POISONING OF FIREMEN BY IRRITANT FUMES

CADMIUM AND SULPHONATED CASTOR OIL

BY

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Introduction

Fourteen members of the Fire Brigade were affected to varying degrees as the result of the inhalation of fumes from a small burning bench in one of the machine shops of a Melbourne firm manufacturing motor parts. One of them died.

Although it was not possible to determine with certainty the relative importance of the various ingredients of the fumes or smokes in producing the effects, the cases are of considerable interest from several points of view. The amount of material consumed in the fire was small in relation to the space in the machine shop, the severity and nature of the effects produced. Cadmium was found in the lungs of the fatal case, though there was no knowledge in the early stages of the investigation of any positive source of the cadmium. The period of exposure to the fumes was short, but these fumes included heat decomposition products of sulphonated castor oil.

The first object burned was a small wooden bench which carried a lathe for machining small bearings for motor cars. The bench was approximately 7 feet long by 3 feet wide and was made from hardwood (eucalyptus) and oregon pine.

The machine shop in which this bench was situated was on the first floor and approximately 100 feet long by 60 feet wide, and 12 feet high. Only the bench, a small area of floor beneath it, and the articles on it, consisting of a small box of motor car bearings (which will be referred to later), and a tin containing about 1½ pints of cutting-fluid, were burned. The cutting-fluid had the following composition:

Mineral oil	..	..	26 per cent.
Sulphonated Castor Oil	9.6	..	..
Oleic Acid	..	..	3.6
41 per cent. Caustic Soda			
Solution	..	..	0.8
Water	..	..	60

The bench, which stood about half-way along the shop and about 10–12 feet from one wall, was not completely destroyed.

The majority of the firemen, all of whom were experienced men, some with more than 30 years' experience in the fire brigade, and some of whom

were senior officers, considered that the effects of the fumes were different from those of fires to which they were accustomed and more severe than would have been expected from the size of the fire.

It was considered at first that the fire was a minor matter and one of the first men to enter the building approached the bench and endeavoured to put the fire out with a hand chemical extinguisher of the sodium carbonate-sulphuric acid type; but after a few minutes it was found necessary to retire from the shop and to attack the fire from outside the building by means of hoses through the first-floor windows.

The fire took place on April 27, 1945, but it was not until May 2nd that it was brought to the notice of the senior author as a result of the death of one of the firemen. It was thought at first from a description of the symptoms of the deceased and others that the cases were due to poisoning by cadmium fumes, as it was known that the firm concerned manufactured and used in their bearings soft metal alloys containing cadmium. However, it was soon found that cadmium-bearing metal had only been used in this machine shop on one occasion. This was in December 1944, when a small amount of cadmium had been melted in a furnace situated in a part of the shop partitioned off from the rest. The product of this melt was machined on two lathes at a considerable distance from the lathe bench which was burned.

The cadmium melting-plant of the firm was situated on the ground floor and in a separate building which was about 100 feet away from that in which the machine shop was situated. The melting-pots were adequately ventilated by a suction exhaust system, the duct of which discharged into the open air above the roof of the next-door building. A solid wall without windows was situated between this discharge chimney and the machine shop.

As there seemed to be no chance of cadmium fumes having been the cause of the symptoms, other sources were looked for. It was found that a can of cutting-fluid containing about one and a half pints of sulphonated castor oil had been burned in the fire. All metalwork on the machines in the shop was heavily rusted on the morning after the fire. The extent of this rusting, in the opinion of the fire brigade officials, was greater than could reasonably

be attributed to the water used to extinguish the fire. The windows of the shop, and of an office partitioned off from one corner of the shop, were heavily coated with a greyish deposit. Rubbings of this were secured and submitted to chemical and spectrographic analysis. Chemical analysis showed the presence of sulphate and traces of sulphite and cadmium. Spectrographic analysis showed the presence of iron, copper, tin, lead, and cadmium as major elements; and among minor elements antimony and nickel were found. The sulphate was probably due to sulphur trioxide caused by the heat decomposition of the sulphonated castor oil.

Spectrographic analysis of the ash from the lungs of the fatal case indicated the presence of tin, cadmium and nickel in excessive quantities. The amount of cadmium, was calculated as 4 milligrams for the two lungs.

Samples of urine were collected from cases 2-13 inclusive (about 500 c.cm. from each), concentrated, mixed, and evaporated to near dryness. The residue was tested spectrographically. A large sample of urine from case 14 (between 1 and 2 litres) was treated similarly. Unfortunately, owing to the time which had elapsed before the occurrence was brought to the author's notice, these samples were not collected until 5 days after the fire. No differences were detected between the elements present in these samples and those in the residue from normal urine treated similarly.

Although the early investigations did not reveal any source of the cadmium, it was subsequently discovered that a wooden box (about 20 × 10 × 10 inches), containing used bearings manufactured by other firms than the one in whose shop the fire occurred, was standing near the bench and was destroyed in the fire. A similar box of miscellaneous bearings derived from various makers was found; and from this, ten bearings were picked out at random and tested for cadmium. Two showed the presence of cadmium, the amount in the soft metal lining of one bearing being 96.0 per cent. There seems little doubt, therefore, that the source of the cadmium was the box of bearings which had been burned.

Case Reports

The fire had been going for a short but unknown length of time before the arrival of the fire brigade at 9.10 p.m. on Friday, April 27, 1945. All the men, with the exception of Case 1, who died, were questioned on Wednesday, May 2nd, and Thursday, May 3rd, by one of the authors (D.O.S.) who was notified of the incident on May 2nd. A number of the firemen who were concerned with the fire were subsequently present at another fire in a picture theatre on Sunday, April 29th, at 1 a.m. This second fire was fought entirely from the outside and from neighbouring buildings. Careful questioning indicated that there were no noxious fumes or smokes encountered on this occasion.

Any influence attendance at this second fire could have had on the effects of the first was due to the muscular exertion involved.

