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Donaldson, Coalminer's lung

Title James Craufurd Gregory and 19th Century Scottish physicians and the link between occupation as a coal miner and lung disease.

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Abbreviations

CWP Coalworker's Pneumoconiosis

PMF Progressive Massive Fibrosis

M_{cPMF} Melanoptysis of cavitating PMF

M_{clearance} Melanoptysis of clearance

Abstract

By the mid-19th century about 200,000 miners were employed in a UK coalmining industry still growing with the advance of the Industrial revolution. Coalminers were long known to suffer poor health and a shortened lifespan but the link to inhaling dust in the coalmines had not been made. In 1813 George Pearson was the first to suggest that darkening of lungs seen in normal individuals as they aged was caused by inhaled soot from oil, candles and coal burning that were the common domestic source of heat and light. In a published case report in 1831 Dr James Craufurd Gregory of Edinburgh first described black pigmentation and disease in the lungs of a deceased coalminer and linked the two to pulmonary accumulation of coalmine dust. Gregory hypothesised that the black material seen at autopsy in the collier's lungs was inhaled coal dust and this was confirmed by chemical analysis carried out by Professor Sir Robert Christison, the Edinburgh physician and toxicologist. Gregory suggested that the coal dust was the cause of the disease and warned physicians in mining areas to be vigilant for the disease in their patients. This first description of what came to be known as Coalworker's Pneumoconiosis sparked a remarkable intellectual effort by physicians in Scotland, culminating in a large body of published work which led to the first understandings of this disease and its link to coal-blackened lungs. The present paper sets out the history of the role of Scottish physicians in gaining this understanding of Coalworker's Pneumoconiosis in the 19th century. It also describes Gregory's case and the lung on which Gregory based his landmark paper, which has recently been discovered in the pathology collection of the Surgeons Hall Museums, Edinburgh, where it has lain un-noticed for over 180 years.

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In the 19th century coal was the principal fuel needed by the ongoing industrial revolution in the UK. Physicians who saw symptoms of chronic lung disease in coalminers commonly described this disease as miner's *phthisis*, miner's asthma or miner's consumption. However, its relationship to *Phthisis pulmonalis*, a generic term used to denote a wasting disease seen in the general population and centred on the lungs and generally caused by tuberculosis (TB), was not known. In the early 19th century physicians, whilst aware of a few diseases linked to occupation, did not consider that coalmining was linked to a specific lung disease or that exposure to coalmine dust might be harmful.

Darkening of lungs in the general population

The increase in autopsy, along with the development of anatomical pathology and morbid anatomy in the teaching of medicine from the 16th century onwards, meant that lungs were being dissected. Anatomists observed and commented upon the black pigmentation of the pulmonary tissue and lymph nodes in ordinary citizens, becoming more marked as they aged. The burning of oil and candles for lighting, which was the norm in buildings prior to the 20th century produced a very smoky environment both indoors and outdoors in cities as noted by contemporary authors (1;2) . Inhaling this smoky air on a daily basis would have resulted in pulmonary deposition of considerable amounts of black particles. Dr George Pearson (1751-1828) who was a graduate of Edinburgh University, in a paper to the Royal Society in 1813 (3), was the first person to correctly suggest in the open medical literature that an exogenous, airborne source was the cause of black pigmentation of lungs in the general public. In his paper to the Royal Society in 1813 he describes the exogenous source of the black pigment as:-

'..sooty matter taken in with the air at respiration and accumulated in proportion to the duration of life..'

(3)

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Despite Pearson's paper, when Laennec addressed the issue of the origin of black pigment in the human lungs in his famous treatise on auscultation six years later (4) he generally believed that the 'black matter' he saw in lungs and bronchial glands was derived from the blood. He comments that the lung black matter is so common that:-

'...we can hardly consider it as a morbid production. It is found more or less abundant in the lungs of almost every adult, and seems to increase with the age of the individual..' (4)

Laennec did not discuss coalminer's lungs but the later English translators of his work (4) have appended notes to Laennec's section on the origin of the 'black pulmonary matter' referring the reader to Pearson and Gregory/Christison as well as Carswell (see later).

