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EDITORIAL

THE LEGEND OF CAMELFORD: MEDICAL CONSEQUENCES OF A WATER POLLUTION ACCIDENT

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INTRODUCTION

THE small Cornish town of Camelford in the South West of England, named after the winding river on which it is built, has two claims to a place in English folklore. The first, is as the site of Camelot of Arthurian legend, and the second is its reluctant prominence at the centre of 'The most serious water pollution emergency in Britain for decades' [1] which took place on 6 July 1988.

THE ACCIDENT

The accident involved the inadvertent deposition of 20 tonnes of aluminium sulphate, a chemical used in water purification, into the reservoir distal, rather than proximal, to the purification plant. As well as adding a massive aluminium load to the domestic water supply of 12,000 residents and 8000 holiday visitors in the district of Lowermoor, north Cornwall, the acidity of the compound leached other metal salts including those of copper, zinc and lead from water bearing pipes. For 2–3 days, residents were subjected to acid tasting, milk-curdling and sometimes grossly discoloured water. Immediate symptoms included nausea and vomiting, skin rashes and mouth ulcers [2]. Others noted that their hair, skin, or finger nails had been stained blue or brown. Soon, rumours were spread by local and national TV and radio, of shoals of dead fish in local rivers, widespread sickness in farm animals and disruptive behaviour in school children. Complaints were made to the water company responsible, South West Water who were slow to act, and when they did, gave false reassurance [3]. Public fears and outrage grew although, curiously, few residents were sufficiently concerned to consult their General Practitioners at the time (only 2 out of 600 consultations) [3]. A commission of enquiry was set up by the Department of Health, headed by Professor Dame Barbara Clayton, a chemical pathologist, which published the first of two reports in July 1989 [3].

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OFFICIAL REPORTS

The first report detailed the composition of serial water samples taken at different sites and showed how that for 3 days, the European Community maximum admissible concentration—established for largely aesthetic reasons—for aluminium (0.2 mg/l) had been exceeded several hundred fold. However, the committee cited evidence that very little of the normal dietary aluminium (average 5–10 mg/day) is absorbed and only a fraction of this passes the blood–brain barrier. Little is known about the effects of aluminium poisoning in general but experience of one relevant clinical scenario was called upon, namely, 'dialysis dementia'. This refers to an encephalopathy first noted in patients with chronic renal failure undergoing dialysis due to accumulation of aluminium in the brain often worsened by aluminium-containing medications. The committee concluded that acute short-lived intoxication was so unlike the circumstances of long-term renal dialysis that there was little cause for concern. The same reasoning was applied to the bone disorder (osteomalacia), again, formerly associated with aluminium toxicity in dialysis patients. Nevertheless, the Clayton report was careful to acknowledge the natural anxiety provoked by the incident and commented on the loss of trust between consumers and providers of water. Further health monitoring of the population at risk was considered unworkable as well as unnecessary—other than through routine health statistics—since the results of any survey would, '. . . *be heavily influenced by people's fears of long term effects*' [3].

Far from being the last word, that was only the beginning. Certain residents of Camelford were not convinced; a cover-up was suspected. The Clayton committee was not seen as independent, and furthermore, people were still experiencing symptoms, in fact, increasingly so. Litigation was well under way, with local meetings arranged for potential claimants. Local people decided to organize their own enquiry and formed a pressure group [4]. Adding insult to injury, newspapers misquoted the report as claiming that residents' complaints were due to hysteria. A powerful television programme made by the reputable BBC '*Horizon*' team entitled 'Camelford: a bitter aftertaste' was broadcast in the spring of 1991 and showed the ravages of supposed aluminium dementia. A review in the *British Medical Journal* communicated the disquiet to the wider medical profession [5].

A number of unfortunate coincidences fanned the flames of indignation and may have steered the focus of health-related attention to the nervous system and the neuropsychiatric domain. First was the epidemiological study by Barker and colleagues [6] published in January 1989, suggesting that aluminium in drinking water was a risk factor ($RR = 1.5$) for Alzheimer's disease. This seemed to tie in with the well known aluminium content of senile plaques and tangles seen in the brains of Alzheimer victims. Could drinking aluminium containing water lead to Alzheimer's disease—several years later? Quantifying such long-term risk presents formidable challenges to epidemiologists, thus tending to leave the element of doubt. Similarly, the potential risk of 'Mad Cow Disease' (Bovine Spongiform Encephalopathy), a prominent preoccupation in early 1990 [7], has the same irrefutability problem: could eating infected meat lead to a few cases of Jakob-Creutzfeldt dementia decades later? Whatever the causes, cognitive symptoms came to dominate the Lowermoor clinical picture, especially memory loss, poor concentration, plus mental and physical fatigue.

