Death in the beer-glass: the Manchester arsenic-in-beer epidemic of 1900-1 and the long-term poisoning of beer

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For an unknown number of years running up to the end of nineteenth century, beer-drinkers in Manchester and the north-west of England suffered an insidious assault to their health - a form of paralysis termed peripheral neuritis. The condition was the result of consuming dangerous quantities of arsenic contained - unsuspected and undetected - in their beer. The source of the poison was contaminated barley malt used unwittingly by brewers, with the extent of the contamination sufficient to provide Manchester hospitals with a steady throughput of severely debilitated beer drinkers. However, the ongoing failure of brewers to realise that they were slowly poisoning many of their customers was facilitated by the systematic misdiagnosis by local medical practitioners of these same beer-drinkers. Their clinical eye prejudiced by assumptions about the habits and honesty of poor working-class drinkers, and by a largely unquestioned belief in the special symptomatology of local alcoholism, these medical practioners consistently attributed the cause of the neuritis to the excessive consumption of alcohol. Thus, the innocent victims of poisoned beer were routinely categorised as the culpable victims of chronic alcoholism.

The misdiagnosis continued even as prevalence of the condition burgeoned into epidemic. It was only at this point, in the Autumn of 1900, and a dramatically more extensive source of arsenic in beer - contaminated brewing sugar - that a number of local medical practitioners began seriously to question the diagnostic paradigm that had hitherto led them habitually to alcoholic, rather than arsenical neuritis.

With this reappraisal came the revelation - and public announcement - that a substantial proportion of the local beer supply was dangerously contaminated with arsenic. As such, local brewers were rendered immediately culpable, and thrust into the public arena to remedy the problem of arsenical beer in the marketplace, to institute safety precautions to prevent a recurrence of the problem, and to stage a defence - both commercial and legal - for their obvious failure to protect their customers.

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The Manchester arsenic-in-beer epidemic of 1900-1 was a serious and widespread food poisoning outbreak affecting several thousand people across the North-West and Midlands, with many cases proving fatal. It exposed lax quality control and dangerous levels of complacency amongst brewing companies, and revealed that many brewers had been poisoning their customers, unnoticed, for many years prior to the epidemic. Ultimately, the brewing industry emerged from the crisis largely unscathed. This resulted in part from the speed and openness of the industry in working with local authorities to remove contaminated beer from the market place, in part by generating their own positive publicity material to reassure anxious drinkers, in part by instituting new analytical safeguards, and in part by transferring the weight of legal repercussions - and hence 'official' blame - onto the manufacturer of the contaminated brewing sugar.

However, the epidemic was not just a 'wake up' call for the brewing industry. The epidemic opened up a debate about the existence of a medical condition that had hitherto been regarded as endemic to the Manchester region. Ultimately, the elimination of arsenic contamination (or rather the reduction of the contamination to non-hazardous levels) from local beer resulted in the effective elimination of alcoholic neuritis from the hospital wards - and in doing so revealed the extent and duration of the previous misdiagnosis.

Previous accounts have focused on the Manchester epidemic either as an episode in the history of brewing,[1] agriculture,[2] consumer protection legislation,[3] or environmental poisoning.[4] What these approaches have in common is that they have effectively excused the medical profession for its role in facilitating the epidemic - and, more seriously, for the long-term misdiagnosis of chronic arsenic poisoning as alcoholic. This article, however, argues that local brewers and medical practitioners were jointly culpable for the long-term poisoning of poor Mancunians, the former for producing contaminated beer, and the latter for failing to question their assumptions about the consequences of working-class drinking, and in so doing, allowing the poisoning to go undetected for so long.

An epidemic of alcoholic neuritis

In the late summer and autumn of 1900, Dr Ernest Septimus Reynolds of the Manchester Royal Infirmary (M.R.I.) and Manchester Workhouse Infirmary (M.W.I.) began to see an unusually high number of out-patients complaining of numbness and 'pins and needles' in the feet and hands, loss of strength and painfulness of the limbs, and the appearance of itchy rashes on the skin.[5] Some of these cases he diagnosed as erythromelalgia, a condition characterised by redness and burning pain in the extremities, and some as Addison's disease, the symptoms of which included weakness, fatigue and discolouration of the skin. Reynolds also

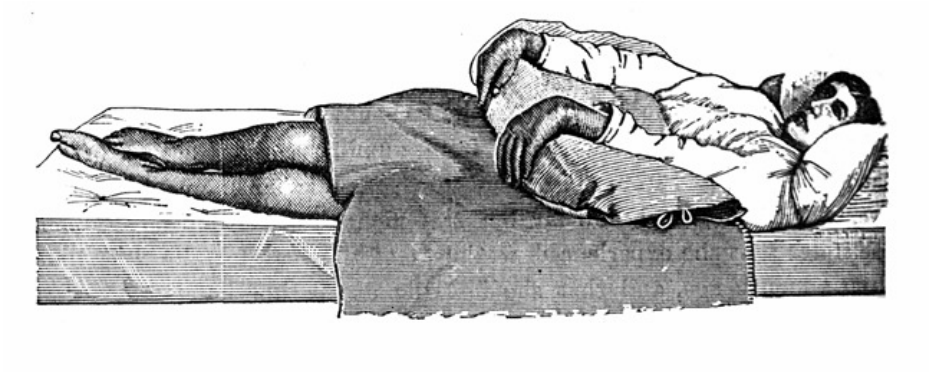


Figure 1. '"Jane H", a typical case of alcoholic neuritis.' Medical Chronicle*, February 1890.*

noted a 'remarkable' increase - perhaps as much as four-fold - in cases of herpes zoster (shingles), characterised by a painful cutaneous rash and blistering around the lower torso.[6] These conditions were commonest in his pauper patients, but what all sufferers had in common was a history of beer drinking. The others, running into several hundreds, he judged as being clear cases of 'alcoholic peripheral neuritis'.[7]

Alcoholic peripheral neuritis was a common condition amongst the poor of late-nineteenth century Manchester. Characterised by progressive paralysis, 'dropping' of the hand and feet, and extreme tenderness of the soles and leg muscles, the condition provided a stark demonstration of the degenerative effects of alcoholism.[8] Indeed, the condition was so common as to be regarded as a special feature of Mancunian public medicine. As Dr T.N. Kelynack of the M.R.I. later noted:

> Multiple neuritis has long been recognised as a common penalty borne by those who indulge in alcoholic drinks in Manchester and district.[9]

Indeed, to demonstrate that this long-held belief was more than anecdotal, Kelynack conducted his own investigation into the frequency of alcoholic neuritis in Manchester compared to other large population centres, the results of which were published in the July 1901 edition of the *Medical Magazine*. Based upon statistics supplied from hospitals around Britain, Kelynack found that Manchester did indeed occupy an 'unenviable prominence' in terms of both the frequency and severity of cases.[10] He calculated that during the period 1892-1898 alcoholic peripheral neuritis

accounted for approximately 1.2% of all admissions to the M.R.I.; this was two to three times the rate for London hospitals, and five to ten times that of Belfast, Cambridge and Dundee.[11] The reason why alcoholic neuritis was far commoner in Manchester hospitals than anywhere else in Britain was unclear, and as Kelynack informed the Society for the Study of Inebriety in July 1901, seemingly unquestioned other than by speculation:

> I have long been in the habit of teaching that either the drinking habits of the people or the character of the drinks taken led to more distinct pathological results in Manchester and district than elsewhere.[12]

Manchester aside, the condition had a long and distinguished history, traceable to antiquity and the writings of Seneca who first made the connection between the characteristic symptoms and excessive consumption of wine.[13]

By November 1900, alcoholic neuritis accounted for a nearly a quarter of acute admissions to the M.W.I., and an equally high proportion of out-patient cases at the M.R.I. - a vast increase on the usual average incidence of approximately one percent.[14] Inquiring locally, Reynolds discovered that colleagues working in Salford and other districts of Manchester were faced with comparable increases in the incidence of alcoholic neuritis. At the nearby Ancoats Hospital, for example, nearly half of all out-patients showed the characteristic symptoms.[15] Reynolds concluded that they were facing an epidemic of alcoholic peripheral neuritis, accompanied by a smaller, but nevertheless marked increase in the occurrence of several other conditions characterised by distinctive skin discolouration.

