PETROL-VAPOUR POISONING

BY

READ AIDIN, M.D., D.C.P.

Pathologist, Worthing Hospital

In view of the widespread use of petrol it is surprising that deaths from inhaling the fumes are so rare. Petrol or gasoline vapour has been known for many years to be poisonous. In the reports of the Chief Inspector of Factories for 1937-47 four deaths due to gassing by petrol vapour have been recorded (Ministry of Supply, 1952).

I have been unable to find an account of death from petrol vapour in British medical journals, and so think the following accident worth recording, as many workers both inside and outside the medical profession are unaware of the lethal properties of the vapour.

Case Report

At a non-medical research laboratory in which petrol was used experimentally it was the custom for laboratory assistants to go to a hut 20 yards (18 metres) from the main building and fill a 2-gallon (9-litre) can with petrol. The petrol was kept in a poorly ventilated hut in a 40-gallon (180-litre) metal drum. The drum stood 33 in. (84 cm.) in height and was kept covered with a tightly fitting metal lid.

At 2.30 p.m. on a hot day a youth of 17, 5 ft. 6 in. (167 cm.) in height, was sent to fetch petrol. As he had not returned at 3.15 p.m. a messenger was sent, who found him dead, slumped with his head, shoulders, and arms over the edge of the drum. There was 9 in. (23 cm.) of petrol in the drum, and the youth had apparently placed the can inside the drum and had been ladling petrol with a beaker into the can.

At necropsy irregularly shaped areas were present on the right forearm and wrist where the superficial epithelium had disappeared, leaving dark-red areas. These excoriations involved areas on the upper arm and round the elbow, and resembled the raw areas seen on the body of a macerated stillborn foetus. Several similar smaller areas measuring up to 1 cm. in diameter were present on the forehead. There was slight bruising over the right anterior-superior iliac spine and on the right side of the abdomen, probably caused by pressure from the edge of the drum.

The body was pale and cyanosed; there were no external haemorrhages. The stomach contained semi-digested food, and the oesophagus and the trachea regurgitated stomach contents. Nothing else abnormal was found. There were no petechial haemorrhages in the lungs, heart, or thymus, nor were there any other signs of asphyxia. The internal organs were not congested. No smell of petrol was noted from the lungs. Microscopically the lungs, liver, heart, and kidneys were normal.

Inquiries showed that other workers, when getting petrol from the drum, left the can on the floor and ladled petrol out without stooping down and putting their heads into the drum. The dead lad could not do this, as he was not tall enough, but apparently had to bend his head and shoulders into the drum.

Discussion

Machle (1941) exhaustively reviews the subject of death from inhalation of petrol vapour and gives many references to American and Continental sources.

Petrol is a mixture of hydrocarbons of varying volatility and toxicity. Octane, which is present in high proportions in some petrols, is very toxic. N-octane is about seven times as toxic as pentane (Lawrence, 1945). Some commercial petrols contain benzene and other aromatic hydrocarbons which are of still greater toxicity. In Great Britain

petrol contains 0.8 ml. of tetra-ethyl lead (T.E.L.) per litre (Ministry of Supply, 1952).

Poisoning by petrol vapour occurs rapidly by inhalation but slowly by ingestion. There is probably some skin absorption, but this is generally masked by the accompanying absorption through the lungs, which is the main factor in poisoning. Vigdortschik (1933) states that a concentration as low as 110 to 450 parts per million may produce symptoms in susceptible persons. Fieldner *et al.* (1921) found that 7,000 p.p.m. when inhaled produced definite intoxication in five minutes. Rambousek (1913) states that higher concentrations of the vapour cause almost immediate unconsciousness and death from respiratory arrest.

Machle (1941) records that men exposed to 1,000 p.p.m. showed drowsiness, dullness, and numbness at the end of 15 minutes, followed by vertigo, ataxia, and slight nausea after an hour. Symptoms occurred much more quickly after exposures to 3,000 p.p.m., while exposures to 7,000 p.p.m. caused intoxication in five minutes. Little is known about the concentration of petrol vapour that causes death in man after a short exposure. Machle records that a concentration of 10,000 p.p.m. is rapidly fatal in experimental animals. If the concentration of the vapour is high, absorption through the lungs may be very rapid and symptoms may appear within a few minutes of exposure. The type of coma appears to depend on the chemical nature of the petrol; paraffin hydrocarbons produce quiet coma, as shown by animal experiments, while unsaturated hydrocarbons cause convulsions (Haggard, 1920). The inferential evidence seems to be that the vapour contains a poison affecting the central nervous system. After a few minutes' exposure to a high concentration of vapour the victim passes into coma and dies without any respiratory struggle or post-mortem signs of anoxia (Machle, 1941).

The day of the accident was warm, favouring the formation of petrol vapour. Since the vapour is heavier than air, it seems certain that a heavy concentration formed in the closed drum. This was confirmed by the following test. In the early afternoon of a warm day in circumstances similar to those of the day of the accident an operator about the same height as the dead youth was equipped with an antitox hose mask apparatus; the free end of the breathing hose was placed well outside the hut. The concentration of the petrol vapour was measured by a Poole combustimeter. So as to obtain samples of air from a wider area than that occupied by the end of the rubber suction tube the end of the tube was attached to a 2½-in. (6.3-cm.) funnel. The funnel was then attached to the operator so that the opening of the funnel was within a few inches of where his mouth and nose were when covered with a mask. The lid was removed from the drum, which contained 9 in. (23 cm.) of petrol. The operator then proceeded to fill the can with the beaker. To do this he had to keep his head and shoulders well in the drum. Fifteen seconds after the lid was removed the petrol-vapour concentration was found to be 25,000 p.p.m. During the next 80 seconds, while the can was being filled, the concentration of petrol vapour varied from 8,000 to 35,000 p.p.m., averaging about 20,000 p.p.m. The higher values were obtained when the operator was pouring petrol into the can and some spilt in the process. Four minutes after the lid had been removed and one minute after it had been replaced the concentration of petrol vapour 3 ft. (91 cm.) from the drum was found to be 2,500 p.p.m.

