

A story of heartache: the understanding of angina pectoris in the pre-surgical period

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ABSTRACT This paper traces the understanding of angina pectoris over two centuries from its first description by William Heberden, emphasising the Scottish dimension in this process. Such a retrospect is appropriate at a time when natural disasters in several parts of the world are drawing attention to their effect in increasing the incidence of heart attacks.

KEYWORDS History, angina, heart disease, cardiology, Scottish medicine

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In the aftermath of recent natural disasters – earthquakes in Christchurch, New Zealand, floods in various parts of Australia and the triple tragedy in Japan – a rise in the local incidence of acute cardiac events can be confidently expected. Cardiologists and emergency services become skilled in anticipating and managing the flow of patients that these disasters provoke. The evidence of such a rise is so far essentially anecdotal: newspaper reports of increased transport of heart attack victims to hospital following each of the three major earthquakes in the Christchurch series and similar accounts from Japan. It remains for the phenomenon to be documented in suitable detail.

It is timely, therefore, to consider the evolving understanding of angina. Although William Heberden and his contemporaries are credited with the earliest descriptions of the condition, Scottish medicine contributed substantially to the later elucidation of a puzzling condition.

DESCRIBING THE CONDITION

The understanding of heart disorders and their management has been challenging over several centuries. Credit for the first description of angina is given to William Heberden (1710–1801), a Cambridge graduate who practised in London for 34 years before retiring to Windsor, and attended King George III during his illness in 1788. In 1768 he read a paper on angina to the Royal College of Physicians of London and in his *Commentaries*, written in that same year for the guidance of his son and namesake (but not published until soon after his death), he described how:

Those who are afflicted with it, are seized while they are walking (more especially if it be uphill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or to continue, but the moment they stand still, all this uneasiness vanishes... The pain... very frequently extends from the breast to the middle of the left arm.¹

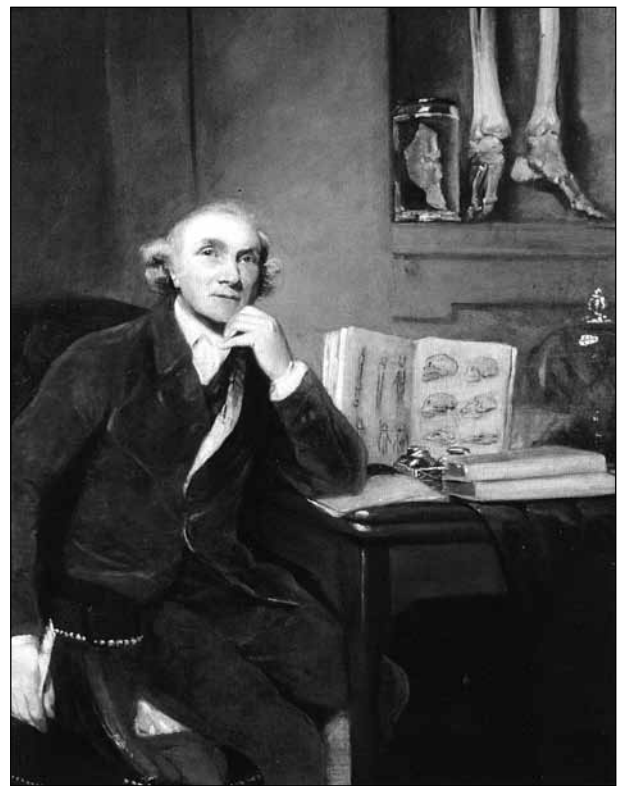


FIGURE 1 John Hunter (1728–93). Copy of original portrait by Joshua Reynolds. Artist unknown. Courtesy of the Royal College of Surgeons of Edinburgh.

The disorder, Heberden recognised, is progressive.

After it has continued a year or more, it will not cease so instantaneously upon standing still, and it will come on not only when the persons are walking, but when they are lying down, especially if they lie on the left side... The termination of the angina pectoris is remarkable. For if no accident intervene, but the disease go on to its height, the patients all suddenly fall down, and perish almost immediately.¹

In his written account, he referred only to 'a disorder of the breast' and did not, it seems, fully appreciate the origin in coronary artery disease of the condition he described.

That understanding we may credit to Edward Jenner (1749–1823), who corresponded with Heberden on the subject. 'How much the heart must suffer,' he wrote, 'from the coronary arteries not being able to perform their functions I need not enlarge upon.'² From Berkeley in Gloucestershire, Jenner also corresponded with his Bath colleague Caleb Parry, whose 1799 text on angina advanced well-documented opinions with due acknowledgment to Jenner.