What Reynolds did not know was that this epidemic extended far beyond Manchester and its immediate district. Manchester had the highest concentration of sufferers, but other medical practitioners working in northern England and the Midlands were also experiencing dramatic rises in cases of alcoholic neuritis. At the Mill Road Infirmary, Liverpool, for example, Dr Nathan Raw recorded a four-fold increase in cases during 1900.[16] In common with Reynolds's experience, some of these exhibited marked skin pigmentation and eruptions, which Raw also diagnosed as Addison's disease. Similarly at Stourbridge in Worcestershire, and Seaforth in Lancashire, local medical practitioners were perplexed by a steady increase in admissions for alcoholic neuritis with the complicating symptoms of scaly skin and sores. The sufferers were all drinkers of beer. This pattern was repeated as epidemic alcoholic neuritis was recorded across central and northern England.[17]

The diagnosis of alcoholic neuritis, even on such a large scale, was supported by the fact that all the sufferers admitted to drinking beer on a regular basis. In almost every local epidemic, this was recognised as the universal feature; as one Poor Law medical officer remarked, ‘it was soon abundantly clear that the one thing common to all was beer drinking’.[18] Next to a history of beer drinking, the

most common factor in the various epidemics was social class. The overwhelming majority of sufferers were working class, mostly from the lower echelons - labourers and paupers. Together they constituted the 'drinking class', and hence those naturally likely to be afflicted by alcoholic neuritis; thus, the diagnosis fitted both demographically and symptomatologically. But if the diagnosis of alcoholic neuritis was correct, the question was, how had it developed into an epidemic in a relatively short period of time? - after all, it was not a contagious disease. The simplest answer had to be that a corresponding increase in drunkenness had taken place, whose harmful effects were now showing in the bodies of drinkers.

That hard drinking was widespread amongst the working class was readily apparent to those medical practitioners who dealt with them on a regular basis. This was especially true in Manchester, where patients exhibiting the physical symptoms of alcoholism were far commoner than anywhere else in Britain. Why this should be was not clear, but prior to the epidemic this was regarded as a local medical peculiarity rather than a cause for special concern.

The existence of this alcoholic residuum did not, however, explain why within a few months cases were reaching epidemic numbers; what could, was if a spate of acute 'binge' drinking was superimposed onto the background of chronic intemperance. And two recent events were blamed for dramatically increased drinking - the Boer War and the general election of 1900.[19] War fever was seen by some as an item of particular concern, especially the drunken celebrations that followed British victories such as the relief of Mafeking, or the safe return of volunteers.[20] Indeed, so notable were the Mafeking night celebrations that the term 'maffick' was quickly coined as being figurative of wild rejoicing. The so-called 'khaki' election of October 1900 was called early by Lord Salisbury, the Conservative Prime Minister, who hoped to be carried back into power on a wave of extreme patriotism. Irrespective of any special circumstances, many critics regarded elections as an open invitation to excessive drinking. Liberal politicians and members of the anti-drink lobby frequently, and without any evidence, accused a supposedly powerful lobby of brewers and publicans of bribing working-class electors with free drinks.[21] The election of 1900 was judged to be no different.

More puzzling though, was that while some patients admitted drinking, they nevertheless insisted that their daily alcohol intake was much lower than that usually deemed capable of causing neuritis. Throughout the previous decade, and well into the early stages of the epidemic, any such claims to moderation were not regarded as problematic in diagnosing the condition due to its very nature. Since those suffering from the condition were assumed to be heavy drinkers, and hence thought liable to

dissembling, concealment, mental derangement or confusion, it was deemed entirely appropriate for the physician to doubt or even discount the veracity of their narratives - more so if they were also poor and working class. Thus, the doctor/patient relationship was framed by the understanding of the disease, not vice-versa. Potential anomalies could not be allowed to undermine the certainty of the diagnosis; rather patient testimony was interpreted in a manner that maximised their consumption. Indeed, the condition's explanatory framework insisted that such assumptions were a necessary part of orthodox diagnosis.[22] A pseudonymous letter to the *Times* revealed the extent to which social diagnosis was colouring clinical investigation:

> As the name implies, their condition was considered to be undoubtedly due to the excessive use of alcohol, and, though the sufferers invariably denied this.., they were naturally not believed.[23]

But as more and more sufferers claimed moderation, it became harder to disregard their testimony.

> Although it is difficult to get to know the exact amount of beer taken a day [Reynolds noted] yet I am convinced after careful enquiry that in some cases at least not more than four glasses a day have been consumed.[24]

In some cases, trust seems to have been made easier by the social status of the patient. As Dr J.W. Crawshaw wrote of one middle-class sufferer:

> The amount [of alcohol] taken has been most carefully inquired into, and, as the patient fully understands the importance of the inquiry, her answers may be relied upon.[25]

Similarly, Dr R.T. Williamson of the Ancoats Hospital, was prepared to accept claims to moderate consumption, even though 'unable to prove [them]'.[26] If these patients were telling the truth, the unified diagnosis of alcoholic neuritis was no longer sustainable. And so, independently, several months after the epidemic had begun, and at least ten years since the modern description of the condition, a number of medical practitioners started to reappraise their diagnosis.

The search for a different cause

In his search for the cause of the epidemic, Ernest Reynolds turned to the principal medical textbook, Judson Bury and James Ross's *A Treatise on Peripheral Neuritis* (1893), and the detailed categorisation of the various different categories of neuritis. One key symptom of alcoholic neuritis was extreme tenderness of the muscles, especially the large muscles of the limbs. This was a particularly pronounced symptom amongst Reynolds's patients at the M.W.I. Indeed, he remarked that he was able to perform a perfunctory diagnosis simply by grasping their leg muscles and observing the 'sudden expression of pain' on their faces.[27] Based on Bury and Ross, this muscular tenderness was associated with only

three known causes of neuritis - alcohol, beriberi, and arsenic.

However, Reynolds's major breakthrough was to connect some of the cases of neuritis with some of the cases of skin discoloration. Hitherto, and elsewhere, the two sets of symptoms had been regarded as unrelated. Reynolds, however, noted that some patients at the M.W.I. had both skin and neuritis symptoms, which he now reassessed in terms of them possibly being part of a single common condition. According to Bury and Ross, only one substance was known to cause the characteristic symptoms of peripheral neuritis and extreme muscular tenderness and skin discolouration - arsenic.

