# **C2100**

neumokoniosis

PNEUMOCON

This exhibit, GC2100, on show at Surgeons' Hall Museums in Edinburgh, is a whole lung preserved in a cylindrical specimen jar, whose cut surface shows heavy pigmentation with black coal dust. A piece of wood was tied to the top of the lung by two ligatures and attached to the lid of the jar to hold it suspended in the preservation fluid.

# The back of the ba

he discovery of a disease that is new to medicine is a rare and significant advance. However, the chances that the new disease was found by observing the pathology

of a single autopsy specimen, one that has been preserved and can be seen today, is even more unusual, if not unique.

Yet just such a specimen was recently discovered in the pathology collection of the Surgeons' Hall Museums in Edinburgh. The disease in question is coal worker's pneumoconiosis, the scarring lung disease that arises in coal miners as a result of the accumulation of inhaled coal dust in their lungs.

The lung was removed from the body of John Hogg, a local coal miner with symptoms of chronic lung disease who had been admitted into the care of Dr James Craufurd Gregory in the Royal Infirmary of Edinburgh in 1831. Hogg died three weeks after admission and, as described in the groundbreaking paper he subsequently wrote during Hogg's autopsy,1 Gregory was startled to find that "...when cut into, both lungs presented one uniform black carbonaceous colour, pervading every part of their substance. The right lung was much disorganised, and exhibited in its upper and middle lobes, several large

'Disorganised' was the term used then for the presence of pathological change, seen then with the naked eye, as microscopic analysis of the tissue was not then standard. The fluid-filled holes or cavities in the lung tissue were where severe scarring had broken down, leaving large craters where lung tissue used to be. By the 1830s, the number of UK coal miners had grown to about 200,000 to meet the needs of the industrial revolution, and most were working in confined and dusty conditions. The idea that they might accumulate harmful amounts of coal dust in their lungs under these circumstances is not surprising to us today, familiar as we are with the idea of excessive dust inhalation being harmful. Modern literature abounds with instances of lung disease in dusty trades, among which coal, silica and asbestos rank as those causing the greatest degree of global ill-health.<sup>2</sup> However, doctors in the early 19th century, while familiar with some work-related disorders, had not linked coal mining with any specific disease. In his paper, Gregory goes on to describe how, having seen the blackened lungs and the concurrent lung disease, a question immediately occurred to him: was the black pigment melanin (called "the peculiar







long-forgotten medical powerful history

irregular cavities. These cavities contained a good deal of fluid".

### Pathology

Coal worker's pneumoconiosis

## **Christison reported** back that the black pigment was coal

matter of melanosis" by Gregory), produced by the body, or coal dust that he had been inhaling on a daily basis at his work?

"The question here immediately presented itself, whether this ought to be considered as a case of infiltration of the substance of the lungs by the peculiar matter of melanosis? Or whether the black colour of these organs depended merely upon the habitual inhalation of a quantity of the coal dust with which the atmosphere of a coal mine must be constantly charged ... "

Fortunately, one of the few people at that time who might have been able to answer that question, Professor Robert Christison, worked across the road from the infirmary, at the University of Edinburgh. Christison was Scotland's first toxicologist, having studied in Paris in the 1820s under Mathieu Joseph Bonaventure Orfila (1787–1853), the Spanish physician often referred to as 'the modern father of toxicology'.

> This specimen was rediscovered and removed from its container after 180 years



Christison had become famous for his lectures on poisons and as an early expert on forensic toxicology in court cases such as the Burke and Hare murder trials in 1829. He was the obvious candidate to determine if the black pigment in Mr Hogg's lungs was melanin or coal dust, and reported back to Gregory that the black pigment was coal. Christison based his conclusion on the finding that, unlike melanin, the black colour of the pigment from Mr Hogg's lung was not lost on boiling with nitric acid. Burning and distilling the products of combustion finally sealed the identification.

Having found the lung of a coal miner blackened by coal dust and also diseased, Gregory became the first to propose that the presence of coal dust in the lungs had actually caused the disease to develop: "...whether ...coal dust... remaining unabsorbed and acting as a foreign body, had led ultimately to disorganisation of the pulmonary tissue..."

