

News, Notes and Queries

JAMES HARDY AND THE DEVONSHIRE COLIC.

JAMES HARDY figures little in most reports of the Devonshire Colic. Instead, authors have concentrated mainly on the role of Sir George Baker in the discovery of the cause of the disease. ^{6, 18, 19, 28, 29} The work of Baker is undoubtedly of the first importance when considering the history of the Devonshire Colic and his essays published in 1767 and 1768, ^{3, 4} discussing the results of his experiments on the Devonshire cider are rightly described as masterpieces of inductive logic.⁸

Baker showed irrefutably that the disease was a form of lead poisoning and he postulated that the poisoning resulted from the drinking of cider contaminated by lead in the cider pounds and presses. The incorporation of lead into the cider-making apparatus was, according to Baker, almost solely confined to the county of Devon and explained why the disease only seldom occurred in the other cider counties of Hereford, Worcester, Somerset and Gloucester.

Baker's 'leaden hypothesis' aroused considerable resentment in Devon. The cider makers were then recovering from the ill-effects of the Cider Tax which had been repealed in 1766, the year before Baker's first essay was published, and they were anxious in case his report resulted in another decline in trade. A number of tracts were prepared purporting to disprove Baker's theory and show that the Devonshire cider was uncontaminated. ^{1, 2, 9, 11, 12}

As was customary in the eighteenth century, the debate was conducted in a forthright—often libellous—manner. According to one contemporary source, it was 'combated with a Degree of warmth, almost unaccountable to those, who deeming themselves uninterested in the Dispute, do not consider that the Opponents believed the Honour of their County depended on the Confutation of Sir George Baker's arguments.'²⁶ There is no doubt that the opponents of Baker's theory felt compelled to act in the main for financial rather than scientific reasons. They were often remarkably candid on this point. Thomas Alcock, one of Baker's principal adversaries wrote:

I am afraid the principal point advanced in this essay will tend . . . to injure . . . our property; will either frighten the cities of London and Westminster, and other distant towns and places, as well as many of our own people, from drinking any cyder at all: or send them to Hereford and its adjacent Counties for all the liquor of this kind, which they may chuse to purchase. It seems as necessary, therefore, for the good of our West Country Plantations, to get this doctrine speedily refuted, as it was to get the cyder act repealed.³

Modern historians have been inclined to suppose that the opposition to Baker's theory was ineffective, but there is some evidence to suggest that this was not the view of Baker's contemporaries. For example, after the publication of Alcock's second tract,³ the *Monthly Review*, which had previously supported Baker, wrote: 'Upon the whole we think the accusation which has been brought against the Devonshire cyder, is rather plausible, than supported by the clear authority of facts.'²⁰ And a few years later the *Review* had this to say: 'Readers cannot be unacquainted with the controversy between Sir George Baker and Dr. Geach and Mr. Alcock. . .

The testimony adduced by the others (Geach and Alcock) against the possibility of . . . an impregnation in many instances where the colic had appeared, seemed rather to give a preponderancy to their side of the question.²¹

It was in an attempt to settle the issue decisively that James Hardy reinvestigated the cause of the Devonshire Colic. The results of his work were set out in a tract published in 1778.¹⁴

Hardy began his investigation by supposing that although Baker had assigned the true general cause to the Colic, that is, lead poisoning, he had been wrong in suggesting that the lead came into the cider from the machinery. Geach and Alcock had, said Hardy, 'proved beyond all contradiction . . . that the general cause of the endemial colic of Devonshire could not arise, or be produced, from any particles of lead conveyed into the cyder from the pounds or presses.'

It was evident that another general means had to be found whereby lead could contaminate the cider and Hardy proposed that it was due to the almost universal use of lead glazed earthenware in Devon. The amount of lead in the glaze was considerable, one ounce of lead-ore being used in each quart of glaze.

