Notes and miscellanea

Acute mercury pneumonitis

A. SEATON¹ AND C. M. BISHOP²

From the ¹Asthma Research Unit, Penarth, South Glamorgan, and the ²Health and Safety Executive, Employment Medical Advisory Service, Swansea, Wales

Although poisoning by mercury has been recognised since the time of Ramazzini (1713), inhalation of sufficient mercury vapour to cause toxic pneumonitis is still an uncommon industrial accident. We report one such accident, which affected four men repairing a condenser in a power station, and following which we were able to make serial physiological and toxicological measurements.

Description of the accident

Four contractors were sealing expansion leak joints inside the steam space of the low-pressure condenser of a turbogenerator at a power station. The space in which they were working measured 24 × 8 × 6 feet (≈ 7.2 × 2.4 × 1.8 m). The turbine was being cooled by a flow of compressed air, controlled by a valve across which was placed a Kent ST/4 universal industrial manometer containing about 200 ml of mercury.

At 3 00 pm on 12 October 1974 the control valve was adjusted to supply more cooling air, at a temperature of 90°C and a flow rate of 500 cu ft/min (≈ 14.2 m³/min). This caused mercury to be blown out of the manometer, vaporised and carried into the intermediate pressure cylinder which had walls at 260°C. Some of this air then passed to the low-pressure condenser in which the men were working at a temperature of 20°C. Although the loss of mercury was noted by the operator (and later established to have been approximately 100 ml), no action was taken at this stage because the spilled mercury was separated from the condenser by a 20 foot (≈ 6 m) vertical pipe and also because it was assumed that the air flow was vented to the outside. However, it was subsequently found that the entire

¹Present appointment, and requests for reprints to Dr Seaton, Director, Institute of Occupational Medicine, Roxburgh Place, Edinburgh EH8 9SU.

Received for publication 8 November 1977
Accepted for publication 14 February 1978

airflow was passing to the condenser, as the valves to the outside were not registering any flow. Twenty hours after the spillage a mercury level of greater than 2 mg/m³ was recorded in the condenser space where the men had been working. This level was the maximum recordable by the Draeger tube that was used. (The TLV for mercury is 50 µg/m³.)

Case histories

Patient 1 smoked 20 cigarettes daily but denied any history of cough, sputum or respiratory illness. At 4 00 pm, one hour after the mercury leak, he noticed tightness in the chest and shortness of breath. He continued working until 8 00 pm, by which time he felt ill, with increased dyspnoea, a paroxysmal cough, and anorexia. He went to bed but was unable to sleep because of fever, profuse sweating, tremor, restlessness and severe dyspnoea. He attributed his symptoms to oxygen lack through working in a confined space and did not seek medical advice immediately, but after a sleepless night he was still ill with dyspnoea, tight chest and cough and he therefore consulted a doctor. He was transferred to hospital that day, 30 hours after the initial exposure.

On admission to hospital he was short of breath at rest and would develop a paroxysm of coughing after taking a deep breath. His pulse rate was 100 beats/min, temperature 37.5°C and respiratory rate 30 breaths/min. Bilateral repetitive inspiratory crackles were present at the lung bases. No neurological or other abnormalities were found and the tremor had disappeared.

Initial investigation showed a total white cell count of 12.0 × 10⁹/litre with normal differential and an ESR of 41 mm/h. His blood chemistry and renal function were normal and the sputum did not contain any pathogenic organisms.

Patients 2 and 3 smoked more than 20 cigarettes daily and *Patient 4* smoked occasionally. All denied previous respiratory symptoms. All were working with Patient 1, but spent less time actually inside the condenser. Their histories were similar to those of Patient 1, although less severe. They developed cough, chest tightness, tremor, restlessness and dyspnoea 1–3 hours after the onset of exposure. Their symptoms kept them awake through the night, lasting between 12 and 15 hours, apart from some residual cough and chest tightness which persisted for about 48 hours. No physical abnormalities were found on admission to hospital 30 hours after initial exposure; chemical, renal and haematological tests were normal.