Case No. 1. Fatal. Aged 56 years. This patient felt unwell at the second fire on Saturday night. On the following Sunday he was short of breath and had pain in the chest. He felt very ill on the Monday while attending court to give evidence. He later had an haemoptysis and went to bed in the afternoon. He was seen by a doctor at 5 p.m. and again by another doctor between 9 and 9.30 p.m. He died at about 7 a.m. on Tuesday, May 1, 1945.

Post-mortem examination showed the following abnormal conditions:—

The heart cavities were dilated and contained fluid blood and a little red clot. The valve rings were dilated but the cusps were normal. The coronary arteries were thickened, atheromatous and narrowed. The aorta showed moderate atheroma. There were small haemorrhages in the visceral pleura of both lungs. The lungs were large, congested and showed an intense oedema in all parts. There were numerous haemorrhages in the mucous membrane of the trachea and bronchi. The mucosa throughout was congested and the lumen showed a little fluid. The liver was congested. The spleen was congested and firm. The kidneys were congested and firm. The brain and its membranes were congested. Death was due to pulmonary oedema following the inhalation of an irritant gas.

Case No. 2. Age 65 years. Arrived at the fire at 9.10 p.m. Entered the machine shop five minutes after the men who opened the shop. Symptoms were not felt at the fire. The fire was quickly subdued and this officer was back at Brigade Headquarters at 9.36 p.m. When taking off his boots he felt giddy and stayed on the floor to kick off his trousers. He then felt constriction in his chest—lack of power to take a deep breath, pains in his ribs, and icy coldness. He was shivering and his teeth were rattling. He was giddy while lying in bed. During the night he had aches in his thighs and back. In the morning he had intense thirst, but no appetite for food. Nausea, lack of appetite and cold shivers persisted until 2/5/45, together with shallow breathing.

Case No. 3. Age 53 years. Described the smoke on going into the machine shop as not thick. He immediately noticed a 'burning' sensation in his chest more intense than that caused by ordinary hot air. Intense uncontrollable coughing occurred almost at once. After reaching headquarters his breathing was restricted and painful, and he had intense thirst. He noticed marked retraction of the intercostal tissues at this time. He went to bed and had intense throbbing in the head. At 2 a.m. the pain in his chest had eased. He got up and fell to the floor with an attack of giddiness. He was sleepless, and later he again got up and retched. He felt faint on several occasions. At 7 a.m. he got up, but could not stand and went back to bed. Then he had pains in all his limbs, buttocks, and thighs, which lasted till 8 p.m. on Saturday, April 28, 1945.

Between 1 and 2 a.m. on Sunday he arrived at the other fire, and was on his feet the whole of Sunday. He had aches all over, no appetite, and a bad taste in his mouth on this day. He continued to have 'cold shivers' until the morning of 2/5/45, when all the symptoms had cleared up.

Case No. 4. Age 47 years. Entered shop at same time as case No. 2. The first symptom he noticed was a feeling of bells ringing in his ears, followed by a lack of strength in his arms. Later he had a feeling of constriction in his chest described as being 'not due to the density of the smoke.' At midnight he could not sleep—at 4 a.m. he drank about half a gallon of water. He had 'cold shivers' all night. On Saturday morning he could not rise from his bed, and had intense pain in the

spinal region for a few seconds. He had no energy all this day. He attended the other fire at 1 a.m. on the Sunday, and while there he 'retched' for 2 hours. After returning from this fire at 4 a.m. he was unable to straighten himself owing to the severe retching. He had a severe epistaxis. He was seen by a doctor at about midday on Saturday, and then complained of pain in the region of the bases of the lungs. Apparently there were no signs as he was not put off duty. On 2/5/45 his appetite was excellent but he still felt weak, and had a 'cold in the head' and dryness of his throat; the pains in his limbs had vanished.

Case No. 5. Age 40 years, had been 18 years in the fire brigade. He entered with case No. 3. He noticed symptoms as soon as he entered the machine shop. He stated that the smoke did not appear excessive in amount. It had a 'biting' effect and took his breath away. He did not feel any pressure or pain in his chest. He retired almost at once from the shop to the fresh air, when he immediately had a severe fit of coughing and felt nauseated. His breathing was then rapid and shallow.

He returned to headquarters at some time between 9.30 and 9.45 p.m., and sat in a chair until 2.30 a.m. as he had a similar feeling to when he had attacks of asthma as a small boy. He went to bed at 2.30 a.m. and slept propped up until 6.30 a.m. He had a throbbing headache throughout Saturday and Sunday. After breakfast and dinner on Saturday he vomited and he felt nauseated all day on Saturday and Sunday. This nausea lasted until the following Wednesday. He had no pains in the limbs but had pain in the epigastrium. On Wednesday he still felt light-headed if he tried to take a deep breath. He had been affected by smoke before but never to the same degree. He had not regarded the job as any worse from the point of view of smoke or fumes than some others he had attended.

Case No. 6. Age 55 years, had been 34 years in the fire brigade. This officer used a chemical extinguisher (sodium carbonate and sulphuric acid) on the fire. He described the smoke as being more pungent on the stairs leading to the first floor than at the burning bench, and stated that the smoke was very heavy. He had no symptoms while at the fire and was inclined to make light of the whole occurrence. On Saturday morning, at 6.45 a.m., he started to shave and found that he could not raise his hand to apply the shaving-brush to his face. He felt faint, sat down, and broke into a most profuse perspiration. His legs felt too weak to hold him. At about this time he started to feel soreness in the chest, his breathing being restricted. He had no pains in his limbs and no vomiting. He noticed that the veins of his arms and hands were extremely hard and felt sore. His head felt as though it were being squeezed. He had no thirst at any stage. On Tuesday he felt well in the morning, but very tired later in the day. On Wednesday he felt very miserable when he awakened. At this stage he still complained of shortness of breath on exertion, which lasted for 10 days after the occurrence.

