William O’Shaughnessy and the forgotten cure for cholera in the 1832 British epidemic

GEOFF GILL*

Introduction

The diseases commonly prevalent in 19th century Britain are like something from a modern textbook of tropical medicine. They included malaria, smallpox, tuberculosis, typhoid, typhus, dysentery and cholera. Many medical, social and climatic factors were responsible for these disease processes, but overcrowding and poor sanitation were the most important factors. The majority of these infective illnesses were endemic i.e. they were constantly present, but some were epidemic, occurring in sudden and time-limited outbreaks. Of these, cholera was by far the most important, dramatic and feared.¹ Part of the reason for this was that cholera was unknown in Britain until late 1831, when the disease was introduced into Britain from the European mainland. Between then and 1866, there were four major cholera epidemics, following which the disease disappeared from Britain. The epidemics were dramatic, spread rapidly, and were marked by significant morbidity and mortality.

This paper will examine cholera as a disease, particularly in the context of the British 1831-32 epidemic. It will then focus on the therapeutic inertia which pervaded the medical treatment of cholera, and the emergence of what may be regarded as a “lost cure”, discovered and subsequently forgotten.

What is cholera?

Though many theories existed, the cause and mode of spread of cholera were unknown in early 19th century Britain. It is now known to be due to a bacterium known as Vibrio cholera. This is transmitted usually by drinking infected water. In the small intestine, the bacteria produce toxins which lead to a severe and sudden diarrhoea. This is often of such severity that huge volumes of watery stool are passed, leading to rapid dehydration and circulatory collapse

* Address for correspondence: Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK. e-mail: G.Gill@liverpool.ac.uk

¹ There are several useful secondary source books on 19th century cholera, for example: R.J. Morris, Cholera 1832: the social response to an epidemic (London, 1970); M. Pelling, Cholera, Fever and English Medicine 1825-1865 (Oxford, 1978); N. Longmate, King Cholera – the biography of a disease (London, 1966); M. Durey, The Return of the Plague. British society and the cholera 1831-32 (Dublin, 1979).
which can be fatal. The stool is characteristically similar to water with an off-white colour, and is known as the “rice-water stool” (Figure 1). Though diarrhoea and dehydration are by far the commonest features of cholera, other symptoms may be nausea, vomiting, and abdominal pains.

Though vaccination and antibiotics have some place in treating and preventing cholera, the most important treatment is vigorous rehydration, which may be oral or intravenous. This treatment alone can massively reduce cholera mortality, and currently in well-managed cholera outbreaks, mortality rates of below 1% are usual. However, though treatment may be effective, cholera epidemics continue to occur regularly, though nowadays they are confined to the tropics and sub-tropics.

**The 1832 cholera epidemic**

In the 19th century, cholera was frequently encountered in the Indian subcontinent, but rarely spread outside. However, in the early part of the 19th century it began to slowly move west, presumably spread by travellers. By the mid-1820s the disease had reached Europe, and its entry into Britain retrospectively became inevitable, delayed only by Britain’s island status.

Though the epidemic is generally referred to as occurring in 1832, the first cases occurred in late 1831. The initially diagnosed patient was in Sunderland, and the attending doctor had previously worked in India and correctly made the diagnosis of “Asiatic cholera”. This term was used because the word “cholera” was used generically for any diarrhoeal illness – often in Britain known as “summer cholera”. The first patient diagnosed died within 24 hours, and it was this rapidity of movement from good health to death which struck fear in the British population, as the disease rapidly spread throughout the country. The mode of death was also far from pleasant and quite different from other endemic infections. Severe dehydration led to the body taking on a skeletal appearance as the skin and subcutaneous tissues shrank. Pre-terminally, the low blood pressure led to a blue appearance, due to cyanosis, or de-oxygenation of the blood. The medical journal *Lancet* published a print of a young girl in “the blue stage of spasmodic cholera”, demonstrating vividly this premorbid appearance (Figure 2). The shocking print caused consternation amongst both doctors and the lay public.

