A Landmark Case in Asbestosis

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The first published account of disease attributed to occupational asbestos exposure was that of Nellie Kershaw, who died in 1924. The circumstances relating to that case are described and explanations are given for its not having a greater impact on policy at that time. This case, starting in 1898, is set in the context of missed opportunities for preventing a major public health hazard. The effects of this hazard are still being witnessed today.

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THE FIRST death due to pulmonary asbestosis described in the medical literature was reported by Cooke.[1] A more detailed record of the findings was subsequently published on December 3, 1927.[2] It was this latter study that gave the disease its name, "pulmonary asbestosis"; the previous short note was titled "Fibrosis of the Lungs due to the Inhalation of Asbestos Dust." The unfortunate patient's name was Nellie Kershaw. There had been other cases of illness and death attributed to asbestos inhalation before hers, but the observations had either been sequestered in a British departmental committee's minutes[3] or governmental papers[4,5] or patients had been seen by practicing physicians in areas near asbestos plants and the cases clinically noted but not made the subject of scientific reports.[4]

Nellie Kershaw's case was somewhat different. Her illness had been diagnosed during life by her physician, Dr Walter Scott Joss, and he had certified it as "asbestos poisoning" to an official medical benefits agency that referred the case to the asbestos company for which she worked. Second, the medical diagnosis was influential in having a postmortem examination done and the findings reported.

The case is reviewed herein after a passage of some 70 years and is set in the context of the long-playing tragedy of asbestos, a story of missed opportunities for avoiding a worldwide disaster.

THE LIFE AND DEATH OF NELLIE KERSHAW

The story, pieced together from a patchwork of surviving published and unpublished records, is that of Mrs Nellie Kershaw of Rochdale (near Leeds, England). Born to Elizabeth and Arthur Kershaw in 1891, she lived her short life in Rochdale, marrying Frank Kershaw, a tiler's laborer. In the manner of the time, she had left school in 1913 at the age of 12 and had gone to work in a cotton mill. After about 5 months, she moved to a local asbestos factory (Garsides). There she stayed for 14 years, transferring to Turner Brothers Asbestos Company on December 31, 1917, at the age of 26 years, where she worked in the textile factory at a roving machine. Such facilities were rather new; commercial exploitation of asbestos in Britain started around 1880, with three or four kinds of goods being made. By 1892, over 100 varieties were in production.[6] Environmental control was poor and health complaints were common.

From the accounts given by her family and her physician, Mrs Kershaw's health had been "quite good" while working at the cotton mill and at Garsides. While working at Turners, after her daughter was born (around 1920 at the age of 29), she started being treated by her panel physician for a lung condition. For 2 or 3 years her pulmonary symptoms waxed and waned but she continued to work until the week ending July 22, 1922, when she was issued a National Health Insurance sickness certificate of unfitness for work that gave entitlement to payment of benefit under specified circumstances. (The textile works' manager later stated that during her 4 1/2 years of employment she was absent for 24 months or so, but this may have been due to maternity absence.)

Being too ill to work, Mrs Kershaw applied for National Health Insurance benefits to the Newbold Approved Society (a Rochdale Friendly Society approved under the National Health Insurance Act, to which she had contributed). Unfortunately, as the sickness certificate given to her by her physician, Dr Joss, bore the diagnosis "asbestos poisoning" rather than a nonoccupational cause that would have entitled her to sickness pay, she was not qualified for benefits from that source. The president of the society wrote repeatedly on her behalf to persuade Turners that they should compensate her as her condition should have qualified under the Workmen's Compensation Act (J. Blomley, letter to Turner Brothers Asbestos Company, September 13, 1922). Under the Workmen's Compensation Act, employees who contracted certain diseases, as determined by the government, were entitled to payment by their employers. It was general practice for employers to take out insurance, although it was not, strictly speaking, compulsory. At that

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turer provides to *any* source (exempting the Department of Veterans Affairs); manufacturers will not be able to raise prices to Medicaid faster than the rise in the Consumer Price Index (*New York Times*, November 6, 1990:D-2).