Case No. 7. Age 30 years. This fireman was one of the first to enter the shop. He almost immediately noticed irritation of the throat and lungs—with difficulty in breathing—and a burning sensation in his chest. He vomited while at the fire. On returning to headquarters his throat and chest felt raw. The following morning and during the whole of the Saturday he had aches and pains in his back and soreness in his throat. He vomited again after breakfast. He had shivering attacks all day on Saturday. On Wednesday he stated that his chest was still sore, and he could not take a deep breath. Smoking was unpleasant.

Case No. 8. Age 31 years, had been 6 years in the fire brigade. He entered the shop with case No. 4.

He started to cough immediately, and could not get his breath and had to retire in a few moments. On the Wednesday he had pain in the chest on attempting to take a deep breath and his appetite was poor. For two days after the event he had aches all over body and limbs.

Case No. 9. Age 53 years. This fireman was one of the first to enter the shop, and he noticed that there was something other than wood smoke present in the smoke. He soon retired owing to irritation of his respiratory tract by the smoke, which made him cough. He experienced no further effects other than soreness in the throat and coughing. By midday on Saturday he had recovered.

Case No. 10. Age 29 years. This fireman went in at the start of operations and experienced no immediate effects, except a few coughs, but he thought there was something unusual in the smoke. He had been at fires in paint and oil works but the smoke and fumes in this case were different from those he had previously experienced.

At 3 a.m. he had an extremely severe headache, tightness in his chest and shortness of breath. He had difficulty in taking a deep breath owing to what was described as a 'cutting short' of the breath, but no actual pain. The tightness in his chest and shortness of breath persisted throughout the Saturday. On the Sunday these effects gradually grew less, and on the Monday he felt normal except for a dry, hacking cough.

Case No. 11. Age 51 years. This fireman went in at the start of operations and immediately noticed soreness and irritation in his throat and shortness of breath. He retired for a while and then returned to the machine shop and stayed until operations ceased.

About an hour after returning to the fire station he felt unwell; he had a feeling of pressure in the head, a very sore throat and rapid breathing. After a hot bath and a drink of hot milk and brandy he went to bed and slept soundly until 1 a.m. when he awoke feeling very hot and dry and then broke into perspiration. During the day he felt drowsy and tired. On Wednesday he still suffered from shortness of breath on exertion.

Case No. 12. Age 29 years, had been 3½ years in the fire brigade. This fireman was orderly to case No. 1. He stated that the smoke caused a choking feeling and tightness in the throat and chest. He retired after about two minutes and vomited twice, which he had not previously done at a fire. He had had a severe post-orbital headache on the right side since the fire, never having had a headache previously. The headache commenced while in bed on the Friday night and had continued since. On the Saturday morning he had an ache in the throat and chest. He was short of breath on exertion, for some days after the fire.

Case No. 13. Aged 27 years, had been 6 years in the fire brigade. He entered the shop at once on arrival. He stated that the smoke smelt like burning an aluminium or a magnesium incendiary bomb. In about 5 minutes he commenced to feel nauseated and started retching, and was short-winded. He considered the smoke had quicker effect than other smokes he had encountered. After returning to the fire station he had a drink of milk and vomited some of it. He went to bed feeling short-winded and with pain on deep breathing. In the morning (Saturday) his knees were stiff and painful and he could not bend them until they had been massaged. He had a headache for 24 hours after the fire. He felt very weak but attempted to play football in the afternoon, but had to have oxygen administered owing to his breathlessness; he could not run ten yards. He wisely decided to give up the attempt to play, although a second administration of oxygen was advised, apparently with the object of enabling him to carry on the game. At 10 p.m. on Saturday night he had a severe fit of coughing and choking when going to bed. He shook all over

and was unable to drink a mouthful of water. In spite of this he turned out to the second fire, and felt very tired on his return. On Sunday he lay down all day. If he attempted to get up he nearly fell over, feeling dizzy and coughing. He felt very nauseated, but could not vomit, and had no appetite. He could not walk upstairs without holding to the rail, and was very breathless. When seen on Thursday he was much better—his appetite was then normal, but he was still breathless on exertion. He was then in bed, where he had been kept for some days by his medical adviser.

Case No. 14. Age unknown, had been 15 years in the fire brigade. This fireman was a patient in the Royal Melbourne Hospital, having been admitted as a case of an atypical pneumonia. He stated that he did not notice any particular symptoms at the fire. After returning to the fire station he felt some tightness in his chest, but had practically no cough. He was sleepless and had a severe throbbing frontal headache on the Friday night. On the Saturday he had a headache all day, and shortness of breath on slight exertion.

He went to the second fire at 1.3 a.m. on Sunday, and was short of breath when climbing the ladders. The headache was not so severe on Sunday, but on Monday it was very severe again, and there was shortness of breath even while in bed. He was seen by a medical officer on Monday night and again at 8.30 a.m. on Tuesday, and was transferred to hospital. When seen on Thursday he was improving. His cough on occasions had produced blood-stained mucus. He eventually recovered.

Consideration of Various Possible Toxic Agents

Carbon Monoxide. Two rats were found dead on the floor of the machine shop on the morning after the fire, but were unfortunately discarded. Later, two small rats were found dead in a nest behind a partition. It is probable that they died as a result of fumes from the fire. They were somewhat decomposed, but their livers were examined spectroscopically for carbon monoxide. It was absent. While it is possible that some of the firemen did suffer some effects from carbon monoxide, the nature and time of occurrence of the symptoms indicated that this gas could not have played an important part in producing them.

Nickel Carbonyl. The inhalation of this vapour causes among other effects oedema of the lungs. The possibility that nickel carbonyl might have been responsible for the effects was considered. This substance is formed by the action of carbon monoxide on freshly reduced, finely-divided nickel at ordinary temperature. It decomposes at 180° C. Hence it is not likely that nickel carbonyl, even if formed, escaped from the region of the fire into the atmosphere of the room.

Nickel was present in the wipings from the windows in small amount as compared with the cadmium. The relative amounts of nickel and cadmium present in the bearings destroyed in the fire are not known. The white metal usually machined at the bench was composed of antimony 5 per cent., copper 3.5 per cent., nickel 0.5 per cent. and tin the balance, but the compositions of the miscellaneous bearings destroyed in the box are not known.

