ABSTRACTS


Among the returning men and women we shall feel a special sense of obligation to those whose war service has caused lasting disability. They will have to overcome serious physical and psychological handicaps as part of the process of adjustment to ordinary civilian life. The job is made easier than after the last war: first, because great strides have been made in surgery and therapies, i.e. in the field of rehabilitation; second, by the corresponding progress in the field of resettlement, including wide-awake legislation of the Disabled Persons (Employment) Act, 1944. This Act provides for industrial rehabilitation courses of 6-8 weeks to ‘tone up’ disabled workers. (The first centre for this has been opened by the Ministry of Labour at Egham.) It also provides vocational training courses of 6-12 months at Government Training Centres, and at special centres (such as St. Loyes College, Exeter, and Queen Elizabeth’s Training College, Leatherhead) for the seriously handicapped. There is also to be a Register of the Disabled; compulsory employment of a quota of registered disabled; and the formation of a non-dividend-paying company to provide sheltered employment. A National Advisory Council is already in operation to advise the Minister of Labour on these matters, and District Advisory Committees are appointed to advise locally.

Rehabilitation, training, placement in suitable jobs, and, in certain cases welfare follow-up, are therefore all within the scope of the Government scheme. While this is good in theory, in practice something is going wrong. Placement in the right job is becoming difficult; there should be better welfare follow-up; there may be inadequate co-operation between hospitals and the Ministry of Labour. The Disablement Rehabilitation Officer—the DRO—is generally criticized; it is alleged that they are insufficiently trained, have inadequate knowledge of industrial work, have little understanding of the man’s background, a too casual approach, and have not the right type of education or experience for the work. The field of recruitment should therefore be widened. Selected ex-servicemen, after a certain amount of training, would be specially acceptable to men from the Forces. Welfare and labour officers of the Ministry of Supply, now seeking other employment, might be suitable. There is need for better co-ordination of the national scheme, need for publicity, and the responsibility of industry itself must be more readily accepted by employers.

Memorandum on Dermatitis. (1944). LEADS JOINT COUNCIL ON INDUSTRIAL MEDICINE.

Dermatitis is a general term indicating inflammation of the skin which may result from external or internal causes. Industrial dermatitis is the term applied when such inflammation results from contact with particular dusts or liquids capable of causing damage to the skin and encountered in the course of industrial occupations. Similar dermatitis is caused by other dusts and liquids encountered outside industry as in household duties. It may arise because of the irritant properties of the dust or liquid, from inadequate protection or from inadequate cleansing after work. It may also arise from idio-syncresy or from sensitiveness of the skin to a dust or liquid normally harmless. Such personal predisposition can to some extent be determined by medical examination before employment.

Industrial dermatitis is responsible for much loss of man-hours, but it is, in the main, avoidable. It is not a disease which can be passed from one individual to another, is not contagious, and there is no reason why it should not clear completely if appropriate measures are taken. Industrial dermatitis is the reaction of a sensitive skin to some substance of which it is intolerant. The potential irritant properties of substances naturally vary. It is essential to appreciate that if the skin of a particular individual is provoked to a rash by a particular substance with which he makes contact in his work then as a rule he will recover if removed from that contact or if he is effectively protected from contact. Return to contact with the offending substance will cause a return of the rash unless effective protection is afforded and should not be permitted without medical sanction.

To avoid industrial dermatitis it is of prime importance to have a clean industry and a clean worker. Unnecessary contact between the worker and the dusts or liquids with which he works should be avoided. Dust extractors, ventilators, splash guards, washing facilities and provision of suitable ‘cleansers’ are important. Thought should be given to the method of drying the skin after washing, e.g. by hot air dryers and automatic individual towels. Clothing should protect as much as possible of the worker from dusts and liquids, and should resist and throw off dust and liquid and not absorb them or bring them into more intimate contact with the skin. In some cases a change of clothing is necessary, e.g. in powder factories or mines. The skin that must be exposed may be effectively protected by the use of appropriate barrier creams applied to the skin of exposed parts before starting work. These leave on the skin an invisible and imperceptible film which remains intact until the end of work when washing will remove the cream with the dust or liquid. Appropriate barrier creams are made to protect against almost all industrial hazards. If barrier creams are not employed the use of appropriate cleansers after work may be indicated, e.g. sulphonated castor oil with 2 per cent. wetting agent.

Attention is drawn to the danger of using such things as paraffin, turpentine, thinners, suds, abrasives, strong caustics, for cleaning the skin, or using any cleansers other than those recommended for the particular process.

Where these measures are employed care should be taken to receive and convey exact instructions concerning their use as they may be ineffective when improperly applied. It must be obvious that some individuals by reason of their sensitive skins or for other reasons are unsuited to certain trades and in such cases no protective measures will render them suitable.

Publications*:

Memoranda:

Prevention of Dermatitis. Factory Form 1863.
Prevention of Oil Rashes. Factory Form 296.
Dermatitis among French Polishers. Factory Form 1797.
Dermatitis in the Boot and Shoe Trade (Stock Rooms). Factory Form 1814.
Prevention of Industrial Dermatitis, with special reference to the use of Barrier Substances. Factory Form 330.
Prevention of Industrial Dermatitis: Dermatitis from Glues used in Aircraft Construction. Factory Form 331.