While public costs have gained increasing attention, aggregate public benefits have not been analyzed. They include, at the least, the reduced risk of contracting an *untreatable* rare disease or condition, potential health care cost savings, and the fuller societal participation of people with orphan diseases. As just one example, patients with Parkinson's disease who can slow their disease progression with the orphan drug selegiline may save the public $10 million per week by delaying the initiation of disability payments and by providing tax revenues while they continue to work.[60]

To the extent that Medicaid, Medicare, and private carriers cover the cost of orphan product prescriptions, the larger issue of costs and benefits to the public may become more prominent and should be considered.

CONCLUSION

The Orphan Drug Act has resulted in new products developed for patients with rare diseases and conditions. This has been achieved at a cost to some companies, and to some patients, that was not intended and that has added issues of affordability to the debate. These issues will need to continue to be examined as changes evolve in technology, third-party payer coverage, and possibly in product protection. If major changes in the Act are proposed in the future, they should be viewed within this larger context to assess their relative costs and benefits for companies, patients, and the public.

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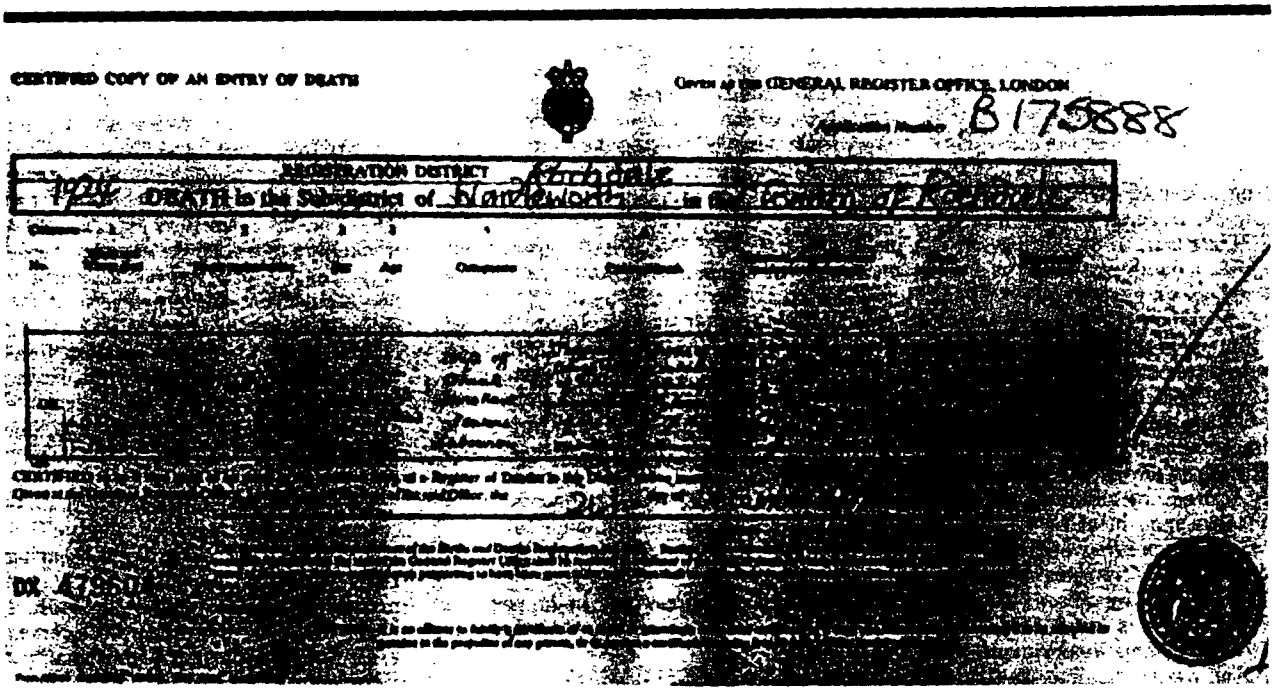
CERTIFIED COPY OF AN ENTRY OF DEATH

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B175888

REGISTRATION DISTRICT Rochdale

1924 DEATH in the Sub-District of Blatchworth in the County of Rochdale

DX 47950

Death certificate issued April 2, 1924, for Nellie Kershaw.