Having put forward a hypothesis, Hardy, like Baker before him, devised a set of experiments to test it. In all he conducted twenty-five separate experiments in which the effects of standing and boiling cider and other liquids in a series of earthenware, glass, tin and tinned vessels were studied. In particular, the uptake of lead from the various vessels was measured using a test solution of orpiment and quick-lime in water. When this solution is applied to one containing lead, the lead is precipitated out of solution as lead sulphide. The test is sufficiently sensitive to detect the presence of lead in a concentration of 1 mgm./litre. Hardy was a careful experimenter who understood well the necessity of proper controls and the following experiment serves to illustrate the methods he employed in his investigations.

Experiment I. A quart of must, fresh from the pound, stood in a glazed earthen vessel, without being agitated, six hours. Upon the application of a few drops of the test to a glass of the must, a reddish cloud was produced. After standing nine hours, the like application produced a deeper cloud. After standing twelve hours, the cloud was yet more deep; and in a little time, the must became opaque. After twenty-four hours, a deep, almost liver-coloured cloud was produced; which, on being stirred with a small piece of wood, instantly occasioned that cloud throughout the whole.

I remarked that it did not seem of much importance, whether the test was used in the quantity of only five or of ten drops: As the degree of colour it produced, it seemed to depend, solely, on the quantity of mineral particles with which the liquor was impregnated.

No adulteration was produced by an addition of the like quantity of the test to a glass of the same must, which had been preserved in a bottle.

After completing his experiments Hardy noted that when the cider was allowed to stand in glazed earthen pots it became rapidly and heavily impregnated with lead and that the uptake of lead from the glaze occurred more quickly if the liquid was agitated or boiled in the pot. He found that several other liquids could also take up lead from the glaze, including water containing cream of tartar and lemon juice, small beer, grape juice, vinegar-water, honey-water and sherbet. In each case the impregnation was heavier if the liquid was agitated or boiled in the vessel.

The results of his experiments led Hardy to conclude that 'the certain general cause of the endemial colic of Devonshire, is by them clearly demonstrated'.

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As a result of his investigations Hardy felt that he was able to give explanations to several puzzling features of the disease. For example, why some families in a particular locality contracted the disease and not others; why the poor generally suffered from the disease more than the rich and why Devonshire was more afflicted than her neighbours. It was simply that glazed earthenware was used in one family and not another, that the rich tended to use glass and stone vessels and the poor glazed pots, and that the use of glazed vessels was more frequent in Devon than the other cider counties.

Hardy did not wish to claim originality for his hypothesis, indeed several authors had already pointed out the dangers inherent in the use of glazed earthenware. Amongst these was Sir George Baker who had examined this point in the second of four remarkable papers which he read before the College of Physicians in 1767 and which were subsequently published in the *Medical Transactions of the College* the following year. Examining the several ways in which lead might enter the body Baker wrote:

Vinegar, boiled with the glass of lead, or in the glazed earthen vessels, the glazing of which is principally lead, becomes strongly impregnated with the pernicious qualities of the metal, and yields, on evaporation, a true *saccharum Saturni*. But this glazing is very considerably acted upon even by cold vinegar. And hence it is manifest, that the custom, which I apprehend to be too common, of keeping pickles in such vessels, cannot but be dangerous to health.⁵

However, according to Hardy, little attention was paid to this paper of Baker's, or indeed to the others he published that year, because Baker's hypothesis concerning the cause of the Devonshire Colic had been overthrown by Geach and Alcock. Polwhele, in his *History of Devon*,²⁴ remarked that Baker had drawn ridicule upon himself by the publication of his 'leaden hypothesis' and it was Hardy's principal aim to show that Baker *had* shown the true general cause of the Colic. Hardy's own tract was intended only to serve as a commentary on Baker's essay by demonstrating the true manner in which the lead hazard arose. However, Hardy's tract is of more significance than this because of the attempt he made to present evidence which was not speculative, but based on experimental evidence.