None of the four patients had any other recent

Table 1 Lung function tests in four workers exposed to mercury vapour

Patient no.	Date of test	*Results of lung function tests as percentage of predicted normal				
		PFR	FEV _{1.0}	FVC	TLC	T _{Lco}
1	15 Oct 1974	43	48	53	70	65
	18 Oct 1974	90	85	89	80	78
	22 Oct 1974	98	99	110	98	92
	25 Oct 1974	98	102	112	103	95
	29 Oct 1974	102	103	117	99	95
	29 Oct 1975	95	117	123	106	89
	6 Oct 1976	96	126	128	107	111
2	17 Oct 1974	89	73	82	75	73
	30 Oct 1974	91	76	84	77	76
	29 Oct 1975	90	75	85	78	66
	6 Oct 1976	85	75	78	80	69
3	17 Oct 1974	97	85	95	93	79
	29 Oct 1974	95	85	98	92	79
	29 Oct 1975	93	89	110	89	82
	6 Oct 1976	82	84	95	98	83
4	17 Oct 1974	88	93	95	89	114
	30 Oct 1974	83	93	95	84	114
	10 Dec 1975	93	99	97	84	100
	6 Oct 1976	89	92	93	82	103

*PFR = peak expiratory flow rate; FEV_{1.0} = forced expiratory volume in one second; FVC = forced vital capacity; TLC = total lung capacity; T_{Lco} = transfer factor for carbon monoxide.

exposure to mercury, either industrially, as medicine or as dental amalgam.

METHODS OF INVESTIGATION

Chest radiographs of all four patients were reported upon by one observer without knowledge of the history of the patient or of the date of the film.

Serial urine mercury levels were measured in duplicate in two laboratories, using a cold vapour technique and atomic absorption (Rathje, 1969). The urine samples were voided at home at the same time of day on each occasion and frozen in transit to the laboratories. Corrections for specific gravity were made in calculating the mercury levels. Serum mercury levels on admission to hospital were measured by the same technique.

Serial measurements were made of ventilatory capacity (McDermott dry spirometer), lung volumes (helium dilution) and transfer factor (single breath carbon monoxide test). Values have been expressed as a percentage of the predicted normal (Cotes, 1968).

MANAGEMENT

Patient 1 was treated with oxygen and prednisone (40 mg/day for one week, 20 mg/day for a second week) because of his respiratory difficulty at the time of admission. No patient was given specific antimercurial therapy, because of the time lag between exposure and admission to hospital. Patients 2, 3 and 4 did not receive any treatment.

PROGRESS

Patient 1 recovered completely in one week. His dyspnoea lasted four days, while his cough and chest tightness persisted for a week. Patients 3 and 4 remained symptom-free from the second day of hospital admission. Patient 2 developed dry cough and mild exertional dyspnoea which is still present two years after the episode. Nevertheless, he remains active and at work.

Results of tests

CHEST RADIOGRAPHY

The initial radiograph of Patient 1 showed irregular patchy opacities in both lower zones and diminution of radiographic lung volumes compared with subsequent films. The opacities cleared within one week and the volume increased over three weeks.

The initial radiograph of Patient 2 showed indistinct bilateral basal nodular shadows and Kerley B lines. Subsequent films showed no change up to two years later. No radiographs taken before exposure were available for comparison.

All radiographs of Patients 3 and 4 were considered to be normal.

PULMONARY FUNCTION

Results of these tests are given in Table 1. Patient 4, the only light smoker, had normal results throughout. Patient 1 showed restriction with low transfer factor initially, and also evidence of airways

obstruction. These values reverted to normal within a week. Patient 2 also showed slight restriction and reduction in transfer factor and these changes have persisted for two years, while Patient 3 had only a slight reduction in transfer factor, which subsequently improved.

SERUM AND URINARY MERCURY LEVELS

The results of measurements of urine mercury are shown in Table 2. Serum mercury measured on admission was 1.0 µg/litre in Patient 1, 2.0 µg/litre in Patients 2 and 3, and 20 µg/litre in Patient 3; 95% of the normal unexposed population have serum mercury levels of less than 30 µg/litre (International Committee Report, 1969). Whole blood mercury was not measured.