Coalminers, melanoptysis and phthisis

In the early 19th century the industrial revolution was in full swing in the UK and coal was required as its principal fuel. By the 1830s, the period under consideration in this paper, about 200,000 British miners were producing around 36 million tons of coal per year (5). Coalworker's pneumoconiosis (CWP) is the scarring lung disease seen in coalminers as a consequence of the accumulation of coal-dust in their lungs (6). Whilst the subtleties of the pathobiology of CWP or its cause were not known to physicians in the 19th century they were aware of the health impacts associated with coalmining including cough, dyspnoea, cachexia and especially a particular condition known colloquially as 'black spit' or melanoptysis (7;8). In coalminers in fact melanoptysis can be of two quite distinct types. The melanoptysis of the classic 'black spit' is only found in a miner with severe CWP where the fibrous lesions of Progressive Massive Fibrosis (PMF) develop, then undergo necrosis and cavitation. If the cavity erodes into an airway, coal-dust and liquefied lung tissue may then enter the airways from where it is expectorated as the copious black spit (9;10), hereafter this is referred to as M_{CPMF} i.e. the melanoptysis of cavitating PMF. In a miner with healthy lungs, or at least not suffering from cavitating PMF, the

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melanoptysis is different, with the sputum more-or-less normal in quantity and grey in colour. This colour is imparted by coalmine dust deposited in the lungs which is undergoing normal clearance by the muco-ciliary system; this form of haemoptysis is hereafter referred to as M_{clearance}.

Making the link between working in a coalmine, black lungs and CWP; the Gregory paper

In a paper published in Edinburgh in 1831 (11) James Craufurd Gregory MD, FRCPEd, FRSE, (1801-1832) was the first to make the link between working as a coalminer, black lungs and the development of a scarring and cavitating lung disease. All major authorities give Gregory primacy in making this link e.g. (5;6;9;12). The original lung specimen on which Gregory based his famous paper was recently discovered by the authors in the Surgeons Hall Museum collection of the Royal College of Surgeons of Edinburgh.

The paper, (11) begins with what amounts to a public health warning confirming that Gregory immediately understood the implications of his finding.:-

'..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..' (11)

Gregory then goes on to describe John Hogg, a 59 year-old man who had worked locally as a miner and who came under Dr Gregory's care in the Royal Infirmary of Edinburgh on the 29th of March 1831. His symptoms were typical of chronic lung disease like CWP and its cardiac sequel *cor pulmonale*. His health continued on a downward trajectory and by early April his peripheral oedema worsened and his dyspnoea became more urgent. His sputum became much more copious, and *'..of a peculiar dark gray*

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or nearly black colour..' a classic description of M_{CPMF} indicating underlying cavitating PMF. He died on 18th of April 1831, 3 weeks after entering Dr Gregory's care.

An autopsy was carried out and Gregory noted the striking appearance of the lungs :-

'..When cut into, both lungs presented one uniform black carbonaceous colour, pervading every part of their substance..' (11)

Gregory's description is of the typical appearance of advanced complicated CWP with lesions of PMF with cavitation and nodules throughout the lungs:-

'..the right lung was disorganised .. with several large cavities .. a considerable portion of the pulmonary substance ..was dense, hepatized and friable..the rest of the lung was somewhat condensed and oedematous...minute hard points could be felt in various parts of both lungs..' (11)

Having found the pervasive black pigment throughout the lungs and accompanying lung disease in Mr Hogg's lung, Gregory then describes his thought process:-

'..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,..' (11)

The role of Professor Sir Robert Christison Fortunately, at that time, Professor Robert Christison (Figure 1) worked close to the Royal Infirmary of Edinburgh in The University of Edinburgh. Christison was one of the few people in that period who could justifiably call himself a toxicologist and was famed at that time for his involvement as a forensic witness in the Burke and Hare murder trials 2 years previously. He had studied toxicology in Paris under Mathieu Joseph Bonaventure Orfila (1787 – 1853) a Spanish physician often referred to as 'The Modern Father of Toxicology' (13). He had also been trained in analytical chemistry by Pierre Jean Robiquet whilst in Paris (14) and was currently Professor of Medical

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Jurisprudence in Edinburgh University. Christison was ideal for the task of identifying the black pigment from Mr Hogg's lungs as endogenous melanin or exogenous coal dust. Christison undertook analytical chemistry experiments, the results of which he reported back to Gregory, confirming that the black pigment was not melanin but coal:-

'..Concentrated nitric acid boiled on it did not alter the colour.' – whereas melanin loses its colour on this treatment and on heating and distillation of the products :-

'..In the products of this experiment, it is scarcely possible not to recognize the ordinary products of the distillation of coal. ,; .'(11)

Gregory goes on to insightfully invoke a mechanism for the pathogenic action of coal dust on the lungs based on the fact that the dust can be viewed as a foreign body:-

'..the coal-dust , which, remaining unabsorbed and acting as a foreign body, had led ultimately to disorganization of the pulmonary tissue ..'(11)

The lung of Mr John Hogg in the Surgeons Hall Museums The final sentence of the Gregory paper reads

:- *'..the lungs themselves, have been preserved..“*

A search of the General Catalogue (G.C.) of the RCSEd museum was carried out and a lung with the number GC.2100 was identified, entered into the museum collection in 1840 by Prof Sir Robert Christison, with no detail regarding the donor and the description in the 1840 catalogues of 'Carbonaceous melanosis of the lung with caverns'. The description of the lung given in Gregory's paper, with its heavy pigmentation and cavities in the upper lobes, closely matches the appearance of GC.2100 (see below). Christison submitted GC.2100 into the collection in 1840 and the 9 year gap may be explained by the fact that Christison stored it for teaching and realising its historical importance some years later submitted it to the museum in 1840. The entry for GC.2100 in 'Catalogue Royal College of Surgeons Edinburgh Vol III 1903', which marked an update of the catalogue, has a new concluding

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sentence, added since its first entry in 1831, which demonstrates that its historical significance was recognised by 1903 :-

'This is probably the specimen from which the condition of anthracosis was first described by Drs Sanders and Christison.'

