usually after a period of several months. He autopsied 6 and found the heart enlarged in most; pericardial fluid surrounded one heart; and in several other the pericardium adhered to the surface of the heart (Dundas 1809).

Wells encountered his first rheumatic patient with heart disease in 1798. Enlightened by Baillie’s description, Wells suspected heart disease because the eighteen-year-old boy complained of an “oppression in his chest.” Wells consulted with Pitcairn, who confirmed his diagnosis. The boy subsequently died, and at autopsy Wells discovered an enlarged heart. Wells observed his second case of rheumatism associated with heart disease four years later. This time his patient, Martha Clifton, recovered after eleven weeks.

Is it possible that Pitcairn, Wells, and Dundas simply observed what others had plainly overlooked in the past? I think not; rather, it is at least possible that the cardiac involvement was new at the end of the eighteenth century. Let us imagine that there was a clinical spectrum of heart involvement in rheumatism:

1. heart not involved
2. heart involved but the patient asymptomatic
3. heart involved, patient initially asymptomatic, but patient later developed significant (and symptomatic) heart disease years after a bout with rheumatism
4. heart involved and patient symptomatic (congestive heart failure, pericarditis, chest pain)
5. death

Even an astute observer could miss the first three categories at the onset of rheumatism. A careful physician, however, would not miss the last two categories. Even Haygarth, van Swieten, and Cullen—who did not associate rheumatism with heart disease—nevertheless did remark on these “unusual” symptoms. In the eighteenth century, patients with rheumatism were not discomfited except from their joint pains, and they did not die. A close look at individual case reports from Wells and Dundas leaves little doubt that these early patients had cardiac symptoms.

*Example 1* (Wells). In the beginning of August, shortly after remaining some time in a cold cellar, she was seized with pains, swelling, and redness of her joints, and fever. These symptoms lasted only ten days. Immediately upon their ceasing, her heart began to beat with considerable violence. Her right hypochondrium soon after became painful, and about the same time she began to complain of a pain in the tops of her shoulders. The palpitation of the heart, which had never ceased from its first appearance, was distinctly felt in every part of the thorax, to which my hand was applied. In the arteries, only a shaking was perceivable, which could not be divided into distinct pulsations. The strokes of the heart were one hundred and ninety in a minute; she frequently complained of a great and indescribable anxiety in her chest. The external jugular veins were swollen, and alternately rose and fell. After her death, the following are the principal morbid appearances, which, as I was afterwards informed, were observed: The whole of the internal surface of the pericardium was attached to the heart.

*Example 2* (Dundas). The patient complains of great anxiety and oppression at the praecordia; has generally a short cough, and a difficulty of breathing, which is so much increased by motion or by an exertion, as to occasion an apprehension that a very little additional motion would extinguish life. There is also frequently an acute pain in the region of the heart, but not always.

The observation that took Wells and Dundas years to repeat became commonplace in the nineteenth century. For example, a physician at St. Bartholomew's Hospital, P.M. Latham, tabulated that 13 percent of all patients admitted between 1836 and 1840 with the diagnosis of acute rheumatism suffered from pericarditis (Church 1887). The proportion of patients with pericarditis as part of their rheumatism grew to 22 percent at Middlesex Hospital between 1853 and 1859 (Bury 1861) and to 24 percent at Guy's Hospital between 1870 and

1872 (Pye-Smith 1874). The prevalence of endocarditis in some hospitals was even higher. Following Wells's lead, a trickle of practitioners began to examine the heart in all cases of rheumatism, whether or not there were cardiac complaints. For example, Laennec (1821, 266) listed "gouty or rheumatic affections" as an occasional cause of pericarditis. Such case reports increased to the point where a Parisian medical student could sustain in 1824 a twenty-four page thesis for graduation from medical school, entitled "Considerations sur la Rhumatisme de Coeur" (Itard 1824).