After the discussion of his experiments, Hardy devoted the major part of the remainder of his tract to a scholarly account of the history of the adulteration of wine by lead. Of particular interest is his account of this practice in Ancient Rome. Wine was commonly evaporated in leaden vessels to sweeten it although the ill-effects of such a practice were well known, Pliny writing, for example, that 'from the excessive use of such wine, arise dangling, enervated knees and paralytic hands.'^{*}

Hardy noted that an endemic disease, not unlike the Devonshire Colic, had been described by Paulus Aegineta as having raged throughout Italy and other parts of the Roman Empire, but he did not discuss the possible effects that the widespread drinking of lead-contaminated wine might have had on the subsequent history of the Roman Empire. It is interesting, nevertheless, to read Hardy's account in the light of a recent 'new hypothesis' which attributes the Fall of Rome to just this cause.¹³

At the time Hardy was writing, an endemial colic was prevalent in the province of

^{*} Hardy's translation, contained in the 1778 tract.¹⁴

New Castile in Spain. Since it was the practice in Spain to boil wine, and since glazed earthen vessels were widely used, Hardy suggested that the disease might have origins similar to those of the Devonshire Colic. To test this hypothesis, Hardy suggested that a Royal Commission might be set up. 'Their most Christian and Catholic Majesties would confer on their subjects, the most humane and essential obligations, by appointing a committee of physical gentlemen, with express directions to examine into the cause in those provinces where the disease is most prevalent, and to report the actual fact, in order to guard them effectually in future.' It is doubtful, however, that this novel idea of Hardy's was acted upon.

Hardy considered that the adulteration of wine with lead, whether accidental or intentional, was so widespread that he was sure many cases of paralysis and gout which presented themselves to the physicians would, on close investigation, show themselves to have this single cause. He was careful, however, to point out that he was not wishing to ascribe *all* paralytic complaints to the same cause. Nevertheless, in those cases where paralysis was preceded by obstinate constipation and bowel pains and by pains in the limbs, then he was convinced that 'the admission of lead into the body, in some shape or other, is the only absolute original cause of the disease, and its paralytic consequences'.

It was widely held by the eighteenth-century physicians that the gout was caused by putrid bile gaining admission to the blood stream. This had been suggested as the cause of the Devonshire Colic by Huxham¹⁷ and was still held to be true by Baker's opponents. Hardy was scornful of this theory, based as it was on pure speculation. Like Baker, Hardy was concerned to base conclusions on experimental evidence and to this end he had another revolutionary idea to suggest, namely:

That a certain course of experiments be tried on convicts, under approved limitations, sufficient to demonstrate the truth, or falsehood, of what has been here advanced . . . Then we shall learn, that the *primary* causes of the gout, are infinitely less complex, than they have hitherto been supposed; and then we shall have the melancholy satisfaction of knowing, that had our predecessors employed themselves, in the arrangement and investigation of facts only, instead of raising a temporary brainsick theory, this formidable and painful disease would have been many ages since almost annihilated.

Hardy's suggestion that convicts might be used experimentally provoked a sharp rebuke from a writer in the *Monthly Review* who said, 'We should be very unwilling to have the Faculty loaded with the Odium of racking a man with the colic, or crippling him with the palsy or gout.'²¹ Nor was the reviewer impressed with any other part of Hardy's thesis. He thought it doubtful that the Devonshire Colic was caused through the use of glazed earthenware or that the other cider counties were spared from the disease because the glassware was used more extensively than in Devon, a fact for which the reviewer saw little evidence. Hardy's proposition that lead-contaminated wine caused gout was also unfavourably received and was likened to a syllogism of the form:

Lead has always been used to contaminate wine;
Wine drinkers have always been susceptible to gout;
Ergo, lead causes gout in wine drinkers.

A lengthier denunciation of Hardy's view concerning the origin of the gout was

contained in a pamphlet published in 1778²⁵ by Francis Riollay.* Riollay writes that Hardy's observations on the Devonshire Colic 'throw . . . upon that disorder a greater degree of light than it had ever received before' and deserves 'the approbation of Physicians, and the thanks of the Public.' But as for drawing an analogy between the cause of the Colic and that of the gout, Riollay says, 'to me, indeed, it is a subject of wonder, that you did not reject the idea as soon as it presented itself to your imagination'.

Riollay, of course, had no experimental data on which to base his objection to Hardy's theory and his tract consists of a number of subjective arguments. He notes that most people take wine daily, the greatest part of which he supposes to be adulterated, and yet many of these never succumb to the gout. On the other hand, in Burgundy, where the inhabitants take great pains to ensure that the wine is unadulterated by personally supervising all aspects of its production, still they are visited by the disease 'early, often and long'.