Jenner's interest in the subject may be attributed in part to the cardiac history of his old teacher, John Hunter (1728–93) (Figure 1). Hunter's first heart attack was in 1772, about the time when Jenner was proposed as naturalist for Cook's second voyage but elected instead to return to general practice in his native Berkeley; the next was in 1776, when Hunter was 'extremely ill' and took himself to Bath to convalesce, just as he did the following year, when Jenner came over to see him. John Lettsom, a pioneer of resuscitation, described a later attack, in 1785:

He declares he was the other day dead for four minutes, not having pulsation in the heart or arteries.³

Jenner forbore to discuss his views on angina with Hunter, out of consideration for his old chief but Hunter himself saw his life as being 'at the mercy of any rogue who cares to anger me',⁴ which suggests that he was more aware of his situation than Jenner had believed.

When Hunter died in October 1793, his autopsy was performed by his brother-in-law Everard Home (1756–1832), who reported to Jenner that 'the coronary arteries had their branches... in the state of bony tubes which were with difficulty divided with the knife'. In his biographical sketch, included in the posthumous (1794) publication of Hunter's *On the blood, inflammation and gun-shot wounds*, he reiterated these findings.⁵

Home would also have discussed these appearances with his fellow-executor, Hunter's nephew Matthew Baillie (1761–1823). Baillie's own publication *Morbid anatomy* had its first edition in 1793; it may be conjectured that Home's information was responsible for the inclusion of the statement that 'ossification of the coronary arteries would seem to produce the symptoms of angina pectoris' in the second, 1797 edition. Baillie's work has earned the comment of that fastidious anatomist-bibliophile Kenneth Russell: 'his book is one of the greatest contributions made to pathology by an Englishman.'⁶

An Englishman: if we reflect that Baillie was descended from Lanarkshire farming stock on his mother's side, and

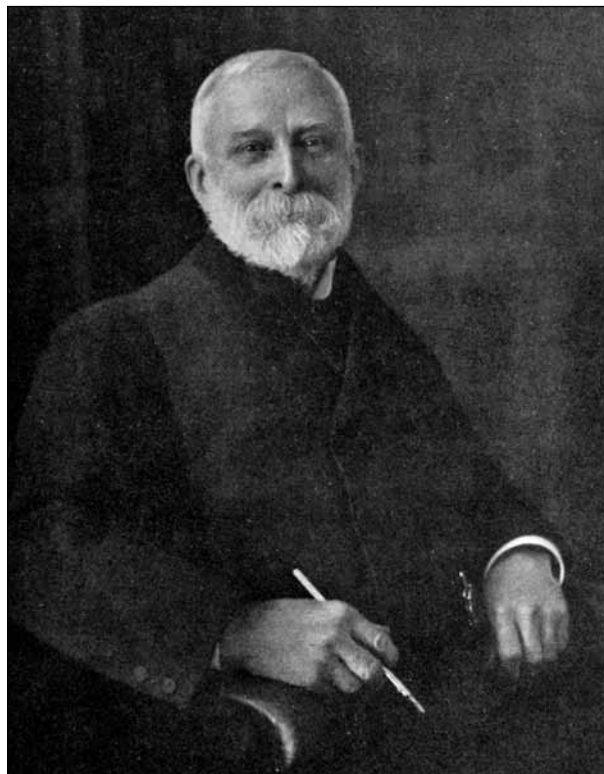


FIGURE 2 Thomas Lauder Brunton (1844–1916). RCPE Collection.

that his father was professor of divinity in Glasgow, we have to perceive a certain looseness of terminology even in that most meticulous of Australians. Indeed a significant role in the unravelling of the puzzle of angina has been played by the Scots, even though a number have, like John Hunter himself, migrated south as part of the great Scottish takeover.

