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CLEARING OF X-RAY SHADOWS IN WELDERS' SIDEROSIS

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With illustrations on plate

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IN 1936 we described the abnormal X-ray shadows in the chest films of some electric-arc welders and emphasised that they were found in men who were apparently in good health (Doig and McLaughlin 1936). We were unable at that time to come to a conclusion about the cause of the condition, but suggested that the iron-oxide particles in the welding fume, when inhaled, "might be opaque to X rays and produce the picture without the associated presence of fibrosis and congestion." Our original observation has been amply confirmed by many investigators, including Enzer and Sander (1938), Britton and Walsh (1940), Koelsch (1941), Cramer (1942), Humperdinck (1942), Groh (1944), and Jones and Lockhart (1944).

Enzer and Sander's article, illustrated with excellent photomicrographs, contains the only published account of the pathological investigation of the lungs of a welder who had died after an accident and who during life had shown the characteristic radiographic changes.

Apart from lobar pneumonia in the left lung, both lungs showed a finely distributed black pigmentation throughout and there was no tuberculosis. On histological examination the lymph-glands showed irregularly distributed pigment almost all of which was within large macrophages. In the lungs there was much amorphous pigment, mainly in peri-arterial but also in peribronchial lymphatic channels and alveolar septa. No fibrosis was found either in the glands or in the lung tissue; the blood-vessels were not obliterated, even where the pigment was most dense, and the walls were not thickened. Chemical analyses of two samples of the lungs showed 0·096 and 0·089 mg. of silica per 100 g. No quantitative analysis for iron was made, but both in sections of the lungs and glands and in the residue left after micro-incineration the Prussian-blue reaction for iron was obtained.

Enzer and Sander conclude that the radiographic appearances were caused by the deposition of iron oxide in the lungs resulting from inhalation of the welding fume, and point out the apparently inert nature of the pigment and the absence of functional impairment.

The condition can be called siderosis, not in the sense in which Zenker originally used the term as implying the presence of fibrosis, but simply meaning the presence of iron dust in the lungs. Subsequent investigations have shown conclusively that iron oxide in its pure form does not set up fibrosis in the lungs. McLaughlin et al. (1945) described siderosis in 4 silver finishers who had inhaled iron oxide (rouge) at work for many years. One of these men died after a gastric operation, and no fibrosis of the lungs was found. Three confirmatory necropsies were described by Barrie and Harding (1947). It was also shown by experiments with sponges that iron-oxide dust is opaque to X rays. Harding (1945) and Harding et al. (1947) introduced iron oxide into the lungs of rats both by intratracheal injection and by inhalation and obtained radiographs comparable with those obtained in human siderosis. Histological studies showed that fibrosis did not develop in relation to the deposits of iron-oxide dust in the lungs.

Since 1936 we have continued our examinations of some hundreds of welders (electric-arc, carbon-arc, and oxyacetylene) and have found siderosis in each group, but we have delayed publication in the hope of an opportunity, which has so far not presented itself, for necropsies and histological studies. The fact that only one necropsy in a case of welder's siderosis has been reported (death was due to an accident) is evidence that the condition is not morbid. We have been constantly on the look-out for necropsy material.

FOLLOW-UP OF ORIGINAL CASES

In October, 1945, we re-examined 15 of the cases described by us in 1936, and in some respects the results are instructive. All except 2 of them had continued to work full time as welders, and all had remained in good health. One man has given up welding entirely, and the other has become a welding instructor. These 2 cases are described in greater detail below. Of 7 men who showed no specific radiographic changes due to dust in 1936, 5 (average age 33·3 years, average welding exposure 14·8 years) still show no abnormal changes; 1 (aged 35, sixteen years welding) was now classed as suspicious; and 1 (aged 30, fifteen years welding) showed a definite but slight degree of radiographic reticulation. Classed as suspicious in 1936, 2 welders showed in 1945 a definite picture of welders' siderosis; their ages were 29 and 31 years and their welding experience fifteen and seventeen years. Of 6 men who previously showed definite inhalation changes 5 continued to do so in 1945. In 4 of these (average age 55·25, average welding experience 32·25 years) there was no change in the intensity of the abnormal X-ray shadows. In one case (case 2 below) there was a considerable clearing of the shadows. The other man (case 1) had a normal chest film where previously he had shown pronounced radiographic changes due to inhalation of dust.