The problem with such symptoms is that they are subjective and difficult to verify without the subject's awareness that verification is taking place. Primary care and community studies have established that these symptoms are both common and associated with psychological distress and psychiatric disorder [8–11]. It was therefore of considerable interest that an 'objective test' of aluminium induced intellectual impairment was available to Camelford residents [1]. The test was of visual evoked potentials and had been applied in research on 10 dialysis patients [12]. The paper described significant correlations between poor performance on an automated psychological test battery, the Bexley and Maudsley Automated Psychological Screening Battery (BMAPS) [13]. Similar findings were being claimed at Camelford. Detailed examination of the testing and its interpretation will be given below.

The second, longer Clayton report [14] heard evidence from a wider range of experts. Their conclusions were stronger than before: there was no evidence of long-term effects on health as a result of the water contamination. Such symptoms which were being reported by up to 400 people, were put down to anxiety. The report was, rightly, wary of dismissing such symptoms and indeed affirmed the *'real mental and physical suffering in the community'* (p. 31) following the accident, but attributed this to non-permanent, non-physical factors. Despite its thoroughness and measured tone, commentators anticipated that the report would still fail to satisfy some individuals [15]. Psychological 'damage' can lead to successful compensation claims, provided it is shown to have resulted in a defined psychiatric disorder, as in the well-known example of post-traumatic stress disorder [16], but it was clear that at Camelford a physical attribution was required by sufferers. Finally, nearly 5 yr after the event in July 1994, the many litigants had to be satisfied with a modest out of court settlement.

SENSITIVITY AND SPECIFICITY

The main evidence regarding cognitive impairment came from the same automated psychological tests, neuropsychological assessments and evoked potentials, mentioned above. The inconclusiveness of this was raised in the final Clayton report. It was pointed out that psychological disturbances such as anxiety and depression may complicate the interpretation of abnormalities on psychological tests. The report highlighted the difficulty of extrapolating evidence from research laboratories to clinical populations, especially relevant to neurophysiology where there is much within- and between-laboratory variation and where the entire 'normal range', adjusted for age and gender is seldom delineated. For example, the BMAPS was devised to *screen* for cognitive impairment in 103 alcoholic patients with approximately 60 normal controls drawn from hospital employees available for comparison. The authors suggested in their manual, that subjects scoring less than one standard deviation below the mean, as the level below which *further testing is indicated* [13]. If this cut-off is used as indicating *pathology*, it will, by definition, include one sixth of the population.

The abnormal evoked potentials recorded on some of the symptomatic consumers of the contaminated water require explanation. The technique involves presenting two types of visual stimulus: simple light flashes and a checker-board pattern. A negative cortical potential is usually recorded in response to a flash, after around

120 msecs, and positive after the pattern at 100 msec, giving the latter potential its common name, the P100. The technique introduced by Wright and colleagues [17] to study dementia, and adapted by Altmann and colleagues [12], took as its main dependent variable the difference between these two potentials. As well as chronic renal patients, prolongation of this difference has been found in Alzheimer's disease as well as other causes of cognitive impairment [18] i.e. the test is not highly specific. As Philpot and colleagues [19] demonstrated, the flash-pattern difference is no better at distinguishing Alzheimer's disease from other conditions than the average clinician unassisted by investigations. While the use of a difference measure allows a control for individual differences in response time, it may obscure increases or decreases in either (or both) of the underlying parameters. Furthermore, the image of evoked potentials as entirely 'objective' may be confusing. Like most physiological measures they are influenced by such things as anxiety and hyperventilation [20] as well as being open to deliberate deception (such as focusing in front of the display) [21]. Indeed, the more sensitive the measure, the more prone it will be to such influences.

THE 'DENOMINATOR PROBLEM'

Another shortcoming of the data presented to the committee arose from it being gathered from a biased and self-selected group of subjects, often undergoing investigation because of physical and mental symptoms, and inevitably influenced by the adversial atmosphere that accompanies on-going litigation. Without knowing the base rates of the various symptoms in the population (i.e. 'the denominator' in epidemiological parlance) it is impossible to determine whether the rates have changed. Not only is it important to have an estimate of such rates before the incident in question, but also before awareness of the possible dangers of the incident have been disseminated. Occasionally, such data are available (see below) but otherwise GP and hospital records may fulfil this function. The possibility of a small group of individuals who habitually present, with undiagnosed physical symptoms, distorting levels of an apparently new complaint must also be born in mind.