MANAGING THE CONDITION

Contemporary with Jenner, and like him a country general practitioner, was the physician to whom we owe the earliest specifically cardiac drug: William Withering (1741–99) was the son of a Shropshire country doctor, who graduated from Edinburgh in 1766. Guthrie wrote of him:

Like Jenner... the inspiration for his great discovery came from folk medicine. Withering was aware the country folk of Shropshire used a decoction of foxglove (foxglove tea leaves) as a cure for dropsy. He showed that the dropsy might be due to cardiac disease, and that digitalis, if carefully used and stopped if any nausea was caused, was an excellent remedy.⁷

Allan Burns (1781–1813) was, like Matthew Baillie, a Glaswegian, who documented the progression of angina through the further stages of dyspnoea and heart failure, in 1809.⁸ But thereafter, it seems understanding of the significance of angina progressed very little for a century. Its management likewise remained empirical, but in 1867

a young Edinburgh houseman, Thomas Lauder Brunton (1844–1916) (Figure 2) observed that bloodletting eased anginal symptoms. Brunton, who progressed to St Bartholomew's Hospital and a baronetcy was apt to be ahead of his time; his later suggestion, in 1902, that mitral stenosis might be treated surgically was received with derision.⁹ He was compassionate as well as far-seeing. Lord Lister fell into a state of melancholy after Lady Lister's death. To rescue him from this, Brunton secured for him the office of Foreign Secretary of the Royal Society, in an exercise of great diplomacy.¹⁰

Brunton's observation of the benefit of bloodletting led him to speculate that lowering the arterial blood pressure would be beneficial and in turn this prompted him to apply the finding of his colleague Arthur Gamgee (1841–1909), that inhaling the vapours of amyl nitrite lowered the blood pressure in an animal. Administration of amyl nitrite to Brunton's anginal patient was highly effective.¹¹

Equally effective was the explosive nitroglycerine (glyceryl trinitrate) though its potential for causing severe headache affected its acceptance. So did its explosive qualities, until it was appreciated that these could be avoided by the use of fatty or oily, not alcoholic, solutions. Adjusting the dosage dealt with the problem of headache and nitroglycerine became the drug of choice, though as late as 1952, *Martindale: the extra Pharmacopoeia* counselled that 'its use should be avoided in coronary thrombosis'.¹²

There is a phenomenon in medicine in which a condition whose mechanism is not understood will be derided as functional. It would appear to be this process that led Byrom Bramwell (1847–1931) to class angina under 'cardiac neurosis'¹³ in *Diseases of the heart and thoracic aorta*, published in 1884 not long after he had moved from Newcastle back to his alma mater, Edinburgh. Yet he was described as 'a born diagnostician and a brilliant clinical teacher' and in spite of his scepticism in the matter of angina, he was knighted in 1924.

A SUBJECT FOR SPECULATION

Writing on the evolution of cardiology in 1968 in the centenary volume of *The Practitioner*, Sir Ian Hill (1905–1982), a past president of the Royal College of Physicians of Edinburgh, noted that it was Herrick in 1912 and 1916 who first described myocardial infarction as a clinical entity, and recalled that Osler:

... lecturing on the subject [of angina] in 1912, said that the disease was not one about which one should lecture to undergraduates or recent graduates, since a doctor was likely to have attained the Fellowship of his College before he saw his first case.¹⁴

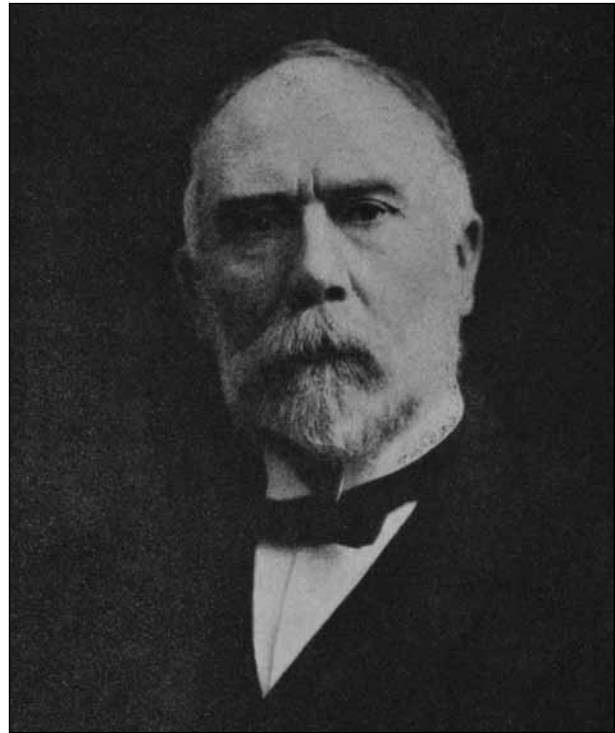


FIGURE 3 Sir James Mackenzie (1853–1925). RCPE Collection.

