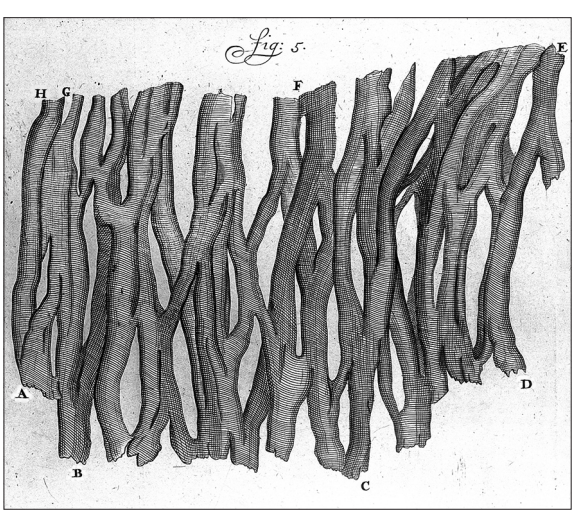
Case histories
Acute myocardial infarction

For more on **Case histories** see **Comment** *Lancet* 2016; **387:** 211 and **Perspectives** *Lancet* 2019; **393:** 2113

"You need a heart before you can have an attack", growls the gumshoe Eddie Valiant in the 1988 cartoon-noir *Who Framed Roger Rabbit*. Valiant has a point, and not just the obvious one: our contemporary concept of an acute myocardial infarction (AMI) makes little sense outside a series of historical frames in which we have come to understand the function of the heart, the connection between symptoms and underlying pathologies, and the tensions between abstract analyses of risk and the experience of a heart attack.

One strand of this story begins with the English physician William Harvey's *De Motu Cordis* (1628), in which Harvey described the circulation of the blood, drawing an analogy between the heart and a mechanical pump. If the heart was indeed a kind of pump, what happened if its pipes were blocked or its components seized up? Another strand can be traced back to 1768 and a lecture given by another English physician, William Heberden, in which he described a "disorder of the breast", typically afflicting men older than 50 years. If this "chest-choking" condition—angina pectoris—followed its usual course, "the patients all suddenly fall down, and perish almost immediately". Some of Heberden's contemporaries observed calcification in the coronary vessels of those who had suffered angina, and a new consensus emerged. Angina was caused by an obstruction, likely a clot, in narrowed coronary arteries, and such a blockage was invariably fatal.

Over the next century, this consensus became the subject of intense controversy. Pathologists noted no clear correlation between symptoms of angina and infarction of the coronary arteries at autopsy, while the effectiveness of amyl nitrite—known to be a vasodilator—in treating angina suggested a more complex aetiology. In 1912 the American physician James Bryan Herrick, more famous for his work on sickle cell anaemia, described a patient who had apparently survived AMI—an impossibility according to received clinical wisdom. Herrick suggested that the condition might be a generalised affliction of the coronary vessels rather than the result of a single blockage, but this idea (in his words) "fell flat as a pancake".



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Within a generation, however, practitioners of the emerging discipline of cardiology began to draw a distinction between AMI and coronary thrombosis. The Framingham Heart Study, begun in 1948 in Massachusetts, USA, identified a set of "cardiovascular risk factors" such as high blood pressure, smoking, and obesity. The "social medicine" developed at Oxford and Yale in the 1930s and 1940s, meanwhile, framed AMI as the most severe outcome of heart disease, a chronic condition with many causes. Newspapers, novels, and films of the 1950s linked AMI to the post-war world of consumer capitalism, an occupational hazard for stressed, sozzled, smoking, sedentary executives.

Statistical evidence of rising rates of heart disease across the industrialised world raised a new set of questions for clinicians, governments, and citizens. How to diagnose a disease that could be asymptomatic until a lethal crisis? What could be done to help those with significant risk factors for AMI, and could it be treated once it had begun? Initial Framingham reports recommended a low-fat diet, and studies of smokers showed that quitting significantly reduced the risk of AMI. Coronary artery bypass grafting, along with percutaneous techniques like angiography, balloon angioplasty, and stenting, offered the prospect of repairing damage to the heart vessels themselves, while statins and antihypertensives could be used for prevention and to protect the heart after an attack.

In the last generation, views of AMI and its aetiology have shifted once more, as clinical attention has moved towards the "vulnerable plaque" whose rupture causes downstream thrombosis. New antithrombotic therapies dissolve clots in situ, while secondary prevention strategies, including dual antiplatelet therapy and lipid-lowering therapy, are important. Social studies, meanwhile, have shown that the typical AMI patient is not wealthy and overworked but poor and deprived, and have highlighted the need to improve care for women. In most high-income nations, rates of AMI are falling, but the central challenge of this condition is that of so many chronic diseases. Researchers have identified effective interventions, but they require commitment, persistence, and—most importantly—access to services and resources.

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Further reading

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