PUBLISHED RESEARCH

The Clayton committee had to digest and summarize much research, little of which reached publication and hence wider scrutiny. One of the first published reports to emerge was of a survey of farm animals. It confirmed the transient appearance of dead fish in the two rivers draining the contaminated reservoir but refuted any rise in illness or deaths to livestock [22].

Local community medicine specialists attempted to gather data on consumption, symptom prevalence and consultation behaviour from a postal questionnaire sent to a random sample of 500 households within the district exposed to the aluminium increase and controls drawn from a neighbouring district [23]. Changes in water taste were reported more in the exposed group as were all 18 symptoms on the questionnaire (which did not include cognitive dysfunction). Approximately one-quarter of the exposed group accounted for the bulk of the symptoms, most of which pre-dated the incident, and none were present in more than that proportion. Joint pains emerged as especially liable to occur in the exposed group (21.9% vs

2.6%; relative risk 8.3, 95% CI 4.8–14.3). A few (13%) reported taking a week or more off work.

There were a number of weaknesses in this study. Response rates were disappointing (around 45%) but were comparable for 'cases' and controls with a suggestion that respondents were biased towards those with symptoms. More importantly, data were recorded retrospectively. Households were mailed in November 1988, some 4 months after the incident and respondents were asked to record symptoms and behaviour in the preceding months of July and August. Adverse publicity surrounding the incident was proposed as a major contributing factor to some of the positive results.

The possibility of teratogenicity was examined by child health specialists who found no significant increase in serious congenital abnormalities in the 88 pregnancies in women at risk in comparison to controls [24].

However, a report of two cases in whom bone biopsies were taken 7 and 14 months after the incident, confirmed the presence of abnormal levels of aluminium on the first occasion—compatible with an increased aluminium load—which were not seen at follow-up. There was thus no doubt that at least in some subjects, exposure resulted in excess absorption of aluminium [25].

Of most interest in the current context are reports of psychological functioning, both by McMillan and colleagues [26, 27]. The first detailed a number of clinical, psychometric tests of overall intelligence, memory, concentration and verbal fluency on nine self-referred, symptomatic residents from Camelford, including the two who had bone biopsies. The tests were repeated one year later. All were tested for the first time at least 12 months after the accident, with the exception of the biopsy cases who were seen, at 6–7 months and again, 19 months post-accident. There was a slight, non-significant discrepancy between estimated pre-morbid IQ and measured IQ on both occasions. Tests of verbal and visual memory were impaired in eight and five out of nine at initial testing, respectively, and this poor performance persisted at follow-up. A test of paced auditory information processing was reported as showing a mean decline for the whole group, which again was maintained. In addition, the group was rated on standardized scales for anxiety and depression. Curiously, one subject scored in the depressed range on the Beck Depression Inventory (BDI) [28] at first assessment but some 2 yr after the accident, four of six subjects tested were moderately or severely depressed (range 19–29). Similarly, employment status deteriorated: eight out of nine were reported to be in full-time work at the time of the accident, yet only one was in full-time and three in part-time work at initial testing, with two returning to part-time employment by the second testing session. Significant correlations between test performance and anxiety/depression were not found.

Other tests included plasma aluminium levels, MRI brain scans, lung function tests and radio-isotope bone scans, all of which were normal. GP records were available in seven cases and one was found to have a previous history of 'anxiety over poor physical health'. Interpretation of these results is complicated by the biased nature of the sample and the presence of emotional disorder, as the authors acknowledge, but also the fact that:

> '. . . the possible impairment of memory . . . led the patients to request assessment . . . since several *believed that these symptoms might be due to water contamination*'. Italics added (McMillan *et al.*, 1993, p. 37) [27].

Such beliefs are bound to influence a subject's test performance.

The other paper [26] concentrated on intellectual performance of all 39 local Camelford school pupils, aged 8–9 years, and had much stronger methodology. Test scores had been routinely gathered 2 months prior to the incident. These were repeated 10 and 22 months after the accident. Schools serving similar populations nearby were studied (there were 64 control children). The authors applied a number of sophisticated statistical techniques to the data and showed that, in general, the educational tests used were valid and reliable and that there was no evidence of intellectual decline in the Camelford children. Furthermore, there was no increase in the number of children referred for specialist help because of behavioural problems.

In summary, the available evidence, and the considered judgement contained in the final Clayton report give no support to material damage to health as a direct consequence of the sudden, temporary increase in aluminium consumption caused by the water purification error. So why then did some people suffer and complain for years after the event?