These initial reports resulted from external inspection of patients followed by confirmatory autopsies. The stethoscope changed this. While Laennec's introduction of the stethoscope in 1816 has been well studied by historians, a great deal less is known about the reception by practitioners of this technological breakthrough. The acquisition of skills among practitioners that correlated sounds at the bedside with structural changes at the autopsy table took time. The best historical accounts demonstrate that the stethoscope received a slow but steady welcome from clinicians, especially among those physicians who had been trained in Paris. Initially, sounds emanating from the lungs received attention, and only by the 1830s did clinicians begin to sort out which abnormal heart sounds came from a particular chamber or valve of the heart (Davis 1981; Smith 1978).

Using the stethoscope, Jean-Baptiste Bouillaud (1796–1881) a controversial Parisian clinician, argued forcefully that there was a "constant coincidence either of endocarditis or of pericarditis with acute articular rheumatism."

"In auscultating the sounds of the heart in some individuals still laboring under, or convalescing from acute articular rheumatism, I was not a little surprised to hear a strong, full, saw or bellows sound . . . such as I had often met in chronic or organic induration of the valves, with contractions of the orifices of the heart" (Bouillaud 1837).

What the stethoscope permitted Bouillaud to discover was a greatly increased number of patients with asymptomatic heart disease (categories 2 and 3 above) and to locate more specifically which part of the heart was affected. In a short monograph devoted entirely to the subject, Bouillaud gave a systematic approach to examining the heart of patients with rheumatism. Like other members of the "Paris school," Bouillaud made extensive use of percussion and auscultation,

and he followed unsuccessfully treated cases to the autopsy room. Hospital-based, Bouillaud saw enough cases to estimate that nearly one-half of people with acute rheumatism suffered from either pericarditis or endocarditis (or from both), either symptomatic or asymptomatic. By midcentury then, the technology and experience were available to diagnose whether the pericardium or endocardium was involved; the stethoscope was not, of course, as helpful in determining myocardial damage.

Less speculative were the distinct shifts in clinical thinking about acute rheumatism in the nineteenth century, changes brought about by the very complexity of the disease. Each patient with acute rheumatism experienced the disease differently. A variety of joints were swollen, inflamed, and painful, but rarely in any perceived pattern. Some patients had rashes, others fever, but in no common sequence. Some behaved peculiarly. Some were mildly discomfited; others died rapidly in great pain. In some families, several would be sick at one time. In other families, many generations suffered. Some were sick for days, others weeks or months. Some suffered one bout, others more than ten. Some were noticeably sick from other illnesses before rheumatism struck (scarlet fever, for example), others not. Some responded to therapy, others got better without therapy. Still others died despite all measures.

Rheumatic fever required physicians to sort out common and useful patterns in a disease that was complex in its presentation. This sorting out occurred in three distinct phases, clearly reflected in clinical reporting. Early in the century, practitioners wrote about individual cases that stressed elements peculiar to that patient. Schematically, the format was similar to those case reports of Wells and others (table 1).

The focus centered on the joint disease, but other manifestations, such as cardiac damage, were added to the picture in a supporting role (Stille 1840; Bright 1839; Fowler 1880; Barlow 1881; Garrod 1888). In midcentury, hospital-based physicians in Great Britain and France, with access to hundreds of patients with rheumatism, were able to describe a "statistically average" case. An example was George William Fleetwood Bury's (1861) analysis of 476 patients admitted to the Middlesex Hospital with the diagnosis of rheumatism between 1853 and 1859. Bury found that 253 suffered heart disease (138 with endocarditis, 35 with isolated pericarditis, 71 with both). Using a

TABLE 1  
Elements Accompanying Rheumatic Fever

Fever, Arthritis +	Carditis Chorea Erythematous rashes Subcutaneous nodules Tonsillitis Pleurisy/Pneumonia Hyperpyrexia
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numerical approach, he noted that women and younger patients of both sexes were more likely to suffer heart disease in rheumatism (table 2).

At Guy's Hospital, medical registrar Philip Henry Pye-Smith assembled the records of 300 hospitalized patients with rheumatism and an additional 100 outpatients seen from 1870 to 1872. He showed that fully one-half of the patients were under twenty years of age (table 3). Pye-Smith also noted that over one-half (227 of 400) suffered heart damage (table 4). What is apparent from these tables (only three from hundreds) is that the literature had shifted from individual cases to statistical averages based on large numbers and that heart damage had surfaced as the most significant clinical manifestation of the disease.