It is evident from the first few lines of his paper that Gregory was well aware that he was opening up the prospect of a whole new disease to the scrutiny of the medical profession, and that he specially wished to

The cavities, marked with a C, show substantial loss of lung tissue. The cavities mark where the blood supply has been compromised because of coal dust

The tissue had fibrosed, died, broken down and liquefied

warn physicians whose patients included coal miners to be vigilant for cases of the "I am induced to publish the following

case, partly because I have not hitherto met with the record of any similar affection; and partly with a view of calling the attention of those practitioners who reside in the vicinity of the great coal mines, and who may have charge of the health of the miners, to the existence of a disease, to which that numerous class of the community would appear to be peculiarly exposed."

new disease.

Gregory's paper is recognised by multiple authorities to be the first paper that alerted the world of medicine to this new occupational disease that, although unnamed by Gregory, eventually came to be known as coal worker's pneumoconiosis (CWP). The final sentence of the paper states that "the lungs themselves have been preserved".

The anatomy museum of the Royal College of Surgeons of Edinburgh did not open formally until 1832, in the new Playfair Building close to the university and old infirmary, but the college had been accumulating specimens since about 1804.

While obviously being preserved in 1831, Mr Hogg's lung was not formally entered into the museum's collection by Christison until 1840, as specimen number GC2100. It may have remained in Christison's possession or in one of Edinburgh's many private anatomy schools as a teaching aid from 1831 until 1840. Realising its historic importance, Christison may then have formally submitted it to the museum.

In the 1903 catalogue, GC2100 has an additional sentence added to its notes of 1840 stating: "This is probably the specimen from which the condition of anthracosis was first described by Drs Sanders and Christison." By 1903, therefore, the importance of the specimen was recognised, anthracosis being the old name for coal-blackened lungs, although a 'Dr Sanders' is credited instead of Gregory.

When the specimen was removed from its container for the first time in more than 180 years, the lung was found to be well preserved and the nature of the pathology was more clearly seen, with large cavities at the top of the lung showing substantial loss of lung tissue. These deep cavities mark where, due to the sheer quantity of coal

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and liquefied.3

Museums in Edinburgh



The fact that the lung can still be seen is remarkable

dust in the tissue, the blood supply has been compromised. The tissue first fibrosed, then died, broke down completely

This black liquid was spat out as the copious 'black spit' often seen in severe coal worker's lung disease and produced by Hogg in his final days. "The sputa became much more copious and of a peculiar dark grey or nearly black colour."

A single tissue block was taken from the lung and sent for paraffin histology (see above). The normal lacy appearance of the alveolar part of the lungs is replaced with a large nodule of scar tissue, with incorporated coal dust, present in the centre of the field. Around the nodule there is loss of alveolar tissue characteristic of emphysema. The lung, therefore, shows the typical appearance of advanced CWP.4 The disease first recognised by Gregory is now diagnosed as classical advanced CWP, and the publication of his paper drew a significant response from physicians in the Scottish coalfields, who began to seek and identify this disease in their patients and describe its essential features.

This culminated in a first understanding of the condition, most importantly distinguishing it from the disease called phthisis pulmonalis - now known to be tuberculosis - which is similar in some of its symptoms. Debate as to whether coal dust was the only cause, however, continued for the next 100 years, as poor housing, overcrowding, stone dust (silica), tuberculosis and cigarette smoking in coal miners were all advanced as possible contributory factors, too.5

Meanwhile, the rapid growth of the coal industry to meet the needs of the industrial age meant that many millions of men around the world were exposed to coal dust and developed CWP before control of the dust in coal mines to safe levels became mandatory in the mid-20th century.

Gregory's groundbreaking paper initiated the long process of fully understanding and preventing this deadly new disease, and in so doing saved lives. The fact that the lung can still be seen today in the Surgeons' Hall Museums in Edinburgh<sup>6</sup> is remarkable.

Tragically, Gregory died within a year of the publication of his paper, aged 31, from typhoid fever contracted from one of his patients in the Edinburgh Royal Infirmary. The perceptive nature of his 1831 paper suggests that, had he lived, he might well have made other significant contributions to medicine.

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