LAM 019062

time, asbestos-associated disease was not specifically scheduled and, consequently, Mrs Kershaw's employers were not compelled to compensate her. Her condition might have been accepted under the loose classification of "silicosis." Turners rejected this application and alerted its insurance company, expressing the opinion that it would be "exceedingly dangerous" to accept "any liability whatever in such a case." They felt that they ought to do all in their power to "repudiate the claim" (Turner Brothers Asbestos Company, letter to the London and Lancashire Insurance Company, September 21, 1922).

Mrs Kershaw wrote to the company:

> What are you going to do about my case? I have been home 9 weeks now and have not received a penny—I think it's time that there was something from you as the National Health refuses to pay me anything. I am needing nourishment and the money, I should [have had 9] weeks wages now through no fault of my own. (Mrs N. Kershaw, letter to Turner Brothers Asbestos Company, 1922)

No record has been discovered of how the Kershaws managed during the last 20 months of Nellie Kershaw's life. There is a record that Turners told Mrs Kershaw of the insurance company's negative response in September 1922. In her last 5 weeks, her husband stated that she gave up going to a physician since she saw no improvement (*Rochdale Times*. March 15, 1924). She died at 6:30 AM on March 14, 1924, at the age of 33.

THE CORONER INVESTIGATES

The very diagnosis of asbestos poisoning made by her panel physician, Dr Joss, attracted the attention of the coroner of Rochdale, one of whose duties was to inquire into unnatural deaths. The coroner, E. N. Molesworth, started an inquiry promptly on the day of death but adjourned it for a postmortem to be carried out by the local police surgeon. An obscure 24-line account of the first inquest under the heading "Married Woman's Death: Tired of Going to See Doctors," was spotted by Turners, who suggested to their insurance company that it would be in their interests for them both to be represented at the inquest (letter, March 17, 1924). After the postmortem, although death was stated by the pathologist, Dr F. W. Machichan, to be due to pulmonary tuberculosis and heart failure, a further adjournment was made to allow for microscopic examination of the lungs for a definitive diagnosis. Following this, the inquest was resumed on April 1, 1924. It was reported on the next day at length in the local newspaper (*Rochdale Times*. April 2, 1924:8). Far from making a simple inquiry into the death, the coroner held a full inquest with a jury and was advised by Dr Machichan as coroner's pathologist, Mr Barrett as HM Inspector of Factories, Dr S. A. Henry as Medical Inspector of Factories, and Dr W. E. Cooke, pathologist at Wigan & Leigh Infirmaries, also attending. The husband was represented by a solicitor, and the company was represented by a barrister advised by Dr W. H. Bateman, a local practitioner. Extensive information was provided by Dr W. E. Cooke, who had carried out microscopic examination of the lungs. He had found healed tuberculosis (old scar tissue in the left lung and a calcified tuberculous gland). Such findings were "extremely common in town dwellers and dwellers in urban districts." There was fibrosis in the lungs, and in some sections, particles of mineral matter were seen. The pathologic findings, with old scar tissue in the apex of the left lung, suggested completely healed tuberculosis. Second, there was "fibrosis of the lungs due to inhalation of mineral particles." Dr Cooke had received samples from Dr Henry of settled dusts that were "similar in size and shape to those found in the lungs of Nellie Kershaw." The pathologic findings and microscopic examination of the asbestos dust led to the conclusion that the mineral particles in the lung originated from asbestos and were, beyond a reasonable doubt, the primary cause of the fibrosis of the lungs and therefore of death.

Dr Joss also testified, and stated that

the reason he certified that the deceased was suffering from asbestos poisoning was that she had worked with asbestos "and he had previous experience of such a lung condition for many of his patients, who were asbestos workers." He stated that he saw 10 to 12 cases a year, occurring in persons working with asbestos (Dr W. J. Joss, written statement to HM coroner, 1924). Following the inquest, a death certificate was issued on April 2, 1924, stating that Nellie Kershaw, female, 33 years of age, died of "fibrosis of the lungs due to the inhalation of mineral particles" (Figure).