Riollay also contrasted the gradual onset of the gout with the suddenness of an attack of lead poisoning. He was quite unable to accept that lead poisoning might manifest itself as a chronic illness, indeed he even doubted that lead was absorbed from the gut at all since, he wrote, its properties were 'to coagulate and constrict, and thence its deleterious effects in the alimentary canal'. Thus, remarks Riollay naively, 'as long as our liquors produce no complaints in the stomach and bowels, we may account ourselves perfectly safe from every effect of adulteration'. A more ill-conceived and dangerous notion can scarcely be imagined! Riollay concluded that adulterated liquors could never be reckoned as the *sole* cause of the gout (a proposition Hardy had not, in fact, put forward) instead, it was caused by the 'ennervating influences of fermented or spiritous liquors in general when indulged in too plentifully'.

Riollay's attack on Hardy embodied all the most conservative elements of eighteenth-century medicine, being founded on pure speculation, and he stands in sharp contrast to Hardy who, taking Sir George Baker as his mentor, strove to base hypothesis on experiment. But the *Monthly Review* which had damned Hardy, greeted Riollay's tract with enthusiasm: 'When Dr. Hardy printed his hypothesis that lead taken internally was the cause of the gout, we thought it too manifestly extravagant to excite any public notice. 'It costs [Riollay] little pain to refute Dr. Hardy's idle notion; which he does by a few remarks.'²²

Hardy, naturally, produced a counter to Riollay's attack.¹⁵ It consisted mainly of a point-by-point refutation of Riollay's arguments, but from it emerge some indications that Hardy's insight into lead poisoning was well in advance of most of his contemporaries, and yielded little to that even of Sir George Baker who was the acknowledged master of the profession.

Hardy had no difficulty in accepting the concept that individuals differed in their susceptibility to lead as Heberden¹⁶ had earlier pointed out, and—more significantly—he was well able to conceive that lead poisoning could be caused by the most minute doses of the metal.

* Francis Riollay (1748–1797) was of French origin. He was born in Brittany and educated at Trinity College, Dublin. He practised in Newbury and received his M.D. from Oxford in 1784; the following year he was elected a Fellow of the Royal College of Physicians.²³

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If the 30th part of a grain of that wholesome mineral iron, diffused or dissolved in one pint of water, and taken daily for a few weeks, can produce the most salutatory effects, by its insensible operation on the human body; does it seem absurd or even difficult to believe, that one half of the same quantity of a noxious mineral suspended or dissolved in wine, and taken occasionally for a number of years, should ultimately prove injurious? We cannot demonstrate by what means the inconceivably minute portions of the first mentioned mineral bring about their salutatory effects; yet no one doubts the fact, because repeated observation has confirmed the truth: why then should we not extend the like mode of reasoning to the action of noxious minerals?

A considerable part of the opposition to Baker's theory concerning the cause of the Devonshire Colic was brought about because the detractors of the theory were not able to believe that the small amounts of lead which Baker found in the cider were sufficient to produce the dramatic symptoms of the disease.* Hardy was one of the few who found no difficulty in accepting that they could.

Practically nothing seems to be known of Hardy apart from the information to be obtained from his two tracts. Even the dates of his birth and death are lost. The only mention of him in a work of reference is an unkindly one contained in Chalmers' biographical dictionary where the author alludes to him in a discussion of the Devonshire Colic saying that the arguments Hardy put forward were 'rather feebly supported'.⁷

There is one further reference to Hardy in the *Devonshire and Cornwall Notes and Queries* where it is stated that John Watkins, in his *Universal Biographical Dictionary*, alleged that Samuel Badcock wrote one of the tracts attributed to Hardy.¹⁰ I can find no trace of this statement in any edition of Watkins' *Dictionary* which I have examined and it is difficult to believe that there is any truth in this curious remark.

In his second tract Hardy wrote that he had intended to publish some observations on the cure of the Devonshire Colic and the gout, to which end he had been working for twelve months. Since it seemed that almost every patient required a peculiar mode of treatment, Hardy was not confident that he would be able to fulfil this ambition, and certainly no such tract appears to have been published.

