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Case reports of eye injuries due to splash burns from chloroacetophenone, chloroacetophenone solution, bromobenzylcyanide, phosgene, white phosphorus, sulfur trioxide chlorosulfonic acid, titanium tetrachloride, and thermite are presented. Treatment is discussed.—*Author's summary.*

HEMOGLOBIN CRYSTALS, CASTS, AND GLOBULES IN THE RENAL TUBES OF GUINEA PIGS FOLLOWING CHEMICAL HEMOLYSIS. R. C. Dunn and S. H. Webster. *Am. J. Path.*, vol. 23, pp. 967-981 (Nov., 1947).

Single one-hour exposures to stibine, a hemolytic gas similar to arsine, were administered to guinea pigs, wide ranges of concentration being used with different groups of animals. Hemoglobinuria was observed frequently with concentrations above 65 parts per million. Under conditions which are described, hemoglobin crystals were formed in the renal tubules of 23 of 97 guinea pigs. The crystals were found only in animals dying or killed on the second through the sixth day following the exposure to 92 p.p.m. or higher. All of the animals showing crystals had clinical or microscopic evidence of hemolysis, or both. In guinea pigs succumbing during the above time interval, the usual clinical picture before death was that of dyspnea, weakness, often a shock syndrome, and hemoglobinuria followed by coma. In addition to crystals, many of the tubules showed hemoglobin casts and globules. The association of these three forms in relation to time and stibine concentration is indicated. Other pathologic conditions, such as tubular dilatation, coagulation necrosis, cellular casts, fat, and glomerular changes, were sometimes noted. Hemosiderosis in the kidney, although of frequent occurrence, was not found in animals with three to five weeks of survival time except after exposure to high concentrations of stibine. There are factors, other than stibine concentration, which may influence the deposition of one or more forms of hemoglobin in the tubules. The data presented are sufficient to give a picture of the chronologic development of hemoglobin globules, casts, and crystals in relation to concentration and time.—*Author's summary.*

THE EFFECT OF ASBESTOS ON TISSUE CULTURES: A COMPARATIVE STUDY WITH QUARTZ AND COAL DUST. T. H. Bell, J. Friedmann and E. J. King. *J. Path. and Bact.*, vol. 59, pp. 159-164 (1947).

(1) Phagocytosis, even of small particles of asbestos, took place slowly in tissue cultures and a considerable portion of the asbestos dust, approximately 50 per cent, remained untouched, though within range of phagocytes throughout the experiments. Compared with coal or quartz, asbestos was less readily phagocytosed, particle size being equal. (2) Asbestos particles in small amount had little or no toxic effect on living cells in tissue cultures. The growth of fibroblasts was neither retarded nor accelerated and no necrosis of cells was

observed as a result of contact with asbestos particles, while fatty change appeared to occur more slowly in asbestos phagocytes than in coal phagocytes. (3) Asbestos particles underwent no visible change, and in particular no visible diminution in size, after having been ingested by phagocytes for as long as five weeks. (4) The motility of wandering phagocytes was decreased after the ingestion of asbestos particles. This effect, not necessarily of a toxic character, may be deleterious in the case of cells within the lungs, since it tends to restrict the migration of cells and so favor the retention of particles within the lungs. (5) Particles under 5 microns in length restricted cellular motility more than larger particles, for the reason that they were more readily phagocytosed. It would seem that wandering phagocytes are rarely able to ingest particles larger than 10 microns. Giant cells sometimes formed in tissue cultures, apparently by fusion of two or more ordinary phagocytes, and were then able to engulf larger particles or clusters of particles. (6) There was no evidence of the formation of asbestos bodies in these experiments, nor have we noted the appearance of brown granular pigment like that seen in the lungs of experimental animals.—*Authors' summary.*

TISSUE REACTION TO SERICITE AND SHALE DUSTS TREATED WITH HYDROCHLORIC ACID: AN EXPERIMENTAL INVESTIGATION ON THE LUNGS OF RATS. E. J. King, Margaret Gilchrist and M. V. Rac. *J. Path. and Bact.*, vol. 59, pp. 324-327 (1947).

