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The recognition of lung disease in coal workers: The role of Gough–Wentworth whole lung sections

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Abstract

From the identification of a specific lung disease caused by coal dust exposure in miners in 1831 until the demonstration of the association of that exposure to risk of emphysema in 1984, there was continuous argument about the harmfulness of coal dust. Ill health in miners was attributed variously to tuberculosis, quartz exposure or cigarette smoking. An acceptance that coal dust was harmful only started with investigative radiology and pathology in the 1920s, and physiology in the 1950s. Most of the early investigations were in South Wales, the centre of the most important coal field in Great Britain. Among the investigators was Professor Jethro Gough who, with his technician James Wentworth, introduced a technique for making thick sections of whole, inflated lungs on paper backing. Here, we describe this method and its central role in understanding the relationships between coal dust exposure, pneumoconiosis, emphysema and lung dysfunction in miners.

Keywords

coal workers' pneumoconiosis, emphysema, tuberculosis, coal miners, fibrosis

The recognition of coal workers' pneumoconiosis

Lung disease in coal miners, known originally as miners' phthisis or miners' asthma, was first formally described in Edinburgh by Gregory in 1831, and its essential features were defined by the mid-nineteenth century.[1] However, in the late nineteenth and early twentieth century, before the advent of chest radiographs and based on the relatively good health of coal miners as opposed to urban dwellers and other miners, medical authorities judged the disease to have all but disappeared. Subsequently, some experts asserted that coal dust exposure was harmless, other than the effects of the quartz in it. Quartz was known to be a highly pathogenic mineral that caused disease in coal miners if, unusually, it was present at high proportion in the dust they breathed.[2] However, the introduction of radiology to medicine in the 1920s and the recognition of high death rates from lung disease among coal miners in South Wales re-awoke attention to a widespread silicosis-like disease among them, leading pathologists to investigate their lungs at postmortem.[3] Because of the historic understanding of the role that quartz plays in one type of pneumoconiosis – silicosis – the low percentage of quartz always present in the air of coal mines was suspected by some as being the primary noxious agent in dust in the air of coal mines. This belief was strengthened when an episode of rapidly progressive silicosis was described in 1927 among men who were tunnelling in a coal mine in South East England.[2] However, in the same year, Edgar Collis, who was originally sceptical of the harmfulness of coal dust and had become a professor of public health in the Welsh National School of Medicine (WNSM) in Cardiff, along with a tuberculosis (TB) specialist James Gilchrist, tested this theory by carrying out a study of coal trimmers working in ships' holds in the docks.[4] These men were heavily exposed to coal dust without significant quartz content and were found to develop significant radiological pneumoconiosis.

Collis's colleague, Lyle Cummins, Professor of Tuberculosis at the WNSM, was also originally sceptical about a separate pneumoconiosis attributable to coal dust. However, his pioneering studies showed that the radiological

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Figure 1. Professor Jethro Gough appears before the Senate Labour Subcommittee on Coal Mine Safety and Health, Washington May 1969 holding two Gough–Wentworth paper-mounted whole lung sections.[12]

and pathological features of diseased coal miners' lungs, whilst similar, differed in some specific ways from those of classical silicosis.[5] He questioned the quartz theory on the basis of this experience, noting that, 'in the South Wales coal field, cases of a more or less disabling lung condition are frequently met with which cannot conscientiously be fitted into the category of silicosis'.[6] As a response to the likely prospect of having to pay compensation to thousands of coal workers and the importance of those men in preparations for the looming Second World War, in 1936 the UK Government requested the British Medical Research Council (MRC) investigate lung disease in Welsh coal miners. Under the auspices of the MRC, a widespread radiological and dust sampling campaign was undertaken in the Welsh coal fields by Philip D'Arcy Hart and Edward Aslett. Jethro Gough, a pathologist in the WNSM, was recruited to investigate the pathology in deceased coal miners and coal trimmers. The brief of the MRC was to investigate 'the incidence and characteristics of disabling pulmonary diseases affecting workers in coal mines and the conditions which give rise to them'.[7,8]

The outcome of the MRC studies was unambiguous in showing that exposure to dust as a consequence of employment in coal mines was responsible for causing a form of pneumoconiosis that was different from silicosis. It was eventually named coal workers' pneumoconiosis (CWP) in order to include individuals who worked on the surface screens and in ships, and underground miners.[8,9] The authors did however confirm that silicosis could occur in coal miners who worked in developing the seams and were exposed to silica dust from the drilling of rock, so-called 'hard-headers'.