In fact the entry should read *'..first described by Drs Gregory and Christison.'*; there is no paper by Sanders and Christison.

Macroscopic appearance The lung was preserved in a cylindrical glass jar (Figure 2 a) and the top of the lung was ligated to a piece of wood which was attached to the lid of the specimen to hold the lung suspended in the preservation fluid. The lung was black on its cut surface and the pleural surface was also black in keeping with marked anthracosis (Figure 2 b). Centrally on one side of the specimen the central airways and large vessels were seen. On the other side the cut surface showed a mottled pattern of anthracosis. The tissue felt generally firm but no definite palpable nodules were identified. There was evidence of extensive cavitation towards the apex of the upper lobe (Figure 2c) with the largest cavity measuring up to 10 cm with further smaller cavities in the adjacent lung tissue. The cavities were consistent with the clinical description of M_{CPMF} given by Gregory and a major airway opens into a cavity, possibly the seat of the M_{CPMF} . Several lymph nodes are present at the hilum and these are also anthracotic. Minimising loss of this historic specimen, a single tissue block was taken from the periphery of the lung, including pleura and parenchyma and sent for paraffin histology.

Microscopic appearance The histological section taken from the specimen, conventionally stained with haematoxylin and eosin, shows moderate uptake and staining with eosin but no nuclear staining can be seen (Figure 3). DNA is the target to which haematein, the active principal of haematoxylin binds (15) and so loss of DNA over time would result in non-staining of the nuclei. Figure 4 shows pleura and underlying alveolated lung tissue and a fibrous nodule with surrounding emphysema. There is evidence

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of widespread anthracosis with deposition of black pigment in the pleura and along the fibrous septae of the lung. In places there are small aggregates or macules of particle-laden cells which are presumed to be macrophages. Elsewhere the presence of rather irregular nodular areas of fibrosis are identified within the lung parenchyma associated with further deposition of anthracotic material. Examination under polarised light reveals very occasional small fragments of crystalline material suggestive of silica but this is not prominent or widespread. These appearances are in keeping with CWP. In the histological sections available, these fibrous nodules measure less than 10 mm in diameter and could be regarded as constituting simple pneumoconiosis. However as noted in the macroscopic description of the lobe there is evidence of cavitation and clinical description of $M_{\text{C}_{\text{PMF}}}$ suggesting that the fibrosis is more extensive than identified in the section taken and would be consistent with complicated coal workers pneumoconiosis or progressive massive fibrosis (PMF).

Further publications from Scotland

The appearance of the Gregory paper in a popular medical journal resulted in increased interest amongst the Scottish medical community and thinking on lung disease in coalminers gathered pace. This response was focussed almost entirely in physicians in the Scottish coalfields and although sometimes confused and even wrong, over the succeeding 30 years, in about 10 papers, these Scottish physicians clarified the aetiology, clinical picture and pathology of lung disease arising in coalminers.

In May 1834, Dr George Marshall from Cambuslang, Glasgow published a paper entitled 'Cases of spurious melanosis of the lungs or of *phthisis melanotica*' (7). In describing this lung blackening in coalminers as 'spurious melanosis', Marshall recognises that this is not true melanosis i.e. not melanoma, but is a different entity unique to coalminers to which he gives the entirely new name of *Phthisis melanotica*. He describes 2 patients who were colliers, initially diagnosed from their non-specific symptoms (cough, dyspnoea etc), as having *Phthisis pulmonalis*. However, in the advanced

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stages of disease, both patients developed the unmistakable sign of M_{CPMF} . At autopsy he saw severe anthracosis, hepatisation (PMF) and cavitation in the dissected lungs of both miners. He therefore revised the original diagnosis of *Phthisis pulmonalis* to *Phthisis melanotica* in these miners. Importantly however this could not be done until the sign of M_{CPMF} had occurred, which Marshall perceptively suggested coincided with onset of cavitation.