We suggest that the most likely explanation of the Camelford findings is that the perception of normal and benign somatic symptoms (physical or mental) by both subjects and health professionals was heightened and subsequently attributed to an external, physical cause, such as poisoning [29].

This mechanism is the final common path fed by a number of routes:

1. *The normal levels of somatic symptoms in any community*

Some symptoms such as fatigue, headache and poor concentration are far more common than many realise [30, 31]. In one early survey, only 14% of a community sample reported having no symptoms at all [32]. An American study of healthy university students taking no medication found that no fewer than 81% had experienced at least one somatic symptom during the previous three days [33]. During a 6 week period normal American women reported at least one somatic symptom on 43% of days—the figure for men being only slighlty less at 31%. Fatigue was one of the commonest somatic symptoms encountered, second only to headache [34]. A recent study using the Epidemiologic Catchment Area Program (ECA) found that of 26 somatic symptoms studied, no fewer than 24 had been a problem for more than 10% of respondents at some stage of their life, and for the vast majority such symptoms had interfered with routine activities, or caused them to attend a physician [11].

2. *Anxiety related symptoms following the incident*

There have been numerous reports of substantial, often dramatic, increases in psychiatric morbidity following either documented environmental toxicity [35], or even rumours of such exposure [36, 37], all of which can easily be amplified by media attention [38]. The natural widespread fear and concern following an incident can result in anxiety-related symptoms (cognitive failures, altered bowel habit, muscle tension etc.).

3. *Litigation*

Although the notion that individuals frequently invent symptoms in order to gain compensation is now discredited, there can be no doubting the substantial effect that the prospect of litigation has on distress, particularly of the ill-defined sort present at

Camelford. The need to demonstrate persistent disability, the experience of disbelief, and the adversial nature of the British legal process, all act to perpetuate disability.

4. *Somatization disorders*

In any such incident it is probable that among those afflicted are a few individuals with pre-existing somatization or abridged somatization disorders [39]. These are individuals with long histories of multiple physical complaints attributed to various physical causes, which were then diverted to fall in line with the prevailing complaints of the affected population.

Other more general influences also played a role in turning an incident like that at Camelford, into a legend. We have already noted the chance association between the water pollution incidence and the publicity accorded the report of a possible link between aluminium and Alzheimer's disease, still hotly debated in the scientific community. There is also the modern preoccupation with the state of our environment, reflected in general concerns over pollution, chemical toxins and other 'green' issues. These widespread concerns, which many share, can often appear in disease idioms, especially in such controversial fields as environmental medicine and clinical ecology [40].

Finally, we echo Shorter's observations on the changing nature of somatic symptoms [41]. As medical diagnostic techniques become more sophisticated, so do lay models of bodily functioning. Hence paralyses and movement disorders are less common while memory failure and fatigue more common. These are correspondingly more difficult to quantify and explain by clinical investigators.

THE REAL LESSONS FROM CAMELFORD

To this day it is impossible to quantify the morbidity, be it primarily psychological or physical, arising from the water contamination incident. The need for population surveys in response to public concern has been raised [42], but clearly these are expensive. Only in retrospect is it clear that such a response should have been mounted immediately in north Cornwall. By the time the Secretary of State had acted, it was clearly too late. Litigation, community action, self-appointed experts, consumer opinion polls, a media frenzy and accusations of a conspiracy, had already gathered momentum. To carry out epidemiological surveys after every potential scare—even on a fraction of the population at risk—would be expensive and would probably generate more anxiety than it alleviated. National data on levels of morbidity, collected in conjunction with the 1991 census will provide a valuable bench-mark in the event of further disasters.

Nevertheless, the response to the recent Shetland oil spill was exemplary [43]. The tanker *Braer* ran aground off the coast of the Scottish Shetland Isles and spilt crude oil from 5th to 11th January 1993. A survey including non-exposed controls was started on the 13th and completed on the 21st. A transient increase in headache, sore throat and itchy eyes only was found, and that seems to be the end of the matter.* Although locals may cite dour Gaelic stoicism as shaping such an outcome,

* An increase in psychological morbidity was recently reported 6 months after the grounding of *Braer*, in those exposed [44].

this speedy public health response must have obviated harmful speculation. Both the islands and north Cornwall depend on tourism and farming, and both would wish to convey an image as healthy, scenic, non-urban environments. Unlike Cornwall, the doctors in Shetland were lucky not to have had to deal with the irresponsible reporting which so inflamed passions in Camelford. These and other variables which turn an incident into a legend, merit serious study. Future investigations of environmental incidents should recall that social and cultural factors are as important as medical ones.

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