The innocuous nature of South Wales shales and sericites in the rat lung is confirmed. Preliminary treatment of shale and sericite dusts with hydrochloric acid resulted in their acquiring pathogenic properties, which led to the production of fibrous nodules in the lungs of rats.—*Authors' summary.*

ACCIDENTAL ELECTROCUTION: WITH DIRECT SHOCK TO THE BRAIN ITSELF. W. E. Carnegie Dickson. *J. Path. & Bact.*, vol. 59, no. 3, pp. 359-365 (July, 1947).

1. The naked-eye and microscopical findings in the brain of an accidentally electrocuted woman aged 45 years are described.

2. A 240-volt alternating current from the main was, owing to a short circuit, applied through an electrically-lit retractor directly to the right temporal lobe, resulting in immediate unconsciousness and death after 21½ hours.

3. The outstanding lesion was a widespread disruptive process in both grey and white matter, demonstrated in the illustrations, and believed to have been produced by the action of the electric current in liberating bubbles of steam and gas as a result of the intense heat and electrolytic action.

4. No description of an extensive cerebral lesion of this nature has been found in the literature of electrical injuries.—*Author's summary.*

THE GENERAL PRACTITIONER AS AN INDUSTRIAL SURGEON. F. E. P. *Ind. Hyg. & Toxicol.*, vol. 4, no. 3, pp. 293-304 (Sept., 1947).
A number of ways in which the general practitioner can aid and cooperate with the industrial surgeon are discussed. In addition to rendering aid in the case of minor defects which impair the efficiency of the worker, the general practitioner can provide efficient placement of handicapped workers, and a realistic evaluation of working capacity in the case of workers with prognosticating disabilities, occupational health hazards. The author stresses objectivity and care in all aspects of the general practitioner's role in the case of the worker. The smoothness of his cooperation with the industrial surgeon is emphasized.—*James H. Jandl.*

THE WORKING OF AN INDUSTRIAL NEUROPSYCHOLOGICAL UNIT. Maxwell Jones. *Occup. Ther.*, vol. 1, pp. 213-221 (Aug., 1947).

The author describes the organization and therapeutic procedures employed in the neurosis unit at Sutton Emergency Hospital. The unit is at present in process of being transferred to a general neurosis center of the hospital, and is treating only patients with minor neuroses. To be admitted to the unit, a patient must be registered under the Employment Act, and is usually referred to the unit by the industrial department in the London area, the final decision being made by the hospital. In addition to the unit, any part of the country on referral to the unit is possible.

The unit contains 100 male patients. A full psychiatric history taken and a full employment history form is completed for each patient.

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IDENTIFICATION OF FREE SILICA IN INDUSTRIAL DUSTS. W. H. Gilzen. *Anal. Chem.*, vol. 20, pp. 267-268 (March, 1948).

The author precipitated free silica from dusts directly on thin celluloid with a molybdenum precipitator, then cut strips of the celluloid and graphed the particles by a method which distinguishes between silica and alumina. The mounted preparation was further treated by being dissolved and volatilized, the alumina being unaffected.

No claim is made that this method of analysis will work on ground

and, instead of a somewhat granular and disseminated nodular appearance followed by concentration and breaking down of coalescing areas of nodulation, there seem to develop large pseudo-tumorlike masses. Such shadows may be found in silicosis without tuberculosis, and are then usually central. When tuberculosis is added, they are apt to be less symmetrical and to be found also at the apex or base, and to be accompanied with the other indications of tuberculosis already listed. Tuberculosis has been twice as frequent among the miners in Belgium, especially in workers over 40, as it has been among other laborers. The present underfed and exhausted state of many miners indicates probable increase in the number of open cases. Most of the miners work in hard rock, but even those who have not apparently been as directly exposed to silica dust develop some silicosis, possibly from siliceous dust which has settled in the bottom of the mine. Some of these men have previously worked in other mines where they have been directly exposed to silica. Compensation should be available for silicosis developing in workers in any of the mines.—*Am. Rev. Tuberc.*

ARCHITECTURAL CHARACTERISTICS OF VARIOUS TYPES OF PNEUMOCONIOSIS STUDIED BY MEANS OF PLASTIC MODELS. G. Mottura and R. Dulbecco. *Bull. Histol. Tech. micr.*, vol. 24, p. 11 (1947).