Having identified arsenic as a possible cause, Reynolds's next deductive leap was to find a means of transmission. It was an established 'fact' of the epidemic that all the sufferers were beer drinkers. But Reynolds had also noted that people who drank only spirits remained unaffected by alcoholic neuritis, in spite of the quantities of alcohol consumed. For Reynolds, this confirmed that alcohol per se was not the cause of the sickness, and suggested where the arsenic was most likely to be coming from. Beer drinking was the only factor common to all sufferers, therefore this must be the source of the arsenic. Reynolds justified his hypothesis in almost Holmesian language:

> Improbable as this hypothesis at first seemed, yet it was a valid hypothesis, for it was not known to be untrue; it explained all the facts and it was easily capable of proof or disproof. This hypothesis I imagined on Nov. 15th 1900.[28]

Reynolds accordingly obtained specimens of beer from outlets frequented by some of his patients. On 18th November he tested the beer samples and found arsenic: 'thus, the hypothesis became a fact'.[29] His analysis was confirmed two days later by Professor Dixon Mann of Owens College, Manchester, who passed on news of the arsenic contamination to Dr C.H. Tattersall, medical officer of health for Salford. Reynolds announced his findings at a meeting of the Manchester Medical Society on 21st November, and had them published in the *British Medical Journal* (B.M.J.) three days later. The Manchester public learned of the poisoned beer epidemic one day earlier through the daily newspapers.

Unbeknown to Reynolds, Tattersall had been carrying out his own investigation into the epidemic in conjunction with Dr R.D. Cran, a Salford Poor-Law medical officer, and Sheridan Delépine, professor of pathology at Owens College.[30] Cran suspected beer as early as 9th November, but was unsure of what agent was causing the illness. He also had a large numbers of patients suffering from various skin complaints, but, unlike Reynolds, none were also affected by neuritis, and so he did not consider the two as parts of a common condition. Nevertheless, like Reynolds, he eventually deduced that the cause of the neuri-

tis was some form of poison contained in beer, but could not narrow the range of possible causes any further. On 12th November, he visited James Groves, M.P., the Chairman of Groves & Whitnall, Salford's largest brewing company and informed him of his concerns. Cran obtained samples of beer and sent them to Charles Estcourt, Manchester's public analyst, but Estcourt failed to detect arsenic because Cran and Tattersall were unable to give him a better idea of what toxin he was supposed to be looking for. Tattersall, Delépine and Cran nevertheless concluded that beer definitely was the means by which the toxin was transmitted, a belief supported by the fact that several of Cran's patients were brewery employees. Meeting to discuss the epidemic one week later, they decided to obtain more beer samples for analysis, but before these could be tested, Tattersall was informed by Dixon Mann that he and Reynolds had both found arsenic in beer.

Also finding arsenic in some of their samples, Tattersall and Delépine continued their investigations one step further, obtaining samples of all brewing materials from one of the brewers whose beer had proved arsenical. Tests revealed that all the ingredients were arsenic-free apart from one sample of glucose and another of invert sugar, both of which had been supplied by the same sugar manufacturer - Bostock & Co. of Liverpool. Visiting Bostock's the next day, Tattersall and Delépine procured further samples that linked the poison to sulphuric acid used in the sugar production process. Pursuing their investigation to its logical conclusion they next visited Nicholson & Sons, the Leeds-based manufacturer of the sulphuric acid. Here they discovered that the sulphuric acid was produced from pyrites which typically resulted in acid containing arsenic. Having established the provenance of the poison, Tattersall and Delépine published their findings in the *Lancet*, where they expressed the conviction that not only had they positively identified all the guilty materials, but that they had also effectively eliminated all other possibly sources of contamination.[31]

Equally quick to react to news of arsenic in beer was Dr T.N. Kelynack, who, in common with Reynolds and Tattersall, had witnessed an increased incidence of alcoholic neuritis in connection with his role of medical registrar at the Manchester Royal Infirmary, and honorary medical officer at the Salford Royal Infirmary. Convinced that some form of beer contamination was responsible for the illness, Kelynack sought out the assistance of William Kirkby, a pharmacologist at Owens College, and Dr Forsyth, medical officer to Groves & Whitnall. With the co-operation of James Groves, samples of beer were obtained, this time bottled during the previous three months, in all of which arsenic was present. This proved not only that contamination had been taking place for some time, but also that all possible sources between the brewery and the customer could be discounted. Armed

with the results of his investigations, Kelynack then informed the Manchester and Salford medical officers of health and the implicated brewers. And, as with Reynolds, Tattersall and Delépine and Kelynack and Kirkby were quick to have their role in understanding the emerging epidemic ratified through publication in the Lancet, where Kelynack declared his satisfaction in knowing 'that as a result of … [their] labours the source of the introduction of arsenic … was localised within a few hours'.[32]

News of the Manchester epidemic and its suspected cause was followed by a veritable rash of similar epidemics in other districts of the north-west. Medical practitioners puzzled by inexplicably high incidences of alcoholic peripheral neuritis, realised that they too were facing epidemic chronic arsenic poisoning. The B.M.J., one week after Reynolds's article, carried news of outbreaks in Lancashire, Yorkshire, Cheshire, Leicestershire, Worcester and Staffordshire.[33] Numerous cases were reported in Liverpool at the Mill-road Infirmary, and also across the Mersey at the Tranmere Workhouse infirmary.[34] At Chester approximately thirty cases of a similar nature were announced, with new patients presenting themselves daily. In the West Midlands, similar numbers of sufferers were recorded at both Stourbridge and Birmingham.[35] In Manchester, sufferers remained the most numerous; as well as the Royal and Crumpsall Workhouse infirmaries, further admissions were recorded at the Witherington Workhouse Infirmary and

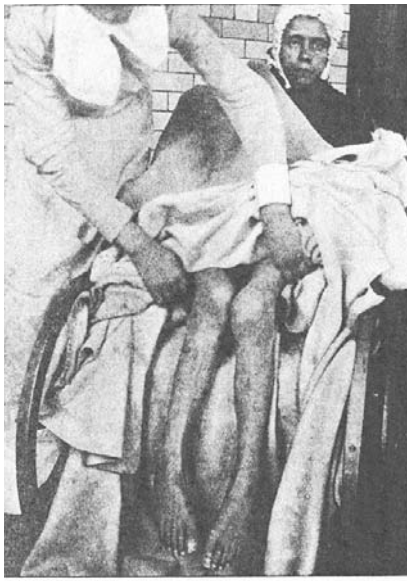


Figure 2. The human cost of the epidemic. Complete paralysis of the lower limbs with much atrophy, nails thinkened, and slight pigmentation of the skin. From Kelynack, T.N., & Kirkby, W. (1901) Arsenical Poisoning in Beer Drinkers*, Baillière, Tyndall & Cox: London, p.39.*

the Ancoats Hospital. Just to the north, in Heywood, it was estimated that two to three hundred persons had been affected.36 According to Dr J. Niven, medical officer of health for Manchester, by early December 1900, the total number of arsenic poisoning cases in the city had risen to between one and two thousand.[37]

Although the nature and source of the poison had been isolated within one day of the public announcement of the epidemic's discovery, in the mass of conjec-

Figure 3. 'Dropped' hands from paralysis of the extensors, with much muscular atrophy. From Kelynack, T.N., & Kirkby, W. (1901) Arsenical Poisoning in Beer Drinkers*, Baillière, Tyndall & Cox: London, p.64.*

ture that followed, the 'facts', as understood by Reynolds, Tattersall and Kelynack, became only one theory among the many which sprang into circulation. Indeed, far from constituting a fait accompli, announcement of the epidemic and its cause triggered increased speculation on what was occurring, why it was occurring, and how it might be prevented in future. Reynolds had originally believed that the arsenic resulted from hops that had been treated with sulphur as an insecticide, while others conjectured that the cause was completely unconnected with beer, being perhaps a new form of enteric fever brought back from South Africa, or else the result of contamination of local water supply.[38] One imaginative Chester brewer suggested that cheap Sinhalese tea contaminated during drying was to blame.[39] Charles Estcourt, the Manchester city analyst, who had earlier failed to detect arsenic in samples sent to him, was initially prominent among those opposed to the idea of beer as the guilty agent. In a letter to the Manchester Guardian he dismissed the arsenic in beer theory as 'improbable', and suggested that pursuing this line of investigation would, 'retard the discovery of the real cause'.[40] According to Estcourt, if beer was the cause, three thousand, not three hundred persons would be affected, which later proved to be a fairer estimate of the number of people affected.