The melting- and boiling-points of cadmium

and of the metals, contained in the white metal machined in this shop, are as follows:—

	M.-P. ° C.	B.-P. ° C.
Cadmium	320.9	767
Cadmium oxide.. ..	—	1000 (Decom- poses)
Copper	1083	2300
Antimony	630.5	1380
Tin	231.85	2260
Nickel	1452	2900

The presence of nickel in small amount as compared with the respective amounts of iron, copper, tin, lead, and cadmium in the window wipings is in accordance with what might be expected from the boiling-points of these metals.

Nickel was, however, detected in the lungs of the fatal case. It may have been inhaled in the form of finely-divided nickel, or its oxide. There is no available evidence (International Labour Office, 1938) of any acute effects on the lungs due to the oxide of nickel, although workers engaged in handling this compound have been exposed to this dust in considerable amount. While there is no certainty that nickel carbonyl was not the cause, or at any rate, a contributing cause of the effects experienced, it is considered unlikely that this substance could have been present in the air inhaled by the firemen.

Antimony. In the fatal case no antimony was detected in the lung. Since death took place several days after exposure, antimony may have originally been present and subsequently removed and distributed to other organs, or eliminated. There appears to be no evidence in the literature that exposure of considerable numbers of people to inhalation of antimonial dust causes acute lung damage.

Copper and Tin. In regard to these metals or their oxides there is again no evidence of acute lung damage to people who are exposed to fumes or dusts, as in bronze casting. Brass founders' ague or 'metal fume fever' is stated by Legge (1934) to be due to the zinc oxide fumes produced in the casting of brass. Casters of bronze, an alloy of copper and tin, do not suffer from this malady.

Impregnants in the Wood. The possibility that smoke and fumes may have contained toxic substances, such as fluorine compounds derived from sodium fluo-silicate used to impregnate the wood was investigated. It was found that no impregnants had been used in the wood from which the bench was constructed, so this possibility can be excluded.

Cellulosic Materials. The possibility that the effects may have been caused by the products of the

thermal decomposition or combustion of wood was considered. The conclusion that this was unlikely was based largely on the experience of the firemen. As already pointed out, these were all men experienced in fighting the varied kinds of fires which occur in a large city containing many manufacturing establishments, and some with over 30 years' service. Numbers of them had been previously affected by smoke on one or more occasions, but the opinion was very general that there was 'something different' about the effects of the smoke on this occasion.

Easton (1945) discusses the effects produced by the vapours from burning cellulosic materials. He reports two fatal cases of pulmonary oedema due to inhalation of smoke from a burning mattress in a prison cell and a bed respectively, and suggests that in the case of the Cocanut Grove fire, in Boston, which killed 492 and seriously injured 100, on November 28, 1942, certain non-thermal respiratory injuries were caused by irritants in the inhaled smoke which was derived chiefly from wood, cotton, and other cellulosic materials. While admitting that smoke from the burning wood may have contributed to the effects produced in the present series of cases, it is felt that other substances played a greater part.

Cadmium

A considerable number of cases of poisoning by inhalation of cadmium fumes have now been reported. Legge (1934) refers to a fatal case due to the melting of cadmium ingots in an inadequately ventilated lean-to shed, and to 2 cases due to welding in a small oil tank with electrodes containing cadmium. Prodan (1932*a, b*) has discussed the history of cadmium poisoning and has carried out experimental work on animals which will be referred to later. Bulmer, Rothwell, and Frankish (1938) report 1 case due to welding cadmium-plated levers in a large open shop, and 14 cases, including two deaths, due to annealing of cadmium-plated rivets in a large room. Spolyar, Keppler, and Porter (1944) report 5 cases, including one death due to the heating of cadmium-plated pipes in an open shop. Ross (1944) reports 23 cases due to ignition of cadmium dust. Wood and canvas filter frames were also burnt, so that the exposure was not only to fumes of cadmium oxide. There were no deaths in this series of cases, but several were off duty for periods from 4 weeks to 2 months. United States Public Health Service Reports (1942) discuss cadmium poisoning and list 58 cases in industry due to inhalation, with eight deaths.

From a survey of the literature it appears that the lethal dose of cadmium for man by inhalation is not known. The minimum lethal dose for man by swallowing is not known either, but Lewin (1929) reports that 30 milligrams of cadmium sulphate (equivalent to 16 milligrams of cadmium) by mouth caused increased salivation, choking attacks, persistent vomiting, abdominal pain, diarrhoea and

tenesmus, and Burdach (1827) produced nausea and vomiting by 33 milligrams of the sulphate (equivalent to 18 milligrams of cadmium).

In view of these figures the finding of 4 milligrams of cadmium in the lungs of the fatal case, in which death occurred several days after exposure, must be regarded as of considerable significance. Prodan (1932*b*) exposed cats for 30 minutes to air containing cadmium fume. One cat was killed 5 hours after exposure and determinations of cadmium in its organs were made. The other cat with similar exposure, died 12 hours after exposure ceased. From the amount of cadmium found, the respiratory rate and volume, and assuming 25 per cent. retention of cadmium, it has been calculated that the concentration of cadmium in the air was 1.2 milligrams per litre. Cats exposed for 24 hours to a concentration which has been calculated to have been 0.0165 milligrams per litre did not die, but sacrificed animals showed very severe damage to the lungs.

Estimated Concentration of Cadmium Fumes. Basing our assumptions on Prodan's work with cats, it is assumed that 30 per cent. of the total cadmium retained was found in the lungs. Drinker, Thomson and Finn (1928) have shown that about 50 per cent. of inhaled zinc oxide fume is retained in the body. Using these assumptions, and assuming a reasonable breathing rate, and knowing the approximate time spent in the harmful atmosphere by the fatal case, it has been calculated that the concentration of cadmium in the air breathed was of the order of 0.14 milligrams per litre (140 milligrams per cubic metre), i.e. fourteen hundred times the permissible limit for continuous exposure. The actual amount was probably higher than this, since the calculation is based on the lung content at death, which did not take place until several days after exposure had ceased. Further, the figures for retention refer to the overall retention in the respiratory tract and do not distinguish between the amount retained in the lungs and that retained in the naso-pharynx and trachea and that swallowed.