These dramatic features of cholera explain many social responses to the

---

4 The print of the girl in the “blue stage” of the “spasmodic cholera” appeared in the 4 February 1832 edition of the *Lancet.*
Figure 1: What looks like a bottle of water or urine, outside a hospital ward in Africa, is in fact the “rice water stool” from a cholera patient.

epidemic. The Church of England regarded this new scourge as a possible “Act of God” in return for past sins of the nation, and in the summer of 1832 the following prayer was read by all English churches –

Oh Almighty God, who has visited the nations with the sudden death of thousands, spare, we beseech Thee, this favoured land

In addition, riots occurred on the streets of Britain (the “Cholera Riots”), protesting against a variety of issues including emergency burial practices (in unhallowed ground), and the ineptitude of the medical profession in responding to the crisis. Indeed, many seriously believed that cholera victims removed to hospital were being deliberately killed by doctors to be used for dissection.\(^5\) Cholera also stimulated the mid-19\(^{th}\) century move for public health reform.\(^6\)

---


Edwin Chadwick drew heavily on data from the 1832 cholera epidemic in his landmark 1842 *Report on the Sanitary Conditions of the Labouring Population of Great Britain*, which arguably was highly influential in leading to the 1848 Public Health Act, widely regarded as a turning point in British public health history.

Yet for a disease which had such far-reaching effects on society and politics, the mortality of cholera on a national basis was remarkably small. Table 1 shows the mortality figures for each of the four main 19th century cholera outbreaks, compared to the total British population. The overall population mortality varied from 0.05 to 0.2%, averaging about 1 in 1000. Compared with tuberculosis, typhus, and pneumonia, for example, these are small numbers. Again, the essential historical importance of cholera was that it was new to Britain, caused death fearfully quickly, and was responded to with futile impotence by the medical profession.

Amongst the many medical misconceptions was the mode of spread of cholera. The overwhelming belief was “miasmatic theory” - that the disease was spread by vapours and smells in the air. The opposing “contagion theory” was generally doubted and the true water-borne cause was not to be demonstrated until John Snow’s masterful epidemiological work in London.

---

during the 1854 epidemic.\(^9\) Strangely, in terms of public health, miasma theory was not an obstacle, since removing the smells required sewage disposal and clean water supply. Chadwick was a confirmed miasmatist, and it is often regarded that he and the political founders of the 1848 Public Health Act “did the right things for the wrong reasons”.

**Table 1: Cholera deaths for each of the 4 British 19\(^{th}\) century epidemics, in total and as a proportion of population.**

<table>
<thead>
<tr>
<th>Epidemic</th>
<th>Cholera deaths</th>
<th>Total population</th>
<th>% mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1832</td>
<td>21,882</td>
<td>24.4 million</td>
<td>0.1%</td>
</tr>
<tr>
<td>1849</td>
<td>55,201</td>
<td>27.4 million</td>
<td>0.2%</td>
</tr>
<tr>
<td>1854</td>
<td>24,516</td>
<td>28.0 million</td>
<td>0.1%</td>
</tr>
<tr>
<td>1866</td>
<td>14,378</td>
<td>30.3 million</td>
<td>0.05%</td>
</tr>
</tbody>
</table>

**Notes:**
1. Population numbers are extrapolated from the census figures before and after the epidemic (eg for the 1832 epidemic, the census figures for 1831 and 1841 were taken, the difference calculated, and 10% added to the 1831 figure)
2. The epidemic dates represent the major, but not entirely the only year, the epidemic occurred. For example, the “1832 epidemic” actually started in late 1831, and ended in early 1833

**Grappling for answers**

With no clear understanding of the cause of cholera, or of its mode of spread, it is perhaps not surprising that medical treatment was illogical and misdirected. However, even accepting these knowledge limitations, at least some of the therapies offered were frankly dangerous. In his 1982 paper on 19\(^{th}\) century cholera treatment, Norman Howard-Jones described cholera therapy as “largely a form of benevolent homicide”, going on to say that “to counter persistent vomiting, the physician came to the aid of nature by administering emetics, and

he exacerbated the intractable diarrhoea that was rapidly dehydrating the sufferer of his vital fluids by drastic purgatives”.