In spite of these other theories, the consensus of expert opinion was that beer was the means by which the poison had reached the public, and brewing sugar, contaminated by impure sulphuric acid was the source of the arsenic. Much as, half a century earlier, John Snow had traced the source of the Golden Square cholera epidemic to the Broad Street pump, so Ernest Reynolds had traced epidemic peripheral neuritis back to the beer-pumps of local public houses; however, in Reynolds's case, preventing further sickness would involve more than simply removing pump handles.[41] Rather, the brewing industry was immediately and inextricably involved in the crisis, and regardless of any further developments, brewers needed to act swiftly to prevent further supplies of poisonous beer reaching the general public.

The brewers' response

Bad publicity aside, the poisoning epidemic carried a number of serious implications for the brewing industry, both immediate and long-term. Most apparent was the fact that they had poisoned a large number of their regular customers, possibly killed some of them, and caused considerable distress, albeit unwittingly. Somewhere in the beer production process a lethal poison had gained entry and passed undetected to consumers. Furthermore, brewers were still manufacturing and distributing poisonous beer, and would continue to do so if special measures were not introduced. The financial impact of the crisis had also to be considered: just one day after news of the crisis broke, a 'considerable falling off' in beer consumption was being reported.[42]

Halting the epidemic as quickly as possible, and instituting measures that would prevent any future recurrence was vital, both to the brewing industry's short-term profitability, and its long-term commercial freedom. As the crisis had entered into the arena of public debate, it would be necessary for brewers to be as transparent in their actions and inquiries as they had been in their failure. Neither would it be to the brewing industry's advantage to sit idly by while other organisations, possibly informed by anti-brewing agendas, carried out their own investigations. Rather, brewers would be better served by conducting a robust defence of their own activities, both before, during and after the crisis.

Having been apprised of the problem several days before the general public, James Groves was provided with an opportunity to co-ordinate action that would go some way toward pre-empting negative publicity. Following Cran's visit on 12th November, Groves travelled to London to discuss the crisis with A. Gordon Salamon, a London-based chemist often employed as a consultant by brewing companies. They agreed that if the problem was as widespread as suspected, Groves should involve the whole industry through the appointment of a commercially independent commission of experts.

At Groves's instigation, a specially convened meeting of the Manchester Brewers' Central Association (M.B.C.A.) was held on 23rd November, where it was resolved that:

> every possible facility and assistance shall be given in the way of elucidating the mystery, and … local authorities (shall) be given every help in arriving at a definite conclusion.[43]

Groves told reporters that in anticipation of the M.B.C.A. appointing its own special investigatory committee, he had 'taken steps to secure the assistance of the most eminent men in the scientific and medical worlds'.[44] Declaring himself 'completely puzzled' by what was occurring, Groves was nevertheless able to demonstrate to anxious Mancunians

that local brewers had reacted swiftly, positively and decisively to news of the epidemic, with the prime objective of identifying and eradicating the source of the poison.[45]

The principal difficulty facing both brewers and local authorities in containing the epidemic was that its source was diffused through an unknown number of breweries. If sugar was to blame, this ingredient came from a single supplier who served a large number of consumers, so it was likely that more breweries were involved than had so far been identified. As the *Manchester Courier* explained,

> if all the cases had been traceable to one brewery, just as an outbreak of scarlet fever can generally be traced to a particular milk supply, the matter would have been probed at once, but several firms were involved.[46]

But, reassured that the source of the epidemic had been isolated, the article concluded that it was now a relatively simple matter for local authorities to halt further sickness: 'the analysts will have no problem in settling the matter one way or the other, and it may fairly be supposed that no further cases of illness will arise.'[47] This prognosis was to prove overly optimistic.

On 26th November, the M.B.C.A. announced the appointment of its expert commission. Senior Home Office analysts Dr T. Stevenson and Dr A.F. Luff were recruited to perform similar functions for the M.B.C.A., as was Sir Thomas Lauder Brunton, the eminent pharmacologist. Already involved in investigating the epidemic, Gordon Salamon was seconded into the expert commission as resident chemist of brewing. The commission was headed by Fletcher Moulton, Q.C., M.P. Chosen by James Groves, Moulton was a specialist in legal cases involving scientific issues, and had amassed a fortune defending patent cases: he would be of special value if, or when, the M.B.C.A. became involved in any legal actions. The commission's depth of medical knowledge was further strengthened by Dr Samuel Buckley who joined several days later. The M.B.C.A. also announced that steps were being taken by brewers to withdraw contaminated stocks of beer from the marketplace, with all new shipments being tested before supply.[48] Signalling its approval, the Manchester Courier considered that in appointing the expert committee the brewers, 'have taken the only wise course open in the matter'.[49] However, the worst of the epidemic had not yet been reached, and within two days of the Courier predicting that no further illness would arise, the first deaths were reported.

Although it was already understood that the use of contaminated sugar was not confined to one brewery, Tattersall's ongoing investigations revealed that Bostock's supplied approximately 200 breweries in the north of England and the Midlands.[50] Reynolds wrote to the newspapers on 27th November, stating that he just bought beer openly from a shop and found it contaminated with arsenic.

Assured that sugar, not hops, was to blame, he felt that the risks were still such that '…it is really necessary to warn the public against drinking beer, especially the cheaper kinds'.[51]

In advising against the drinking of 'cheap' beers, Reynolds was highlighting the fact that beers likely to contain arsenic were those brewed using sugar or glucose, which were generally the cheaper varieties. More expensive beers were typically brewed using malt and hops only and therefore not likely to be contaminated. The link between low price and likelihood of poison was also evidenced by the demography of the epidemic - the vast majority of sufferers were lower working-class, whose purchasing power tended to limit them to the cheap, 'four-penny beers'.[52] This led to immediate speculation that cheapness was a dominant rather than subsidiary factor in arsenic being present in the beer. In an uncharacteristically critical leading article, the *Manchester Courier* attributed blame to

> … the mania for cheapness which taints the whole of commercial life to-day, [which had provided] an object lesson in the evils of drink [by suppliers] … careless of the most elementary principles of honesty so long as they can squeeze a little extra profit out of the transaction.[53]

Similarly, the *Liverpool Mercury* reported that, 'in Liverpool as elsewhere …, everything seems to point to the fact that poor people have been drinking poor beer' - the crucial point being the interrelatedness of cheapness and poor quality.[54]

While the use of sugar in beer production did present brewers with a number of financial advantages, this was a quite different proposition to suggestions that they were indulging in wanton and reckless cost-cutting, of which the eventual result was toxic beer. In large towns and cities, beer prices were governed by a highly competitive marketplace, dominated by large breweries whose extensive consumer bases were sustained by the tied-house system and low prices. And in maintaining low prices, sugar provided brewers with a number of advantages. Sugar used in conjunction with low grade barley-malt was a cost effective substitute for expensive premium-grade barley, while the use of priming sugar reduced conditioning times, thereby improving throughput. Finally - and crucially in terms of de-emphasising the financial advantages of using brewing sugar - brewers claimed that in the period following the abolition of the Malt Tax (1880), public tastes had changed, and drinkers now preferred light, sparkling ales, which could only be brewed with the additional use of sugar.[55]