Cautionsary Notices:

Dermatitis. Factory Form 367. 2d. (3d.)
Dermatitis. Factory Form 397. 2d. (3d.)
Dermatitis. Caution to workers handling flour and dough or sugar. Factory Form 355. 2d. (3d.)
Effects of Lemon and Orange Peeling on the Skin. Factory Form 396. 1d. (2d.)
Effects of Chrome on the Skin. Factory Form 398. 1d. (2d.)
Dermatitis from Glues (Synthetic). Factory Form 366. 1d. (2d.)

* Memoranda obtainable from the Factory Department, Ministry of Labour, St. James’s Square, London, S.W.1. The Cautionsary Notices may be purchased from H.M. Stationery Office (prices in brackets include postage).

Experiments were made to determine the most severe environmental conditions in which men could maintain thermal equilibrium after the second hour of six-hour exposures. The experiments were made in men wearing shorts or army jungle uniforms in air-conditioned rooms with dry-bulb temperatures between 23*C. and 50*C. with various relative humidities. An air movement of 55 m. per minute prevailed during the experiments. Experiments were made with the man (a) sitting, (b) walking at easy pace on the treadmill, (c) performing moderate exercise in the treadmill. Observations were made on the elevation of the heart rate, rectal temperature, skin temperature and rate of sweating. Men walking in shorts with metabolic rates of 188 Cal./m.2 per hour maintained thermal equilibrium for six hours at 34°C. with 91 per cent. humidity and at 50°C. with 21 per cent. relative humidity. With metabolic rates of 136 Cal./m.2 per hour the men could maintain equilibrium at 35°C. with 96 per cent. humidity and at 50°C. with 32 per cent. humidity. With resting metabolic rates of 46 Cal./m.2 per hour men in shorts could maintain equilibrium at 50°C. with 98 per cent. humidity and at 50°C. with 34 per cent. humidity.


This bibliography has been prepared in response to demands from the medical profession, industry, and labour in the U.S.A., 'for a list of material on industrial hygiene.' The authors state that it does not attempt to be a complete list (nevertheless it is comprehensive and will be helpful to those engaged in industrial medical research).


Silver polishers inhale iron oxide from the rouge used on their work. Three workers were examined clinically and radiologically and a fourth who died, following a suspension of gastric ulcer, had a necropsy. In each case the skiagrams of the chest were comparable with that found in electric arc welders. The necropsy showed bulky, heavy lungs with bullous emphysema. There was pneumonitis for six hours to four weeks. Microscopical examination showed extensive acute broncho-pneumonic and well marked emphysema. There was no evidence of chronic inflammation and there was no fibrosis. The exudate in the alveolar walls that enclosed in phagocytic cells found in aggregates under the pleura, and along the pulmonary vessels, this was probably iron oxide; a fine granular pigmentation of the elastic laminae with the lower lung. Chemical examination of the lung showed that ash constituted 10-17 per cent. of the dry weight, and of this ash 72 per cent. was iron (reckoned as Fe2O3) and 6 per cent. was silver (as metal). The x-ray changes were considered therefore to be mostly due to iron oxide but in part to silver. X-ray pictures of normal sponges resemble a lung somewhat. They were soaked in graded suspensions of iron oxide (rouge), ranging from 5 to 60 grams in a pint of water. The excess fluid was squeezed out and they were again radiographed. Stippled shadows appeared in the films of these rouge-containing sponges, varying in density with the concentrations of iron oxide particles. This proved that iron oxide will cause these shadows in the x-ray. Little or no physical disability appeared to be caused to the men by the presence of the iron oxide dust in the lungs, though in one case there was emphysema. A worker


The best performance of fully acclimatized young men on a good daily diet, performing intermittent hard work in the heat, is achieved by replacing hour by hour the water lost in sweat. Any amount of water less than this leads to a matter of hours to several days and eventually to exhaustion. Six men were exposed to a dry heat of 100°F. 30 per cent. relative humidity and a moist heat 95°F. 83 per cent. relative humidity. They maintained their performance at 3-5 min. per mile and eventually rose from microbes introduced into the tissues at the time of injury, were due to Staph. aureus. Those that develop as open lesions, e.g. paronychia and large septic lacerations, commonly yield St. pyogenes. It is suggested that the majority of wound infections are due, in the first place, to Staph. aureus and that St. pyogenes is commonly added later to wounds already infected.
who has inhaled radio-opaque dusts may therefore have an x-ray picture of his lungs simulating silicosis or miliary tuberculosis, and yet have no obvious physical disability.


Eleven grinders were examined. The metal ground in the manufacture of bearings is chrome vanadium and chrome molybdenum tool steel containing about 98 per cent. chromium and not more than 0.2 per cent. silica. Artificial abrasive wheels composed of bakelite, silicon carbide (carborundum) and aluminium oxide (aloxite), have been exclusively used. The dust concentration averaged 4.1 mg/m3. In the air, of which 96.5 per cent. were less than 5 μ and 99.5 per cent. less than 10 μ. The average percentage of silica as quartz in the dust was 0.43 per cent. Four of the workers who had been grinders for 12, 13, 14 and 17 years showed x-ray changes of nodulation. One man showed a coincident pulmonary tuberculosis in the x-ray, though he had no clinical manifestations of active infection. It was considered that these were 4 cases of siderosis occurring in metal grinders.


Inhaled pure graphite dust can produce pneumoconiosis. Five cases are reported. The symptoms and signs may be strikingly mild, even in advanced cases. Case 1, aged 56, had worked with graphite for 20 years. He had cough, sputum and pain in the chest. Skilagram showed patchy infiltration throughout the right lung. Case 2, aged 63, had worked with graphite for 17 years. He had cough and sputum, several specimens of which contained no graphite. Skilagram showed a massive reniform opacity in the right upper zone with a similar but less well marked opacity in the left. Case 3, aged 58, had worked with graphite for 20 years. He had cough, sputum, and pain in the chest. Skilagram showed peripheral fibrosis in the right upper zone, with some nodules and increased linear striation. Case 4, aged 42, had worked with graphite for 18 years. He had no complaints, but a skilagram showed rounded opacities in both intraclavicular regions.