The final third of the nineteenth century witnessed yet another shift in clinical thinking about rheumatic fever. "Individual" case histories showed the immense variability of acute rheumatism; "statistically average" analyses demonstrated in a general way how rheumatism affected populations. Neither approach was entirely helpful to the practitioner when confronted with sick patients.

What emerged in the 1880s was the concept of the "typical" case that allowed for variability yet possessed common elements. This approach, which clustered elements from many cases, permitted physicians to make a certain diagnosis in a disease that was most uncertain in its presentation. Pioneering in this strategy was Walter Butler Cheadle, physician to the Hospital for Sick Children, Great Ormond Street.

The question Cheadle (1889) addressed in his Harveian Lectures was how a practitioner could recognize the "various manifestations of



TABLE 2  
The Relative Frequency of Acute Rheumatism, and Also of Its Heart Complications, at Various Ages

Age	No. of cases	No. in which heart was affected	%
Under 10 years	9	6	66.6 %
Between 10 & 15 yrs	51	31	60.8
Between 15 & 20 yrs	124	85	68.5
Between 20 & 25 yrs	103	59	57.28
Between 25 & 30 yrs	76	31	40.8
Between 30 & 35 yrs	36	19	52.8
Between 35 & 40 yrs	29	8	27.58
Between 40 & 45 yrs	23	7	30.43
Between 45 & 50 yrs	15	5	33.3
Between 50 & 55 yrs	6	2	33.3
Between 55 & 60 yrs	3	—	—
Between 60 & 65 yrs	1	—	—
TOTAL	476	253	

Source: Bury 1861.

the rheumatic state." His solution was to describe a "typical" case, or rather ten of them. These typical cases were not statistical averages. Typical cases did not last 25.5 days, have fevers of 102.78°F., or have a 56 percent chance of heart disease.

Cheadle initially identified the common elements: "The claims of

TABLE 3

Age	Initial attacks	Total attacks
5-10 yrs	22	23
11-20 yrs	179	277
21-30 yrs	118	200
31-40 yrs	34	80
41-50 yrs	8	28
51-61 yrs	4	12

Source: Pye-Smith 1874.

TABLE 4

Pericarditis	70	Endocarditis	
with large effusion	4	Systolic bruit at base	20
with endocarditis	17	Systolic bruit at apex	3
with pleurisy	5	Systolic bruit at precordium	11
		Mitral bruit	78
		Aortic bruit	16
		Presystolic bruit	3
TOTAL	96		131

Source: Pye-Smith 1874.

endocarditis, of pericarditis, of pleurisy, of tonsillitis, of exudative erythema, of chorea, of subcutaneous nodules, will hardly, I think, be seriously disputed." What Cheadle did next was pioneering. He claimed that each element was separate and could appear in nearly any combination or in almost any order. In children, the variation was even more extreme. He called this variation "phases in the rheumatic process or series." Each member of the series—for example, chorea or carditis—had causes other than rheumatism, but rheumatic fever was one of the most common if not the most common predisposing cause (figure 1).

Cheadle's clinical organization of thinking about acute articular rheumatism largely settled the difficult task of diagnosis. Leading physicians quickly adopted Cheadle's scheme, and nearly all discussions at meetings and lectures, in textbooks, and in scholarly papers showed clear evidence that his method swiftly reached the practitioner as well as the prominent workers in the field. Indeed, no one has improved upon his general approach. Even T. Duckett Jones's criteria, which medical students began to memorize after 1944, must be understood as giving simple mathematical precision to Cheadle's earlier clinical triumph (table 5). A diagnosis of rheumatic fever is made if a patient has two of the major manifestations or one major in association with two or more minor manifestations.

