

## Subacute bacterial endocarditis observed: the illness of Alfred S. Reinhart

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Medical practice is concerned with somatic abnormality, typically with disease processes that are for the most part hidden from direct observation. Internal medicine — *innere medicin'* — derives its name from this concern with what must usually be inferred from an imperfect and incomplete set of facts, both subjective and objective. The physician relies largely on what the patient conveys in a language that must itself be interpreted. One disease in which language plays a critical role in awakening the clinician's mind to its presence is subacute bacterial endocarditis (SBE), for the seat of this disease is a vital organ, whereas its manifestations are almost entirely elsewhere.

Descriptions of a particular illness by a physician who has contracted it can be particularly revealing. One example is the description by the surgeon, Sir Zachary Cope, author of a highly regarded monograph on the early diagnosis of the acute abdomen, of his own experience with cholecystitis.<sup>2</sup> However, the physician-patient's medical vocabulary can make the language issue quite complex. The physician's physician needs to be sure when the physician-patient is speaking as colleague and when as patient.<sup>3</sup>

Harvard medical student Alfred S. Reinhart had an unusual ability for self-observation combined with a gift for a vivid and picturesque turn of phrase. His observations of SBE, noted in the journal that he kept until 2 days before his death from this disease at the age of 24, were published in 1942 by Soma Weiss, the eminent Boston internist who was Reinhart's personal physician. Weiss also provided his own case notes in the same report.<sup>4</sup> Thus we can consider, side by side, 2 descriptions of the illness, one by a scientific clinician, the other by a physician-patient.

Reinhart was born in Boston in 1907, the son of eastern European Jewish immigrants.<sup>5</sup> He entered Harvard University in 1924 and worked part time as literary editor of the *Boston Transcript*. After graduating in 1928 in Government and English, he entered Harvard Medical School, where he was still a student at the time of his death in 1931. Rheumatic fever at age 13 had left him with aortic insufficiency. Between April and October 1931, SBE gradually revealed itself to Reinhart and, eventually, to his sceptical physicians. Although vascular and perivascular invasion of the left middle cerebral artery produced most of the manifestations that he experienced in the last days of his life, he

actually died of aortic valve destruction, heart failure and pulmonary edema (see text box).

Rheumatic fever was prevalent at the time. Its survivors lived with the ever-present threat of developing rheumatic heart disease and its complicating SBE. Antibiotics were an unrealized dream, and immunocompromised patients were almost unknown, such that the course of SBE was typically what its name implies: a slow and inexorable worsening.

Reinhart knew that his health was precarious; in medical school he learned about the true nature of the risks that he faced:

It was when I had acquired enough medical information to understand the clinical problems surrounding my particular case, that ... I knew there were ... three octipi [sic] ready to grab me in their tentacles at the first possible opportunity. The first of these was a recurrence of the rheumatic fever, the second was cardiac decompensation, for I was leading a very active life, and the third was subacute bacterial endocarditis.<sup>4</sup>

The description of his baseline functioning leaves us in little doubt as to Reinhart's ability to describe what he was feeling:

For ten years now, I have carried a blood pressure ranging on the average of 160 systolic and 0 diastolic, a fact, which translated into physical emotions means, especially when we consider the existence of the *cor bovinum* of aortic insufficiency, that every ventricular systole is sensed by the patient with no effort on his part, so that I might almost facetiously say that, if I did not sense the heart beating at any time, during the past ten years, I knew I was dead. The physical discomfort of being forced to experience every ventricular systole over a period of long years is not to be underestimated, and I had often felt willing to sacrifice many things in order to feel again how it was to be able to live without feeling my heart beat.<sup>4</sup>

At first he took a scholarly approach to his fears, researching a historical paper on the origins of the concept of rheumatic fever.<sup>6</sup> But a sensation of heaviness over his liver and clubbing of the fingers made him worry about the presence of endocarditis, and in January 1931 he consulted Samuel Levine, the eminent cardiologist, who examined him and reassured him that he did not have endocarditis.<sup>5</sup>

Much later that year, when his SBE had become apparent to all, Reinhart began to keep his own regular notes.