Howard-Jones’ points are well taken, particularly as the degree of induced emesis and purgation was often remarkable. For example, a respected Liverpool physician, Dr D. Baird, wrote that “the great thing to be done is to throw into the bowels, by means of an injecting pump, a quart or three pints of warm water or gruel”. This pressurised enema was to be repeated every half to one hour! Bleeding was also frequently practised – also destined to worsen dehydration and circulatory collapse. Indeed it was often noted by physicians attempting bleeding in the 1832 cholera epidemic that the technique was unusually difficult. Veins were collapsed and blood flowed sluggishly. The response was unfortunately sometimes to proceed to puncture arteries, or even the heart. Less toxic treatments included traditional medicines such as laudanum (opium), brandy, calomel (mercurous chloride), rhubarb and ginger. Cold water or brine baths were also sometimes employed.

It seems hard to justify the use of emetics, purgatives and venesection in patients with severe diarrhoea. However, in defence, medicine was practised empirically in these days, and not scientifically. Therapeutics was also remarkably conservative, with a significant reverence for past practice and past practitioners. Purgation, emesis and bleeding had been part of the physician’s practice for literally centuries. There were also erroneous principles of pathophysiology – “congestion” and “heart strain” were felt to be late effects of cholera requiring reduction in circulating fluid volume. Sadly, the end result of cholera therapy in 1832 was to often increase rather than decrease mortality.

**O’Shaughnessy’s proposal**

The therapeutic tide turned in late 1831 with a proposal from Dr William Brooke O’Shaughnessy, who was a 22 year old Irishman recently graduated from the University of Edinburgh (Figure 3). On the 2 December 1831 he delivered a lecture to the Westminster Medical Society, proposing a new method for treating the “blue epidemic cholera”, which was published in the *Lancet* a few days later (see Figure 4). His idea was to inject fluids and salts to combat the “universal stagnation of the venous system”. The technique of *infusing* fluids was yet to be discovered, but syringes were available which

---

12 Howard-Jones, p 380.
could inject fluids. O’Shaughnessy later adopted the “Read’s Patent Syringe”, which was normally used for blood-letting.\textsuperscript{15}

Soon after his lecture in London, O’Shaughnessy moved to Sunderland. Here he performed a series of chemical experiments on the blood of cholera patients. He showed that the blood was deficient in water, saline and “free alkali”; and concluded that injection of water and salts in cholera patients was rational. The results were published again in the \textit{Lancet},\textsuperscript{16} and as an expanded monograph.\textsuperscript{17} It was also presented in January 1832 to the Central Board of Health in London.

\textsuperscript{16} W.B. O’Shaughnessy, ‘Experiments on blood in cholera’, \textit{Lancet}, 1 (1832), 490.
\textsuperscript{17} W.B. O’Shaughnessy, \textit{Report on the Chemical Pathology of the Malignant Cholera} (London, 1832).
Figure 4: William O’Shaughnessy’s landmark 1831 Lancet paper, leading to the birth of intravenous fluid therapy (reproduced with permission of the Lancet).

O’Shaughnessy’s chemical findings were confirmed by Dr WR Clanny of Sunderland, and Dr Thomas Latta of Leith in Scotland undertook early experiments on saline injections in dogs and later cholera patients. These results were also reported to the Central Board of Health and published in the Lancet. This peer-support led to rapid and widespread interest in O’Shaughnessy’s new method of cholera treatment. Retrospectively his work was remarkable for the time – he made a sensible hypothesis and backed it up with scientific observations. In the next two sections we will trace the rise and fall of intravenous fluid therapy as a treatment for cholera.