Aware that the epidemic was rapidly developing into an item of national concern, and was being fuelled by much unsubstantiated speculation, H.A. Newton, the secretary of the London based Country Brewers' Society, wrote to the *Times*, expressing the hope that until

the facts were known, 'judgement may be suspended'.[56] James Groves made a similar plea for,

> the press and public to reserve their judgement, and continue to give them [the brewing industry], in their exceeding trying, difficult, anxious and heartrending moment, their sympathy.[57]

Elsewhere, the brewing press was quick to condemn what it saw as the anti-industry bias of much of the popular press in which,

> [e]very petty reporter and Grub Street journalist in the land [had] immediately blossomed forth an expert on beer, and with a display of sublime ignorance … forthwith proceeded to blacken and defame an honourable trade.[58]

Worse still, in these same newspapers, 'every wild ridiculous statement… that is damaging to the trade, is readily published and apparently accepted as true'.[59] It was undeniable, however, that the arsenic epidemic had provided the numerous critics of the brewing industry with an ideal occasion for denouncing it publicly that was unlikely to be spurned: 'So excellent an opportunity for the exercise of malice was not to be thrown away'.[60] But if the industry's critics were prepared to use the crisis to defame an honourable trade, then the industry was prepared to be equally scathing of their motives for doing so, variously categorising them as 'teetotal fanatics', 'alarmists', 'veiled protectionists', and 'irresponsible paid agitators, both in and out of the press'.[61] Regardless of the best efforts of its critics, the brewing journals remained defiantly proud of the industry's achievements:

> We emphatically assert that never in the history of the trade was beer produced of a higher standard as regards quality and freedom from adulterants, than it is today.[62]

However, it was not enough for brewers to hope that the press and public would suspend judgement: they needed to end further speculation by publicly identifying the source of the arsenic. Accordingly, on 2nd December, the M.B.C.A. expert commission issued an interim report which identified the hitherto anonymous sugar manufacturer. The commission reported that all materials used in brewing in Manchester were free from arsenic except for certain sugars supplied by Bostock & Co. of Liverpool. The commission recommended therefore, that any beer brewed from Bostock's sugars should be immediately recalled, and, if found to be contaminated, destroyed. Furthermore, no beer was to be sent out without having first been tested, and certificates verifying its freedom from arsenic should be issued with the beer. In order to ensure uniformity of response, the experts proposed that all certificates be issued in the name of the M.B.C.A., which would supervise the testing process.[63] At a meeting of the M.B.C.A. the next day, the expert commission's recommendations were unanimously adopted, with the secretary of the association declaring his satisfaction with their

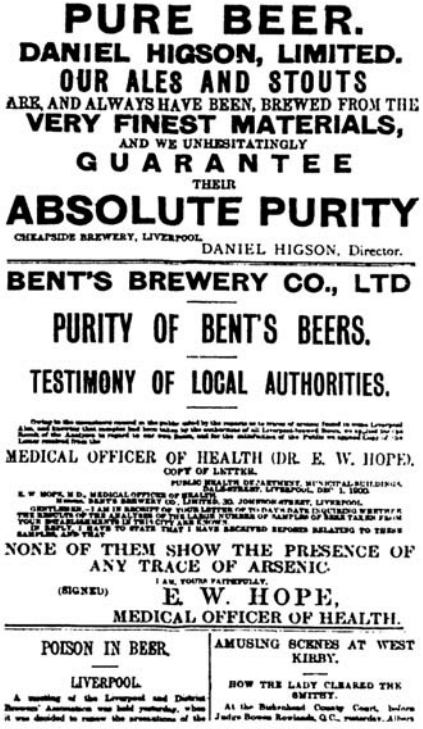
efforts so far: ‘no more critical and complete inquiry could be made than that conducted by the experts’.[64]

In identifying Bostock & Co. as the source of the poison, the M.B.C.A. were doing far more than simply assisting other brewers to confirm whether or not they had used contaminated sugar. Rather, they were providing the press and the public with a name that would allow the industry to distance itself from responsibility for the epidemic. Now that Bostock's had been identified and the association formed, this could be substantially reinforced in legal actions taken by individual brewing companies. The brewers had fulfilled their promise of elucidation, and identified their scapegoat.

Equally beneficial for the brewers was the fact that the epidemic was showing signs of abating. By early December, newspapers reported that at the various hospitals involved in the epidemic new admissions were greatly reduced.[65] This was largely a result of the actions taken in concert by local public health and sanitary authorities and brewers. In Manchester and Salford, as soon as news of Dr Reynolds's conclusions was received, Food and Drugs Act inspectors had begun collecting samples of beer from throughout the region, passing them to public analysts for testing. Contaminated stock found at public houses and other outlets was sealed under the authority of the Food and Drugs Act, and was then withdrawn by the brewers and destroyed. Meanwhile, brewers who had employed their own analysts to ensure that their products were pure were destroying suspect materials without reference to external authority. As well as having tests carried out by their own analysts, many brewers took the additional precaution of sending samples to their local medical officer of health for 'official' confirmation that their products were pure.

By January, 1900, the extent of the arsenic epidemic had been largely realised. Approximately 3,000 persons had been diagnosed as suffering from chronic arsenic poisoning as a result of drinking contaminated beer, with upward of 70 cases proving fatal; the final total number of sufferers was estimated at 6,000.[66] The number of fatalities may also have been significantly higher, as it was admitted by the medical authorities that many deaths attributed to 'alcoholic' neuritis in the preceding months might instead have been due to arsenic poisoning.

As a pro-active counter to the negative publicity generated by the daily newspapers, many brewing companies decided to generate their own positive publicity material. This took the form of leaflets distributed among customers, notices posted in retail outlets, advertisements in local newspapers, and written guarantees sent out with supplies of beer, all declaring the purity of a particular manufacturer's products.[67] In Birmingham, for instance, local brewers issued circulars declaring that they did not use sugar from Bostock & Co. in their products, while in



PURE BEER.
DANIEL HIGSON, LIMITED.
OUR ALES AND STOUTS
ARE, AND ALWAYS HAVE BEEN, BREWED FROM THE
VERY FINEST MATERIALS,
AND WE UNHESITATINGLY
GUARANTEE
THEIR
ABSOLUTE PURITY
CHEAPSIDE BREWERY, LIVERPOOL. DANIEL HIGSON, Director.

BENT'S BREWERY CO., LTD
PURITY OF BENT'S BEERS.
TESTIMONY OF LOCAL AUTHORITIES.

MEDICAL OFFICER OF HEALTH (DR. E. W. HOPE).
COPY OF LETTER.
NONE OF THEM SHOW THE PRESENCE OF ANY TRACE OF ARSENIC
(SIGNED) E. W. HOPE,
MEDICAL OFFICER OF HEALTH.

POISON IN BEER.
LIVERPOOL.

AMUSING SCENES AT WEST KIRBY.
HOW THE LADY CLEARED THE SMITHY.

Figure. 4. Advertisements of beer purity'. Liverpool Mercury*, 5th December 1900.*

Liverpool newspaper advertisements emphasised the fact that brewers' products had been tested by public or independent analysts and had been found to be arsenic-free. Some Liverpool brewers even made use of official replies from district medical officers of health as advertisements for their products, to the annoyance of the city council.[68] In Manchester, Groves & Whitnall began issuing certificates of purity with all beer from 27th November.

This policy was not confined to brewers operating in those areas which had experienced poisoning outbreaks. In the panic that ensued from the epidemic, brewers across the country were presented with the choice of either mounting a public defence of their products, or else risk losing their market share to a competitor who was prepared to use publicity to their own advantage. Those brewers who did not use brewing sugar were especially keen to advertise this fact, emphasising that their product purity did not result from the imposition of analytical safeguards - even though these were now offered as an additional reassurance for customers - but was the result of using 'natural' brewing ingredients.