Table 2 *Mercury in urine of four workers exposed to mercury vapour*

Patient no.	Mercury in urine (µg/litre) at various times after exposure (Months)							
	0	1	2	3	5	6	8	9
1	10	—	66	88	36	30	—	0
2	20	380	54	96	—	36	—	0
3	20	108	—	74	46	26	26	—
4	270	102	—	38	28	24	28	—

The initial level of urine mercury excretion was high only in Patient 4, whereas the other patients showed peaks of excretion 1–3 months after the accident. Mercury was present in the urine of all patients in April 1975, six months after exposure. Mercury excretion in normal subjects is usually zero and rarely above 50 µg/litre (Jacobs *et al.*, 1964).

Discussion

Acute mercury vapour poisoning is an uncommon industrial accident. Nevertheless it has been recognised since 1713 and has been described in the extraction of the metal from the ore (Warren, 1930), the manufacture of tungsten and molybdenum wire (Lewis, 1945), the production of thermometers (Vroom and Greer, 1972) and the cleaning of tanks used for electrolysis (Milne *et al.*, 1970). In a domestic setting, attempts at alchemy and the use of mercury-containing paint on a stove have caused acute mercurial pneumonitis (Matthes *et al.*, 1958; Haddad and Stenberg, 1963; Hallee, 1969). All episodes reported have been associated either with exposure to mercury in an enclosed space, or with heating of metallic mercury, or with both.

The accident reported here seems to have been caused by an unfortunate combination of circumstances in an industrial setting where the possibility

of mercury poisoning would normally be considered remote. While a group of contractors was working in a confined space sealing joints, another group of quite separate workers was engaged in regulating the flow of cooling air into a system of chambers, using specially hired manometers. When mercury spilled from one of these manometers the only concern was whether the mercury would be lost, and because it had to ascend a 20 foot tube this was considered to be unlikely. The possibility that the mercury might be vaporised did not cause concern because it was initially thought that the airflow had an outside vent. Even when this was found not to be the case, and it was apparent that air was flowing directly to the condenser where the contractors were working, the possibility of dangerous amounts of mercury vapour reaching them was not considered. Only when the men complained of symptoms was it recalled that mercury had been spilled and it was then realised that the flow of cooling air at 90°C would provide ideal conditions to vaporise the metallic mercury and carry it directly to the condenser. Confirmation of the high levels of mercury was then obtained, 20 hours after the leak.

The symptoms described by our patients were identical to those in previous reports (such as that by Milne *et al.*, 1970), starting 1–3 hours after exposure and lasting up to one week. Our most severely affected patient, who was also exposed longest to the toxic atmosphere, initially showed a marked abnormality of lung function, consistent with involvement of both airways and interstitial lung tissue. Treatment with corticosteroids was followed by the return of lung function to normal within a week, with no recurrence of symptoms. In contrast another patient, whose initial symptoms were less severe, has developed a persistent dry cough and slight exertional dyspnoea; objective evidence of the presence of interstitial lung disease has been obtained from a restrictive pattern of his lung function. Whether this would have been prevented by administration of steroids is not known, but seems possible, considering that he was less exposed than Patient 1, and had similar smoking habits. No other known cause of interstitial fibrosis was identified in his case and it was thought that his condition was probably attributable to mercury exposure.

Jacobs *et al.* (1964) showed that, although 81% of 778 urine samples from people with no occupational exposure did not contain any mercury, levels up to 50 µg, and occasionally higher, were found. The initial urinary mercury levels of three of our patients could, therefore, have been regarded as normal. However, all these three patients subsequently showed a rise in urinary excretion to definitely abnormal levels, with a peak 1–3 months after ex-

posure, not returning to normal until 6–8 months later. The levels of excretion showed no relationship to the severity of symptoms in our patients. It appears that mercury, initially rapidly absorbed through the alveoli (Kudsk, 1965; Teisinger and Fiserova-Bergerova, 1965) is retained in the body tissues (Matthes *et al.*, 1958) and thereafter is excreted only slowly through the kidneys. While such a sequence of events has been described previously after exposure to organic mercurials (Dinman *et al.*, 1958), there has been no previous report of prolonged sequential studies after a single limited exposure to metallic mercury vapour. It is clear from our studies of urine levels, together with the low initial serum levels, that these measurements are not helpful and may even be misleading at an early stage in the diagnosis of mercury poisoning. However, it is possible that, had we measured whole blood mercury initially, we might have found high levels such as were reported by Berlin *et al.* (1969) in monkeys and rabbits after mercury vapour exposure. We were unaware of this work at the time that the patients were admitted.