**Enthusiasm, doubt and rejection**

By the early summer of 1832, saline injections were being increasingly used successfully. The Lancet of 2 June published letters by several doctors describing 15 moribund cholera victims treated with intravenous saline, of whom 5 survived. An accompanying editorial suggested that this was “one of

---

18 W.R. Clanny, ‘Case of cholera at Sunderland with analysis of the blood taken from the patient’, Lancet, 1 (1832), 505-06.
the most interesting recordings in the annals of the medical profession”. The lay press was more openly enthusiastic – on the same day as the Lancet letters and editorial, the Liverpool Chronicle reported on cholera cases treated by “throwing into the blood vessels a quantity of water, with salt and albumen sufficient to supply the deficiency in the blood”. The results were described as “miraculous … the lips became red … the spasms ceased, the pulse returned”. Interestingly, the article recorded the amount of fluid injected, which was in total “six to ten pounds” (about 3 to 5 litres), but in one case “forty pounds” (about 20 litres). Interestingly, these large amounts are approximately what would be used today in severely dehydrated cholera patients.

Use of saline injection continued to spread in the summer of 1832. In London, Dr Stevens privately published a treatise on fluid treatment, including the use of oral salt solutions. The volumes used were rather small, but again it is of interest that oral rehydration in milder cases of cholera is now current practice.

By the late summer and autumn of 1832, however, doubts were being expressed as to the efficacy of the O’Shaughnessy method. One report from London by Stevens of a 100% cure rate with fluid treatment in 92 patients was reinvestigated and it was believed that none of the patients had genuine cholera. Also, an “unsuccessful trial of Dr Stevens saline treatment” was reported in which only 1 out of 9 treated patients survived. This survivor also had severe phlebitis as a result of the injection syringe. Infection and probable septicaemia were reported in other treated patients, as well as occasional symptoms of “cerebral irritation” – possibly reflecting cerebral oedema (brain swelling) related to excessive fluid treatment.

As well as side effects, a further problem was that intravenous fluids were usually reserved for the more severe cases of cholera – often at the “blue” or preterminal stage. Between 2 June and 14 July 1832, 7 separate reports in the Lancet were published of a total of 31 cholera cases treated with intravenous fluids. Nineteen (19) of these patients died – a mortality rate of 61%, which was certainly higher than overall rates (the mortality for the whole epidemic was about 36%). A curious outcome comparison of various cholera treatments also suggested that intravenous saline was associated with a much worse outcome than traditional methods such as calomel or opium (see Table

---

22 Liverpool Chronicle, 2 June 1832.  
23 W. Stevens, Dr Stevens Treatise on the Cholera (1832).  
27 Durey, p 38.
Again, the issue of case severity was not addressed, and also the overall very high mortality for all treatment is striking. Nevertheless, Mr de Grave had reported one of the first comparative trials of different treatments for a single disease.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Cases treated</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calomel</td>
<td>75</td>
<td>47%</td>
</tr>
<tr>
<td>Opium</td>
<td>81</td>
<td>58%</td>
</tr>
<tr>
<td>Calomel + opium</td>
<td>196</td>
<td>57%</td>
</tr>
<tr>
<td>Oral fluids</td>
<td>51</td>
<td>59%</td>
</tr>
<tr>
<td>Intravenous fluids</td>
<td>20</td>
<td>90%</td>
</tr>
</tbody>
</table>

Rightly or wrongly, by the autumn of 1832, saline injections were rapidly declining in use, and when the epidemic ended in early 1833, it was regarded as a discredited treatment.

**Demise and late re-emergence**

During the later British cholera epidemics of 1849, 1854 and 1866 intravenous fluid injection was only sporadically used, with very occasional reports and discussions in the medical literature. The rare protagonists who had witnessed the potential dramatic effects could not muster support amongst their colleagues. Dr Howlett in 1849 wrote that “if a fair trial were to be given it would be crowned with abundant success”. His words were overwhelmed by nearby pages continuing to extol the virtues of calomel and opium.

A turning point occurred in Germany in 1879, when it was demonstrated that dogs with induced haemorrhagic shock (from experimental bleeding) could

---

28 Editorial, ‘Malignant cholera in London’, *Lancet*, 1 (1833), 599. Though published in early 1833, the data refers to cases from London in 1832. The information was said to be “transmitted by Mr F de Grave”.