The existence of siderosis, or the deposition of iron in the lung, has recently been discovered in arc welders, though the occupation has not hitherto been considered to cause disablement. In order to investigate the question more thoroughly a group of 15 arc welders were examined. The metal used in the manufacture of bearings is chrome vanadium and chrome molybdenum tool steel containing about 98 per cent. chromium and not more than 0.2 per cent. silica. Artificial abrasive wheels composed of bakelite, silicon carbide (carborundum) and aluminium oxide (aloxite), have been exclusively used. The dust concentration averaged 4.1 mg/m3. In the air, of which 96.5 per cent. were less than 5 μ and 99.5 per cent. less than 10 μ. The average percentage of silica as quartz in the dust was 0.43 per cent. Four of the workers who had been grinders for 12, 13, 14 and 17 years showed x-ray changes of nodulation. One man showed a coincident pulmonary tuberculosis in the x-ray, though he had no clinical manifestations of active infection. It was considered that these were 4 cases of siderosis occurring in metal grinders.

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Memorandum on Welding Processes and their Hazards. (1945). LEEDS JOINT COUNCIL ON INDUSTRIAL MEDICINE.

1. Forge Welding. The blacksmith's principle of heat and pressure is used in certain modern electric welding processes such as spot welding, seam welding, projection welding, and butt and flash butt welding. In these no fluxes or slags are used and there is no added metal.

(a) Spot Welding. Used on fairly thin metal sheets.

(b) Seam Welding. The overlapping sheets are pushed through a pair of disc electrodes which gives a continuous linear weld instead of a series of single joints as in spot welding.

(c) Projection Welding. The machine is in the form of a press the flat plates of which are the electrodes. One of the sheets to be welded has small projections of metal welded on its under surface which form points of contact with the lower sheet. As the electrodes are brought together under pressure a series of spot welds are formed at the projections while the sheets are being pressed together.

(d) Butt and Flash Butt Welding. Used for joining lengths of heavy sections of steel end to end. The two pieces are held in electrodes in the form of a vice and as the ends are brought together an arc is struck and welding takes place.

2. Autogenous or Fusion Welding. A local melting and pooling of the metals to be joined takes place with or without the addition of molten metal from a filler rod or electrode.

(e) Gas Welding. Oxy-acetylene—propane—hydrogen—butane—coal gas. Filler rods, non-coated, usually the same but may be of different composition from the metal being welded. A flux may be used with some metals.

(f) Metal Arc Welding. Heat supplied by arc struck between metals to be joined and an electrode. Both parent metal and electrode melt and fill in jointing space. Electrodes are usually designed to deposit similar metal to that of parent metal being joined, and are coated by means of dipping, wrapping or extruding or a combination of these methods. The most commonly used method is (1) Stabilization of the arc. (2) Production of a flux and of a protective slag to cover the hot metal during cooling. (3) To add alloying elements to the weld metal. To provide a gas shield round the arc to prevent access of air to the weld metal.

Examples of electrode coatings: sodium silicate; iron oxides and silicates (e.g. haematite and kaolin); potassium aluminium silicates (felspar, mica, talc); blue asbestos, NaFe(SiO₃)₂; and fluor spar, CaF₂.

(g) Carbon Arc Welding. Carbon electrode carried in holder. Heat supplied by arc and, if added metal is required, filler rods must be used as in gas welding.

(h) Atomic Hydrogen Welding. Arc is struck between two electrodes carried in holder and a stream of hydrogen (or cracked ammonia) is blown into arc. This is in effect a special form of gas welding as the melting is due to the high temperature flame produced and not to the arc.

Hazards

Health risks are confined to the second group of autogenous or fusion welding processes.

(1) Electric Shock. Very small risk with D.C. at 50—100 volts. Greater danger with use of A.C. Factory Department recommend D.C. with voltage not exceeding 60. Nevertheless A.C. is being increasingly employed as for D.C. a rotary converter is required. Electric
shock at voltages employed may have severe effects though seldom fatal, but a shock to a welder may result in a fatal accident by causing a fall when working in dangerous positions as on scaffolding or in shipbuilding.

(2) Explosion risk in Gas Welding.

(3) Radiation Effects. (a) Infra-red—heat effects may give rise to cataract and burns. (b) Light radiation—painful on the eyes. (c) X-rays—on the teeth and bones. (d) U.V. radiation—serious effects on eyes. (d) U.V. radiation—serious effects on the skin. (e) Ionising radiations—casualties to the skin.

(4) Burns from hot metal thrown off from arc during welding.

(5) Eye injuries from chipping off slag from weld after cooling.

(6) Fumes. (a) Iron oxide is known to be deposited in the lung and to give a characteristic x-ray appearance in films but is not known to cause fibrosis or other ill-effects. (b) The exposure, and the rate of respiration of the individual. Increase in temperature, humidity, and CO₂ content of the air, or decrease in O₂ concentration, stimulate respiration and favour absorption of CO. The toxic effects of CO resulting in anoxaemia are due also to the CO haemoglobin further reducing the O₂ supply by interfering with the dissociation of oxygen-haemoglobin available. The formation of CO haemoglobin also results in a reduction of the available reduced haemoglobin and in this way interferes with the transportation and dissociation of CO₂. How far CO also reacts with other "heme"-containing pigments and enzymes needs further study. Concentrations of CO haemoglobin in the blood below 20 per cent. cause only moderate subjective complaints; these become serious with concentrations above 30 per cent. Data concerning the anaesthetic effect of carbon monoxide in the blood have been recorded in many cases of asphyxia.