Whether Hardy had any direct influence on the course of the Devonshire Colic is impossible to say at this distance, but since it is evident that his work was not held in very high regard, it is unlikely. After the publication of the second tract in 1780 little was written about the disease until such time as it had become a matter for the historian to discuss. There is little clear evidence to suggest what factors led to the disappearance of the Colic. It is even questionable how far Baker's views were responsible since he too was widely rejected (on this point) in his own time. It is most likely that the disease vanished as technological improvements were introduced into the cider-making industry—iron engines were already superseding the stone presses in Baker's day, and as glass or pottery took the place of glazed earthenware, contamination from this source would also have disappeared.

However, James Hardy is a figure of some considerable interest to the historian

* Baker extracted lead quantitatively from the Devonshire cider in two separate experiments. The amounts of lead recovered give concentrations equivalent to 4.75 and 14.25 mgm/litre. Although these figures cannot be considered as absolutely accurate, nevertheless, it is interesting to compare Baker's results with one obtained recently when the lead content of some home-made wine which had caused lead poisoning was determined.¹⁷ In this case the lead concentration was 7.5 mgm/litre, very nearly the mean of Baker's results.

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although the picture we have of him is shadowy. He was a product of his time, erudite and well versed in the classics, but, most important, he was keenly aware that the advance of medicine depended on the use of rational, scientific methods. It is doubtful if, as a humble country practitioner—he worked in Barnstaple—he was able to affect the development of medical science to any great degree, but it is important to realize that it is not only in the great centres of learning and influence that men with far-sighted ideas are to be sought. James Hardy is one who thought and worked in relative obscurity, had he been better placed, his contribution might well have been of considerable significance.

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THE ELECTRON MICROSCOPE IN PALAEOPATHOLOGY

INTRODUCTION

THE earliest palaeopathological studies were made in the eighteenth century and were concerned with the results of trauma and disease in fossil animals. In 1825 Granville made a careful macroscopic study of a Ptolemaic mummy and diagnosed ovarian dropsy: this was almost certainly an instance of cystadenocarcinoma of the ovary. Twenty-seven years later Johan Czermak, a distinguished Viennese laryngologist, made the first microscopic studies of ancient mummified tissues. He employed simple but effective methods, teasing out the tissues in a solution of caustic soda. He was also a pioneer, being the first person to use a micrometer in the study of such preparations. The light microscope was therefore employed more than one hundred years ago in palaeohistological investigation.

A long fallow period followed, broken only by some obscure investigations made by Fouquet in 1889 (Moodie 1921) until in 1904 Wilder made sections of Peruvian mummy and dried Utah Amerindian bodies following rehydration of the tissues in a solution of caustic potash. Not long afterwards Shattock (1909) made frozen sections of portions of the calcified aorta of the Pharaoh Merneptah given to him by Grafton Elliot Smith. At about this time Ruffer commenced his classical palaeopathological studies in Cairo (Sandison 1967b). He issued a series of papers from the year 1910 until his tragic death at sea in 1917. Those published in 1909, 1910, 1911 are of histological interest. They may readily be consulted in the collected works edited by Roy Moodie (1921). Ruffer employed a rehydrating fluid which contained alcohol and sodium carbonate and which is still used today (Sandison 1963b).

Further light microscope studies came from Wilson (1927), Williams (1927, 1929), Aichel (1927), Simandl (1928), Shaw (1938), Busse-Grawitz (1942), Gürtler and Lanegger (1942), Graf (1949), Schlabow *et al.* (1958), Rowling (1961) and Sandison (1955, 1957, 1959, 1962, 1963a, 1963b, 1967a, 1967b, 1968). The majority of these studies were made on Egyptian mummies but others included Egyptian Canopic material, and tissues from Peruvian mummies, a Guancho body, Amerindian bodies, German and Scandinavian *Moorleichen*, and Scandinavian and British skeletal material. For an assessment of these investigations reference may be made to Sandison (1963b).

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The light microscope suffers from an inherent limitation, i.e. that although empty *magnification* may be obtained by manipulating the optical system there is a limit to