Dr Matthew Gibson of Govan Haugh, Glasgow, in a paper published shortly after, in August 1834 (16), was quick to dismiss the notion that *Phthisis melanotica* was a truly separate disease entity in coalminers and that it was simply *Phthisis pulmonalis* in a coalminer :- '*I do not consider the black expectoration to be a disease, but quite the opposite..*'. Thus Gibson proposed that M_{CPMF} was in fact normal $M_{\text{clearance}}$ but also declares that the normal clearance of dust involves the breakdown of lung tissue where the dust is residing i.e. cavitation; this would mean that cavitation was not a disease. In fact the diagnosis of *Phthisis melanotica* was generally confounded by the fact that M_{CPMF} was the only pathognomic feature, but M_{CPMF} only arises in severe disease where PMF cavitation happens to involve erosion into airways. Gibson's paper does however highlight the major problem as to whether coalminer's lung disease is, or is not, different from what he calls '*everyday Phthisis*'.

Marshall followed up on his first paper with a second paper in September 1834 (17) which is groundbreaking in its descriptions of the pathology of coal-workers lungs and the events leading up to it. Marshall provides an extensive description of the lungs of a coalminer patient at autopsy, being the first proper description of the pathology of CWP, even though it is based only on naked eye observation. Within this description the main features of the pathology of PMF and its relationship to M_{CPMF} are identifiable and we have restated them in modern terms in parentheses after Marshall's description.

- 1) '*The lung deeply stained..*' (anthracosis)

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- 2) '*..Scattered through it hepatized nodules of greater or less bulk...characterised by their resistance to the knife..*' / '*..General disorganisation of the lungs..*' (PMF lesions)
- 3) '*..Hepatized sections occupied by vomicae..*' '*..The fluid contained in the cavities intensely inky..*' (cavitation within the PMF lesions)
- 4) '*..Bronchiae terminating abruptly and open-mouthed in these excavations..*' (points of erosion of cavities into airways producing M_{cPMF}).

One final conclusion from this remarkable paper by Marshall is that he dismisses the argument that was current in some quarters e.g. (18), that oil soot from lamps caused the condition although this belief continued (see later). He believes the cause to be "*fine coal dust*" based on the sheer amounts of coalmine dust in the air in these pits and also cites Gregory's paper.

Also in 1834, Dr Robert Carswell, born in Paisley and educated at University of Glasgow and in that year Professor of Pathological Anatomy in University College, London, described the Gregory paper at length in his entry under 'melanosis' in the 3rd volume of the Cyclopædia of Practical Medicine (1834) (19). Importantly Carswell mentions that the profession of the patient i.e. a coalminer, assists in the diagnosis; he also points out that one way to discriminate between spurious melanosis and melanoma is that in spurious melanosis there are no black deposits in any other organs, whereas there is invariably metastases with melanoma. Carswell suggests that the low number of cases of disease is due to some underlying condition in some individuals that allows the disease to develop. The true reason was that it could not be diagnosed separately from *Phthisis pulmonalis* unless the miner had very advanced disease with M_{cPMF} . In these pre-lung function and pre-X-ray days, simple pneumoconiosis and less severe forms of the disease simply could not be recognised, leading to under-diagnosis.

Mr Thomas Graham of the Andersonian Institute in Glasgow, in the same year, reported data from specimens from his clinical colleagues where he assessed the chemistry of the black material extracted

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from the lungs of five individuals, including 2 colliers, whose lungs had been examined at autopsy and were found to be coloured black (20). His analysis was that in all cases the black pigment was charcoal and not coal, even in the case of the colliers. The fact that all of the lungs had pigment that analysed as carbon (Charcoal) might be related to the fact that, of course, coalminers and non-miners were all developing environmental anthracosis because of their exposure in their homes to soot. However, the apparent absence of some coal in the analysis of pigment from the collier's lungs is puzzling,

In October 1837, Dr Thomas Stratton, another Edinburgh physician described black lungs at autopsy in a coalminer without M_{cPMF} , confirming that absence of this sign is not a reliable one for discounting a diagnosis of coalminer's lung disease (21). More importantly he suggests correctly that the blackening of the lungs is not necessarily related to any disease :-

'.. anthracosis may exist without any chest symptoms whatsoever ..'

Importantly, Stratton suggests the disease be named 'anthracosis', based on the Latin for charcoal. He therefore ends the complication of using the term melanosis (e.g. *spurious melanosis* or *phthisis melanotica*) in the name of a disease that does not involve melanin.

In the same year William Thomson published a large and comprehensive review in 2 parts concerning lung pigmentation caused by melanoma and by dusty occupations, principally coalmining (8;22).

Thomson made enquiries amongst physicians in coalfields in the North of England and the West of Scotland using a questionnaire. Thomson was also able to co-opt James Young Simpson, famous for his discovery of the anaesthetic properties of chloroform who was then in his early twenties and working in Edinburgh. Simpson made enquires amongst his colleagues in the coalfields of West Lothian and reported, curiously, that the condition was not known in the miners of that district. Simpson did eventually, however, turn up a case in 1833 and one more in 1835. The Thomson paper contains

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numerous meticulous descriptions of the disease, course and symptoms plus autopsy descriptions of anthracotic lungs including the cases already published by Gregory, Marshall and Graham amongst others. The symptoms seen in these cases provide a strong, consistent pattern of cough, dyspnoea, cachexia, M_{cPMF} etc. during life and at autopsy lungs showing blackening, PMF, cavitation and oedema.