Dr Cooke introduced his note of 1924 with the statement that his case was of importance because it was the first in British medical literature to be definitely proved. In addition, he noted that medical men in areas where asbestos was manufactured had long suspected the dust to be the cause of chest disease, a point noted by others.[4,5] In support of his hypothesis he quoted Prof J. M. Beattie's animal experiment, details of which were to be given by Merewether and Price.[7]

COMPENSATION 10 YEARS AFTER NELLIE KERSHAW

There are no records of Nellie Kershaw or her estate's receiving compensation. Ten years later, asbestosis was scheduled as an occupational disease eligible for benefit. By April 1934, 2 years into the new scheme, of 29 claims submitted to Turners, seven were settled at an average cost of what then would have been approximately $370 (F. G. Brook, internal company report to Samuel Turner, April 20, 1934).

MISSED OPPORTUNITIES BEFORE NELLIE KERSHAW

In 1898, a quarter of a century before Nellie Kershaw's death and not long after the early expansion of the asbestos industry, the Chief Inspector of Factories for Britain had included asbestos among the four most hazardous dusts.[8] The report maintained that danger to the health of workers could easily be ascertained and that cases of injury to the bronchial tubes and lungs were medically attributed to employment. Recommendations were made for the application of ventilation to asbestos processes. Two years later the need for ventilation in textile and nontextile asbestos works was still being stated.[8]

In 1899, an asbestos worker was referred to Dr Montague Murray, a physician at the Charing Cross Hospital, with severe nontuberculous pulmonary fibrosis that was shortly to kill him at the age of 33. The patient informed Dr Murray that he had worked with asbestos for some 14 years. The first 10 years were spent in the carding room of an asbestos textile factory, where processes are similar to cotton textile manufacture; of the 10 people working in that room when he started, he claimed to be the last survivor. Unfortunately, this case was not commented on until 1906, during an inquiry of a departmental committee of the British government into the compensation for industrial diseases.[9]

The committee had been charged with investigating which conditions might be added to the list of compensable diseases. Its members had heard of Dr Murray's case and asked him at the inquiry whether he thought that asbestos should be included in the list. He advised against this, judging that now that it was known that asbestos exposure could cause serious, even fatal, disease, appropriate precautions would be taken, and there would be no need for compensation. The report of the committee reads:

> Dr Murray: One hears, generally speaking, that considerable trouble is now taken to prevent the inhalation of the dust, so that the disease is not so likely to occur as heretofore.
>
> Committee Member: Do you think it still may occur?
>
> Dr Murray: If there is dust, certainly.

Dr Murray's advice was taken and asbestos-induced illness was not added to the list of compensable diseases.

A report from the French Factory Inspectorate in 1906[9] mentioned some 50 deaths attributed to asbestos exposure in an asbestos textile factory that had operated for 15 years.

Dr Thomas Legge had discussed the problem with the Inspector of Factories in 1898, and his colleague Dr E. L. Middleton was aware of the circumstances of the Murray case. The Chief Inspector of Factories report for 1910 gave an account of how, after following up information from the Registrar General, five deaths due to phthisis were identified in 5 years among staff at an asbestos factory employing fewer than 40 workers and how exhaust ventilation was recommended and annual medical examinations were instituted.[10] At about this time the Factory Department of the Home Office sponsored a study by Prof J. M. Beattie on the effects of asbestos dust inhalation on guinea pigs. No publication has yet been identified.

The Factory Department's observations and misgivings led it to make inquiry of Canada's Department of Labor as to their experience with asbestos in Quebec, the source of much of Britain's asbestos. They were reassured. An officer of the department had investigated the matter in January 1912 and found no difficulty (*Labor Gazette*. February 12, 1912;761) He visited a large asbestos mine and mill and stated that "all the women found here looked strong and healthy."

While "one of the oldest medical practitioners in Thetford expressed the view that the asbestos dust . . . had a weakening effect on the lungs of those employed," another medical practitioner stated that the employees of the asbestos company affected with lung troubles had been so affected previous to their employment in the mill. It was stated generally that "the record of deaths from lung diseases is not higher in the centers of the asbestos industry than elsewhere."