The progression of clinical thinking shifted emphasis from the joints to the heart. Individual cases showed that carditis was part of rheumatism; statistically average analysis showed that most morbidity and

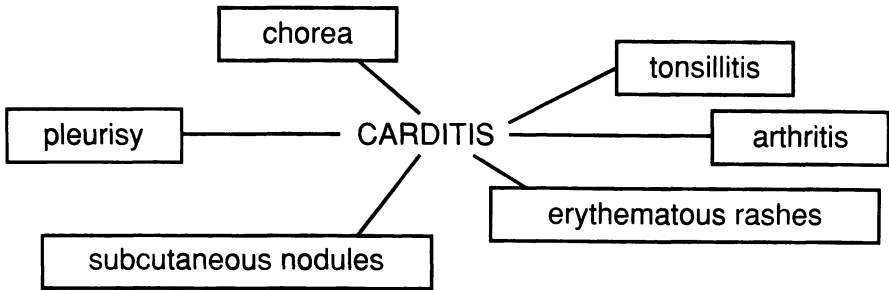


FIG. 1. Cheadle's schema.

mortality resulted from heart disease; the typical case demonstrated that the most crucial element in rheumatism was heart involvement.

Closely associated with these shifts of clinical thinking about rheumatic fever were institutional changes. For the most part, individual practitioners described individual case histories from experiences at the patient's bedside—normally at home. This type of practice dictated the relatively smaller number of cases and the longer period between cases as we saw in the practices of Wells and Dundas. The latter two phases—"statistical average" case and the "typical" case—characteristically were hospital based.

Hospital practice, which normally included hundreds of patients, reinforced the conclusion that damage done to the heart was the clinical event of most concern. Almost certainly, there was a selective bias. Contemporary case reports make clear that only the sickest, and of course the poorest, patients were admitted to hospitals; "sickest" usually meant patients suffering from pericarditis or congestive heart failure. Less-common reasons for admission were extreme joint pain or poorly controlled chorea. In other words, nineteenth-century hospital practice tended to concentrate those patients suffering from heart disease in the hands of hospital physicians who were frequently leaders, authors, and educators. Those dying of rheumatism invariably died from heart complications, findings clearly detected with the autopsy techniques available at midcentury. Pericarditis and endocarditis can be seen easily with the naked eye; they did not require special stains or a microscope. One example of this emerging cardiac prominence came from St. Bartholomew's Hospital where heart disease (valvular disease, mitral stenosis and aortic regurgitation, and pericarditis)

TABLE 5  
Jones Criteria (Revised)\*

Major manifestations	Minor manifestations
Carditis	Fever
Polyarthrititis	Arthralgia
Chorea	Previous rheumatic fever or rheumatic heart disease
Erythema marginatum	Elevated erythrocyte sedimentation rate or positive C-reactive protein
Subcutaneous nodules	Prolonged PR interval
	Plus
Supporting evidence of preceding streptococcal infection: history of recent scarlet fever; positive throat culture for group A streptococcus; increased antistreptolysin-O titer or other streptococcal antibodies.	

Source: Stollerman et al. 1965.

\*Circulation 32:664, 1965.

ranked second—only after tuberculosis—as the leading cause of death from 1830 to 1872 (table 6). While there were causes other than rheumatic fever for each of these pathological entities, acute rheumatic fever certainly played a large role.

Climate and geography may also have played a role in the rise of rheumatic fever. As is obvious from this account, much of the writing on rheumatic fever came from Britain or Northern France—northern latitudes with a generally cold and damp climate. Was this just coincidence? August Hirsch in his *Handbook of Geographical and Historical Pathology* (1881) found evidence of rheumatic fever at all latitudes and climates, but he confirmed the generally held view that rheumatism was more prevalent in northern, cold, and damp locations. Studies in the early twentieth century confirmed this uneven geographical distribution. James Faulkner and Paul Dudley White (1924) gathered together published statistics from 28 hospitals around the world. Those with the coldest and wettest climates reported the most rheumatic fever. Similarly, Tinsley Harrison and S.A. Levine (1924) confirmed the geographical variation: At the Peter Bent Brigham Hospital in Boston, 1.85 percent of all admissions were for rheumatic fever; this compared with 0.73 percent at Johns Hopkins in Baltimore

TABLE 6  
Deaths Attributed to Cardiac Causes at St. Bartholomew's Hospital

<u>1839-1842</u>	<u>1849-1852</u>	<u>1859-1862</u>	<u>1869-1872</u>
82/1461	100/1918	136/2439	240/2237
5.6%	5.2%	5.8%	10.7%

Source: Forbes 1983.

and 0.3 percent at Charity Hospital in New Orleans. That rheumatic fever occurred most frequently in a region of the world that both introduced the stethoscope and organized the practice of medicine in hospitals could only have reinforced the clinical and pathological recognition of heart damage.