Part 2 (22) contains a lengthy review of historical descriptions of black lungs caused by melanoma and 'black matter' and from inspired air containing carbonaceous particles. The paper finishes with communications from a number of physicians who report various amounts of the disease from none to occasional cases in their coalmining region. These draw a clear difference between tubercular *Phthisis* and miner's *Phthisis*, including for coalminers phthisis, slower progress, presence of M_{cPMF} , absence of purulent expectoration and lack of fever. Thomson also notes that miner's *Phthisis* was found to exist in colliers whose death had been caused by accidents, and who during life were robust and healthy, had no cough nor M_{cPMF} , nor exhibited any chest symptoms. This echoes Stratton's statement about lack of disease in black lungs and highlights a constant finding in later research that high attained coalmine dust burdens can be associated with minimal CWP with little or no functional respiratory impairment. Put into toxicology terms, this classifies coalmine dust as 'low toxicity' and is important in that the regulated safe levels finally allowed in coalmines were achieved by virtue of the fact that they were quite high, much higher than for a high-toxicity dust- typified by quartz/silica which need to be kept to a lower level in the air to produce a safe working environment (23). Thomson also refers to a communication from Dr Dewar of Dunfermline in which he claims that the majority of '*most fatal disease*' is not in colliers but in stone workers who remove the stone to clear the seams. This raises the important issue of silicosis, a more aggressive pneumoconiosis, alone or superimposed on CWP to which the stoneworkers may be prone because of silica dust from the stone.

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By 1842 official recognition of the presence of disease in coalmines is to be found in a report by Dr S. Scott Alison of Tranent near Edinburgh on the health of colliers in the South of Scotland (24). Regarding this population of colliers he remarks that '*Spurious melanosis, or "the black spit" of colliers, is a disease of pretty frequent occurrence among the older colliers... .., it invariably ends in the death of the sufferer..*'

Like Dr Dewar, Alison blames stone dust i.e. quartz /silica as causing the disease. CWP was conclusively shown to be a separate entity from silicosis, but not until 1940 (25). In similar vein Dr J.B. Thomson of Perth in 1858 advanced the theory that the black spit and its accompanying lung disease was not caused by coal dust but by personal predisposition and by oil smoke, which was present in some pits (18). This theory had been broached in early papers and was still put forward in his review of coalminers anthracosis by Dr J. W. Begbie of the University of Glasgow as late as 1866 (26) in what can be seen as a transitional paper in trying to reconcile earlier and later theories. Although he admits that the dusty environment might also be important, Begbie principally blamed soot from the burning of oil in lamps. Begbie also implicates silica, since he believes that '*workers at the stone wall*' were particularly susceptible. He also suggests that after inhalation of a certain amount of inhaled carbonaceous material has accumulated in the lungs :-

'.. there occurs a tendency which gradually increases to the arrestment of carbon or carbonaceous pigment in the lungs and its removal there from the blood..' (26)

This seems to be an attempt to reconcile the stance of Virchow (see below) and the early authors like Laennec regarding an endogenous source for the 'pulmonary black matter' with the accumulating evidence in favour of inhaled carbonaceous particles as the source.

In 1846 Dr Archibald Makellar of Edinburgh reported on his experience with 10 cases of what he called Black phthisis, a condition he considered due to the inhalation of carbonaceous dust 'by coal miners and other operatives' (27;28). The paper itself gives meticulous descriptions in 10 cases of coalminers who in general display symptoms and autopsy findings identical to those described by

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Thomson in his series described above. Makellar makes the mistake of implicating the soot from lamps and gunpowder smoke rather than coal-dust as the source of the black carbon and also mentions stone-dust as being especially harmful, as do other authors, raising the perennial question of silicosis. In this regard Makellar mentions an incident that sounds like an outbreak of acute silicosis in Tranent at the colliery of the Messrs Cadell :-

'..where a great number of young, vigorous men were employed at stone-mining... every one of whom died before reaching the age of thirty-five years..'

Makellar also has his own theories about carriage of carbon in blood and especially the generation of particulate carbon by the lung in advanced disease; this latter is an attempt to explain the ongoing production of black spit in miners who are long-retired :-

'..a striking peculiarity of this disease, that when the carbon is once conveyed into the cellular tissue of the lung, that organ commences the formation of carbon..'

In fact the progressive nature of PMF and cavitation fully explains the generation of black spit in the absence of ongoing exposure. This paper is rich with descriptions that include bronchial hypersecretion, the role of heredity and the action of 'tubercular phthisis' in a miner with black phthisis and although much of it is conjecture that is mistaken, there are occurrences of impressive scholarship and thinking on coal miner's lung disease.