No physical examinations were done or mortality data provided, and the Factory Department, with this reassuring reply to their inquiry, did not pursue the matter further, despite the appearance in the US medical literature of a report in 1918, that radiological abnormalities were found in roentgenograms of asbestos workers[11] and another that noted that insurance companies refused to give policies to such employees.[12] Following the 1906 inquiry, Legge visited the factory concerned twice and alerted the Medical Officer of Health.[13] No clinical examinations were made during investigations over the next 20 years, "which alone would have revealed its serious and insidious nature . . . looking back in the light of present knowledge, it is impossible not to feel that opportunities for discovery and prevention were badly missed."

THE LEGACY OF NELLIE KERSHAW

One can only speculate on what might have happened if Turners, which was a prominent company in the industry, had acknowledged Mrs Kershaw's claim and had recognized the hazard presented by asbestos. Employees, factory floor supervisors, and management would have been alerted to the hazard and would have begun to seek effective controls. Turners, through the Trade Association, would have ensured that other members would follow suit; insurance companies might also have insisted on such measures to decrease their liabilities; public awareness would gradually have come into being; public health authorities would have responded with appropriate regulations and rules; engineers would have developed means of avoiding exposures; and men and women could have worked with much less risk and have suffered much less disease. All these eventually came to pass, but it took years, decades.

Instead, the factory went on as before. When additional deaths began to be reported in medical journals, the Factory Department returned to Rochdale. A survey found many instances of asbestosis among the workers. It was observed that, after 20 years, 80% would have evidence of fibrosis.[7]

The Factory Department met with Turners and other asbestos manufacturers, and prepared a set of regulations (1931).[14] For reasons not yet known, however, the regulations were not to apply to most users of the asbestos products, such as construction workers, insulators, and shipyard employees. The general requirement was that manufacturing processes should not release any asbestos dust into the working environment, though there was the proviso that this might occur if unavoidable. There is evidence of schemes of dust control proposed for asbestos from earlier publications in 1898[5] and 1906,[8] but the extent of their implementation has not been ascertained.

In the first periodic examinations of scheduled workers, of the 326 workers employed for 10 or more years, 26 (8%) were suspended and 64 (20%) found to have "distinct fibrosis," roentgenographic and clinical signs of asbestosis (Dr C. L. Sutherland, Chief Medical Officer of the Silicosis and Asbestosis Medical Board, report to under Secretary of State, Home Office, 1933). Commenting on the second report, Merewether noted:

> If they [the Medical Board] had accepted this fact [that there is always present a greater degree of asbestosis than there appears to be, judged by accepted standards in silicosis] . . . it would have been forced to suspend a very large number of workers . . . which would have raised a panic in the industry [Dr E. R. A. Merewether, letter to Dr J. C. Bridge, HM Senior Medical Inspector of Factories, 1934].

Little was reported about the use of the regulations within the plants. *Lancet*, 40 years later, wryly summarized their application as having been "a pious aspiration."[15] Perhaps that explains Turner's workers continuing to have much disease, and having later been the source of a wealth of clinical and epidemiologic data, helping to define the dimensions of asbestos-associated disease. This included a study in 1955 that demonstrated that lung cancer was 10 times as frequent among its long-term employees as among British citizens in general[16] and another that reported that, 50 years after Mrs Kershaw's death, asbestosis was widely prevalent among workers in the factory.[17]

Would a different approach have made an appreciable difference? There is evidence that this could have been so. First, we know that with improvements in exposure control within the plant, death rates significantly decreased.[18] Second, the scientific contributions derived from the unhappy experiences of Turner's workers were an important part of the preeminent contributions of British scientists to our understanding of asbestos-associated disease and its control. These were, at least in part, the outcome of efforts of Dr John F. Knox, a capable and conscientious physician who after the Second World War monitored the work force as medical officer to Turner Brothers Asbestos Company and collaborated in the analyses of causes of death.[19,20] He did as much as he could to speed the application of exposure control measures.