(7) Heat effects and anoxaemia in confined spaces.

Precautions

D.C. under 60 volts. Ventilation—general and localized exhaust. Protective clothing, aprons, gloves, face shields and goggles. Gloves not only protect from burns but are the welder's main protection from electric shock. These are usually of leather and are protective when dry but are conductors when wet. A better material than leather is required but has not yet been discovered.

REFERENCES:


CO poisoning is characterized by the formation of CO haemoglobin. The amount of CO haemoglobin formed depends on the concentration of CO in the air, the duration of exposure, and the rate of respiration of the individual. Increase in temperature, humidity, and CO₂ content of the air, or decrease in O₂ concentration, stimulate respiration and favour absorption of CO. The toxic effects of CO resulting in anoxaemia are due also to the CO haemoglobin further reducing the O₂ supply by interfering with the dissociation of oxygen-haemoglobin available. The formation of CO haemoglobin also results in a reduction of the available reduced haemoglobin and in this way interferes with the transportation and dissociation of CO₂. How far CO also reacts with other "heme"-containing pigments and enzymes needs further study. Concentrations of CO haemoglobin in the blood below 20 per cent. cause only moderate subjective complaints; these become serious with concentrations above 30 per cent. Data concerning the anaesthetic effect of carbon monoxide in the blood have been recorded in many cases of asphyxia.


Di (2-ethylhexyl) phthalate has the trade name of 'Flexol' Plasticizer DOP. It is widely used in the production of flexible films and tubing. It is a light-coloured liquid, insoluble in water, but miscible with most organic solvents. Single doses of it (in a 1 per cent. solution) were administered by stomach tube to 130 rats and rabbits, and the animals were observed for 14 days following the dose. The lethal dose (for 50 per cent. of the animals) was calculated to be 30-4 g./kg. for rats and 33-9 g./kg. for rabbits. Chronic treatment with higher concentrations. Concentrations between 60 and 80 per cent. are dangerous to life. Analysis of the clinical picture and pathological findings indicates that the sequelae of CO poisoning are more serious than would be expected from mere anoxaemia of a similar degree; there may be a specific primary or secondary effect of CO on "heme"-containing pigments and enzymes, and possibly on the organism. In treatment, a prompt elimination of CO from the organism is vital: this is best done by administration of O₂, accompanied, if necessary, by artificial respiration. The elimination of CO₂ is facilitated by artificial respiration and the administration of O₂. The value of the principles of the dithizone method have been previously discussed by several investigators.


By the method to be described a concentration of HCN as low as 0-2 gamma in 10 litres of air (1 part in 50,000,000) can be measured. It has been used repeated for over a year with consistently satisfactory results. A measured air sample is bubbled through a solution consisting of three parts of 0-005 M Na₃HPO₄, 12H₂O and one part of reagent (made by adding 1 ml. of 0-5 per cent. phenolphthalein in absolute ethanol to 99 ml. of 0-01 per cent. CuSO₄.5H₂O solution). One part of 0-1 per cent. KOH solution is added, and the red colour which develops is measured after one minute with a photoelectric colorimeter. The HCN concentration is then determined by means of a curve prepared from readings of standard KCN solutions.

The phosphate buffer solution eliminates the effect of other gases in the air. Five tables of data are recorded showing the accuracy of the method under various conditions, and information is given about substances in the air which may interfere with the reaction.


This paper outlines the procedures necessary for the polarographic determination of atmospheric fume samples of lead, zinc oxide, cadmium, chromium and manganese. Substantially the same operations are performed for each of them, the method being a rapid one, suitable for routine determinations. Moreover, two or more toxic metals can be determined simultaneously. In general, about a cubic metre of the air is sampled by means of the modified Greenberg-Smith impinger or the electrostatic precipitator. The sample is treated with volatile acids and evaporated to dryness. The salt is taken up with a suitable volume of the proper supporting electrolyte solution, and the polarogram is recorded photographically. The concentration of fume is determined, and the fumes are not measured by the fume content of the polargram with a calibration curve prepared by photographing a series of standards. The sensitivity of the polarographic method for estimating lead fumes is considerably greater than that of the dithizone-iodometric method, though not so great as with the dithizone method. A number of technical details are given for sampling each of the fumes mentioned, but they are not suitable for abstracting. The principles of the dithizone method have been previously discussed by several investigators.

It was stated by Barthelmy, as the result of ten years' experience in the manufacture of viscose rayon, that no morbidty was observed if the CS₂ air content was kept below 30 p.p.m. and the H₂S below 20 p.p.m. Other investigators reported psychic symptoms, especially extreme irritability and insomnia. The present investigation was made over a period of four years on 100 individuals, of whom 78 were on a three-shift system and the remainder on day shifts. Air samplings were made regularly, and they showed a range of 1-26 p.p.m. of CS₂ (mean, 12.2), and of 1-0-5-5 p.p.m. of H₂S (mean, 2.5). The subjective symptoms indicated that 32 per cent. of all the workers suffered from fatigue at the end of the day's work, and that 26 per cent. of the shift workers were tired on awakening, as contrasted with 9 per cent. of the day workers. An occasional blurring of vision was experienced by 26 per cent. of the shift workers, and impaired appetite by 5 per cent. of them. The tendon reflexes were increased in 23 per cent. of the entire group, whilst corneal reflexes and hearing were in each case diminished in 16 per cent. of them. The investigators concluded that the symptomatology is vague and difficult to evaluate, and that there was not any sign or group of signs indicative of chronic intoxication.