Models were constructed from sections of normal lungs containing the usual quantities of carbon, from the lungs of an ironstone miner, and from the lungs of foundry sandworkers. Inert dusts, carbon, and iron were most concentrated around respiratory bronchioles, especially at their first point of division. Around infundibula there was dust only at the points of contact with arterial branches. No deformation of the bronchoalveolar structure resulting from anthracosis or siderosis was discovered. (Emphysema is not mentioned).

In silicosis the dust is found around respiratory bronchioles, around the arterial branches that accompany the bronchial divisions, and in the interlobular septa. The distribution around respiratory bronchioles is similar to that of inert dusts, but is accompanied by marked proliferation of the connective tissue. There is a constant mantle of hyperplastic connective tissue around the arterial branches; characteristic hyaline silicotic nodules are formed almost exclusively in the adventitia of the very small arteries close to branches of the respiratory bronchioles. The very small arteries run through these nodules. In some cases, especially in the subpleural zones, there is considerable proliferation of dense connective tissue in the interlobular septa. This is laminated and not nodular, and it encircles branches of the pulmonary vein and sends small prolongations between the adjacent infundibula.

The fibrosis resulting from silica often produces distortion with narrowing of the bronchioles.

The authors conclude that silicosis differs from anthracosis and siderosis not only in the marked connective tissue proliferation at the usual sites of dust accumulation, but also in the presence of hyaline nodules against the arterioles and of a laminated perivenous fibrosis in the interlobular septa. They think that the formation of typical silicotic nodules is evidence of a local tissue disposition in the arterial adventitia, and they explain the interlobular fibrosis as resulting from the strain placed upon the secondary lymphatic channels connecting the periarterial to the perivenous lymphatics when the main normal paths become blocked. This is especially evident in the sub-pleural layers, where these anastomoses may provide a collateral lymph circulation because of their connection with the pleural lymphatic network.

(The illustrations are not useful).—*Brit. J. Indust. Med.*

ASBESTOSIS AND CANCER OF THE LUNG. R. Desmuelle, L. Rousseau, M. Giroux and A. Sirois. *Sem. Hôp.*, vol. 23, p. 1820 (1947).

These four papers come from Quebec, Canada, where several workers in the asbestos mines have been studied. The Quebec school disagrees with the statements of Gardner that pneumoconiosis due to asbestosis is not an important factor in the production and progress of pulmonary tuberculosis, and that the presence of asbestos bodies in the sputum has no value in the diagnosis of asbestosis. Rousseau gives brief descriptions of 4 patients, some of whom are also reported in the following papers. One had worked in the asbestos industry for 18 years, and experienced respiratory symptoms only during the last 3. He complained of cough and an asthma-like dyspnea. Asbestos bodies were found in his sputum, but no tubercle bacilli. Radiography is said to have shown a ground-glass appearance in his lungs (unfortunately the illustration is too poor to demonstrate this). This is the only case seen at this hospital in which it has been possible to make a diagnosis of asbestosis during life. Another patient, who had been exposed to asbestos dust for 25 years and who died from some unrelated cause, showed no clinical or radiological signs of any abnormality in his lungs during life.

Giroux exposed guinea-pigs, rabbits, and a dog to crude asbestos dust in a chamber daily for several months. The rabbits, killed after periods of from 3 to 12½ months, had no asbestosis and no asbestos bodies. The dog showed no lesions after 10 months' exposure: only the guinea-pigs showed lesions in the lungs and asbestos bodies. In these animals he traces the stages of formation of asbestos bodies: fibers reaching the alveoli are rapidly covered by phagocytic cells; movement of the fibers results in microscopic bleeding

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damaged alveolar walls; the hemoglobinized red cells is taken up by the phagocytes which already enclose the asbestos fibers. These are then extruded by means of intratesticular injections into the suspensions of asbestos in olive oil resulting in the production of granulomatous lesions by the asbestos bodies. When however, the suspension is introduced into an area into which 1 ml. of blood had been introduced immediately before, asbestos bodies were not extruded. He considers that the presence of asbestos bodies in sputum has significance, since it indicates damage in the lungs.