COMMENT

The story of Nellie Kershaw is one of unmitigated tragedy. At the personal level, the death in poverty of a young mother struggling for 20 months against progressive asphyxia from respiratory failure is painful enough. That the circumstances of her death did not at least provide stimulus and guidance to help protect others adds to the tragedy. This lost opportunity tells us again that "science is necessary but not sufficient."

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Brief Report

The Prevalence of Gallstone Disease in Very Old Institutionalized Persons

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We present the results of a study that was undertaken at a large geriatric nursing home to assess the prevalence of gallstone disease in very old institutionalized persons. One hundred seventeen residents underwent ultrasound examination of the gallbladder. Two thirds of 82 women and half of 35 men had gallstone disease. When stratified for age, 80% of women and men over the age of 90 years were positive for the disease. In summary, the prevalence of gallstone disease in our very old nursing home population was found to be unexpectedly high.

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PERSONS 85 years of age and over (very old) currently constitute the most rapidly growing portion of the American population; this is also true for most other industrialized nations.[1,2] Cholecystitis with cholelithiasis is the most common acute abdominal surgical emergency in patients over the age of 50 years[3] and in the elderly.[4] Because of the above and our frequent finding of unsuspected gallstones at necropsy, the difficulty in diagnosing abdominal disease in the elderly,[4,5] and the ease of ultrasound examination, we decided to study the prevalence of gallstone disease in our nursing home. The sensitivity and specificity of ultrasound is 95%, which is higher than any other investigative method for gallstone disease.[6-8]

Methods

The study protocol was approved by the hospital ethics committee. The category of gallstone disease was used for residents in whom gallstones were found or who had undergone a previous cholecystectomy. One hundred seventeen residents of Maimonides Nursing Home (Montreal, Quebec) were studied with abdominal ultrasound. Most were admitted because of inability to care for themselves, due mainly to neuropsychiatric and/or physical disorders. Another 19 residents were not entered into the study because of refusal (five), poor compliance (three), or an inconclusive examination (11). The mean age was 83.8 years (SD, 9.0) and 12 residents were over 95 years of age. The residents were Jewish, middle class, 50% were born in Russia or Poland, and 20% were born in Canada. Ultrasonography of the gallbladder was performed by two experienced radiologists (A.L. and M.R.), using a 5× real-time ultrasound unit (Siemen's Sonoline) with a 3.5-MHz rotary mechanical sector scanning transducer. A diagnosis of gallstones was made by well-accepted criteria: echogenic opacity in the gallbladder with acoustic shadowing. For gallstones less than 3 mm where acoustic shadowing was not seen, the movement of the opacity with change in position was the main criterion for diagnosis. Patients who had undergone a cholecystectomy documented by history, who had a right upper quadrant scar with no demonstrable gallbladder on ultrasonography, and those who had stones were classified as having gallstone disease.

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Table 1.—Prevalence of Gallstone Disease in Study Population

Gallstone Disease	Women	Men	Total
No	28	17	45
Yes	54	18	72
Total	82	35	117

Table 2.—Prevalence of Gallstone Disease by Age and Sex in Study Population

Age, y	Women (%)	Men (%)
50-70	3/5 (60)	0/3 (0)
71-79	9/16 (56)	5/9 (56)
80-89	22/36 (61)	9/18 (50)
>90	20/25 (80)	4/5 (80)

Results

Fifty-four (66%) of 82 women and 18 (51.4%) of 35 men had gallstone disease (Table 1). Of the 82 women, 28 had a normal gallbladder, 29 had undergone cholecystectomy, and 25 had gallstones. Of the 35 men, 17 had a normal gallbladder, eight had undergone cholecystectomy, and 10 had gallstones. Although the gender difference with gallstone disease was not significant, the 95% confidence interval for the difference in proportions ranged from −0.05 to 0.34. The power to detect gender differences in gallstone disease was low (0.32) due to a relatively small sample. A two-way analysis of variance revealed a significant difference in mean age between those with gallstone disease, 85.6 years (SD, 8.0), compared with those who were disease-free, 80.8 years (SD, 9.8) ($F[1,113] = 7.20$; $P<.01$). No signifi-