Another estimate of the concentration of cadmium fume is based on the probable weight of cadmium volatilized (2 lb.) and the volume of the shop (72,000 cu. ft.). This gives 440 milligrams per cubic metre, assuming no air changes. In the cases reported by Bulmer 7 lb. of cadmium were volatilized into a space of 32,890 cu. ft. during a period of 90 minutes. Assuming six air changes in this time the concentration would be 562 milligrams per cubic metre. It is not suggested that there was uniform distribution of the cadmium fumes throughout the air space in either of the above cases, but the figures are given merely to afford a rough measure of comparison.

The fire was of short duration, lasting about an hour, and occurred after working hours; the doors and most of the windows were shut. The number of air changes, therefore, would have been less than would occur during working hours. If there were three air changes during the period which

elapsed from the commencement of the volatilization of the cadmium and the entry of the firemen the concentration may have been of the order of 150 milligrams per cubic metre. This compares satisfactorily with the concentration deduced from the lung content.

Discussion of Symptoms

Spolyar, Keppler, and Porter (1944), from a review of the literature in regard to 43 cases of cadmium poisoning, compiled useful tables of the frequency of symptoms and the time of their onset. In the following table their compilation is compared with

TABLE 1

Symptoms	Median Time of Occurrence of Symptom		Average Time Present investigation
	Spolyar, Keppler and Porter (1944)	Present investigation	
Irritation of throat	4-8 hours	Few minutes	Few minutes
Headache and dizziness	6-10 hours	5 hours	5½ hours
Chills	10 hours	3 hours	5 hours
Vomiting	10 hours	6 hours	7½ hours
Cough	20 hours	5 minutes	8 hours
Pain in chest	24-36 hours	2-9½ hours	9 hours
Dyspnoea	30 hours	1½-6 hours	7 hours
Nausea		10 minutes	3½ hours
Weakness		10 hours	9½ hours
Diarrhoea		—	—
Aches in limbs		10 hours	9 hours
Thirst		7 hours	5½ hours
Pain in epigastrium		—	—
Smoking unpleasant		—	—
Tightness in Chest		½ hour	1½ hours
Retraction between ribs		—	½ hour
Fainting		8 hours	8 hours

The most striking differences indicated by this Table are the much more rapid onset of irritation of the throat, of cough, and of dyspnoea in the present series of cases.

TABLE 2
THE PERCENTAGE DISTRIBUTION OF SYMPTOMS

Symptoms	Spolyar, etc. (1944)		Present Investigation		Ross's (1944) Cases		
	No. reported	Per cent.	No. reported	Percentage of 13 cases unless otherwise stated	Symptoms	No. reported	Percentage of 23 cases
Irritation of throat	22	51	6	46	Constriction of throat	6	26
Pain in chest	22	51	8	57 (14 cases considered)	Headache and vertigo	4	17
Headache and dizziness	14	32	9	64 (14 cases considered)	Cough	9	39
Cough	13	30	7	50 (14 cases considered)	Dyspnoea	21	91
Dyspnoea	11	25.6	12	85 (14 cases considered)	Gastric pain	16	70
Vomiting	10	23.2	6	46	Nausea	16	70
Nausea	9	21	3	23	Vomiting	14	61
Chills	7	16.3	5	39	Ague or fume fever	16	70
Weakness	5	11.6	6	46	Prostration	16	70
Diarrhoea	3	7	—	—			
Aches in limbs	—	—	6	46			
Thirst	—	—	3	23			
Pain in Epigastrium	—	—	1	8			
Smoking unpleasant*	—	—	1	8			
Tightness in Chest	—	—	5	39	Præcordial constriction	21	91
Retraction between ribs	—	—	1	8			
Fainting	—	—	2	15	Pneumonia	1	4
					Irritation of eyes	2	9

* In Spolyar's (1944) own cases one complained of smoking being unpleasant, but the rest were non-smokers.

the results found in regard to the 13 firemen concerned in the present investigation. (The details of the symptoms of the fatal case are not included.)

In Bulmer's cases, which are included in the tabulation by Spolyar and his colleagues, there were, however, some individuals who suffered from irritation of the throat after only 10–30 minutes exposure, but none who suffered from this symptom after such a short exposure as produced it in our cases.

In Bulmer's (1938) cases, tightness in the chest was reported in 40 per cent., irritation of throat in 60 per cent., dyspnoea in 33 per cent., and cramps in 13 per cent. of the 15 cases. These cases were probably included in the survey by Spolyar, Keppler, and Porter (1944).

Of a group of individuals exposed to exactly the same risk, which is not rapidly fatal, not all will suffer with the same kind of symptoms nor will they be of the same severity. Where the risk differs for different individuals in the group one may expect wide differences in the percentage distribution of symptoms suffered by one group when compared with those suffered by another group exposed to different risks.

The number of subjects in the present group is small, but with this reservation it appears that there was considerably higher incidence of dyspnoea, chills and weakness than were noticed in the cases quoted in Spolyar's (1944) survey, and there was a higher incidence of dyspnoea than in Bulmer's (1938) cases. In Ross's (1944) series of cases the percentage of those who suffered from dyspnoea, gastric symptoms, and weakness was much higher than in the cases reviewed by Spolyar. This may have been due to greater concentration of cadmium fumes, or to the presence of other substances, such as the decomposition products of wood and canvas.

Evidence has already been adduced to show that the concentration of cadmium in our cases was

probably not as high as occurred in Plant 2, as reported by Bulmer (1938). These differences in percentage incidence of symptoms and particularly in the time of onset of symptoms may, we think, be attributed to the presence of other irritating agents than cadmium in the air. In the cases reported by Bulmer (1938) and by Spolyar, Keppler, and Porter (1944), there appears to be no doubt that the toxic agent was cadmium oxide fume, and nothing else. In the present series of cases there were, in addition to the metallic compounds, thermal decomposition products of wood and of sulphonated castor oil.