COMPLETE CLEARING OF X-RAY SHADOWS

This man was case 2 in our 1936 published series. He was first seen in 1934, when he was 35 years old and had been an electric-arc welder for eleven years. He had not been previously exposed to dust. His welding experience had included about half his time in various types of storage tanks with an average capacity of 500 gallons. Nearly all his work was on uncoated mild steel, and he had not used bare wire electrodes or the carbon-arc method. Except for a slight cough and occasional morning sputum he had no symptoms of ill health.

Clinical examination revealed a chest expansion equal on both sides but rather less than normal. The percussion note was not impaired; the breath sounds were harsh, with prolongation of the expiratory murmur over both lower zones, especially in the mid-axillary area; and post-tussic crepitations were heard at the bases. Examination of the sputum disclosed large numbers of macrophages packed with fine granules of iron oxide giving the Prussian-blue reaction. No asbestos bodies were seen.

Radiography showed a well-marked reticulation with a tendency to early nodule formation (fig. 1). On being informed that his radiograph was not normal he gave up his work as

a welder to become a storekeeper and thus had no further exposure to welding fume. Radiography sixteen months later showed that the shadows were still present, but that the reticular strands had become blurred, suggesting congestion in the areas of dust deposition. Subsequent films until 1938 showed that the blurring had persisted, but that there was a definite decrease in the intensity of the shadows.

Follow-up.—The man was not seen again until 1945, when he looked healthy and said that he felt very well. He had no regular morning cough or sputum, chest movements were of moderate degree, and percussion and auscultation revealed no abnormal signs. A radiograph of the chest showed the complete disappearance of the blurring, reticulation, and mottling, the film being normal except for a slight residual accentuation of the normal linear markings (fig. 2).

PARTIAL CLEARING OF X-RAY SHADOWS

The man in whom the X-ray shadows became less intense was case 1 of our 1936 series. He was first seen in 1933 when he was 44 years of age. Because of slight staining of the sputum he was sent to a tuberculosis clinic and there seen by one of us (A. T. D.). His father and grandfather had both died of silicosis. He himself had worked as a blacksmith for eighteen years, occasionally doing a little arc welding, and after that for eleven years he had been a full-time arc welder doing half his work with covered electrodes in enclosed spaces. When seen he had no pronounced symptoms except a slight occasional cough and he had the appearance of a perfectly fit man, with excellent build, musculature, colour, and nutrition.

Physical examination revealed few abnormal physical signs except somewhat harsh breath sounds and a few fine post-tussic crepitations at the bases. He continued to work as a welder in the same factory but under improved conditions of ventilation.

Follow-up.—We made periodical examinations until 1938, and these continued to reveal a satisfactory state of general health, with no change in the degree of siderosis. He was lost sight of in 1938 but was traced in 1945. It was found that since 1940 he had given up full-time welding and had become a welding instructor at a technical college. This meant that his exposure to welding fume was a great deal less than when he was working at the factory. He estimated that only a quarter of his time was spent in actual welding, and that only in short breaks, in demonstrating the technique of the work or in correcting faults. No work in confined spaces was done at the technical college. We could not examine him ourselves and are indebted to Group-Captain J. G. Somerville, No. 3 R.A.F. Rehabilitation Unit, who made a clinical examination in June, 1945, and reported: "The man says he feels very well and has no complaints. There is no cyanosis and no dyspnœa on exertion, and no abnormal clinical signs can be found in his chest." The radiograph taken at the time was sent to us. It still showed a definite degree of siderosis, but compared with those taken before 1938 there was considerable resolution as shown by diminution in the intensity and concentration of the lesions. The film now shows no nodulation, but merely a rather coarse reticulation with some blurred "beading" of the bronchi (figs. 3 and 4).

DISCUSSION

Siderosis is one of the benign pneumoconioses, a term applied by Pendergrass and Leopold (1945) to those cases in which inert dusts are deposited in the lungs causing characteristic radiographic changes but no fibrosis or disability. Etymologically, the word pneumoconiosis merely means "dust in the lungs" and does not carry with it the concept of fibrosis, but textbooks usually define it as fibrosis of the lungs due to dust. In the Workmen's Compensation Act, 1943, and in the National Insurance (Industrial Injuries) Act, 1946, pneumoconiosis is defined as "fibrosis of the lung due to silica dust, asbestos dust and includes the condition of the lungs known as dust reticulation." For compensation purposes the term must necessarily be restricted to a disabling condition. Recently, however, there has been a tendency to restrict it still further to those cases showing radiographic reticulation of the lungs in contrast with those showing nodulation, and there seems to be little justification for this practice. The difference between the medical and legal concepts of pneumoconiosis should be kept clearly in mind.