Gough's[10] study of coal trimmers, following the observations of Collis and Gilchrist,[4] was especially influential and the unique circumstance of the exposure of coal trimmers was key. They handled only washed coal with a very low quartz content as they were not exposed to quartz from cutting through rock, in contrast to miners working underground. Gough's[10] report that coal trimmers developed a form of pneumoconiosis pathologically identical to the CWP seen in the coal miners confirmed that coal dust alone could cause CWP, adding crucial pathological support to the findings of Hart and Aslett[9] from clinical and radiographic data. In response to this, in 1943 the Coal Mining Industry (Pneumoconiosis) Compensation Scheme began, finally recognising that exposure to coal dust could be linked to the development of a disabling pneumoconiosis.[11] Despite this recognition of CWP as an occupationally linked disease in the UK in the 1940s, the US authorities were slow to reach the same conclusion. As late as 1969, the legislature of West Virginia, a state central to the US coal mining industry, still denied the existence of CWP.[12] However, the US Mine Workers' Union promoted a special meeting in 1969 of the Federal Judiciary Committee and its counterpart in the House of Delegates to debate the matter. Gough was called as an expert for the miners' case and argued for the existence of CWP. He recounted the UK experience of finding the link between lung disease and coal mine dust, using his large lung sections to provide pathological confirmation of the condition (Figure 1); by the end of that year, a compensation plan was produced.[12] This, and a series of strikes, led to the passing of the 1969 Federal Coal Mine Health and Safety Act (1969 Act), which lowered the regulated levels of dust in US coal mines with the aim of reducing disease incidence. It is of note that this dust standard was based on British research, namely that carried out for the nationalised Coal Board by the Institute of Occupational Medicine in Edinburgh.[13]

Gough–Wentworth paper-mounted sections of whole lung

Professor Jethro Gough and his technician James Wentworth developed the eponymous large lung section technique now named Gough–Wentworth sections (GWSs) in the WNSM. Their goal was to 'obtain representative sections of the entire lung for comparisons with radiographs taken during life,[14] seeking to use the sections for side-by-side comparison of lung pathology and radiographs, in order to validate radiological diagnosis. This work was associated with a parallel effort by the MRC unit to quantify the radiological appearances that led eventually to the ILO classification of pneumoconiosis radiographs.[15] Gough and Wentworth's[16] original methodology as described involves fixing the whole lung by inflation with 10% formalin to full distension and then suspension/immersion in formalin. Once fixed, a large knife is used to cut a 1.5-cm thick slice in the sagittal plane with the aid of a rack to ensure a parallel cut. This slice is washed to remove the formalin and immersed in a dilute, embedding gelatine solution and the lung allowed to absorb the gelatine; the efficiency of embedding can be aided by using a vacuum oven. The cooled slice is cast as a block, which is then attached with gelatine to a chuck suitable for a large sledge microtome. The gelatine block containing the embedded lung plus the chuck are frozen and thick, 200–600 μm, sections cut as the block thaws. The cut sections are mounted onto paper and dried flat to produce the final section (Figures 2–5). The lung tissue and