Virchow, the 'Father of cellular pathology' was a strong believer in the endogenous source of black pulmonary pigment – even in coalminers - believing that inhaled particles, although depositing in the lungs, would not be retained (29). He therefore saw M_{cPMF} as evidence of a fully-functioning defence

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system i.e. $M_{\text{clearance}}$. Even after being sent coalminers lungs from Scotland and elsewhere Virchow stated that :-

'.. it is extremely probable that we have here to do with pigmentary changes resulting from extravasations of blood and the subsequent transformation of hematin and not with absorption and deposit of carbonaceous matter inhaled into the lungs..' (29).

Not until 1866 did Virchow finally accept that the black matter in lungs could be carbonaceous and that anthracosis was a true and separate disease entity (30).

The role of the publications from Scotland

By the 1860s, 30 or so years after the Gregory paper, it was generally accepted that working as a coalminer was associated with a distinct lung disease caused by inhaling the air of coalmines and whose features included blackened, fibrosed and cavitated lungs and M_{CPMF} . This was a direct consequence of the pioneering clinical and pathological research of the Scottish physicians and their publications described in this paper. In so doing these physicians can be seen to have produced the first modern aetiological, clinical and pathological picture of CWP. Armed with empiricism, the chief intellectual achievement of the Scottish enlightenment and the European anatomical-pathological tradition, they sought evidence from autopsy and analytical chemistry to confirm the hypotheses they derived from their clinical experience. In discussing these Scottish papers the authors have not cherry-picked them – there are no other important papers addressing this area over this period other than the Scottish ones, as succinctly put by Makellar in his 1845 paper (27) :-

'.. It does not appear, as far as I can ascertain, that any of the Continental physiologists are familiar with the disease now under our consideration .. It is therefore unnecessary to refer to them in general ..' (27)

Before Gregory's seminal paper in 1831 the relationship between symptoms of 'ordinary' *Phthisis*, miner's *Phthisis*, black spit, black lungs and occupation as a coal miner was not understood. Despite

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Pearson's paper the origin of the 'black matter' of the lung was not generally known and was not considered 'morbid' by many authorities e.g. Laennec, Virchow. Thirty years later and after publication of the 10 or so seminal papers from the Scottish physicians starting with the Gregory paper, it had become clear that inhaling particle-laden air in pits lead to the black pigmentation and that the black pigmentation was carbonaceous and therefore, in essence, coal dust. The link between high lung burdens of this dust and fibrosing lung disease with cavitation had been described and the link between cavitation and the pathognomic sign M_{cPMF} , was recognized. Most importantly, the difference between *Phthisis pulmonalis* and miners' *phthisis* was clearly drawn. In addition, the idea that both coal dust and stone dust (silica/quartz), can be produced in mines and that these might differ in their pathogenic potency had been raised, heralding the vexatious issue of silicosis and its role in coalminers which still remains an issue today (31). Whilst there were still conflicting opinions on some aspects, as might be expected at the dawn of such a new understanding, the essential observations and conclusions regarding the aetiology, symptoms, course and pathology of CWP had been made and committed to print. The process of accumulating evidence and a full understanding could then proceed, although as with other environmental disease, this had to await the advent of X-ray, lung function, refined exposure measurement, improved epidemiological methods and quantitative pathology.

Tragically, James Craufurd Gregory died on 28th December 1832 at the age of 31, within a year of the publication of his landmark paper, from typhoid fever contracted from one of his patients in the Edinburgh Royal Infirmary. Gregory was unmarried so left no children and we were unable to find any contemporary images of him. The insights in his 1831 paper suggest that he had much to give in terms of intellectual, as well as practical contribution to occupational and other aspects of medicine. His obituary in the London Medical and Surgical Journal of January 1833, included the following :- " Dr.

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Gregory was one of the finest figures we ever saw. His features were regular, and, indeed, truly handsome, and displayed an intelligence and a benevolence rarely observable." (32)