Arsenic epidemics occurring elsewhere in the north-west and midlands, generated similar policies of co-operation between local authorities and brewers, based upon the systematic analysis, withdrawal and destruction of contaminated beer and brewing ingredients. In Liverpool, for example, a special meeting of the L.B.A. was called on 28th November, which resolved unanimously to appoint an expert committee to investigate the crisis, while rendering every assistance to public officials in their inquiries.[69] A press release two days later confirmed that the L.B.A. had engaged their own expert commission, and that 'severe analytical … tests [were] being applied to all ingredients'.[70] Not only would they destroy all impure beer without hesitation, but also any 'which is in the least suspected'.[71]

On 3th December, the Manchester Courier expressed the hope that the epidemic was at last on the wane, a fact confirmed one week later by what it described as a 'marked decrease' in the number of new cases of peripheral neuritis.[72] Deaths attributable to arsenical poisoning continued, but these were considered to be the result of poisoning that had occurred prior to the cause of the epidemic being understood, or during the first few days afterward.

Confidence was also growing that the region's beer was again safe, with one 'leading Manchester brewer' prepared to state that 'no infected beer is now on the market'.[73] Mr C.K. Redfern, the secretary of the M.B.C.A., confirmed that £40,000-50,000 worth of beer had been run into the sewers in order to secure a safe supply for the city.[74]

If brewers and public health authorities now believed that they had successfully identified and contained the source of the epidemic, further developments in Manchester were threatening to prove them premature in their assumptions. A letter to the *Times* from Charles Estcourt, the Manchester city analyst, revealed that he had uncovered a new and apparently unsuspected source of arsenic contamination. After examining various samples of barley-malt, he had discovered that several of them were contaminated with arsenic in quantities sufficient to register in the finished beer.[75] Although he had not finished his investigations, Estcourt felt sufficiently confident to recommend that brewers should extend their analysis of raw materials to malt as well as sugar. Writing again five days later, Estcourt, confirmed his earlier findings, alleging that certain samples of contaminated barley-malt were capable of polluting beer well above the level deemed dangerous to health.[76] Estcourt concluded that the arsenic was not natural to the barley, but had been transmitted onto its surface during kilning. Typically, malt grains were dried in a kiln where they were exposed to the hot vapours of a coal or coke fire; if the fuel contained arsenic, which it often did, this was then carried into the combustion fumes, and thence onto the malt grains. Estcourt attributed different degrees of contamination to variations in drying times and arsenic levels in the fuel used.

With the epidemic now under control and the number of sufferers diminishing daily, the M.B.C.A. expert commission presented a further report to the Association.[77] Although it added little to what was known about the epidemic, the report was nevertheless important in further reinforcing public association of guilt with Bostock & Co. rather than any individual brewing companies. The commission also reported that their recommendations regarding the testing and certification of all new beer supplies had been fully implemented, and, as all beer brewed with Bostock's sugar had now been destroyed, no further danger arose from this source. The recent discovery of arsenical malt meant that their work was not yet over, but by apportioning blame

for the source of current epidemic and instituting safeguards against its reoccurrence, they had effectively fulfilled their original terms of reference. By rendering their investigations and conclusions public, the M.B.C.A. had demonstrated that brewers had nothing to hide.

The M.B.C.A. expert commission continued its investigations to their conclusion, presenting its final report in early May, 1901.[78] The report confirmed that beer brewed using arsenical malt was capable of endangering human health - indeed, dangerous levels of arsenic had been detected in several samples of beer brewed using malt and hops only. However, now that regular and systematic testing of ingredients had been instituted by brewers, arsenical malt was as easily allowed for as arsenical sugar. The commission's final recommendation was that brewers should insist upon written guarantees of purity with all purchases of brewing materials. In fact, the discovery of arsenical malt was ultimately of greater long-term consequence to the medical profession than it was to the brewing industry.

Legal repercussions

Suffering of the nature and scale witnessed in Manchester was bound to result in some form of legal process, and, possibly, for retribution to be meted out on those deemed responsible. Until now the brewing industry's defence had been based upon the public denouncement of Bostock & Co., casting themselves as honest tradesmen temporarily disappointed by an incompetent supplier. According to the brewers, as soon as the problem was realised, they had done everything within their power to halt it. Now the courts would provide an opportunity for them to demonstrate to the public and the Government exactly what they had done, and that it had been enough. Equally, the courts could damn them as reckless, avaricious poisoners, defiling the national beverage with noxious chemicals in pursuit of ever greater profits. The successful defence of any legal action was thus of the utmost importance. In common with this objective, counsel representing the brewing interest would be superintended by Fletcher Moulton, Q.C., M.P., who was already enrolled on the M.B.C.A. expert committee.

The first prosecutions connected with the epidemic were of retailers from whom contaminated beer samples had been obtained, and which were judged to constitute offences under the Sale of Food and Drugs Act (S.F.D.A., 1875). Initially, local authorities had allowed beer sellers an amnesty whilst all efforts were directed at identifying and removing contaminated beer from the marketplace. But, by the end of November, this forbearance was being withdrawn. On 30th November, the sanitary committee of Manchester Corporation gave notice that any samples now taken that tested positive for arsenic would result in prosecution of the vendor. Similarly, Dr Tattersall, issued a circular to every drink

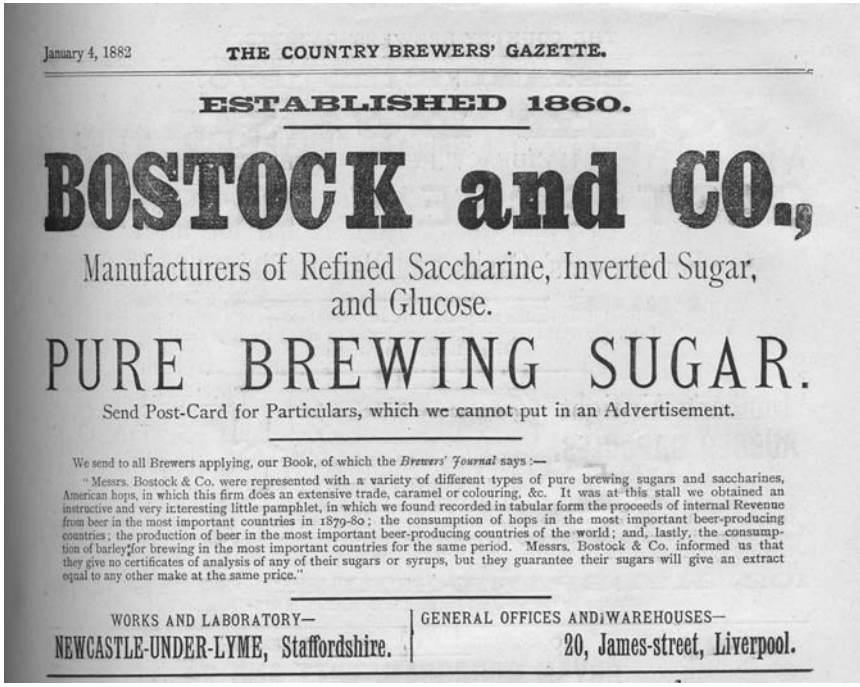
seller in Salford, informing them that from now on, it would be considered an offence to sell contaminated beer.[79] This hardening attitude reflected the desire of local authorities to deflect criticism that in spite of widespread suffering, they had yet to institute any legal proceedings. Reiterating points raised during a 'heated debate' of Manchester City Council, the Manchester Guardian asked why, two weeks after discovery of the poison, prosecutions of those involved had not begun - a sentiment echoed in other local newspapers.[80]

The first conviction at Manchester was of Elizabeth Goulder, a liquor license holder, with a further 17 retailers convicted and fined for contravening the S.F.D.A.[81] Similar convictions were secured across the north-west and midlands. However, aside from negative publicity, the material threat to brewers of such prosecutions was not great since the maximum penalties allowed by the S.F.D.A. were low in business terms - £50 and £20 respectively.[82]

Potentially far more serious for the incriminated parties were coroner's court inquests into the deaths of beer drinkers certified as having resulted from arsenic poisoning. A verdict of criminal neglect, culpable negligence or reckless indifference could lead to criminal prosecutions for manslaughter and the possibility of custodial sentences and hefty fines.