Desmuelle, Giroux, and Richard reported 44 years, in whose family there was tuberculosis. He had worked as a bag maker for 20 years: after 10 years he began to experience dyspnea on effort, accompanied by cough and sputum. He became more severely affected in the months before his death, which was due to asbestosis. The authors argue that their case preceded and paved the way for the recognition of asbestosis. They admit that they have no proof of this connection of tuberculosis and asbestosis is generally accepted in Britain but is doubted in the United States. Desmuelle, Rousseau, Giroux and Sirois give brief details of 2 patients who had asbestosis and carcinoma of the lung. (The claim that carcinoma with asbestosis has been recorded before cannot be allowed. Nor will there be any agreement with the statement that silicosis plays an important part in the pathogenesis of primary carcinoma).—*Brit. J. Indust. Med.*

CLINICAL REPORT ON MINERS' NYSTAGMUS. Adams Campbell. *Brit. J. Ophthalmol.*, pp. 193-198 (April, 1948).

A clinical survey of miners' nystagmus of the general types of the disease: asymptomatic nystagmus with symptoms of head aches, giddiness and oscillation of underground work; are unfit for underground work); as psychoneurosis in whom ophthalmological examination was added to the above. It is recommended that the examination be made by a thalamic surgeon and a psychiatrist; suitable work as soon as possible, and unemployment; that those unfit for therapy plus suitable rehabilitation be aided by a medico-social worker; compensation be made making it financially attractive; and that the disease be recognized by not requiring miners to work in awkward postures.—*Paul*

THE DARK ADAPTATION OF COAL FROM NYSTAGMUS. Forbes W. Ophthalmol., vol. 32, no. 4, pp. 199-201

is resulting from silica often produces with narrowing of the bronchioles.

Authors conclude that silicosis differs from and siderosis not only in the marked tissue proliferation at the usual sites of alveolar, but also in the presence of hyaline in the arterioles and of a laminated fibrosis in the interlobular septa. They explain the formation of typical silicotic nodules as a local tissue disposition in the arterial and they explain the interlobular fibrosis from the strain placed upon the secondary channels connecting the periarterial to the lymphatics when the main normal paths are blocked. This is especially evident in the layers, where these anastomoses may lateral lymph circulation because of their with the pleural lymphatic network. (Conclusions are not useful.)—*Brit. J. Indust.*

AND CANCER OF THE LUNG. R. Desmeules, M. Giroux and A. Sirois. *Sem. Hôp.*, 20 (1947).

papers come from Quebec, Canada, workers in the asbestos mines have been Quebec school disagrees with the Gardner that pneumoconiosis due to is an important factor in the production of pulmonary tuberculosis, and that the asbestos bodies in the sputum has no value as evidence of asbestosis. Rousseau gives brief details of 4 patients, some of whom are also described in the following papers. One had worked in the asbestos industry for 18 years, and experienced symptoms only during the last 3. He had cough and an asthma-like dyspnea. Asbestos bodies were found in his sputum, but no radiography is said to have shown a calcification in his lungs (unfortunately the poor to demonstrate this). This is the first case at this hospital in which it has been possible to make a diagnosis of asbestosis during life. The patient who had been exposed to asbestos and who died from some unrelated cause showed no clinical or radiological signs of any asbestos in his lungs during life.

In guinea-pigs, rabbits, and a dog to be kept in a chamber daily for several months, killed after periods of from 3 to 10 months, no asbestosis and no asbestos bodies were found. No lesions after 10 months' exposure: the lungs showed lesions in the lungs and in these animals he traces the stages of asbestos bodies: fibers reaching the lungs, covered by phagocytic cells; move- ment results in microscopic bleeding

from damaged alveolar walls; the hemoglobin from the liberated red cells is taken up by the phagocytes which already enclose the asbestos fibers. Subcutaneous or intratesticular injections into guinea-pigs of suspensions of asbestos in olive oil resulted in the production of granulomatous lesions but no asbestos bodies. When however, the suspension was injected into an area into which 1 ml. of blood had been injected immediately before, asbestos bodies were found after a month. He considers that the presence of asbestos bodies in sputum has significance, since it implies tissue damage in the lungs.

Desmeules, Giroux, and Richard report a patient, aged 44 years, in whose family there was no history of tuberculosis. He had worked as a bagger of asbestos for 20 years: after 10 years he began to experience dyspnea on effort, accompanied by cough and expectoration. He became more severely ill some 9 months before his death, which was due to tuberculosis and asbestosis. The authors argue that the asbestosis preceded and paved the way for the tuberculosis, but admit that they have no proof of this. (The association of tuberculosis and asbestosis is generally admitted in Britain but is doubted in the United States).