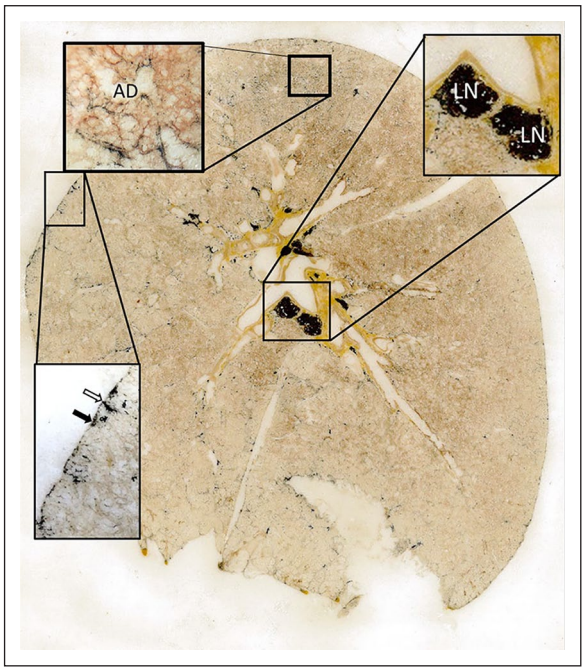

Figure 2. Gough–Wentworth section GC.13159 from the Surgeons' Hall Museums collection. Record states 'Whole section. Normal lung from a female aged 83 years, a citizen of Cardiff. Slight general enlargement of air spaces. No destructive emphysema. Died from chronic nephritis. Presented by Professor J. Gough'. Callout at right shows anthracotic peribronchial lymph nodes. Callout top left shows a terminal bronchiole and alveolar duct showing mild anthracotic pigmentation. Callout on the left shows pigment in sub-pleural lymphatics.

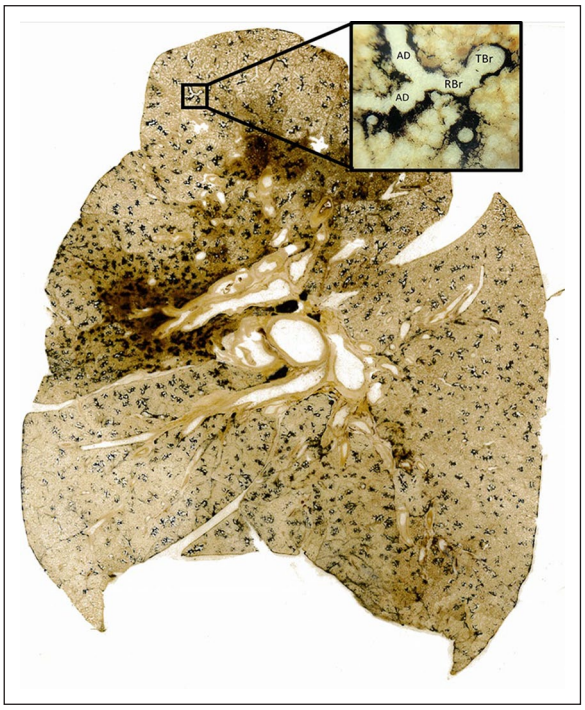

Figure 3. Gough–Wentworth section presented to one of the authors (AS) by Professor Jethro Gough. On pencil on the reverse is written '204/48 Tricuspid, aortic and mitral stenosis. Brown induration of lung. The lung shows clear macule formation'. The callout shows a terminal bronchiole that shows interstitial anthracosis in the terminal bronchiolar (TBr) respiratory bronchiolar (RBr) ductal (AD) and alveolar septal walls.

the accumulated coal dust are very clearly seen, contrasted against the white paper. Any pathology, fibrosis or emphysema, is also very clearly shown, allowing it to be quantified using morphometric techniques.

Gough and coal workers' pneumoconiosis: Pathological and radiological comparisons