Perchloroethylene degreasers of the non-condensing type are now being used which rely on electricity as a source of heat and upon a pair of electrically operated liquid and vapour thermostats to control their operation. Tests made by Morse and Goldberg on 11 machines of this type yielded vapour concentrations in the breathing zone of the worker averaging 221 p.p.m., as compared with 135 p.p.m. for conventional non-ventilated condenser-equipped trichloroethylene degreasers, and 96 p.p.m. for ventilated degreasers of the same type. The accepted maximum limit for trichloroethylene and perchloroethylene is 100 p.p.m., and even this figure may be too high. In the present inquiry fifteen perchloroethylene degreasing tanks of the non-condensing type were investigated, and air tests made at eight of these tanks showed that perchloroethylene in the breathing zone of the operator averaging 180 p.p.m. when objects such as large and small castings were being put in the tanks, and 484 p.p.m. when they were being removed. It is therefore recommended that for safe operation this type of tank be used only when (a) equipped with condenser and local exhaust ventilation, and (b) operation should be performed in a temperature, according to all other generally accepted standards for safe degreasing operations.


It is well known that ingestion of fluoride compounds in abnormal quantities generally results in the extensive storage of fluorine in skeletal tissues, and it is stated that endemic dental fluorosis (mottled enamel) may arise if the domestic water supply contains more than 1-0-1-5 p.p.m. of fluorine. The present investigation was made on five young men who spent eight hours each day of the five-day test periods in an experimental chamber which was either maintained at the 'comfortable' temperature of 84° F. and of 50 per cent. relative humidity, or at the 'hot-moist' temperature of 100° F. and 68 per cent. humidity. The daily food ration provided contained 0-5-0-9 mg. fluorine, together with additional supplements in food and water providing 3-2-5-0 mg. The fluorine excreted in the sweat and body washings, and in the urine and faeces, was determined, and it was found that the two contrasted sets of environmental conditions had a marked influence on excretion. Of the fluorine eliminated by the kidneys and skin, 77 per cent. of that absorbed in the 'comfortable' periods appeared in the urine and 24 per cent. in the perspiration, whilst in the 'hot-moist' periods the averages were 49 per cent. in the urine and 44 per cent. in the perspiration. The (largely visible) sweat collected in the chamber which contained 0-1 mg. of fluorine, while the insensible perspiration of the comfortable periods averaged 1-2 p.p.m. The absorption of fluorine from bone meal, cryolite and calcium fluoride (in food) as a general rule was less than 0-1 mg. fluorine in food or water, so it is evidently dependent on the solubility of the fluoride. The investigators found that the elimination of absorbed fluoride via the urine and perspiration is practically complete when the quantities absorbed do not exceed 4-0-5-0 mg. daily, so they consider that any drinking water which does not contribute more than 3-0-4-0 mg. daily is not likely to create endemic cumulative toxic fluorosis.


An investigation into the relationship of fluorine ingestion and height, weight and bone fracture experience of selected groups of 1458 high school boys and 2529 young adult men entering U.S. armed forces was carried out. The fluorine content of the water was obtained from 0 in Washington, D.C., and New Hampshire to 5-0 p.p.m. in Texas. Texas men averaged 69-6 in. in height and 149-0 lb. in weight, Washington, D.C., men averaged 69-3 in. and 151-2 lb., and those from New Hampshire 67-3 in. and 149-6 lb. These height-weight figures show no relation to fluorine exposure. Among high school boys the number of fractures per 100 boys varied from 21-3 to 32-4. At induction centres for the armed forces bone fracture experience was from 25-0 to 30-0 fractures per 100 men averaging about 15-25 years of age. While these data on bone fracture experience do not permit final conclusions, they do suggest that no serious impairment in skeletal performance, as might be manifest in the number of broken bones, seems related to exposure to fluoride domestic waters.


Pooled specimens of urine from men examined at U.S. army induction centres were examined. Those obtained from men coming from fluoride areas were compared with those from men from non-fluoride areas. In Washington, D.C., and New Hampshire, the fluoride content in domestic waters was 0, in Indianapolis 0-2 p.p.m. and in Hendricks County, Ind., 0-6-1 p.p.m., whereas in Amarillo, Texas, it was 4-3-5-1 p.p.m., in...
Acute and Subacute Toxicity of DDT (2, 2-bis (p-chlorophenyl)-1, 1, 1-trichloroethane) to Laboratory Animals.


DDT is acutely toxic by mouth to rats, mice, guinea-pigs, rabbits and chicks in doses ranging from 150 to 750 mg./kg. The symptoms produced in those animals which did not die consisted of anorexia, nervousness, sensitivity to stimuli and fine tremors. When given in small amounts in the diet (0.05 per cent. for rats and mice, 0.1 per cent. for guinea-pigs and 0.05 per cent. for growing chicks) for periods up to 20 weeks, signs of subacute toxicity arose. They consisted of a decrease in hyperexcitability, an improvement in drinking water and fluorine content of the drinking water. In Hendricks County the fluorine present in the urine is 1.0 p.p.m., in Amarillo 4.0 p.p.m., in Lubbock 4.2 p.p.m. and Joliet 1.0. The correlation between fluorine concentration in drinking water and fluorine content of urine suggests that the presumed hazard of cumulative toxic bone fluorosis surrounding certain water-borne sources of fluorine in the United States is greatly reduced by this relationship.