It would appear that the fatal case was exposed to a concentration of cadmium oxide fumes intermediate between that which was fatal to cats after a half-hour's exposure, and that which caused severe lung damage to cats by an exposure for 24 hours.

Table 3 summarizes the data on cadmium exposure in this case, in some of the cases reported by Bulmer (1938), in the experimental results of Prodan (1932*b*) and Otto (1925) and the guinea-pig experiments reported later in this paper.

Cat 4 in Prodan's (1932) series, which weighed 1·7 kilos, showed 0·39 mg. of cadmium in the lung, 5 days after exposure ceased. This amount was not fatal, and would correspond to 16 mg. for a 70-kilo man, and compares with 4 mg. in the case described here. There is the possibility that the amount of cadmium found in the lungs of this man may have resulted from a shorter exposure to a higher concentration. This may be more harmful than a longer exposure to a lower concentration.

Animal Experiments

Two guinea-pigs were also exposed to cadmium oxide fumes obtained by heating in a silica tube a small amount of cadmium scraped from the motor bearings (96 per cent. cadmium). Air was passed at a known rate through a silica tube in which

TABLE 3

Subject	Concentration (Determined, or estimated approximately) (Milligrams per litre)	Time (Minutes)	Results
Cat 1 (Prodan)	1·2	30	Death
Cat 4 (Prodan)	0·018	1440	Severe lung damage
Cat (Otto)	2·0	15 (on 4 occasions)	Death
Guinea-pig	2·0	60	Death
Guinea-pig	0·4	60 on two occasions	Death
Man, Bulmer's Case 1	>0·56	90	Death
Man, Bulmer's Case 2	>0·56	90	Death
Man, Bulmer's Case 3	>0·56	30	Affected seriously
Man, Bulmer's Case 4	>0·56	10 + unknown time	Affected in less than 10 minutes. Total exposure caused serious effects
Man, Bulmer's Case 5	<0·56 (50 feet away from furnace)	20 + unknown time	Affected in less than 20 minutes. Total exposure caused serious effects
Man, Bulmer's Case 6	<0·56 (75 feet away from furnace)	5	Affected seriously
Man (present case)	0·14	20·	Died.

cadmium bearing fittings were heated, gently at first, and then at bright red heat. The approximate concentration was determined from the air-flow, and the original and final weight of the cadmium.

Guinea-pig 6 was exposed for 60 minutes to a fume concentration of 2 mg. of cadmium oxide per litre. The effects were excessive salivation, and marked increase in respiration rate, but otherwise the animal appeared normal. On removal from the exposure the increased respiration rate was maintained for at least 1–2 hours, subsequently decreasing slightly. Death occurred within 24 hours. Necropsy showed the lungs to be congested and haemorrhagic, and a blood clot was in the trachea. The kidneys were enlarged, but the other organs appearing normal. Microscopically the trachea showed patches of erosion and traces of blood clot. The lungs showed large areas of emphysema, intense congestion with blood, extravasation of blood into the alveoli, fibrinous exudate in the bronchioles,

Lungs. G.-P.6. Exposed to fumes of cadmium oxide.

and a large area of red hepatization. The kidneys gave evidence of glomerulo-tubular nephritis shown by cloudy swelling in the tubules and loss of cellular outlines, congestion, and necrosis in some areas. The glomeruli showed congestion in the capillary tufts and destruction of the lining epithelium.

Guinea-pig 7 was exposed for one hour on two successive mornings to a fume concentration of 0.4 mg. of cadmium oxide per litre. During the first exposure only a slight increase in the respiration rate occurred, continuing after removal for 2 to 3 hours, and being approximately normal 24 hours later. During the second exposure the increase in the respiration rate was more marked, was maintained for a considerable time after removal from the exposure, and was faster than normal 24 hours later. Death occurred within the next 48 hours.

At necropsy the organs appeared normal. Microscopically the lungs showed widespread thickening, intense congestion and rupture of the alveolar walls, emphysema, and destruction of alveolar epithelium. In large areas the alveoli and

bronchioles were blocked by fibrinous exudate. The kidneys showed cloudy swelling, and blockage of the lumen of tubules by cellular debris and fibrinous exudate, and necrosis. The glomeruli showed engorged capillary tufts, and necrotic epithelium. In some cases the lumen was completely blocked, and in others the capillary tuft was

Kidneys. G.-P.7. Exposed to fumes of cadmium oxide. The circular dots in 2, 4, 5, 6 are artefacts.

small and contracted. In some parts the glomerular structure was completely disorganized. There were patches of haemorrhage. The condition was a severe glomerulo-tubular nephritis. Table 4 summarizes the results of these animal experiments.

Effects of Decomposition Products of Sulphonated Castor Oil

At present the thermal decomposition products of sulphonated castor oil or of castor oil are not known. They include sulphur trioxide in the case of the sulphonated oil, and probably include acrolein. It has been suggested that under certain conditions alkyl sulphates, such as dimethyl sulphate, are formed. All these substances are irritant to the respiratory tract and may produce oedema of the lungs. It is well known that acrolein is a lachrymator as well as a lung irritant—the lachrymatory effect being produced by concentrations of 0.007 milligrams per litre. Ten minutes exposure to 0.35 milligrams per litre is lethal. Dimethyl sulphate is about as toxic as phosgene, and also is a powerful irritant of the conjunctivae. It is readily hydrolysed, forming sulphuric acid.

While these substances may have been present in the atmosphere, none of the firemen complained of irritation of the eyes, or of lachrymation. It might be argued that this was due to the fact that the firemen were so used to lachrymation and eye irritation caused by smoke that they hardly thought them worthy of comment. However, enquiries elicited the fact that lachrymation and eye irritation were noticeably absent or less than would have been expected from the amount of smoke.