The clinical and physiological changes shown by our patients are consistent with the known pathological effects of mercury vapour. Initially all four patients suffered from a general febrile illness with breathlessness and cough suggestive of acute toxic damage to airways and lung tissue. The physiological tests in Patient 1 supported this diagnosis, in keeping with the pathological changes described in fatal cases of both bronchial mucosal and alveolar damage (Matthes *et al.*, 1958; Tennant *et al.*, 1961). Subsequently Patient 2 developed physiological evidence of interstitial pulmonary disease, an outcome similar to that in a patient previously described, in whom lung biopsy showed a fine interstitial fibrosis (Hallee, 1969).

Our report draws attention to the need for vigilance whenever metallic mercury is spilt in a confined space, especially if it is hot, and to the need for anyone using manometers to be aware of the danger of mercury spillage. Furthermore, the progress of our patients suggests that early use of corticosteroids, even in those patients who appear to be only mildly affected, may prevent the progression of the acute syndrome into interstitial fibrosis.

We are grateful to the Director of the Central Reference Laboratory and Dr C. H. B. Binns of BP Chemicals for the mercury assays.

References

- Berlin, M., Fazackerley, J., and Nordberg, G. (1969). The uptake of mercury in the brains of mammals exposed to mercury vapor and mercuric salts. *Archives of Environmental Health*, **18**, 719–729.
- Cotes, J. E. (1968). *Lung Function*. Blackwell Scientific Publications: Oxford.
- Dinman, B. D., Evans, E. E., and Linch, A. L. (1958). Organic mercury. Environmental exposure, excretion and prevention of intoxication in its manufacture. *Archives of Industrial Health*, **18**, 248–260.
- Haddad, J. K., and Stenberg, E. (1963). Bronchitis due to acute mercury inhalation. *American Review of Respiratory Diseases*, **88**, 543–545.
- Hallee, T. J. (1969). Diffuse lung disease caused by inhalation of mercury vapor. *American Review of Respiratory Diseases*, **99**, 430–436.
- International Committee Report (1969). Maximum allowable concentration of mercury compounds. *Archives of Environmental Health*, **19**, 891–905.
- Jacobs, M. B., Ladd, A. C., and Goldwater, L. J. (1964). Absorption and excretion of mercury in man. *Archives of Environmental Health*, **9**, 454–463.
- Kudsk, F. N. (1965). Absorption of mercury vapour from the respiratory tract in man. *Acta Pharmacologica et Toxicologica*, **23**, 250–262.
- Lewis, L. (1945). Mercury poisoning in tungsten-molybdenum rod and wire manufacturing industry. *Journal of the American Medical Association*, **129**, 123–129.
- Matthes, F. T., Kirschner, R., Yow, M. D., and Brennan, J. C. (1958). Acute poisoning associated with inhalation of mercury vapour. *Pediatrics*, **22**, 675–688.
- Milne, J., Christophers, A., and De Silva, P. (1970). Acute mercurial pneumonitis. *British Journal of Industrial Medicine*, **27**, 334–338.
- Ramazzini, B. (1713). *De Morbis Artificum Diatriba*. Translation by W. C. Wright. University of Chicago Press, Chicago, 1940.
- Rathje, A. O. (1969). Rapid ultraviolet absorption method for the determination of mercury in urine. *Industrial Hygiene Association Journal*, **30**, 126–132.
- Teisinger, J., and Fiserova-Bergerova, V. (1965). Pulmonary retention and excretion of mercury vapors in man. *Industrial Medicine and Surgery*, **34**, 580–584.
- Tennant, R., Johnson, H. J., and Wells, J. B. (1961). Acute bilateral pneumonitis associated with inhalation of mercury vapor. *Connecticut Medicine*, **25**, 106–109.
- Vroom, F. Q., and Greer, M. (1972). Mercury vapour intoxication. *Brain*, **95**, 305–318.
- Warren, V. A. (1930). Toxicology of mercury. *Veteran's Bureau Medical Bulletin*, **6**, 39.