An efficient urinary elimination of fluorine appears to be characteristic of individuals residing in certain fluoride areas of the United States. The metabolic function of the kidneys is a normal function of the human body and seems characterized by a condition approaching metabolic equilibrium.


When dogs, rats and guinea-pigs were exposed to 54.1, 12.44 and 6.22 mg. of DDT per litre of air in a static atmosphere for 45 minutes, no toxic signs or symptoms resulted. In the aerosol containing more than 6 per cent. sesame oil mice tolerate concentrations of 6.22 mg. of DDT per litre of air. If there are higher concentrations of either, after a few hours increased excitability, tremors and clonic convulsions develop, and later death. The DDT contaminates the fur and enters the body through the animal licking the fur and by absorption through the skin. Daily exposure of monkeys and mice for 45 minutes over a period of 5 weeks to an intermittent concentration of 0.183 mg. DDT per litre of air caused no toxic effects. When the exposure was repeated three times daily for 4 weeks, no toxic effects were noted and mice showed a definite signs of poisoning. Exposure of two human subjects to 0.035 mg. of DDT per litre of air for 1 hour daily for 6 days, produced no toxic effects, nor did three times this concentration. DDT sticks firmly to any surface and therefore it was not possible to build up a higher concentration. Daily exposure for 3 hours over a period of 4 weeks to concentrations of 12.48 mg. of DDT dust per cubic metre of air caused no toxic effects in dogs. Daily insufflation of 100 mg. per kg. of pure DDT caused primary injury to the liver and kidneys and subsequent nervous manifestations resulting in tremors. The threshold of three dogs at a blood concentration of 100 mg. per kg. of pure DDT over a period of 7 weeks caused no toxic symptoms in dogs beyond a moderate increase in the daily volume of urine. Exposure of rabbits to 1 per cent. DDT heavy mist for 48 minutes daily resulted in irritation of the eyes and upper respiratory tract caused by the solvent. Pathological findings in animals exposed to high concentrations of DDT included fatty degeneration of liver and kidneys in mice, showers showed a slight degree of centro-lobar changes in the liver. Most of the animals showed a moderate degree of haemoderosis and the anterior motor neurones showed evidence of degeneration. The liver of one dog out of four which had symptoms of DDT poisoning showed a healing sub-acute yellow atrophy of the liver. One chemical and one spectrophotometric method for the determination of DDT are described. The X-ray diffraction pattern of DDT is also described.

The Percutaneous Absorption of DDT (2, 2-bis (p-chlorophenyl) 1, 1, 1-trichloroethane) in Laboratory Animals.


Powders containing DDT 5 per cent. and tale 95 per cent. applied to the skin produced no toxic symptoms. Thirty per cent. and 25 per cent. solutions of DDT in dimethyl and dibuthyl phthalates are absorbed through the skin and produced symptoms of anorexia, loss of weight, hyperexcitability, and nervous tremors at a dosage level of 9-4 c.c./Kgm. Higher dosages produced toxic symptoms of death and in some cases paralysed death after 30 days. Animals with the most severe symptoms showed a moderate polymorphonuclear leucocytosis. There were wide individual variations in susceptibility.


A man of 30 allowed small quantities of an acetone solution of DDT to evaporate on the back of his hand. He developed a feeling of heaviness and aching in all the limbs, with weakness in the legs and spasms of extreme nervous tension. After 3 weeks sleep became impossible, and after another 6 days involuntary muscular tremors occurred over the whole body. His recovery was slow and was not complete after one year.


The concentration of isopropyl alcohol was determined in blood and body fluids by distilling off the alcohol under reduced pressure into a solution of potassium dichromate in strong sulphuric acid. The reduced dichromate was estimated iodometrically. The disappearance of isopropyl alcohol from the blood stream of dogs after oral administration does not proceed at a constant rate and is not directly proportional to the blood concentration. It is dependent upon the functional efficiency of all excretory organs. High concentrations were found in saliva, urine and stomach contents. It is rapidly absorbed from the gastro-intestinal tract and is widely distributed throughout the body in less than two hours. The narcotic action is twice that of ethyl alcohol.


Tri-ortho-cresyl phosphate poisoning is described clinically and pathologically. More than one hundred patients have been examined in the late stage of the palsy; and material for histological examination was obtained from 36 patients. The pathology may be divided into neural and extraneural. The small arteries, particularly the capillaries and pre-capillaries of muscle had extremely narrow lumina because of thickening of all of the coats of the vessels. The intima was involved to a greater degree than the media and adventitia. The larger arteries were relatively normal. The picture is similar to thrombo-angiitis obliterans and two patients had to have an amputation of the thigh for gangrene of
ABSTRACTS

of acute feature.
of the spinal cord. There was moderate involvement of Goll's column, and a rim of comparatively light staining of myelin sheaths about the circumference of the cord, and a striking loss of anterior horn cells were the most marked changes. All of the small arteries of the viscera were affected by endothelial proliferation. One patient died of bulbar palsy. The rarity of this manifestation may be related directly to the limited amount of the exogenous toxin ingested. The single patient who died having taken 15 times the amount of the poison ingested by the ordinary patient. She died after 50 days, unable to talk or swallow. From the clinical standpoint poisoning from tri-ortho-cresyl phosphate resembles amyotrophic lateral sclerosis.