Gough's involvement in the MRC South Wales studies followed the pioneering studies of Cummins, who had described the first classification of the radiological appearances.[6] The essential pathological description of the two forms of CWP were summarised by Gough in 1949: simple CWP and complicated or 'infective' pneumoconiosis, also called progressive massive fibrosis (PMF). The latter is characterised by large masses of dense fibrous tissue, and in the time of Cummins, it had been apparent that this lesion was likely to cause impaired lung function, but its aetiology was in doubt.[17] Tuberculosis was then endemic in Britain, especially in South Wales, and Gough believed that TB infection was the essential co-factor that caused the PMF to develop.[14,17] Others, notably Cochrane in the MRC Pneumoconiosis Unit, were more cautious as to the role of TB, and this led to a rift between the MRC unit and the WNSM.[2,18] Nevertheless, more remained to be elucidated once the GWS technique and a radiological classification were available. First, the correlation between estimation of lung disease from radiographs and the actual pathological change needed to be verified. Secondly, given that emphysema could not be quantified on chest radiography, determination of any relationship between respiratory impairment and emphysema in coal miners required a pathological technique such as the GWS. Gough addressed this in a ground-breaking study of 76 coal miners' lungs in 1949. The aim was described thus: 'The investigation has consisted of a comparison of the pathological changes in the lungs of miners with the radiographic appearances of the chests of the same individuals during life. For the demonstration of the pathology a new technique has been used in which representative sections of the entire lung have been cut and mounted on paper'.[14] Taking the GWSs as the 'gold standard', the authors set out to estimate the value of radiology with respect to:

> the recognition of the presence of pneumoconiosis
> the estimation of the extent of disease as the profusion of coal nodules present
> the estimation of the degree of macroscopic emphysema

The authors reported that with slight degrees of pneumoconiosis visible in the GWS, there may be no recognisable changes in the radiographs, whereas with moderate or severe degrees of pneumoconiosis, there were distinctive radiographic changes, nodules being present in almost all

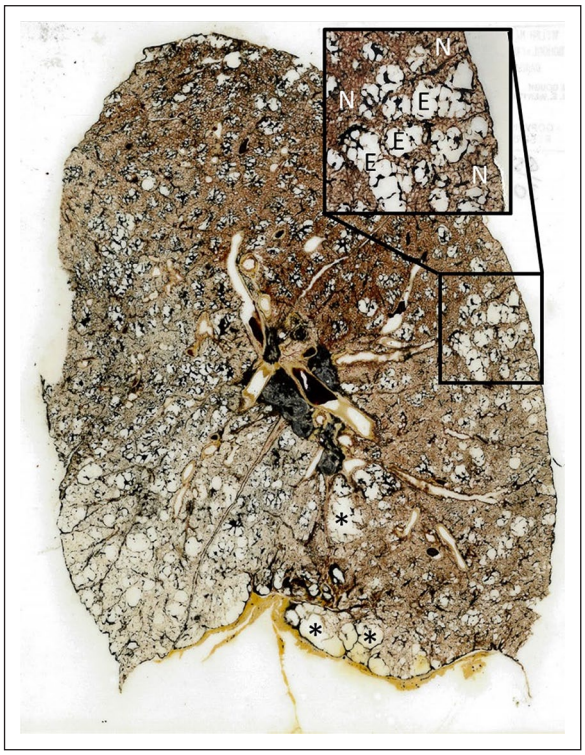

Figure 4. Gough–Wentworth section GC.13157 from the Surgeons' Hall Museums collection. Record contains 'Whole section. Focal dust emphysema. There is limited deposit of carbon throughout the lung. Early pneumokoniosis'. Presented by Professor Jethro Gough. The callout enlarges an area with numerous areas of centrilobular emphysema (E) in comparison with nearby areas of normal parenchyma (N). Typically present in the lower part of the section and centrally, are some areas more typical of panlobular emphysema (asterisk).