Desmeules, Rousseau, Giroux and Sirois record brief details of 2 patients who had asbestosis and cancer of the lung. (The claim that carcinoma of the lung with asbestosis has been recorded before in only 1 case cannot be allowed. Nor will there be general agreement with the statement that silicosis is admitted to play an important part in the pathogenesis of pulmonary carcinoma.)—*Brit. J. Indust. Med.*

CLINICAL REPORT ON MINERS' NYSTAGMUS. Dorothy Adams Campbell. *Brit. J. Ophthalmol.*, vol. 32, no. 4, pp. 193-198 (April, 1948).

A clinical survey of miners' nystagmus shows 3 general types of the disease: asymptomatic nystagmus; nystagmus with symptoms of headaches, photophobia, giddiness and oscillation of underground lights (these are unfit for underground work); and nystagmus with psychoneurosis in whom blepharospasm whenever examined was added to the above symptoms. It is recommended that the examination team be an ophthalmic surgeon and a psychiatrist; that men return to suitable work as soon as possible, preferably without unemployment; that those unfit for work be given eye therapy plus suitable rehabilitation; that follow-ups be aided by a medico-social worker; that adjustment in compensation be made making return to work financially attractive; and that the disease be prevented by not requiring miners to work in poor illumination and in awkward postures.—*Paul R. McCurdy.*

THE DARK ADAPTATION OF COAL MINERS SUFFERING FROM NYSTAGMUS. Forbes W. Sharpley. *Brit. J. Ophthalmol.*, vol. 32, no. 4, pp. 199-204 (April, 1948).

Observations of the final rod threshold of dark adaptation on certified and uncertified (for disability) miners with and without active nystagmus or symptoms and on a group of normal non-miner controls showed consistent elevation of the threshold of all miners above that of the controls. Those with active nystagmus and symptoms showed little essential difference from those miners who were without either nystagmus or symptoms (control miners). Highest threshold was in miners certified but without actual nystagmus. No conclusions are drawn.—*Paul R. McCurdy.*

A COMPARISON OF DARK ADAPTATION IN MINERS WITH THEIR NUTRITIONAL STATE. Dorothy Adams Campbell and Eva Tonks. *Brit. J. Ophthalmol.*, vol. 32, no. 4, pp. 205-208 (April, 1948).

Nutritional evaluation of miners and control surface workers by means of history, plasma carotene and vitamin A levels, serum alkaline phosphatase, and thymol turbidity liver function tests showed that the raised threshold for dark adaptation constantly found among miners is not due to any obvious nutritional disturbance.—*Paul R. McCurdy.*

THE PSYCHIATRIC EFFECT OF MINERS' NYSTAGMUS. 1. Edward S. Stern. *Brit. J. Ophthalmol.*, vol. 32, no. 4, pp. 209-224 (April, 1948).

Psychiatric observations on a group of miners, with and without nystagmus or symptoms and certified and uncertified (for disability) and on a group of normal miner controls show that miners' nystagmus is a psychosomatic affliction arising principally from emotional stresses peculiar to their precarious life (economically and physically) plus added factors of working in the dark and in awkward positions for long periods. Fatigue of convergence normally occurring on attempting to see in the dark and fatigue of upward gaze often required of miners are the mechanisms of lateral and vertical nystagmus, respectively. Both combined produce rotatory nystagmus. More than 10 and usually 25 to 34 years of work are required to produce this psychosomatic affliction. Better selection of miners, even if possible, would therefore be impractical as a control measure.

The author recommends that miners' nystagmus as a term be abandoned and that cases should be seen early by a psychiatrist; that such cases be found work above ground in good light without periods of compensation, although short periods of rehabilitation may be necessary; that they be kept under psychiatric supervision; that conditions of lighting and posture under which miners work be improved; that optimal physical and economic security be given them; and that optimal working hours be worked out scientifically rather than politically.—*Paul R. McCurdy.*

A COMPARISON OF DARK ADAPTATION WITH THE PSYCHOLOGICAL STATE IN MINERS. Dorothy Adams