Mozart's Death—The Case for Complications of Rheumatic Heart Disease

The tragedy of Mozart's premature death was compounded by his ignominious funeral and burial in an unmarked grave in St. Mark's cemetery. Ever since, the cause of death at the height of his creative powers has been a reason for continual speculation.

In most ways, Mozart was a very ordinary man. He was impetuous, fickle, and careless, but he also could be charming, cheerful, and co-operative. He was garish in his dress, pompous in his appearance and often tactless in his behavior. He was a bit of a flirt, but was not lecherous. He was ambitious but not greedy, casual but not slovenly, extravagant but not wasteful. He was also an infant prodigy who matured as a composer with few peers. He was able to visualize a complete symphonic work and then record it with few errors. During the latter years of his life he produced a major work on average once every two to three weeks.

Ever since his death at the height of his creative power, there has been almost continuous speculation as to the cause. Many have been fascinated with the possibility of deliberate poisoning, although an accidental overdose seems a more likely possibility. Other diseases suggested have included various infections, such as tuberculosis and syphilis, chronic renal disease with terminal uremia, malignant hypertension with cerebral hemorrhage, as well as leukemia, Henoch-Schönlein purpura, polyarteritis nodosa, amyloid disease, and cirrhosis. The real facts available regarding his illness and death are relatively meagre. The most reliable information

comes from letters by various family members, particularly those of his father and Mozart himself. Little was recorded at the time of his final illness, and virtually all of the information concerning this was obtained years afterwards. Although the actual cause of death will never be known, the information available points more clearly in some directions than in others.

Recently at an annual meeting of the American Heart Association in Anaheim, California, Dr. W. Virgil Brown, the president of the association, stated in his opening address that Mozart had rheumatic heart disease and almost certainly died of bacterial endocarditis (Brown 1969). While this is an over-interpretation of the facts, there is a good case to be made for rheumatic valvular disease and its complications as the cause of death. Before outlining this case, I will review the information that was available concerning this disease in the years immediately before, during, and following Mozart's death, and will make a few comments about the diagnosis and management of vascular disease at that time.

We now know that rheumatic heart disease is an entity which involves the whole heart in an inflammatory process during the acute phase, and the valves predominantly during the chronic phase. Death occurs as a result of interference with the mechanical function of the valves, emboli associated with the valvular abnormalities, or infection of the damaged valve structures. Milestones in the history of our understanding of this condition include the initial recognition of the presence of abnormalities of heart valves, the relation of these abnormalities to rheumatic fever, identification of the spectrum of involvement, separation of rheumatic from non-rheumatic valvular disease, and the identification of the specific changes related to infection of the valves.

The initial observations concerning abnormal heart valves were made in the eighteenth century. In 1705 William Cowper described thickened, narrowed aortic valves. One of his illustrations shows irregular masses associated with the valve which appear more like clots than vegetations. His observations led him to believe that these valves became incompetent making him one of the first to describe aortic insufficiency. In 1761 John Baptist Morgagni identified bony aortic valves in a patient who died with palpitations and dyspnea. He felt that such changes would lead to obstruction, or to use his words, "increase the obstacles to its exit,"

meaning the exit of blood. Jean Nicholas Corvisart in 1806 noted narrowed right-sided, as well as left-sided heart valves.

The connection between valvular disease and rheumatic fever was not made until after Mozart's death. In 1810, Wiliiam Charles Wells wrote on rheumatism of the heart, and described subcutaneous nodules, skin rash and fever, in addition to arthritis. He observed the relationship between rheumatism and the symptoms of palpitation and shortness of breath, as well as autopsy findings of pericarditis with effusion, and of "excrescences" attached to heart valves and to the lining of the heart. In 1810, René Laennec described the lesions of heart valves more fully. He suggested that there were two types: verrucous and globula. The first type

are extremely like warts, especially those of venereal origin. They sometimes resemble small cherries, and occasionally merely give the parts on which they are situated a rough or rugged surface. They adhere sometimes so strongly as to be only separable by incision, but more commonly they are easily removed by scraping. (Willius 363)

The second or globular type "resembled little balls or cysts the size of a pea to a pigeon's egg. These may contain liquid resembling half coagulated blood, more opaque matter, or yellow fluid like thick pus." These were located on walls rather than on valves. Jean Baptiste Bouillard described the pathology of endocarditis in 1835. He noted three stages: firstly, a red inflamed stage with some thickening of the endocardium; secondly, fibrinous concretions, either wart-like or globular as previously described by Laennec; and thirdly, osseous change, with or without valve narrowing. Sir William Osler helped to clarify bacterial endocarditis in the latter part of the nineteenth century. He was able to distinguish vegetations of bacterial endocarditis from those of rheumatic fever, and from other non-infective causes.

Thus disease of heart valves was only beginning to be recognized as an entity at the time of Mozart's death. The relationship of rheumatic fever to rheumatic heart disease was not considered for another couple of decades and clarification of the basic pathology of rheumatic heart disease did not occur until a half century later. The separation of bacterial endocarditis from other localized valvular lesions awaited the observations of Osler, 100 years later.