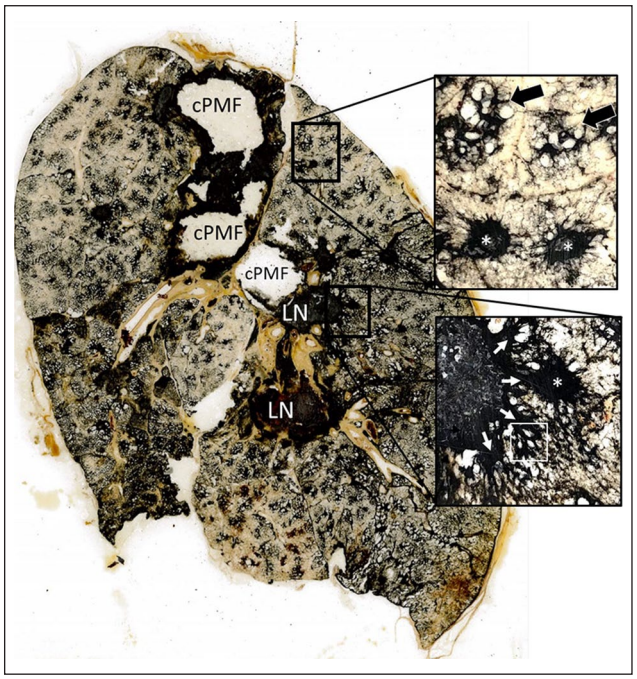

Figure 5. Gough–Wentworth sections GC.13162 from the Surgeons' Hall Museums collection. Record contains 'Whole section of lung. Massive pneumokoniosis from a coal miner, showing cavitation in the upper and middle lobes. The pigmentation is diffuse but particularly marked at the hilum. Presented by Professor J. Gough'. Cavitated progressive massive fibrosis (cPMF) lesions are present in the upper and middle lobes and the lymph nodes (LNs) are highly pigmented. The callout top right shows an area where there is centrilobular emphysema (arrows) and stellate nodules (asterisks). The callout lower right shows areas where the LN capsule may have been breached as a result of the high particle burden and pigment has flowed in to the surrounding parenchyma (arrows), which shows honeycombing (box) and nodule formation (asterisk).

cases. Furthermore, there was fairly good correspondence between the profusion of opacities seen in the radiographs and the numbers of coal nodules found in the section. The terms 'reticulation' or 'granularity' had been used to describe the very early radiographic appearance of coal workers' lungs which, not being clearly nodular, did not satisfy the definition of pneumoconiosis. From the lung sections, Gough and colleagues concluded that these changes were not just due to dust accumulations but reflected the presence of mild early pathological change. This was characterised as a localised accumulation of reticulin fibres, which did not proceed to collagen fibre accumulation.[19] These were a response to the accumulated dust and were the earliest defining lesion of simple pneumoconiosis, the coal macule. More importantly, Gough and co-authors suggested that, when this minimal level of nodule formation was seen on chest radiographs, the sections could still show considerable emphysema. They concluded that emphysema could not be reliably quantified on the chest film, yet '...it is the focal emphysema which may be the cause of the common dyspnoea in coal workers'.[14]

Progressive massive fibrosis

Two theories to explain the progression from simple to complicated pneumoconiosis in the minority of miners who did develop PMF dominated in the mid-twentieth century. These were concurrent silica exposure or TB infection. Given the coal trimmer studies where Gough had seen PMF in the absence of silica exposure, he espoused the TB theory. This was a view held by others such as Fletcher[3] and Nagelschmidt.[20] As TB declined, it became apparent that this was not usually correct, PMF often occurring in the absence of evidence of TB infection. However, later, attention was directed to the role of the lymph nodes (LNs) draining the lungs, which in Gough's time were commonly destroyed by primary TB infection. It now seems likely that the consequential interference with removal of dust from the lungs from diseased hilar nodes, whether from old TB infection or from fibrosis consequent upon accumulation of large amounts of dust, is a significant risk factor for PMF; Gough may well have been at least partially right, if for the wrong reasons.[21]

Gough and emphysema in coal workers

In 1952, Gough made his most important contribution to the general study of the pathology of emphysema when he provided the first descriptions of panlobular and centrilobular

emphysema using GWSs and suggested different aetiologies: 'The patterns suggest that the mechanics involved in the production of widespread chronic emphysema are not always the same and there appear to be at least two perhaps fundamentally different types. In the one, the forces at work appear to affect the lungs as one unit whereas in the other, the disturbance seems to concern more particularly the mechanics of the individual secondary lobules'.[19] With regard to disease in coal workers, it had become apparent that even extensive small radiological opacities were not necessarily associated with impairment of lung function or symptoms, yet many miners with normal radiographs had shortness of breath and reduced function. As early as 1936, Cummins had observed that in CWP the extent of fibrosis 'does not necessarily correspond with the degree of dyspnoea noted'.[6] He also had speculated that, whilst complicated CWP contributed to impaired lung function, it was probable that the majority of miners with both simple pneumoconiosis and symptoms were most likely to have impaired lung function as a consequence of their emphysema.