At Manchester and Liverpool, where the greatest number of deaths had occurred, the question of establishing criminal liability resolved into two test-cases; interest was such that they were reported at length in the *B.M.J.*, *Lancet*, *Brewers' Journal* and *Brewers' Guardian*, as well as the daily papers. The Manchester inquest was into Mary Dyer, who had died at the Crumpsall Workhouse in November 1900; Dyer had been an habitual consumer of Groves & Whitnall's beers. Assisting the brewers in their defence, Dr Tattersall stated that they had been 'perfectly frank and free' in assisting him in tracing the source of the contamination.[83] Building upon Tattersall's testimony, James Groves gave the court a detailed account of how his company had acted swiftly and decisively the moment it was 'suggested' that arsenic might be present in beer.[84]

The inquest next focused on where, between Bostock's and Nicholson's, ultimate responsibility lay. In the event, the history of transactions between the two companies proved to be so confusing as to defy the jury's best efforts to unravel and apportion blame. Bostock's maintained that the fault lay with Nicholson's for supplying them with impure acid when they had always previously supplied pure; Nicholson's, held that since Bostock's contract did not specifically request pure acid, they had been quite justified in supplying impure. What did become clear during cross-examination was that Nicholson's explanation for the change of supply was highly questionable, and Bostock's procedures for testing raw materials and finished prod-



January 4, 1882 THE COUNTRY BREWERS' GAZETTE.

ESTABLISHED 1860.

BOSTOCK and CO.,

Manufacturers of Refined Saccharine, Inverted Sugar, and Glucose.

PURE BREWING SUGAR.

Send Post-Card for Particulars, which we cannot put in an Advertisement.

We send to all Brewers applying, our Book, of which the *Brewers' Journal* says :—

"Messrs. Bostock & Co. were represented with a variety of different types of pure brewing sugars and saccharines, American hops, in which this firm does an extensive trade, caramel or colouring, &c. It was at this stall we obtained an instructive and very interesting little pamphlet, in which we found recorded in tabular form the proceeds of internal Revenue from beer in the most important countries in 1879-80; the consumption of hops in the most important beer-producing countries; the production of beer in the most important beer-producing countries of the world; and, lastly, the consumption of barley for brewing in the most important countries for the same period. Messrs. Bostock & Co. informed us that they give no certificates of analysis of any of their sugars or syrups, but they guarantee their sugars will give an extract equal to any other make at the same price."

WORKS AND LABORATORY— NEWCASTLE-UNDER-LYME, Staffordshire.

GENERAL OFFICES AND WAREHOUSES— 20, James-street, Liverpool.

Figure 5. Advertisement for Bostock & Co. from less-trying times. Country Brewers' Gazette*, 4 January 1882.*

ucts were inadequate. The jury's verdict was that Mary Dyer's death was caused by arsenic poisoning as a result of consuming contaminated beer. On the question of responsibility they returned an open verdict.

Perhaps unsurprisingly, given the near identical testimony presented, the verdicts of the Liverpool jury into the death of Mary Rankin were as inconclusive as those at Manchester. Minor admonishments aside, the brewers had been successful in convincing both inquests that they were unwitting agents in the epidemic, not its perpetrators. As the *Manchester Courier* remarked of the Liverpool verdicts:

> after the frank assistance rendered … in their quest for the source of the evil ..., every fair-minded man will admit that the brewers come out of the inquiry with a clean record.[85]

However, the process of shifting responsibility for the epidemic away from the brewing industry was not yet complete. The method for doing so was the instiga-

tion of civil actions for damages against Bostock & Co. by brewers forced to destroy contaminated beer. As the Manchester Courier explained,

…the result is of the deepest importance to the brewers, not merely from the pecuniary point of view, but as affording another convincing proof that the liability for the arsenic is not theirs.[86]

The first case against Bostock & Co. was heard at Manchester in February 1901; the plaintiffs were Richard Holden Ltd., of Blackburn.[87] Judgement was given against the defendants for £1,980 and costs, less £93 due to the defendants for goods supplied.[88] Following this, and bearing in mind several outstanding actions, Holden's successfully petitioned for the compulsory winding-up of Bostock & Co. By April, the total value of claims against Bostock's had risen to £132,443.[89] Foremost amongst these were Groves & Whitnall, claiming damages amounting to £15,769.[90] However, the payment of any of these claims depended entirely upon Bostock's being able to recover damages from Nicholson's,

for the negligent and wrongful supply of sulphuric acid not made from brimstone and not a pure commercial acid in breach of a contract to supply the plaintiffs with such aforementioned acid.[91]

This action began in January, 1904, with many of the same witnesses called, and the testimony largely a rehash of the earlier inquests. Mr Justice Bruce gave his judgement on 8th March 1904; his opinion was that Nicholson's had breached warranty by supplying impure acid, but if Bostock's had exercised 'ordinary care', they would have discovered the presence of arsenic in the sugar.[92] The award of damages was, therefore, limited to the value of the arsenical acid and the value of the sugar spoilt by using it; crucially, claims for the damages sought by brewers were rejected.

Unsurprisingly, the brewing press was unsympathetic to the plight of Bostock & Co., even if the outcome of the case meant that individual brewers were likely to have their damage claims significantly reduced.

The moral [opined the *Brewing Trade Review*] is that they should have not have relied so entirely on the warranty, but have found out for themselves whether the acid was fit for use or no.[93]

The irony is that the condemnation was equally applicable to the brewing industry. If Bostock's chemists were lax in failing to test their products, then the same was true of the brewers, whose only defence was ignorance and the misplaced assurance that this had never happened before.

However, apart from licensee prosecutions under the Food and Drugs Act, the trade's legal team, under the leadership of Fletcher Moulton, had been successful in protecting the good name

of the trade. From the outset, the brewers had publicly identified Bostock & Co. as the source of the contamination, while presenting themselves as innocent agents doing their utmost to ensure that an episode like this could never happen again. Legal process had confirmed this, and Bostock & Co. were left to sink under the burden of guilt - and writs - that had been placed on them.

The disputed existence of alcoholic neuritis

By mid-December 1900, the consensus amongst medical practitioners was that arsenic was to blame for the epidemics of peripheral neuritis. The evidence was compelling; in virtually every locality affected by the disease, arsenic had been detected in beer, and the classic symptoms of chronic arsenic poisoning identified in the victims. However, the near-universal acceptance of Reynolds's explanation opened up a new debate which questioned the very existence of alcoholic neuritis. The realisation that arsenic, not alcohol was responsible for the Manchester epidemic revealed the extent to which clinical diagnosis was informed by class-based narrative paradigms. To some practitioners this suggested that the disease frame of alcoholic neuritis was fundamentally flawed and required substantial renegotiation - or, more radically, that the condition known as alcoholic neuritis could simply be written off as a medical error.