Seven casualties, three fatal, occurred from exposure to screening smoke following the explosion of smoke generators. One man developed cough with profuse frothy sputum, slight stridor and vomiting. Rhonchi were present. His temperature was 101.8°, pulse 135, and respirations 35. He later developed retrosternal pain and frothy salmon-pink sputum. Two days later blisters containing straw-coloured fluid developed on the ears and hands. The bronchitis resolved in five days, but three weeks later he developed stridor, followed by right basal pneumonia. In the middle of the next month he had marked oedema of arytenoids and ventricular folds with stricture of the glottic aperture. He was treated by tracheotomy and dilatation of the stricture by means of Hegar's dilators.


A man aged 45 swallowed a quantity of furniture cream by mistake at 8.30 a.m. He vomited half an hour later. At 3 p.m. he was drowsy and had marked cyanosis of the lips and conjunctivae. Later 3 pints of blood were withdrawn and he was given a transfusion of 3 pints of Group O blood. The nitrobenzene content of the furniture cream was 5 per cent.


This article gives an account of blood changes found among 180 males engaged in the rotoevaporating industry. Before 1940 a mixture of toluene, xylene and benzene had been used as a solvent in the industry; during the years 1940–43, the use of benzene had to be increased in Sweden, owing to the shortage of xylene and toluene. After 1943 xylene was made available in large quantities. The author, however, does not know when to what extent benzene was substituted for xylene and toluene in the different industries. A diagnosis of chronic benzene poisoning was made when the blood picture showed definite leucopenia or thrombopenia. Sometimes anaemia was the only abnormal feature. Thirty-eight persons showed chronic benzene poisoning, while of the remaining cases there were symptoms suggestive of poisoning in 61 instances, while in 81 there had been neither symptoms of acute intoxication nor evidence of blood changes.

In this series anaemia was present in 25 of the cases, leucopenia in 35 and thrombopenia in 11. No counts showed lymphocyte values over 60 per cent. Eosinophilia was present in 3 cases. In 10 cases red cells showing punctate baso-haemophilia were observed in the peripheral blood. Sternal puncture was done in 6 cases. In each instance the specimen was rich in cells, erythro-poiesis was very active and leuco-poiesis was without any striking change lateral pyramidal tracts with degeneration most severe in the lower segments of the spinal cord. There was moderate involvement of Goll's column, most obvious in the upper segments. Anterior horn cells were numerically reduced. No abnormality was seen in brain stem or brain. Histological studies of one patient who recovered showed changes limited to the spinal cord. Slight demyelination of Goll's column, and a rim of comparatively light staining of myelin sheaths about the circumference of the cord, and a striking loss of anterior horn cells were the most marked changes. All of the small arteries of the viscera were affected by endothelial proliferation. One patient died of bulbar palsy. The rarity of this manifestation may be related directly to the limited amount of the exogenous toxin ingested. The single patient who died having taken 15 times the amount of the poison ingested by the ordinary patient. She died after 50 days, unable to talk or swallow. From the clinical standpoint poisoning from tri-ortho-cresyl phosphate resembles amyotrophic lateral sclerosis.


Dogs were subjected to repeated and prolonged inhalations of trichlorethylene vapour of known concentrations, simulating factory exposure. The exposures were made in a galvanized-iron cylinder fitted with a plate-glass top, and an air-trichlorethylene was drawn through it at the rate of 350 litres per minute. The 17 dogs used were exposed to air containing from 150 to 750 parts per million of the solvent for 5 or 6 days a week over a period of 2–8 weeks. Liver function was measured every 10 days by the bromsulphalein test, and it showed a progressive impairment. The amounts of bromsulphalein retained in the blood and 60 minutes after it was injected were larger than in normal dogs, and the retention was more pronounced when the exposure was more intense. The ability of the liver to conjugate chloral was also impaired, the chloral hydrate being administered by stomach tube. Microscopic sections of the liver taken from the dogs that died or were sacrificed during the period of intoxication showed depletion of glycogen and hydropic parenchymatous degeneration. Other signs of intoxication were anaemia, vomiting and diarrhoea.


A healthy man, aged 61, cleaned some trays with anhydrous ethylene chlorohydrin, and though he wore impervious rubber garments he suffered so much from nausea and vertigo that after about two hours' exposure he had to cease work. A few hours later he became cyanotic, with laboured breathing, and 9 hours after the exposure he died. Tests made at the breathing level of the man, when cleaning, showed that the air contained about 1 mgm. per litre of the solvent. On exposing six white mice to an atmosphere containing 1.2 mg. per litre of the solvent for 2 hours they all became ill, and one of them died. The others recovered and seemed normal 24 hours later. Evidently ethylene chlorohydrin is a highly toxic substance.


An account of three cases of poisoning, one of which was fatal, is given. Three men who had drunk heavily over the weekend worked on Monday in a room measuring 10 × 11 × 50 feet from 8.30 to 11.30 a.m.
Three men who had not had previous alcohol also worked in the room; these men developed no symptoms. They cleaned machinery with rags soaked in carbon tetra-chloride, and used 1-4 litres in the three hours. The smell was well marked. One man felt sick at 10 a.m. and he died; the second did not feel any symptoms until 6:30 p.m. that evening. The third who frequently left the chamber showed no symptoms until the following morning.