In a study of 247 cases by Ryder et al and including Gough in 1970, a correlation was shown between the pathological, physiological and radiological findings of emphysema. The pathological evidence of emphysema was compared with an age-matched non-mining population. Incidence of emphysema was much more common among the coal miners than among the control group non-miners. When emphysema was quantified, there was a linear association between the extent of emphysema and ventilatory impairment, as measured by the forced expiratory volume in 1s (FEV1). The authors concluded 'It is thus clear that the impairment of pulmonary function is closely related to the emphysematous changes...'.

In 1974, Lyons and colleagues[22] including Gough examined the significance of the type of opacities seen on the chest radiograph. Using GWSs, they concluded that the rounded and stellate nodules seen in the GWSs corresponded to rounded and irregular opacities in the radiographs, respectively. Furthermore, the irregular opacities seen in the GWSs were associated more clearly with emphysema and other lung parenchymal changes. The deficit in lung function, as measured by FEV1, was directly related to the number of irregular opacities and not to the profusion of rounded opacities, again suggesting a role for emphysema.

The pathogenesis and aetiology of emphysema in coal workers

Gough published a number of papers exploring the aetiology and pathogenesis of emphysema in coal miners using GWS. In 1952, he noted that the most common form of emphysema seen in coal workers was within the centres of the secondary lobules, focussed around coal dust deposits.[19] In 1965, he described two types of emphysema occurring in the centres of the lobules, which he named in a rather confusing way.[23] One of these was the predominant lesion seen in coal workers in the centres of lobules, which he named focal dust emphysema, and one where there was little pigmentation associated with the lesions, which he named Centrilobular Emphysema (capitalisation of centrilobular emphysema is used to delineate the pathological entity identified by Gough from centrilobular emphysema, the generic anatomical position of one type of emphysema). The confusion arose because both lesions are found in the centre of the lobule, but only one is called Centrilobular Emphysema. He considered Centrilobular Emphysema to be found mainly in smokers in the general public and to be caused by '...an inflammatory process that causes disruption of the wall of respiratory bronchioles'. By contrast, he considered focal dust emphysema, found in coal miners and other dusty trades to be caused by dust and he described it as a '...a group of dilated respiratory bronchioles occurring in and around a focus of dust such as coal', with no inflammatory component. Gough recognised the close morphological similarity between the two types of emphysema, and the potential confusion between them: 'Confusion has arisen in descriptions because the two forms of emphysema, Centrilobular Emphysema and focal dust emphysema, commence in centres of lobules. The importance of distinguishing them is, however, that the emphysema due to the dust has less effect upon the respiratory function than the centrilobular emphysema due to bronchiolitis'.[23] This statement was grist to the mill of those who believed that emphysema was not a significant consequence of coal dust exposure. In fact, it turned out to be at odds with the observations he obtained later, regarding the impact of centrilobular emphysema on respiratory function in coal miners.[24]

By 1968,[25] Gough was describing three kinds of centrilobular emphysema in his GWSs: (1) focal dust emphysema due to industrial dust-like coal; (2) soot emphysema caused by soot in atmospheric pollution in non-smokers of the general public; (3) Centrilobular Emphysema, due to bronchiolitis caused by exogenous agents such as smoking, infection, cadmium fumes, etc. With the benefit of hindsight, it is clear that Gough did not recognise the common feature of all three, namely the deposition of inhaled particles in the centrilobular region. This now seems self-evident, and the need for three categories is redundant, but at the time it was a source of confusion. Nor did he recognise the imperative for an inflammatory component in all three cases. At that time, the cellular understanding of pathological change was not advanced, and the current appreciation of the role of proteolytic enzymes and growth factors, released by inflammatory leukocytes during lung remodelling, was not available to him.