At the forefront of those questioning the existence of alcoholic neuritis was Ernest Reynolds. He presented his views at the December 1900 meeting of the Liverpool Medical Society, and repeated them some three weeks later before the Royal Medical and Chirurgical Society in London.[94] His contention was simple: that alcoholic neuritis did not in fact exist. Reynolds claimed that he had doubted alcohol was capable of causing peripheral neuritis for over ten years, a view that had been reinforced through his greater experience of the condition. The basis of his doubt was the absence of alcoholic neuritis in spirit drinkers. Now, his earlier opinions seemed to be confirmed by the very feature that had led to alcoholic neuritis being so well described at Manchester - its geographical specificity. The epidemic proper had been tied to a single local supply of contaminated brewing sugar. However, the subsequent discovery of arsenic in local barley malt pointed to arsenical beer having been on the marketplace for many years, although to a considerably lesser extent than during the epidemic. This alternative source of arsenical beer was also restricted to Manchester and the north-west of England, mirroring the normal geographical prevalence of the disease. Arsenic was thus capable of explaining both epidemic and endemic alcoholic neuritis. Reynolds saw the corollary of his hypothesis in the statement of Sir William Tennant Gairdner, the eminent Scottish physician, as to the great rarity of alcoholic neuritis amongst the whiskey-drinking poor of Glasgow - a

group not usually noted for temperance of habit.[95]

Other practitioners refused to accept that alcoholic neuritis had been wrongly constructed out of a series of false premises. On one level this amounted to a simple denial of Reynolds's hypothesis.

> As is usual in such 'scares' [wrote one physician] there have probably been many cases of 'neuritis' reported, in the production of which arsenic had no share whatever.[96]

Similarly, Dr Judson Bury, the co-author of *A Treatise on Peripheral Neuritis*, disagreed with Reynolds's assertion that alcohol alone was incapable of causing peripheral neuritis. Bury was the acknowledged expert on peripheral neuritis; now he was being forced to defend his and the late James Ross's diagnostic frame. Bury insisted that previous to the epidemic he typically treated twenty to thirty cases of alcoholic neuritis yearly, some of which involved spirits only and so could not be blamed on arsenic.[97] Ross also emphasised the importance of subtler symptomatological differences that distinguished alcoholic from arsenical neuritis - greater ataxia with arsenic, greater mental derangement with alcohol, and so on - differences less experienced practitioners might well overlook. He agreed with Reynolds that arsenic was responsible for the Manchester epidemic, but refused to allow that alcoholic neuritis had always really been arsenic induced.

Bury was not alone in insisting that widespread misdiagnosis did not invalidate the disease frame. Dr Nathan Raw, for example, maintained that ‘true alcoholic neuritis’ was a distinct condition, which he had observed in those who drank spirits exclusively.[98] However, the problem with these counter arguments were that they failed to address the crucial issue of geographical specificity. If, as Raw and Bury insisted, alcoholic neuritis did indeed exist as a true form of peripheral neuritis, the question remained as to why it had long been markedly more prevalent in Manchester than anywhere else in Britain; as the *Lancet* noted, never before had the aetiology of alcoholic neuritis been ‘so openly challenged’.[99]

Ultimately, time rather than debate would answer the question. If Reynolds was correct, following the removal of arsenical beer from the streets of Manchester, 'so-called' alcoholic neuritis would accordingly diminish and eventually disappear; if Judson Bury was correct, what would remain was a core of cases of 'true' alcoholic neuritis.

The answer was forthcoming one year later, and was provided by Ernest Reynolds before the Royal Commission appointed to investigate the arsenical poisoning epidemic. Reynolds reported that during the preceding year he had seen a large number of very heavy drinkers, but, of these, only two had presented the symptoms of alcoholic neuritis, both of whom were spirit drinkers: peripheral neuritis in beer

drinkers had completely disappeared.[100] Furthermore, Reynolds claimed that this was the same experience of the hitherto sceptical Dr Judson Bury. Whereas in previous years the M.R.I. would typically see twelve to twenty cases, it had not registered a single case for nine months. Statistically, the incidence of alcoholic neuritis in Manchester was now comparable with that of other British cities. The conclusion was inescapable: the earlier 'normal' occurrence had been due to arsenical malt, and the epidemic to arsenical sugar.[101] Eradicating arsenic from local beer had effectively eradicated alcoholic neuritis. The consensus of expert medical opinion - now including Reynolds - was that a 'true' form of alcoholic neuritis did indeed exist, but that this was a far, far rarer condition than had long been the case at Manchester.

Also forthcoming at the Royal Commission was an explanation for the long-term geographic specificity of 'alcoholic' neuritis in the Manchester region. According to Mr H.A. Taylor, a Hertfordshire maltster, in the north and midlands, gas coke had long been used as fuel in the kilning process, and gas coke typically contained much higher quantities of arsenic than other forms of coke or coal, the result of which was higher levels of arsenic contamination of the finished malt, and hence of local beers.[102] Thus, a geographical coincidence existed between the supply of arsenical malt and arsenical brewing sugar, with the latter ultimately leading to the discovery of the former.

Conclusion

The Manchester arsenic-in-beer epidemic was a dramatic wake-up call for an industry lulled into a false sense of security by prolonged financial growth. During the last two decades of the nineteenth century the brewing industry had progressed commercially and technically. However, the principal cause of the arsenic-in-beer epidemic was not progress, but complacency - the extent of this complacency revealed by the fact that the alarm was sounded by the medical profession rather than from within the brewing industry. Somewhere in the drive for greater profitability, similar progress in consumer protection had been overlooked; the result was 70 deaths and upward of 6,000 non-fatal cases of arsenic poisoning. The epidemic dramatically illustrated that the mass-production of foodstuffs should be accompanied by new levels of consumer protection. Product purity could not be left to chance: it demanded new and rigorous safeguards. While chemical analysis was an indispensable element in controlling the brewing process, it had not been adopted as a safeguard against product contamination. Fortunately, the brewing industry instituted such safeguards so quickly and so thoroughly as to escape the legal consequences of their earlier inaction.

But, equally, the eventual discovery that arsenic, not alcohol, was the cause of both epidemic and endemic peripheral neuritis in the Manchester region revealed the extent to which social

diagnosis had clouded the clinical eye of local medical practitioners. For many years prior to the epidemic, substantial numbers of beer drinkers had been systematically misdiagnosed as suffering from alcoholic neuritis when in reality they were being slowly poisoned by arsenic. The basis for this misdiagnosis was the willingness of medical practitioners to disregard patient testimony in favour of prejudice about the honesty and reliability of the lower working class. Thus, the epidemic was as much of a wake-up call for the medical profession as it was for the brewing industry. The reason the medical profession emerged from the epidemic with greater credit was simply because they happened to discover the source of their long-term error before it was pointed out to them.

The 1900 epidemic and the subsequent stopping of all sources of arsenic contamination resulted in the effective elimination of a medical condition that had long been endemic to the Manchester region. And, by precipitating awareness of the need for rigorous controls over product purity, the crisis taught the brewing industry a valuable lesson, to its benefit, and that of the British beer-drinking public. Following the crisis, the nation's beer supply was probably purer than it had ever been, a key part of which was the discovery that barley-malt was also liable to contamination with arsenic. Although contaminated sugar was responsible for the Manchester epidemic, it was apparent that arsenical malt had been poisoning beer drinkers for a far longer period. If the epidemic had not occurred it is a matter of conjecture as to how long the arsenic contamination of malt would have remained undetected, and how long hospital wards in the north-west of England would have been populated by the misdiagnosed victims of arsenical beer.

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