And of Mackenzie, he commented:

...in his textbook on angina pectoris, refers to some 200 odd patients he had seen. The point is that the increase in frequency of ischaemic heart disease is real. Osler and Mackenzie were not clinical fools: if their patients had had ischaemic heart disease they would have recognised and recorded it. But even these great clinicians were blind to the difference between angina and myocardial infarction... In Mackenzie's book on 'Angina Pectoris', published in 1925, while there are case histories characteristic of myocardial infarction, neither that term nor coronary thrombosis is even mentioned.¹⁴

Sir James Mackenzie (1853–1925) (Figure 3) was born in Scone and educated in Edinburgh and he practised for a good many years in Lancashire. Guthrie has described how:

He applied himself to... the mechanism of pain and the significance of irregularities of the pulse. One of the results of his investigations was to alter the attitude towards abnormal heart sounds (murmurs) and abnormalities of the heart's action, neither of which, he submitted, need be viewed with alarm. The important factor in prognosis was the ability of the heart to react to increased effort; the condition of the myocardium should be the main guide in framing an opinion as to the outlook.¹⁵

Indeed in 1916 Mackenzie had described angina as 'exhaustion of the heart', writing that 'angina develops with great severity and ends speedily in death'. 'On the whole,' he remarked, 'these cases are rare.'

REVELATION

Reverting to Sir Ian Hill's reminiscences:

The first cases described in this country were in 1926 by McNee [Sir John, 1887–1984, regius professor of medicine in Glasgow 1936–53] and again by Carey Coombs, and I personally saw my first patient with myocardial infarction as a house physician in 1928.

The diagnosis was made by a young clinical tutor, A Rae Gilchrist, and the condition was one of which neither my Chief nor I had ever heard.¹⁴

This was indeed the first clinical diagnosis of myocardial infarction confirmed at necropsy in Edinburgh.

On that basis, Gilchrist's 1930 paper on the subject must be regarded as trailblazing. Two decades later Cameron would comment, looking back over the scene:

Jenner must... have been one of the first to give a correct solution of the problem. *Certainty in this matter was reached only as recently as the last twenty years.*¹⁶[my italics]

Gilchrist himself was a notable pioneer of cardiology, and remains a significant figure in the history of his specialty.

He graduated in 1921 and was appointed to the senior staff of Edinburgh Royal Infirmary in 1930, becoming physician in charge in 1939. By 1953 he had established a department of cardiology with its own outpatients. He was a brilliant teacher, and six of his protégés went on to occupy chairs of medicine or of cardiology. He was president of the Royal College of Physicians of Edinburgh from 1957–60, and was made CBE in 1961.

In 1959 he was admitted to honorary Fellowship of the Royal Australasian College of Physicians in its 'coming of age' year when, for the first time, a joint plenary session with the Cardiac Society of Australia and New Zealand was held at the annual Meeting of the College.¹⁷ Cardiology might be said to have 'arrived' in the Antipodes.

The management of coronary disease, however, was to remain exceedingly cautious for a generation or more. In the early 1950s the British Medical Association published a series of articles, subsequently issued in book form, entitled *Refresher course for general practitioners*. The second volume, covering the period 1951–52, included an essay by Gilchrist, at this time Reader in clinical cardiology in Edinburgh. In it he

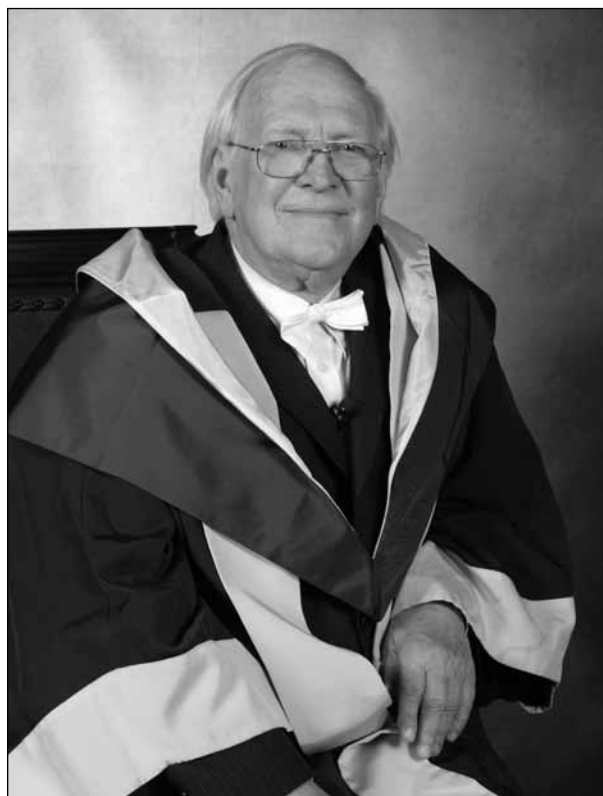


FIGURE 4 Sir James Whyte Black (1924–2010). Image courtesy of Dundee University.