By 1970, these issues had been clarified by measuring emphysema in GWSs of 247 Welsh coal miners who had died between 1965 and 1967, and whose FEV1 and chest radiographs were available.[22] The emphysema seen in the coal miners was found to be predominantly Gough's focal dust emphysema, that is caused by coal dust. There was a linear relationship between increasing focal dust emphysema score in the lung sections and decreasing FEV1. It was evident from this study therefore that the main type of emphysema in coal miners was Gough's focal dust emphysema, a response to deposition of coal mine dust in the centrilobular region, and that this was associated with airways

obstruction. Crucially however, smoking history was not accounted for in this study, weakening any conclusion of a causative link.

Gough died in 1979, but in 1981 his colleagues confirmed, using GWSs, that in both smoking and non-smoking miners, the single most dominant type of emphysema was that related to coal dust deposition in the centrilobular region.[26] In 1984, it was further established that centrilobular emphysema is the form of emphysema associated with coal dust exposure in coal miners,[27] and in 1990, Rom[28] described inflammatory processes underlying emphysema in coal miners and proposed that such processes underlie all emphysema except that due to alpha-1 – antitrypsin deficiency. Today, centrilobular emphysema is seen as the defining form of emphysema that arises in coal miners, smokers and other groups where there is extensive centrilobular deposition of toxic particulate matter.[29]

With hindsight it is clear that, in his early work, Gough advocated an overly complex classification of emphysema. In fact, the lung deposition of all particles including coal mine dust, environmental soot and cigarette smoke is highly focal and maximal in the centrilobular regions. Here, beyond the ciliated airways, the net flow of air becomes zero, leading to efficient particle deposition by diffusion. The fact that inflammation arises at this site is consistent with the basic tenet of toxicology – that the pathological response is seen at those points where the highest dose is focussed, and thus the classical centrilobular pattern of tissue destruction is seen in coal miners. The extent of the inflammation and subsequent degree of disease is modified by the deposited dose and intrinsic toxicity of the particles in question. Some particles, such as asbestos and silica, cause mainly fibrosis, whereas some, such as cigarette smoke and cadmium, cause mainly emphysema, but coal causes both.

Consequences for coal miners

Gough raised the most effective medical voice in the long campaign for compensation for miners disabled by pneumoconiosis, both in the UK and USA. In the UK, CWP was recognised as an industrial disease in 1943. By 1970, the association between the extent of emphysema as measured in lung sections in CWP, and decline in lung function had been recorded.[22] However, opinion was divided on emphysema since most miners smoked and cigarette smoking had been recognised from the 1950s as a major risk factor for development of emphysema. Further research in the 1980s showed that the reduction in respiratory function in coal workers was related to their cumulative dust exposure and raised the obvious issue of emphysema as a compensable condition in coal miners.[30] The presence of emphysema was shown to be related to the extent of dust exposure as a consequence of working underground, but only in miners who showed some degree of fibrosis. This effect was evident in both smokers and non-smokers, although emphysema was more severe in the smokers. In 1993, the British government accepted a recommendation from the Industrial Injuries Advisory Panel that miners who had been employed for more than 20 years underground should be compensated for any chronic bronchitis and emphysema they had developed, as judged by a defined decrement in their FEV1.[31,32]

GWS in the collection of the Royal College of Surgeons of Edinburgh museums

The Museums of the Royal College of Surgeons of Edinburgh have, in their collection, 12 GWSs of whole organs: five lung, two brain, four heart and one liver, gifted to the museums by Professor Jethro Gough in the 1960s. Typical of these is museum catalogue number GC.13157. According to the specimen number of the Pathology Department of WNSM that is entered in pen on the departmental stamp on the back of the section (65/309), the specimen was prepared in 1965, and the sectioning probably occurred shortly after it was logged into the system. The specimens have, therefore, been in the collection of the Surgeons' Hall Museums for about 50 years. During this time, the sections show no sign of physical degradation or breakdown and have retained their colour and structural integrity well, as shown in Figures 2 to 5.