So far as we are aware, no previous reports have appeared in which the disappearance or even retrogression of the lesions of pneumoconiosis are described. Bentzen (1933) records a case of "acute siderosis" in which a man worked for two months at a machine for crushing steel sponge and was exposed to a very light spongy dust which oxidised quickly. The radiographic changes consisted of irregular fine mottling which resolved to some extent later and were interpreted as a miliary pneumonia. In view of the short exposure it seems to us that this term is more applicable than acute siderosis. Our 2 cases provide clinical and radiographic evidence to support the view that some dusts, though producing abnormal X-ray shadows when enough is inhaled, merely lie inertly in the tissues and cause no proliferation of fibrous tissue or other permanent reaction. The normal scavenging mechanism of the lungs does not appear to be impaired. Some of the dust is drained away in the lymphatics to the lymph-glands, and it is conceivable that some might pass through the lymph-glands after being extruded from the phagocytes in which they arrived in the glands and be finally discharged into the blood-stream through the thoracic duct. It is likely, however, that much of the dust is removed from the lungs in the sputum. We showed in 1936 that elimination of the dust by the sputum was actively going on in one case eighteen months after the man had given up welding. This is the man whose radiograph now shows no abnormal shadows. Barrie and Harding (1947), as a result of their necropsies of silver finishers, have also concluded that most of the inhaled iron dust is eliminated in the sputum. It appears therefore that in cases of benign pneumoconiosis, when no more dust is inhaled or when the amount inhaled is less than the amount eliminated, the accumulated dust tends gradually to clear away, with a corresponding improvement in the radiograph.

Though necropsies in cases of siderosis are few, there is a constantly growing body of evidence that it does not seriously affect the worker's health. Clinical examinations (Enzer and Sander 1938, Sander 1947, Britton and Walsh 1940, Humperdinck 1942, Groh 1944, Doig unpublished) show no signs of fibrosis or of diminished pulmonary ventilation. Enzer et al. (1945) found no statistically significant difference between 15 welders with siderosis and a group of normal men in a comparable age-group as regards vital capacity, pulmonary ventilation, and endurance in static and dynamic work. Further evidence of the inert nature of iron in the respiratory tract is found in descriptions of benign pneumoconiosis in occupations, other than welding, involving exposure to fine iron or iron-oxide dust. Otto (1939) mentions a case of siderosis in a man employed in an ochre mine and mill. There were few subjective symptoms. Buckell et al. (1946) found 15 out of 171 persons exposed to iron dust in iron turneries showing radiographic reticulation, but symptoms were few. Pendergrass and Leopold (1945) record 4 cases of benign pneumoconiosis among 50 steel grinders; Sander (1947) describes 3 cases in oxyacetylene cutters; and Hamlin (1947) 4 cases in foundry grinders and burners. In all three series the men worked in rooms where there was also exposure to silica dust as well as iron, but the amount of silica dust was considered too low to cause silicosis. On the other hand, we know of an electric-arc welder who worked in a steel-fettling shop exposed to free silica dust as well as to the iron oxide of the welding fume and who died from silicosis (H. E. Harding, personal communication). But when the exposure to the dust of iron or its oxides is uncomplicated

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by other factors it does not set up a disabling condition. Perhaps this statement should be qualified by saying that, over a 12-year period of observation, siderosis in welders has not been found to cause disability.

SUMMARY

The published work relating to the benign pneumoconiosis or siderosis of welders is briefly reviewed. There is evidence that the condition is not accompanied by fibrotic changes in the lungs or by obvious disability.

In a re-examination of 15 welders examined nine years or more previously 5 of 7 men with no abnormal radiographic changes at the first examination were still normal; the sixth man of this group showed suspicious and the seventh early but definite inhalation changes (reticulation); 2 men who previously had suspicious changes had definite radiographic reticulation; and 4 of 6 men who had originally shown well-marked radiographs of welders' siderosis still had the same condition. All these men had continued to work as welders and had remained in good health.

The cases of 2 men are outlined of whom one had given up welding and the other had spent less time at the job. In the first case the abnormal shadows completely disappeared, and in the second the shadows became less intense.

These 2 cases provide evidence that the radiographic reticulation and nodulation of welders, siderosis is not necessarily permanent, and that the iron-oxide dust can be eliminated from the lung parenchyma after some years.