recommended a week or ten days of bed rest for an episode of coronary insufficiency, but for a frank coronary thrombosis contended that:

Rest in bed must usually be maintained for six weeks or thereabouts, and the more complete the inactivity during the first three or four days the better. During this time the patient should do nothing for himself. He is to be fed and washed; visitors must be excluded and sleep ensured... Among hospital patients, treated as we believe under the best conditions, conservative measures yield a mortality rate of 33% in this country.¹⁸

In 1960, surgical treatment of coronary artery disease began, with no great early success. And into this fertile ground of arduous medical management and hazardous surgical treatment came, in 1964, what has been described as 'the greatest breakthrough in the treatment of heart disease since the discovery of digitalis'.¹⁹

James Whyte Black (Figure 4) was born on 14 June 1924 in Uddington in Lanarkshire; he spent his childhood in Fife and went on a scholarship to the University of St Andrews, attending his classes at University College, Dundee and graduating in medicine in 1946. He lectured in physiology in Dundee and later in Malaya and the veterinary school in Glasgow, where he studied the effect of adrenalin on the heart, especially in angina, before taking a position at ICI Pharmaceuticals to give scope to

this burgeoning interest in pharmacology. His 'invention' of propranolol, a beta-blocker synthesised deliberately for a purpose (rather than being produced and then tested for possible therapeutic effects) was the first of two particular discoveries – the other was cimetidine for peptic ulcer – that established his fame and brought him the Fellowship of the Royal Society in 1976, a knighthood in 1981, the Nobel Prize in 1988 and admission to the Order of Merit in 2000. But one honour in particular he treasured: after his early association with Dundee, it became a university in its own right, and he served as its Chancellor from 1992–2006.

REVOLUTION

The evolution of the management of coronary artery disease over the past half-century, from the rigorously conservative approach of the 1950s through the beta-blocker revolution and on to one in which patients are commonly entrusted to what used to be called surgical interference – or else, more recently, to stenting by an interventional cardiologist – has been so dramatic as to amount to a story of its own.

Another change is in the terminology of heart disease, and in a suspicion of the very term 'angina' which, as Professor Kim Fox is on record as observing, can 'blight a patient's life' because of the sinister overtones it has acquired. It has become fashionable instead for cardiologists to speak of 'ischaemia of cardiac origin' which certainly demands interpretation before it reaches the patient. The change may be regretted, in that 'angina' has a long and hitherto reputable history, coming from the Greek *ankhonē*, meaning strangling, related to *kunankhē*, to throttle a dog, which gave us quinsy (through mediaeval Italian) for inflammation in the throat. Cardiac angina necessarily became 'angina pectoris'.

But if all that sounds puzzling, let the final word on puzzles go to Sir David Weatherall in his account of the challenge that heart disease presents.

The diagnosis is suggested by the patient's story of chest pain. It may be confirmed by changes on the electrocardiogram that are present when the patient first appears. A few days after the episode the products of the damaged heart muscle are shed into the bloodstream, where they can be easily measured. Thus at least the diagnosis of a heart attack should be reliable, based as it is on well-defined principles of electrophysiology and biochemistry. Unfortunately, things are not as simple as this. First, there is no one, infallible test to tell a doctor that a patient has had a heart attack, certainly not if seen shortly after its onset. While the patient may complain of crushing central chest pain, very often the pain is in a different place or has the wrong characteristics, or both. It may, for example, be localised to the abdomen, the

back, the shoulder, or the left side of the chest. Even for the most experienced cardiologist, the distinction between genuine cardiac pain and pain arising from other causes may be extremely difficult or impossible to make. Some patients are unreasonable enough to have heart attacks with no pain whatever.²⁰

Winston Churchill once defined Russia as 'a riddle wrapped in a mystery inside an enigma'.²¹ Something similar might be said of heart pain.

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