In addition, it is well to remember the rudimentary nature of clinical examination and treatment in Mozart's time. Examination of the cardiovascular system consisted chiefly of palpation of the pulse, but almost nothing was known about the causes of its variation. Even Laennec was examining the chest by direct auscultation, as his first observations on the use of the stethoscope were not made until 1819. The importance of renal function was recognized, but examination consisted almost entirely of visual observation of the urine. Treatment methods were more likely to do harm than good. Bloodletting was used indiscriminately to the benefit of few. Drug therapy was entirely empirical and the cause of much scepticism. The physician's motives were often questioned and often for good reason.

The information available concerning Mozart's medical history is sketchy. At age six he had a nodular skin rash (Wheater 586). Engagements had to be cancelled for about a week. At ages seven and ten he had episodes of apparently similar fever and leg pain. These were described by the father at the time of the second attack, in a letter dated 5 November 1766.

He could not stand on his feet or move his toes or knees. No one could come near him for four nights and he could not sleep. This pulled him down a great deal and caused us all more anxiety, since the whole time, especially towards evening, he was very hot and feverish. (Anderson 69)

Mozart's later childhood illnesses probably had no bearing on his final illness. Details of these and of fevers and colics in his third decade are given by Wheater.

He was forced to bed with his terminal illness on the 20th of November 1791. The initial symptom was swelling involving his trunk, hands and feet. His sister-in-law made him a nightgown that was open at the back as the swelling made it difficult for him to move about. There were episodes of profuse sweating as well as diarrhea and vomiting. There was dizziness and profound weakness, possibly localized to one side. Dyspnea was not a prominent feature, although it was more noticeable during the last few days. There was a skin rash as indicated by the diagnosis of "miliary fever" and this was probably confined to the trunk as it was not noted by his relatives. According to his sister-in-law, his spirits fluctuated considerably during the last two weeks of his life.

Two days before his death he felt better, but on the evening of the 4th of December he took a turn for the worse and he rallied only temporarily from this. His mind appeared clear until two hours before his death at which time he became comatose.

The nodular skin rash with reddish lesions on the buttocks and shins that occurred at age six, was almost certainly erythema nodosum, although this condition had not been described at the time. The most common cause in those days was probably tuberculosis, and this cannot be excluded in Mozart's case. However, another common cause is streptococcal infection, and this is a more likely explanation in view of the many sore throats.

The episodes of fever and leg pains that occurred at ages seven and ten were apparently almost identical. Although the description represents only one major and one minor Jones criterion for rheumatic fever, similar information is often all that is available to establish a past history of this disease. In a young man with frequent sore throats, some of which were almost certainly due to the streptococcus, the presence of these symptoms would justify a presumptive diagnosis of rheumatic fever and the recurrence would increase the probability that this resulted in rheumatic carditis with involvement of more than one valve.

Much has been made of the illnesses in the last ten years of his life. The episode of fever and chills that occurred at age 25 may have been an ordinary attack of "flu." The fever, colic and vomiting which occurred at ages 28 and 31 were somewhat more clear-cut. Colic probably means severe abdominal pain, which may have been due to renal lithiasis as has been suggested, but may simply have been a manifestation of acute gastroenteritis. Headaches and toothaches are described in the two years before his death, and the latter suggest a potential source of infection for a damaged heart valve.

The various complaints during the year before his death make it difficult to identify the time of onset of his final illness. There were many symptoms, but at the same time, he had countless professional, family, and financial problems. His letters requesting loans emphasize his complaints, whereas letters at approximately the same time to his wife suggest that he was feeling relatively well, and he was not one who was likely to hide his symptoms from his immediate relatives. He was said to appear tired and sick with heavy eyes in his last portrait, but this was an

exaggeration. Furthermore, his genius was as productive during the final year of his life as at any other time, and he was able to conduct until two days before being confined to bed with his terminal illness. While it may well be that he was feeling somewhat unwell for several months before his death, the final events occurred very quickly over the last two weeks of November and the days of early December 1791.

Although requiring considerable speculation, it is possible to explain most of the final illness on the basis of complications of rheumatic heart disease. According to this scenario, the erythema nodosum was post-streptococcal and rheumatic fever occurred on two occasions at ages seven and ten. The acute carditis was followed by the usual long asymptomatic latent period, with slow development of trivalvular disease. Dental problems were associated with recurrent bacteremia, which eventually caused subacute bacterial endocarditis sometime during the first half of 1791.

Bacterial endocarditis may progress slowly but, if untreated, is always fatal. In mid November, damage to one of his left-sided heart valves resulted in severe regurgitation and left ventricular failure developed, causing edema of the legs and trunk. At the same time, he developed cutaneous manifestations of bacterial endocarditis including Osler's nodes or Janeway lesions on his hands, which became swollen as well. The congestive heart failure was not associated with much dyspnea initially, as there was tricuspid stenosis in addition to the left-sided valvular lesions. Edema of the gut caused abdominal cramps, vomiting and diarrhea. A cerebral embolus from a dislodged vegetation caused a hemiplegia. The profuse sweating was caused by the combination of uncontrolled infection and the relatively acute heart failure. As a result of the extensive edema which kept him confined to bed, he developed thrombophlebitis and had a major pulmonary embolus on 4 December. A second embolus occurred on the following night leading to shock, coma and death.

The nature of the several illnesses which Mozart suffered during his lifetime, and the exact details of the final events will never be fully clarified. However, he almost certainly had rheumatic fever, he probably had rheumatic heart disease, and he quite possibly died of complications related to these.

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