The response of the lungs to dust as revealed in GWS

Figures 2 to 5 comprise three GWSs from the Surgeons' Hall Museum collection, and one GWS presented to one of the authors (AS) by Professor Gough. The four GWSs are shown here to demonstrate how GWSs can very powerfully reveal the exposure and the response of the lungs to coal mine dust. Figure 2 shows a lung from a female who lived in Cardiff and had no occupational exposure to dust, and it is evident from the light level of pigmentation that she was not a smoker. Neither has there been much exposure to environmental particulate in air pollution, as the degree of environmental anthracosis (blackening) is very limited. In that regard, Gough notes elsewhere[24] the 'clean air in Cardiff' compared to London at that time (the mid-1960s). The tracheobronchial LNs are dark but are not enlarged. In the magnified area in the callout at the top, there is a terminal bronchiole and an alveolar duct with some minimal anthracotic pigmentation, and on the left the callout shows sub-pleural pigmentation – a consequence of traffic of lymph, containing particles, to the sub-pleural lymphatics. However, the overall picture is of a very clean lung with regard to particle accumulation with no pathology.

Figure 3 shows a lung with macule formation, the first stage of dust accumulation. The notes accompanying the specimen merely state that 'The lung shows clear macule formation'. These macules are obvious as foci of black dust in centrilobular regions scattered throughout the whole lung section. There is no significant emphysema, although some distension of the alveolar ducts may be present. The callout shows a terminal bronchiole opening out to a respiratory bronchiole and alveolar ducts, which show anthracotic pigmentation in their walls. The two vessels that

supply the lobule are also clearly seen against an accumulation of perivascular pigment. The tracheobronchial nodes are not enlarged, and there is no emphysema.

Figure 4 is a section of lung from a coal miner with 'early pneumokoniosis' according to Gough's own description. The predominant appearance is of centrilobular emphysema, as shown in greater detail in the callout, with little in the way of the classical nodules of CWP. The callout shows the loss of parenchymal tissue typical of centrilobular emphysema. The tracheobronchial nodes are enlarged, but their capsule appears intact in comparison with the nodes as shown in Figure 5. In the lower parts of the lung, there is small amount of panlobular emphysema.

Figure 5 shows an extremely coal-blackened lung with PMF in the upper lobe that has cavitated. There are simple nodules, present along with areas of centrilobular emphysema. The predominant response in this lung is fibrosis, and the relative paucity of emphysema in the section is striking. Failure of the central LNs to retain dust at high LN burden has been advanced as a mechanism in the progression towards complicated CWP.[21,33] In this specimen, the central tracheobronchial nodes are grossly enlarged, and their capsule appears to be breached. Although it is of course impossible to know the active process that is occurring in what is essentially a 'snapshot' of the ongoing pathological process, a strong impression is gained of black pigment and cells exiting the nodes and flowing into the surrounding interstitium, vascular tissue and bronchial walls. Since the role of the LNs is to sequester dust in benign conditions and protect these other compartments, delivery of localised high dose to them when the capsule breaches is pathogenic.

Conclusion

The GWS technique of whole lung thin sectioning was developed by Professor Jethro Gough and Mr James E Wentworth in the WNSM in the 1940s. Using GWSs, Gough was the first to identify the two principal forms of emphysema – panlobular and centrilobular. Gough utilised GWSs to make considerable advances in interpreting X-rays in CWP and understanding and describing the development of simple CWP and emphysema in such workers in the third quarter of the twentieth century. Gough showed that the only available imaging method at that time, X-ray, did not allow quantification of emphysema. Therefore, the anatomical–pathological approach allowed by GWSs was the only one available for understanding emphysema development and its relationship to exposure and lung function decline, although it had the disadvantage that it could only be carried out on lungs post-mortem. The GWS technique enjoyed a peak in the 1960s, and since then its use has declined. Its use predates modern imaging techniques such as the CT scan, which is now used routinely to quantify emphysema. The decline in autopsies also means that fewer lungs are resected and available for whole lung sectioning. Gough's studies in the pathology of emphysema using the GWS method highlighted the role of emphysema in the decline of lung function in coal miners, leading to its classification as a compensable disease. GWSs remain a powerful technique for visualising pathological change, especially emphysema, in dust-exposed lungs.

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