30 Howlett, p 269.
be successfully resuscitated with intravenous saline solutions. Three years later (1882), in England, Dr CE Jennings successfully treated a woman with severe obstetric haemorrhage, using a complex intravenous solution containing many salts, but mostly sodium chloride. A *Lancet* editorial enthusiastically supported this “new treatment”, and its use become more widespread over the next decade. By the end of the 19th century, intravenous fluid treatment was in common use, and 70 years after O’Shaughnessy’s first proposal, had finally received acceptance.

**Conclusions**

One of the early supporters of intravenous fluid treatment for cholera, Robert Lewins, wrote in 1832 that it was “an astonishing medication” that would result in “wonderful changes and improvements in the practice of medicine”. Lewins’ words were indeed prophetic, but he would surely have been staggered to learn just how long it would take for acceptance to occur. Why did this disastrous delay take place – costing the lives of thousands of cholera victims?

The inherent empiricism and conservatism of British medicine in the 19th century was certainly a factor to which we have earlier referred. Medicine was also heavily London-centred. For example, the *Lancet* had a lengthy weekly column of cases from the London hospitals. As an Irishman educated in Edinburgh, one can imagine that O’Shaughnessy’s work may well have been received with scepticism by the London medical establishment (Michael Durey talks of the “citadel of official medicine in London” 35). Fluid injection also ran the hazard of adverse effects: phlebitis, septicaemia and under- or over-infusion. Most importantly, however, was the problem of what we would now call selection bias; that intravenous fluids were almost always reserved for the most advanced cholera cases, condemning the new therapy to a high mortality rate.

O’Shaughnessy’s work, however, remains a landmark advance in medical practice. It was sadly ignored in the 19th century, and even though widely accepted since, the name of its originator is unknown to most modern doctors. It should be said that the idea of fluid injections into the veins in cholera patients probably originated in the work of Hermann in Moscow. 36

---

31 H. Kronecker, S. Sander, ‘Bemerkung uber lebensrettende transfusion mit anorganischer salzsung bei unden’, *Berliner Klinische Wochenschrift*, 16 (1879), 767.
34 R. Lewins, ‘Injection of saline solutions in extraordinary quantities into the veins of cases of malignant cholera’, *Lancet*, 1 (1832), 244.
35 Durey, p 131.
36 Howard-Jones, p 386.
O’Shaughnessy was aware of Hermann’s work;\textsuperscript{37} but his own redefinition of the basic problem of blood volume depletion in cholera, his chemical observations of what was deficient in the blood, and his logical proposed treatment, were indeed “ahead of his time”\textsuperscript{38}.

\textit{Finale}

William O’Shaughnessy did not fade into bitter obscurity at the end of the 1832 cholera epidemic. In 1833 he moved to India with the Bengal Medical Service. His interest in chemistry continued, and he also moved into electronics. At the age of 34, he became Professor of Chemistry at the Medical College of Calcutta and wrote several chemical and pharmaceutical textbooks. He was also elected a Fellow of the Royal Society, and in 1847 achieved a long-held aim of introducing the first overland telegraph service in India. He was also arguably the first doctor to experiment with the use of cannabis for medical conditions.\textsuperscript{39}

All of these achievements (but notably not the introduction of intravenous fluid treatment) led to his being knighted in 1856. He returned to England in 1861 and for unknown reasons changed his name by Royal License to Sir William O’Shaughnessy Brooke. He entered a quiet retirement, and died on the south coast of England (Southsea) in 1899, aged 79 years. He wrote nothing more on cholera after the 1832 epidemic, but it will not have escaped his notice that though intravenous fluid therapy remained rejected during the ensuing cholera epidemics in Britain, at the time of his death it was part of accepted medical therapy.\textsuperscript{40}

\textsuperscript{37} O’Shaughnessy mentions Hermann’s work in his own \textit{Lancet} article in early 1832. See O’Shaughnessy, p 490.
\textsuperscript{38} Foex, p 317.
\textsuperscript{39} Moon, p 284.
\textsuperscript{40} W. Osler, ‘Cholera Asiatica’, In: \textit{Principles and Practice of Medicine} (New York, 1892), pp 118-24.