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PULMONARY TUBERCULOSIS TREATED WITH p-AMINOSALICYLIC ACID

EARLY RESULTS IN 6 CASES

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REPORTS on the systemic treatment of pulmonary tuberculosis with *p*-aminosalicylic acid (P.A.S.) have been published by Lehmann (1946, 1947) and Vallentin (1947), while Dempsey and Logg (1947), in the course of their work on the local administration of this drug in tuberculous empyema, treated a few patients systemically but for short spells only, because of limited supplies (personal communication).

At Colindale Hospital we gave P.A.S. by mouth to 5 patients for sixty days and 1 patient for four weeks, after which the supply of the drug allocated for this trial was exhausted.

As supplied to us, P.A.S. was a white crystalline powder, and from this the sodium salt was prepared, giving, even at a concentration of 30%, a clear solution as described by O'Connor (1948). Preparation of the sodium salt requires patience and accuracy; if the final pH is taken to 7, or if the solution remains exposed to air, it becomes darker but remains translucent. The solution has a bitter but not unpleasant taste, which can be readily disguised with flavouring agents.

All the patients were given 12 g. of P.A.S. daily in divided doses three-hourly, one night dose being omitted. I gave a sixty-day course in cases 1, 2, and 3; Dr. Jannette Owen gave a sixty-day course in cases 4 and 5; and Dr. Grace O'Malley gave a four-week course in case 6. All the patients were men. The results are summarised in the accompanying table. As regards the patients not treated by myself I have interpreted the data supplied to me.

Case 1.—A man, aged 19, was admitted on Aug. 5, 1947, with an exacerbation of pulmonary tuberculosis, which had originally been diagnosed in 1941. He went rapidly downhill and was severely toxic. By December his weight had fallen from 119 to 107 lb. A large cavity in the left apex had increased from $3 \times 3^1/_4$ in. to $3^1/_2 \times 4$ in. and showed a fluid level. Penicillin 9 mega units, in six days, had no effect on his general condition or temperature, and on Jan. 27, 1948, a course of P.A.S. was started.

There was dramatic improvement after the first few days—the toxicity disappeared, appetite returned, and he became interested in his surroundings and asked to be taken back to the general ward from the side ward where he had been kept for two months. He gained 27 lb. in eight weeks.

Radiography (April 19, 1948) showed that the cavity had lost its fluid level and had shrunk from $3^1/_2 \times 4$ in. to $2^3/_4 \times 2^1/_2$ in. There was considerable contraction and hardening of the apical lesions and clearing of the soft mottling in the other zones.

His general condition remained very good three weeks after the completion of the course; but his evening temperature rose above 99°F, his erythrocyte-sedimentation rate (E.S.R.) remained unchanged, the number of bacilli per field increased to 5, and he lost 2 lb. in weight.

Case 2.—A boy, aged 16, with rapidly advancing tuberculous pneumonia involving the whole left lung. By the end of December, 1947, he was severely toxic and rapidly going downhill, with a hectic pyrexia (see figure).

After a course of 9 mega units of penicillin, P.A.S. was started on Jan. 27, 1948. The change in the clinical picture was even more remarkable than in case 1. Toxicity disappeared on the fourth day, and the patient asked to resume the embroidery work he had given up two months previously. He gained 6 lb. during the seventh and eighth weeks of the course.

Radiography showed that the rapid progress of the tuberculosis was arrested after the first fortnight, and films made on April 30, 1948, showed hardening and retraction of the lesions, which were then restricted to the left upper lobe, the other fields having almost completely cleared. The honeycombing in the left upper lobe, noted in January, had given place to clearly defined cavities.

Three weeks after the completion of the course the patient's condition was still very good, with no sign of returning toxicity; and major surgery of the left upper lobe (thoracoplasty or pneumonectomy) could reasonably be considered. But his pyrexia is now gradually increasing, although it is not so high as it was before the course of P.A.S.; there is a slight increase in the number of bacilli per field; the E.S.R. has risen from 8 to 15 mm. in an hour; and the patient has lost 6 lb. in weight.

Case 3.—A man, aged 31, with tuberculous pneumonia and poor resistance, reached a hopeless condition by December, 1947, being very toxic and apathetic, with severe anorexia. He was given penicillin, 6 mega units, after which a sixty-day course of P.A.S. was started on Feb. 29, 1948. Again the improvement in his clinical condition was the first and most striking effect. Cough became much easier, the feeling of tiredness disappeared within a few days, and his appetite rapidly improved. (Five consecutive specimens of sputum were negative for tubercle bacilli.

Radiography (April 30, 1948) showed much fibrosis of the right apex, with displacement of the trachea, well-marked