No discussion of the rise of rheumatic fever can be complete without mentioning the streptococcus. We know that the virulence of the streptococcus, owing in part to the phage-induced M protein, changes with time, sometimes abruptly, and that in general streptococcal diseases (child-bed fever, erysipelas, scarlet fever) were more invasive a century ago. Could it be that the rise of rheumatic fever resulted solely from biological changes within the streptococcus? There is a certain attractiveness in this hypothesis. It would explain a relatively abrupt appearance of rheumatic fever; its brisk rise could be explained as the course followed by any new infection moving through a previously nonimmune or "virgin soil" population, and its decline to a rising immunity within an "experienced" population. Unfortunately, I do not believe that it was so simple. Rheumatic fever is not an infection in the usual sense; rather, it is a host response following a relatively innocuous infection. While it was true that people died of overwhelming streptococcal sepsis following child-bed fever, erysipelas, or scarlet fever, patients did not succumb to streptococcal infection in rheumatic fever. Rather, I think it more likely that the streptococcus changed in a way that induced a host response that damaged the heart. This peculiar host response differs markedly from the host's ability to destroy the streptococcus or to be overwhelmed by bacterial infection (as is the case with child-bed fever, erysipelas, and scarlet fever).

The shift in clinical focus to the heart was not without its ironies. Fever and joint pain were the symptoms that frequently brought

patients to doctors. Yet, it was just these obvious complaints that physicians were asked to ignore, focusing instead on potential dangers, often unperceived by the patient, that had to be detected through new technological devices. This disparity between what was clinically apparent and what was pathologically relevant did not die easily. A look at how clinicians referred to the disease makes the point. Early in the nineteenth century they called it “rheumatism” or “acute rheumatism” (following William Cullen), using a term that clearly focused on joints. Well into the twentieth century (long after heart damage had emerged as the key problem), English writers used “acute *articular* rheumatism,” and French physicians “rheumatism *articulaire* aigu,” again with emphasis on the joints. Only later did “rheumatic fever” and “rheumatic heart disease” emerge as the predominantly used terms.

A similar pattern occurred in therapeutics. In the early nineteenth century, physicians treated fever and joint pain. Both patient and physician were satisfied if these bothersome symptoms were ameliorated. This pattern of gauging successful treatment did not cease with the introduction of salicylates after 1874. Fifty years later physicians still debated whether salicylates benefited carditis, in addition to lowering fever and easing joint pain, in part because there were no carefully crafted clinical studies measuring the value of salicylates in reducing cardiac damage. All investigations had determined dosing and effects on the benefits to the joints and fever.

What I would argue is that rheumatic fever arose in the late eighteenth century as the result of distinct biological changes (organism and host) that led to cardiac damage. Clinicians appreciated this alteration through assimilation of technological changes (stethoscope and autopsy), refinements in clinical thinking (“the typical case”), and the concentration of these invalids into hospitals. Quite possibly, there was also the serendipitous influence of geography and climate.

A final comment on the role of heart disease is the resurgence of rheumatic fever in the last two years. Although rheumatic fever has many clinical components, what catches the practitioner’s eye are transient, migratory arthritis; acquired heart disease; and chorea. Of these, the arthritis is pathologically insignificant. Indeed, temporary arthralgia and arthritis can accompany many conditions, and are generally dismissed. Not so with chorea and acute heart disease. Both

are dramatic and press the clinician into action. In looking at the recent outbreaks of rheumatic fever, 91 percent of children in the Utah epidemic suffered carditis (Veasy et al. 1987); in Columbus, 50 percent (Hosier et al. 1987); in Pennsylvania, 60 percent (Wald et al. 1987). In only one report (northeast Ohio) was the number of effected much lower (30 percent) (Congeni et al. 1987). While no reports specifically commented on the role of carditis in making the initial diagnosis of rheumatic fever, I suspect that acute heart disease is what caught the clinician's attention, in a way that may recapitulate the prominent role of heart involvement in the clinical recognition of rheumatic fever in the nineteenth century.

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