In order to determine the effects of the products of thermal decomposition of sulphonated castor oil, guinea-pigs were exposed to the vapours produced

by heating small amounts of (a) the cutting-fluid, which is a dilute emulsion of sulphonated castor oil, (b) undiluted sulphonated castor oil, and (c) medicinal castor oil. Animals were exposed in a 15-litre bottle to air containing fumes let into the chamber by a side-arm from a test-tube in which a known quantity of the oil was heated. Air was drawn at a known rate, 0.7 litres per minute, over the surface of the heated liquid. In two cases the fumes were passed into the exposure-chamber through a silica tube, a few centimetres length of which was heated to about 500° C.

Guinea-pig 1. This was exposed during a period of 30 minutes to fumes caused by heating 10 c.cm. of the emulsion, the air being passed through a heated silica tube. During the period 0–15 minutes the air contained mainly water vapour and sulphuric acid mist, the fumes from the decomposed oil being evolved mainly during the 15–30 minute period. Considerable condensation of water vapour in the exposure chamber occurred. This exposure was repeated on three successive mornings. The first exposure caused sneezing, coughing and irritation of the eyes and nose and slight increase of respiratory rate. Recovery after change to fresh air was fairly rapid. The second exposure caused similar effects but they were more pronounced, and recovery was slower, the animal being very sluggish next morning.

The third exposure caused very pronounced effects; the gait was staggering, and respiration was slow, deep, audible, and sometimes of the Cheyne-Stokes type. The head was retracted and fore-feet stretched out during inspiration. The hind legs were partly paralysed. Death occurred half an hour after exposure ceased.

Necropsy showed the lungs to be emphysematous, with some patches of congestion. Microscopically, they showed intense congestion, destruction of alveolar epithelium, serous exudate, and emphysema. The epithelium of the bronchioles was necrotic. The pleura was thickened and oedematous.

Guinea-pig 2 was exposed to fumes from 2 c.cm. of heated sulphonated castor oil during thirty

Lungs. G.-P.2. Exposed to fumes from heated sulphonated castor oil.

minutes. The effects were sneezing, coughing, which occurred almost at once, and irritation of the nose and eyes. Respiration soon became rapid and shallow but later it became slow and deep. Difficulty in standing developed, and toward the end of the experiment the animal lay prostrate. The exposure chamber was then filled with fresh air, the animal's condition improved rapidly, and respiration was normal in about half an hour. Death occurred within 24 hours.

Necropsy showed the lungs to be congested and oedematous. Microscopically they showed intense congestion, destruction of the alveolar walls, serous exudate, and emphysema. The bronchioles showed necrosis of the epithelium. The pleura was thickened and oedematous in extensive patches.

Guinea-pig 3. This animal was exposed during one hour to air containing fumes from 1 c.cm. of heated sulphonated castor oil. The air was drawn through an asbestos-packed tower which absorbed most of the sulphur trioxide but allowed the fumes of the decomposing oil to pass. The first effects were irritation of eyes and nose, excessive salivation and very rapid respiration. Later it became slower, deeper and distressed, with some suggestion of Cheyne-Stokes breathing. There was loss of power in the hind limbs. When the jar was filled with fresh air the animal's condition improved slowly; but in the morning it was found dead.

Necropsy showed the lungs congested and there was a blood clot in the trachea. The brain showed some congestion; but other organs appeared normal. Microscopically the effects in the lungs were similar to, but more severe than, those seen in animal 2. There were patches of haemorrhage.

Lungs. G.-P.4. Exposed to fumes from heated castor oil.

The epithelium of the trachea was swollen and degenerated in places.

Guinea-pig 4. This animal was exposed for 60 minutes to fumes caused by the heating of 1 c.cm. of pure medicinal castor oil, the contaminated air being drawn directly into the exposure chamber. The effects were extreme irritation of the nose and eyes, increased salivation, slow, deep, and slightly

convulsive respiration, and marked physical activity. After filling the chamber with fresh air the animal became quieter and respiration gradually improved and was nearly normal after 5 hours. Next morning the animal was still alive but sluggish. It was again exposed to the same conditions. The symptoms during this second exposure were more severe—the respiration was distressed, deep and slow, and physical activity was desperate. After filling the chamber with fresh air the animal became quieter but respiration remained distressed. The animal died.

Necropsy showed that the lungs were solid. Microscopically there were large areas of consolidation, haemorrhage, oedema, emphysema, and necrosis of alveolar and bronchiolar epithelium. The pleura was thickened, haemorrhagic, and oedematous. The damage was more severe than in animals 2 and 3.

Guinea-pig 5. The conditions were the same as for guinea-pig 4 except that the heating was completed in 30 minutes. This animal showed an immediate extreme irritation of the nose and eyes and excessive salivation. Respiration was at first rapid and shallow and slightly convulsive, but later became deep, slow, and laboured. The animal showed marked activity throughout the exposure, becoming desperate towards the end. On removal from the exposure the activity ceased, but respiration continued to be very deep and distressed, and there was unsteadiness in the hind legs. Death occurred 2½ hours after the exposure ceased.

Necropsy revealed that the lungs were congested in the upper lobes, the lower being nearly normal. The brain was congested. The stomach was distended,

Kidneys. G.-P.5. Exposed to fumes from heated castor oil.

and showed haemorrhages, the wall was friable. Microscopically the lungs showed intense congestion, emphysematous patches, and necrosis of the epithelium of the alveoli and bronchioles. The kidneys showed cloudy swelling in the glomeruli, the cellular outlines being lost in many cases. The tubules showed cloudy swelling and necrosis, the central lumen being obliterated in large areas.

The liver showed cloudy swelling and loss of cellular outlines in the lobules. The spleen showed cloudy swelling, and hyaline degeneration in some areas.