Some of them were dictated to a secretary seconded to Reinhart for a short while each day by Weiss, who recognized the young man's perspicacity.<sup>5</sup> The contrast in the introduction made by each man to these notes reveals their different perspectives, although each expresses himself with humility. Weiss uses the spare objectivity of the scientific clinician:

Much has been written on the bacteriologic and immunologic aspects and on the post mortem findings in subacute bacterial endocarditis. The symptoms and subjective behavior of the patient, on the other hand, have received surprisingly little consideration, although the complaints are many and often puzzling. It is indeed a rare opportunity in any disease to obtain an intelligent and complete story in which subjective sensations are well related to objective findings.<sup>4</sup>

Reinhart, in his emotionally sanitized style, is unafraid to say what for him is the stark truth:

#### Reinhart's illness

- 1920 Several attacks of severe tonsillitis lead to tonsil- and-adenoidectomy.
- 1921 Reinhart is admitted to Peter Bent Brigham Hospital with 2-month history of dyspnea on exertion and choreiform movements. In hospital a blowing aortic insufficiency murmur develops; blood pressure is 120/00 mm Hg. Moderate cardiac enlargement is noted.
- 1931 *April:* Acute tonsillitis develops.  
*May:* Salvos of palpitations occur on exertion; petechial shower is observed on left arm.  
*July:* Pain develops in right knee; Reinhart is admitted to Peter Bent Brigham Hospital July 23.  
*August:* Blood culture is positive for *Streptococcus viridans* Aug. 2. Splenic infarction occurs Aug. 24.  
*September:* Reinhart is transferred to Boston City Hospital Sept. 11. Enlarging spleen, anemia, painful cutaneous nodules, showers of petechiae, painful muscles and joints are noted. Another very painful splenic infarction occurs Sept 25.  
*October:* Attacks of transient aphasia begin Oct. 18. Right hemiplegia develops Oct. 21. On Oct. 26 pulmonary edema develops and Reinhart dies.

Autopsy revealed thickened, retracted aortic leaflets. There were numerous soft vegetations on the ventricular surface of the valve extending onto the aortic leaflet of the mitral valve. Three splenic infarcts were seen. There was a subarachnoid hemorrhage over the left frontoparietal region and necrosis in the left internal capsule and lenticular nucleus. Hemorrhage surrounded much of the left middle cerebral artery, and many larger vessels were plugged by older, grayish fibrinous material. Post-mortem blood culture was positive for *S. viridans*.<sup>4</sup>

These notes constitute subjective experiences and observations during the course of a generally fatal disease. It is hoped that here and there, there may be a statement, which may prove of value in the elucidation of some medical problem or of some problem involving the psychology of the sick room.<sup>4</sup>

Each man relates the onset of the SBE to the occurrence of extrasystoles in May 1931. But no other passage so clearly reveals the contrast between the view from without and the view from within as the following. First, Weiss:

In the spring of 1931 ... [Reinhart] developed showers of premature beats which lasted for ten days ... . On examination in July 1931 ... [t]here was marked visible pulsation of the carotid arteries with a thrill and a loud systolic murmur. The apex impulse was 13 cm. from the midline in the sixth space. There was a loud systolic and a high pitched diastolic murmur over the aortic area. The pulse was Corrigan in type. Pistol shots were heard over the brachial and femoral arteries, but Duroziez's sign was absent. The arterial pressure was 140 systolic and 0 diastolic.<sup>4</sup>

Now, Reinhart:

Things went along smoothly until, as I recall it, about toward the end of May. I was studying intensively for final exams at the time, when I vividly recall running across the street to my house to avoid an oncoming automobile, an act which was followed by a procession of extrasystoles unprecedented in their frequency. At the time I thought that this condition would quiet down, but instead it seemed to grow worse. Study became almost impossible, sleep became well-nigh impossible, in fact, life itself was almost intolerable due to the almost never absent extrasystoles. If I had not known exactly what they were, I should readily have diagnosed the condition as auricular fibrillation. The condition was doubly intolerable due to the fact that I was trying to study medicine at the time, and each known fact seemed literally to be hammered into my head by a cannon. I was able to devise no method by which I could obtain relief from these terrible extrasystoles, with the possible exception that at night getting up and walking around, thus increasing the heart rate, might lend some relief.<sup>4</sup>

Shortly afterward, Reinhart observed a shower of petechia on his arm, but he had difficulty convincing any physician of this because the spots had faded before he could obtain a consultation. But at least Weiss recorded what the patient had reported:

[F]ollowing measurement of his blood pressure, he noted a small circle of subcutaneous red spots which did not fade on pressure. The patient suspected subacute bacterial endocarditis.<sup>4</sup>

Reinhart, the physician-patient, never doubted what he had seen nor what their presence portended:

At any rate, at approximately one-quarter to twelve that night, I remember distinctly getting up from my chair and from the table, where my books lay, and taking off my suit coat. No sooner had I removed the left arm of my coat, than there was on the ventral aspect of my left wrist a sight which I shall never forget until I die. There greeted my eyes about fifteen or twenty

bright red, slightly raised, hemorrhagic spots about 1 millimeter in diameter which did not fade on pressure and which stood defiant as if they were challenging the very gods of Olympus. ... I took one glance at the pretty little collection of spots and turned to my sister-in-law, who was standing nearby, and calmly said: 'I shall be dead within six months.'<sup>4</sup>

His physicians thought he had a recurrence of rheumatic fever and suggested readmission. At the Peter Bent Brigham Hospital, he got a sceptical reception from the admitting residents:

As a new patient, I went through the ordinary routine of examination and investigation. The house officer examined me and asked for my chief complaint. I told him 'subacute bacterial endocarditis.' He laughed heartily as did the resident when he came up later. Both told me that they thought very little of my chief complaint and both again made the diagnosis of rheumatic fever, a diagnosis which was becoming increasingly repugnant to me, because there was so little somatically to uphold such a diagnosis.<sup>4</sup>

The residents recorded the findings of aortic insufficiency, noted above, and in addition:

a small reddened streak on the dorsum of the right thumb. No petechia were seen. The rest of the examination revealed no abnormal findings. Rheumatic fever with heart disease and with aortic insufficiency was diagnosed. The possibility of subacute bacterial endocarditis was considered.<sup>4</sup>

Over the next 10 days, the treatment for rheumatic fever failed to bring about any improvement. A splenic infarct occurred and the blood cultures were repeated. The news that returned from the laboratory must have startled his physicians, but for Reinhart it was merely final confirmation of what he knew but dreaded having to acknowledge:

I well remember the morning that Dr. D. and Dr. A. [Levine<sup>5</sup>] approached my bed with sober faces. I rather suspected that something was up when I saw them coming toward me, but I was not exactly sure what that something was. Finally Dr. A. imparted to me that the last blood culture was very suspicious. He all but said that it was positive for streptococcus viridans. Here was the last link in the chain. I had previously had inculcated into me the fact that I was going to die within a comparatively short period of time, but I could always find a leeway out. I could always find a loophole in the evidence here and there howsoever untenable I knew these loopholes to be, but now I was confronted with the *dictum ultimum* from which there was no escape. I do not exactly recall my reaction to this message from the Angel of Death, although as I remember it, I showed no emotional change whatsoever.<sup>4</sup>

In view of his continuing deterioration, Reinhart was transferred to the Boston City Hospital, where once again he was under the care of Soma Weiss:

On September 11 the patient was transferred to the Boston City Hospital ... His temperature varied from 100 to 101°F. The spleen was palpable. On September 14, 1931, tenderness and

slight swelling of the right index finger developed with a vaguely circumscribed red spot. Arterial blood pressure was 130 to 170 systolic and 0 diastolic. The white cell count of the blood was 12,000 to 16,000 per cm. Blood cultures were positive for streptococcus viridans. ... On September 25 he developed an attack of acute pain under the left costal margin which became worse on inspiration. Extreme tenderness was experienced over the spleen.<sup>4</sup>

The significance of the last finding, in the context of the overall illness, is clear to any clinician. (Although we might object that while pain is felt by the sufferer alone, tenderness belongs to the judgement of the physician, who weighs how much pressure is exerted, and where, against the patient's visible reaction and verbal report.) In any case, pain became the centre of Reinhart's universe, and for once he found himself struggling for words:

The second occasion, in which I am forced to the conclusion that my spleen has been infarcted, took place yesterday. ... There was no suddenness or acuteness about the onset. The realization was only gradually forced upon me that I had again pain in the left upper quadrant, and as before, the pain reached its maximum intensity gradually and not suddenly. However, in this second case the pain was very much more severe than in the first ... . It was absolutely impossible for me to lie down in any position, on the left side, on the right side, or on the back. One attempt to lie on the right side caused such a piercing, lancing pain in the region of the spleen that I was left with a fear of even turning on my right side for hours afterwards. It was a pain indescribable in its intensity. It was impossible for me to take an inspiration of any appreciable length. As a result, my respiration increased remarkably both in frequency and shallowness and with it my pulse rate.<sup>4</sup>

Reinhart kept up his chronicle until 2 days before he died. Weiss tells us that he became fearful, demanding and afraid to be alone. Reinhart's notes suggest that he was consumed by pain and was afraid of dying:

I felt sure that I was going to collapse momentarily from sheer exhaustion induced by lack of sleep and the intense pain. I cannot deny that I had visions of the end, nevertheless, I knew that morphine would soon take the edge off my pain ... standing orders permitting me to call for reasonable doses of morphine within proper limits of time as I should find it necessary, proved a great boon in these days. I utilized this privilege comparatively little in the past, but was only too ready to ask for one-quarter of morphine at this time to relieve the intense distress which was gripping me. This dose seemed to take the edge off the pain sufficiently to allow me to lie on my right side for perhaps ten or fifteen minutes during which time I readily fell into a sleep following the exhausting experiences of the past hours.<sup>4</sup>

With all the somatic factors in place for unavoidable death, Weiss did not waste words documenting Reinhart's final week:

Beginning with October 18 the patient developed transient attacks of aphasia and on October 21 complete right hemiplegia occurred. On October 26 he developed pulmonary edema and died.<sup>4</sup>

Today's clinicians, who have had to become familiar with SBE because of the prevalence of injection drug use, central lines and multidrug-resistant bacteria, may find these passages remarkable for their vivid poignancy and evocative description. Reinhart's nobility is also remarkable. Knowing that his life would soon draw to a close, he resolved to leave the best description for anyone who might make use of it. (At the time of the diagnosis of his SBE, Reinhart offered to go to the Rockefeller Institute in New York City to allow experimental investigation on himself<sup>5</sup>).

I find these notes remarkable for an additional reason. There is little reason to imagine that Reinhart's experience was different from that of any other patients, but his account allows the reader to know about it at a level rarely encountered in clinical practice. That open chasm between what the clinicians recorded as useful fact and what Reinhart offered (despite himself) as fact and experience is simply eye-popping. This most revealing account of the experience of SBE is an instance when language allows us to observe an illness almost as though it was ours. Reinhart's language *is* a medical one, but he generally uses it only to describe. On that one occasion when he did indulge in inference — offering a diagnosis as his chief

complaint — it was, apparently, ludicrously obvious to his physicians.

It is a truism that one can only confront the suffering of another with humility, but we physicians are supposed to *know* something about what a patient suffers. We weigh it up and use that assessment in making decisions about diagnostic testing and intervention. After studying Reinhart's notes, I can only wonder how routinely I, for one, fail truly to understand that for which I brazenly propose action.

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