Case histories
Infective endocarditis

Adrian Roots

For more on **Case histories** see **Comment** *Lancet* 2016; **387:** 211, **Perspectives** *Lancet* 2016; **387:** 217, 737, 1265, 1711, 2082, 2495, and *Lancet* 2016; **388:** 228, 649

Sir William Osler—fearsomely learned, immaculately dressed, perhaps the most famous physician of his day—was not a man given to public confessions of inadequacy. In his 1885 Gulstonian Lecture he gave a bravura performance, drawing on two decades of experimental research to construct a new framework for understanding different forms of endocarditis. In his conclusion, however, he emphasised "the outlines of our ignorance" in understanding this protean disease. As Osler knew all too well, few diseases have been transformed so drastically by shifts in medical theory and practice, and few have proved so endlessly resistant to stable classification.

As the historian Alain Contrepois has pointed out, the name "endocarditis"—inflammation of the inner heart—itself encodes a radical new set of ideas about the nature of disease. In the hospitals of Revolutionary Paris young clinicians like René Laennec began to reimagine the body in localist, material terms, as a composite of different tissues and membranes, each of which could suffer distinct kinds of inflammation. These young revolutionaries sometimes found themselves looking backwards as well as forward. In his *De l'auscultation mediate* (1819) Laennec cited a 1646 case from the writings of Lazare Rivière, Professor of Medicine at Montpellier. Rivière had treated a man who complained of palpitations, swollen legs, and an irregular pulse. His patient worsened and died, and at autopsy he found small round outgrowths, "the largest of which was about the size of a hazelnut, which blocked the aortic valve". In 1802 the Parisian physician Jean-Nicolas Corvisart described "excrescences or soft vegetations" in the heart, which resembled the "cauliflower" lesions of some venereal diseases. Could this condition be a hidden consequence of syphilis?

These "vegetations", though, proved to be rare, and physicians found it hard to connect them with other signs of syphilis. The first concrete frame for endocarditis was tied up with later anatomical work on the heart and its structure. In his *Traité des maladies du coeur* (1835) the French physician Jean-Baptiste Bouillaud identified an inner membrane of the heart, naming it the endocardium. Bouillaud argued that this tissue was "the most frequent generator of those multiple organic lesions" identified by Corvisart and Laennec, but he struggled to distinguish cause and effect. Did the friction of the blood against the endocardium cause inflammation, or did inflammation cause the membrane to swell and become an obstruction? Was endocarditis a separate disease, or a consequence of phlebitis or rheumatoid arthritis?

In 1869, the Norwegian physician Emanuel Winge came up with a new answer. A patient of his died after sepsis and acute joint pain, and down his microscope he saw "parasitic organisms" blooming over the "vegetations" he found in the dead man's heart. As was so often the case in late 19th-century bacteriology, the connection of a microorganism with a particular disease was not an end but a beginning, sparking arguments. Was one strain of organism associated with the condition, or several? How did these parasites get into the bloodstream? Did they cause disease in otherwise healthy hearts, or could they only invade tissues already damaged in some way? In his Gulstonian Lecture Osler distinguished two broad forms of the disease—an acute version associated with infections, and a chronic form in which the pericardium became gradually thickened. But he acknowledged that endocarditis challenged the underlying logic of germ theory, which sought to identify one specific agent for each infectious disease. This disease seemed to be associated with several microorganisms and a range of predisposing factors.

From the mid-1940s penicillin provided the first effective treatment for infective endocarditis, but in the same period the clinical picture was becoming even more complex. In 1944 the American physician Thomas Duckett Jones showed that rheumatic fever was symptomatically similar but very different in aetiology, the result of the immune system attacking the joints and the heart after a streptococcal throat infection. Over the next generation, in an age of Cold War paranoia, and with the new specialties of paediatrics and child development seeking to gain professional authority, young patients with rheumatic fever found themselves confined to bed and sedate forms of play for months. In the light of this, we might ask a broader and more unsettling question: what has this long and complex journey of clinical elucidation meant for patients and their lives?

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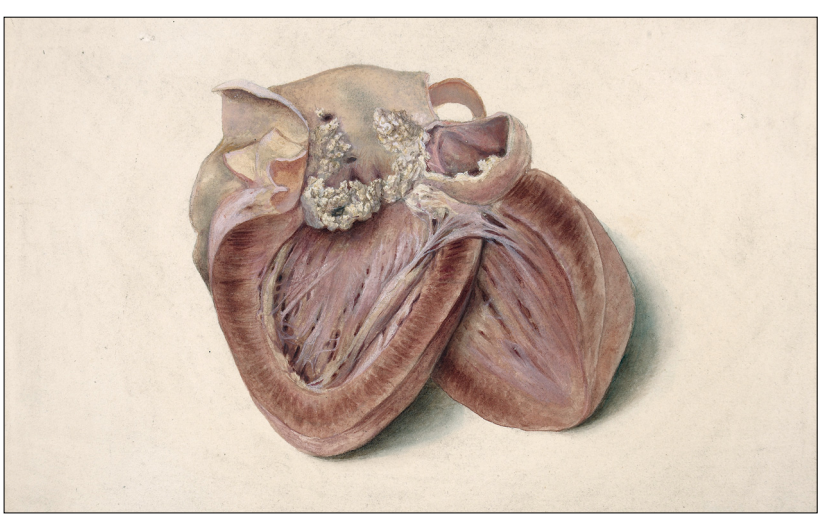
Further reading

Contrepois A. Towards a history of infective endocarditis. *Med Hist* 1996; **40:** 25–54

English PC. Rheumatic fever in America and Britain: a biological, epidemiological, and medical history. New Brunswick, NJ: Rutgers University Press, 1999

Osler W. Malignant endocarditis (Gulstonian Lectures). *Lancet* 1885; **1:** 415–18



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