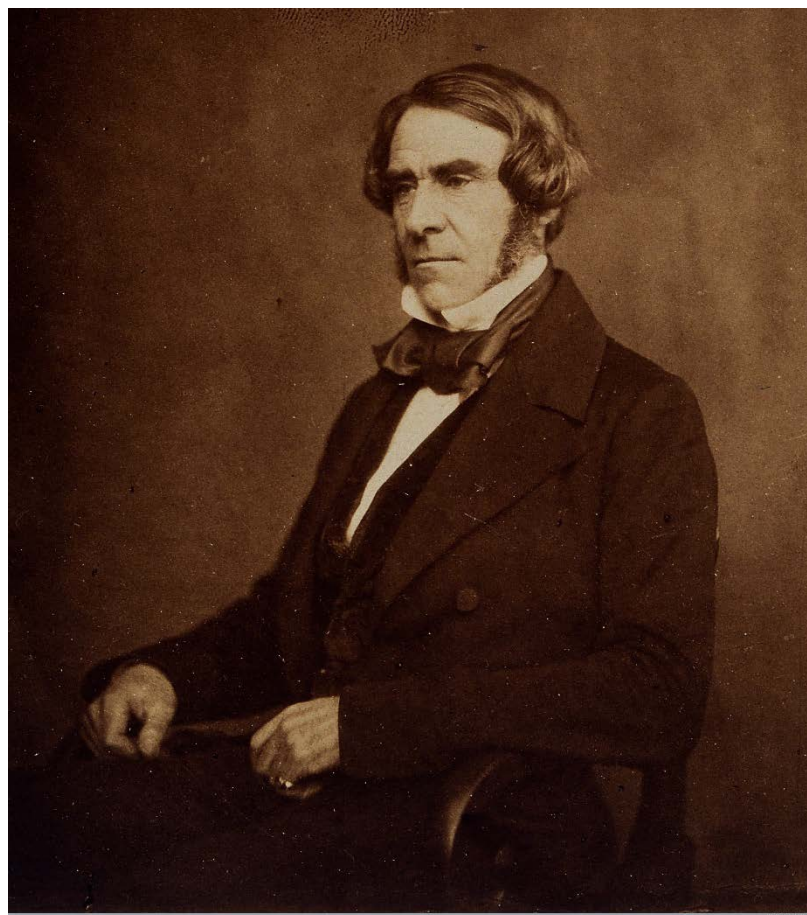


Figure 1 Photograph of Sir Robert Christison; image courtesy of the Wellcome library, London