The main results of exposure to fumes from the cutting-emulsion, the sulphonated castor oil, and the pure castor oil were:—

- (a) Irritation of the nose and eyes and excessive salivation, which commenced almost immediately and were severe in all cases.
- (b) Rapid and shallow respiration with intermittent sneezing and coughing, which became deep and slow with obvious distress. Periods of apnoea and of head retraction during inspiration were also sometimes noticed in the later stages. The course of this respiratory response was similar in all cases; the severity, however, was variable. If death did not intervene temporary recovery appeared usually complete within approximately 1–2 hours.
- (c) Effects on the activity of the animals were very different for the two different kinds of oil. The two animals exposed to the fumes from the heated sulphonated castor oil showed a strong disinclination to move, and when provoked there was a loss of power in the hind legs. In the later stages of the exposure they were lying prostrate. Recovery of activity on removal from the exposure was only partial in each case. On the other hand, the two animals exposed to the fumes from the heated medicinal castor oil showed a very marked activity, becoming desperate in the later stages of the exposure.

Guinea-pig 1 exposed to fumes from the heated emulsion which contained about 1 c.cm. of the sulphonated castor oil was less affected than the other animals, dying subsequent to the third exposure. This may have been partly due to absorption of toxic products in the water which was distilled from the emulsion, resulting in a lower concentration of toxic fumes in the air breathed.

Conclusions

From these rather brief experiments we may, perhaps, draw certain general conclusions:—

1. Exposure to these fumes in the concentration and for the times stated caused severe irritation and respiratory distress. Death resulted within a few hours. The lungs, liver and kidneys were severely damaged.
2. The effects were due mainly to the fumes resulting from the pyrolysis reactions of the vegetable oil, the sulphur trioxide fumes being only of minor significance. It was possible that alkyl sulphates played a part.
3. In addition to the respiratory damage due to such fumes, an agent was present in the fumes from the heated sulphonated oil, but not from the pure non-sulphonated oil, which was toxic to the central nervous system.

TABLE 4

Animal Number	Source of Fumes	Exposure		Results
Guinea-pig 1 ..	Cutting-emulsion 10 c.cm. containing 10 per cent. sulphonated castor oil. Fumes heated in silica tube.	Total concentration of fumes from sulphonated castor oil, approximately 50 mg. per litre. Exposure on successive days. First exposure 30 minutes. Second exposure 30 minutes. Third exposure 30 minutes.		Affected. Affected. Death half an hour after exposure ceased.
Guinea-pig 2 ..	Sulphonated castor oil. Fumes heated in silica tube.	Sulphur trioxide; concentration increasing from 0-22 mg. per litre during 0-15 minutes, decreasing to 12 mg. per litre at the end of 30-minute period. Oil fumes: concentration increasing from 0-110 mg. per litre during 15-30-minute period.		Death within 24 hours after exposure ceased.
Guinea-pig 3 ..	Sulphonated castor oil. Fumes passed through asbestos-filled tower.	Oil fumes concentration increasing from 0-50 mg. per litre during 15-30-minute period, decreasing to 25 mg. per litre at the end of 60 minutes. Heating completed in 30 minutes.		Death within 24 hours after exposure ceased.
Guinea-pig 4 ..	Castor oil.	Oil fumes:— First Exposure: Concentration increasing from 0-25 mg. per litre during 30 minutes and from 25-33 mg. per litre at 60 minutes. Oil heated during 60 minutes. Second Exposure given 24 hours after the first. Concentration and time as for first exposure.		Affected. Death during week-end.
Guinea-pig 5 ..	Castor oil.	Oil fumes:— Oil heated during 30 minutes. Concentration increasing from 0-50 mg. per litre during 0-30-minute period, then decreasing to 25 mg. per litre at end of 60 minutes.		Death 2½ hours after exposure ceased.
		Concentration (approximate) in mg. per litre	Time in Minutes	
Guinea-pig 6 ..	Cadmium metal scrapings	2	60	Death within 24 hours after exposure ceased.
Guinea-pig 7 ..	Cadmium metal scrapings	0.4	(i) 60 (ii) 60	Slightly affected. Subsequent death during week-end.

In the case of exposure to cadmium oxide fumes the response of the animals was different from that caused by the oil fumes. This fume were much less irritant to the respiratory tract so far as the immediate effects were concerned. Weakness in the limbs and the intense activity noticed in the exposure to fumes of sulphonated castor oil and castor oil respectively, were not caused by the cadmium oxide fume. The concentrations of the cadmium oxide fume were much less than those of the oil fume, but the ultimate result was the same—death. The lungs, liver, and kidneys were all severely damaged.

It appeared probable from the animal experiments

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that the cadmium oxide fume was much more lethal than the fume from the cutting-compound since two exposures of one hour each to a concentration of 0.4 mg. of cadmium oxide fume per litre caused death, whereas in the case of guinea-pig 1, two exposures of thirty minutes each to a calculated concentration of 50 mg. per litre of the fumes from the sulphonated castor oil did not cause death. In the latter case the concentration of toxic fumes may have been considerably reduced by solution in the water of condensation, but it was unlikely that such effect would reduce the concentration of this toxic fume to anywhere near that of the cadmium oxide fume.

The number of animal experiments is small, but they do indicate the probabilities in regard to the fire incident. In the case of the firemen the concentration of fume from the sulphonated castor oil contained in the cutting-compound were probably of the order of 0.015 mg. per litre, or about one-tenth of the estimated concentration of the cadmium oxide fumes. It appears, therefore, that cadmium oxide fumes played the important part in producing the effects, though the early onset of irritation of the throat was more likely due to fumes from the heat decomposition of the cutting-compound than to cadmium oxide.

Summary

1. The occurrence of 14 cases of poisoning, with one death, among firemen due to inhalation of irritant fumes is described. The cadmium content of the lungs of the fatal case was 4 milligrams.
2. The probable roles of cadmium oxide fumes and those from a cutting-fluid containing sulphonated castor oil are discussed.
3. The results of experiments on the effects on guinea-pigs of the fumes of the cutting-fluid, of sulphonated castor oil, and of castor oil are described.
4. It is concluded that cadmium oxide fumes probably played an important part in producing the poisoning, the other toxic agents being derived from a cutting-compound containing sulphonated castor oil.

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