**Effect of 3, 4-Benzpyrene on Regenerating Epidermis of Mice.** 


Benzene hastened wound healing by intensifying epithelial proliferation in mice. The mitotic index was increased and the migration of cells into the defect was hastened as compared with control animals. Benzpyrene applied to regenerating epithelium for 5 days hastened healing to the same degree as benzene. The mitotic index rose to ten times normal compared with six times for benzene. If benzpyrene was applied for 4 weeks it was still active when the stimulus to regeneration had ceased. In spite of increased epithelial proliferation, the complete closure of wounds in benzpyrene-treated animals lagged behind that observed in the benzene-treated mice, although it was hastened as compared with wound healing in control animals.

**Influence of Bromobenzene on the Induction of Skin Tumours by 3, 4-Benzpyrene.** 


Batches of 30 mice were treated with benzpyrene and a mixture of benzpyrene and bromobenzene; and it was shown by local applications of bromobenzene to the skin four times weekly that the carcinogenic action of benzpyrene was inhibited and sometimes prevented. That the effect of bromobenzene is local was shown by applying the two substances to different parts of the body when no inhibitory effect took place. The effect is probably due to intermittent interference with sulphur metabolism. Bromobenzene is detoxicated by mercapturate formation in the skin before being excreted in the urine. Glutathione and ascorbic acid levels in the skin are quickly lowered by bromobenzene but recover in a few hours to normal levels.

**Effect of Sodium Citrate on Uranium Poisoning in Dogs.** 


Ten pairs of bitches were studied. One of each pair received intravenously 5 mg. of uranyl nitrate per kilogram of body weight, and the other uranyl nitrate intravenously, and sodium citrate, either orally or intravenously. All the dogs receiving only uranyl nitrate had severe tubular necrosis of the kidneys and died in renal failure. The blood urea rose to 360 mg. per 100 c.c. on the tenth day, the blood creatinine rose to 25.8 mg. per 100 c.c., while the serum chlorides fell to 61.2 milliequivalents per litre; anuria developed on the fifth day. The oral administration of sodium citrate afforded considerable protection against the uranyl nitrate. In these dogs, at the end of 10 days, the blood urea was 9-4 mg. per c.c., blood creatinine 1.2 mg. per 100 c.c., serum chloride 103.2 milliequivalents per litre and urea clearance 42.4. In the urine of 7 dogs given uranyl nitrate only the amount of uranium excreted during the first 24 hours was 15 per cent. of the amount given, while with 58 per cent. in the citrate treated animals. A brown precipitate of uranium was found in necrotic debris obstructing the lumens of the descending segments of the proximal convoluted tubules of the kidneys of the dogs having uranyl nitrate only, while little was found in the kidneys of the citrate treated animals. The protection of the kidneys from uranium poisoning by sodium citrate does not depend on correction of anemia, since the latter follows rather than precedes severe renal damage. It is probably explained by the fact that the citrate facilitates excretion of the poison, thereby reducing its injurious effect. A factor possibly related to increased excretion of uranium concerns the pH at which uranium salts cause precipitation of uranyl proteins. The approximate range lies between pH 7 and 7.

**Mule Spinners' Cancer and Automatic Wiping-down Motions.**


The most certain method of preventing the onset of spinners' cancer is to use only such oils for lubrication as are, as far as is known, non-cancer-producing. Only animal and vegetable oils come within this category, but these are not available at present. The price of such oils should not be a difficulty, for, if non-splash methods are adopted only small amounts need be used in the industry. Mineral oils having certain physical characteristics are found to be least carcinogenic. Such oils, conforming to a certain specification, are now available and are being used by many firms. The specification is given as an appendix to the report, and the authors strongly recommend that only oils conforming to this should be used for the lubrication of spinning mules, until such time as non-carcinogenic oils are again available. That there should be periodic medical examination of all persons engaged in mule spinning is another important recommendation: such examinations to be made every 6 months under conditions of privacy for the worker and the doctor; records are to be strictly confidential but H.M. Medical Inspector of Factories is to have access to them.

**Medical Examination of Employees in Hotels, Restaurants, and Other Food Places.**


An Order in Council, which became effective from July 1, 1944, in Saskatchewan, makes it 'compulsory for the employee of every hotel, restaurant, cafe, lunch counter, ice cream parlour and refreshment room where food or drink is sold to furnish annually to the owner or manager a certificate stating that he or she is not suffering from a communicable disease in a communicable state, and that he or she shows evidence of recent immunity to smallpox, diphtheria, and scarlet fever.' The Order applied to roughly 600 persons in Saskatoon, a town of 42,000.

The first step for each employee was a visit to the venereal clinic at the City Hospital for the presumptive Kahn test and smear. If the Kahn test was positive, it was confirmed by a Wassermann. Smears were taken from the vagina, cervix and urethra of all females, but no prostatic smears were taken from the males.

Following this, the employee received the vaccination for smallpox and the Schick, Dick and tuberculin tests. These were done in the one day. He or she was required to report the next day to have the Dick test read and the reaction to the Schick control noted; on the second day; for reading of the tuberculin test; and in about one week for the reading of the Schick test. The reaction to the smallpox vaccination was noted at each visit. Persons having positive Schick or Dick tests received the innoculations according to the 'usual practice.' Those with positive tuberculin tests had appointments made at the sanatorium for an x-ray.'

Up to date (March 1945) nearly 500 certificates had been issued and a brief analysis of the results of the first 338 examinations is given. No positive TB cases were found; four men and five women were found to have positive Wassermann and Kahn reactions and five women were found to have positive smears for gonorrhea.

The Saskatchewan Government has also passed similar legislation for medical examination of all operators in beauty parlours and barber shops, to be effective as from July 1, 1944.