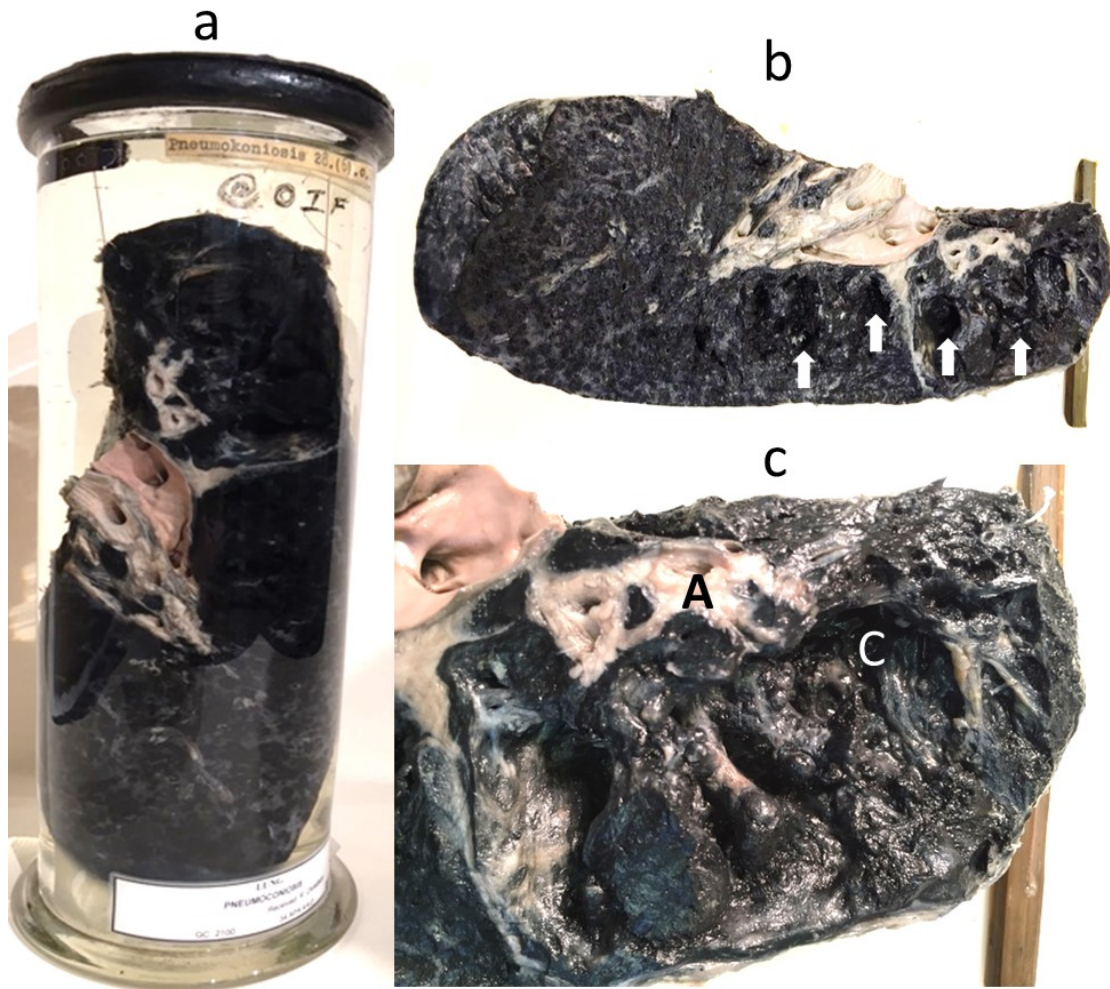


Figure 2 a) GC.2100 in its original preservation jar. b) GC.2100 removed from its jar; the main bronchus and some divisions are clearly seen as is a portion of the pleura at the base of the lung on the left. The lung parenchyma is highly pigmented but the large airways are spared. The arrows indicate areas of cavitation. The wood used to suspend the specimen can be clearly seen at the apex. c) Detail of GC.2100 showing cavitation in the upper lobe. A large cavity (C) adjoins, and appears to be connected to, a large airway (A) which is pigmented; this may be the site of rupture of the cavity into the airway that lead to the sign of H_{CPMF} reported by Gregory.

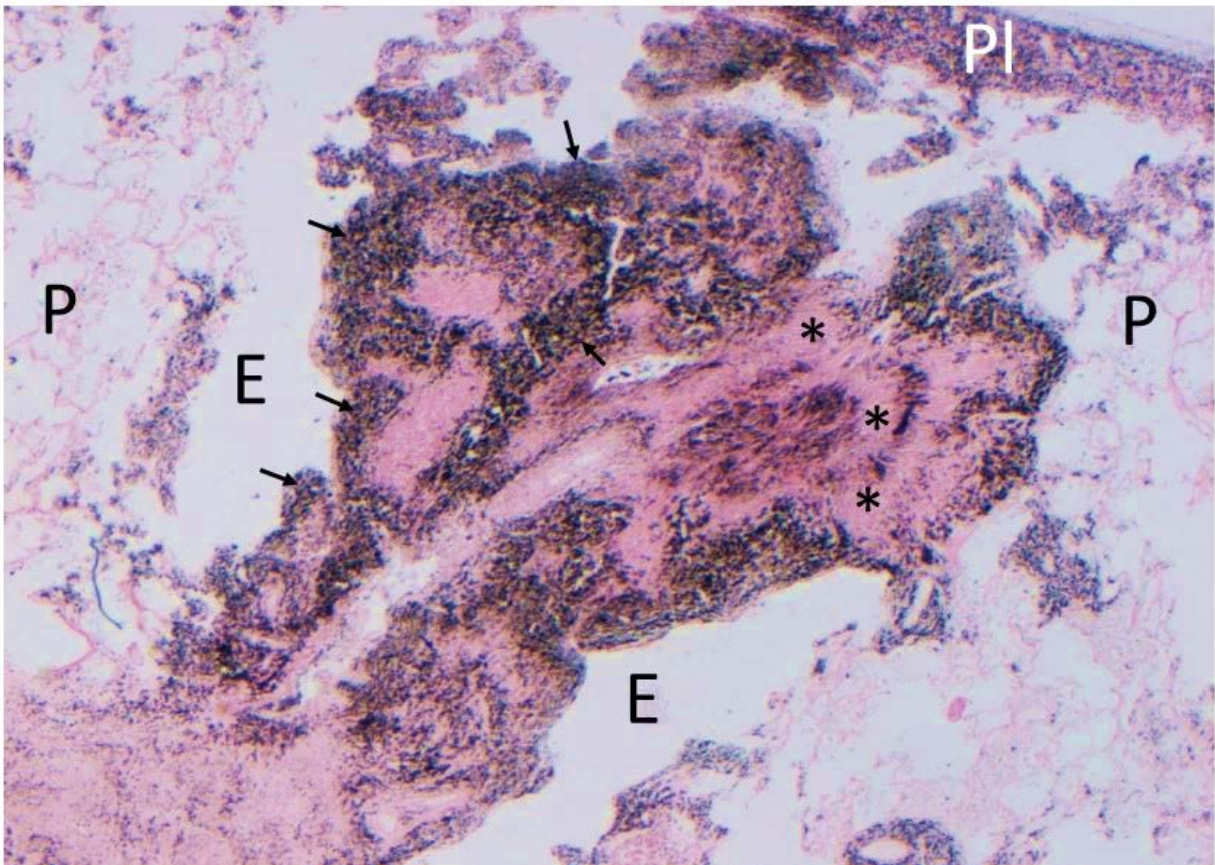


Figure 3 H and E stained section of lung from GC.2100 Pleura is present at the top showing thickening and dust incorporation (Pl). A nodule with extensive incorporation of black particulate (black arrows) occupies most of the central area of the image, with areas of fibrosis (asterisks) and surrounding emphysema (E). Normal alveolated parenchyma (P) is present at the left and right sides

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