At first the cause of death seemed debatable. It was suggested that the lad might have been struck by lightning. About the time of his death there had been a thunderstorm; however, the evidence seemed to be that the storm did not reach the area until after his death. There were no burns such as might be found on a body that had been struck by lightning. The excoriation of the skin was due, it is believed, to clothing soaked in petrol, which may cause severe blistering unless the clothing is quickly removed and the skin well washed. Presumably the process went

on after death. There is some evidence that the skin lesions may have been caused by T.E.L. in the petrol (Ministry of Supply, 1950). When leaded petrol evaporates about half of it evaporates before any of the lead comes off. Absorption is rapid through the lungs and unbroken skin. It is stated that T.E.L. has a specific toxic effect on the central nervous system. Death may occur within 10 minutes of exposure to a heavy concentration. A degree of consciousness and sensation is said to persist almost to the time of death.

Fifteen deaths from this cause are recorded by Bini and Bollea (1947). It seems, however, difficult to determine whether these deaths were due to petrol vapour or T.E.L. Cassells and Dodds (1946) describe chronic poisoning from T.E.L. but mention no acute cases. Machle (1941) states that T.E.L. is not a factor in acute poisoning. The possibility of vasevagal inhibition had to be considered, but there was no evidence of any painful stimulus. Nor was there any evidence of pressure on the carotid body or evidence to support such a questionable diagnosis as status lymphaticus or of death occurring in an epileptic fit.

The pathological changes found at necropsy are not specific in death from petrol vapour. If death is not immediate the lesions may be due to the irritant and lipolytic action of the vapour. There may be damage to the lungs, hyperaemia, petechial haemorrhages, subpleural extravasations, and sometimes gross haemorrhage into the lungs. Bronchitis and pleural effusions may be present. Severe acute exposure will cause sudden collapse; death may follow without loss of consciousness. In view of the great concentrations of petrol vapour, well over lethal level, that were demonstrated when the accident was reconstructed, there seems to be no doubt about the cause of death.

Summary

A case of accidental death due to inhalation of petrol vapour is described; the surprising rarity of this kind of accident is mentioned and the post-mortem findings are recorded. The reconstruction of the tragedy is described, showing that the concentration of petrol vapour must have been at a highly dangerous level. The literature is reviewed.

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A grant of £50,000 has been made by the King Edward's Hospital Fund for London to assist the North-east Metropolitan Regional Hospital Board in their plans for developing St. Clement's Hospital, Bow, as a modern psychiatric hospital. The hospital already provides to some degree for the mentally sick, in that it has an observation ward and a small unit for psychoneurotic cases. The board consider that in future this hospital will no longer be needed to play any part in providing for the general hospital needs of the area. They have therefore decided to extend the psychiatric work undertaken there, with a view to establishing a modern neuropsychiatric centre for East London. The aim is to provide full facilities for the investigation and treatment of all types of acute mental illness, as well as providing teaching and research facilities.

Medical Memorandum

Spontaneous Rupture of Haematocolpos

Atresia of the hymen is a comparatively uncommon condition and receives but brief mention in most gynaecological textbooks. The possibility of spontaneous rupture of the occluding membrane is not considered in any of the standard works of reference; the following case is described in which this very rare occurrence was encountered.

CASE REPORT

The patient, a girl aged 13, was admitted to hospital on April 30, 1957, because of severe vaginal haemorrhage. Since January she had suffered from attacks of acute urinary retention at approximately monthly intervals, but these had apparently settled readily after the administration of a simple diuretic mixture. Accompanying these episodes there had been some complaint of backache and abdominal pain and a feeling of general malaise. On the evening of admission she had suddenly begun to bleed profusely from the vagina.

On examination her face was pale, but the mucosae were well coloured and there was no evidence of shock. She was afebrile, with a pulse rate of 100 and a normal blood pressure. Inspection of the perineum revealed a bluish swelling just posterior to the urethral orifice; this swelling occupied the normal site of the vaginal introitus and from it a small quantity of dark blood was trickling. Closer examination confirmed that the vagina was occluded by a membrane in which there was a tear, about 3 mm. in diameter, immediately proximal to its anterior border.

Under general anaesthesia the membrane was incised in a cruciform manner, releasing a moderate amount of retained blood. The vagina was greatly distended, the cervix being just palpable at the limit of digital examination. Following operation she was given a course of oxytetracycline extending over five days. Her progress was uneventful, and on May 7 she was re-examined under an anaesthetic before being discharged. By this time the vagina had contracted to a size approximating to normal and the uterus could be easily palpated bimanually; it did not appear to be enlarged. In the pouch of Douglas a moderate quantity of soft clot could be felt, but, as she was symptom-free, no attempt was made to evacuate this. She was allowed home the following day, and has remained well since that date.

COMMENT

The preoperative diagnosis in this case might have been suggested by the history of recurrent attacks of urinary retention, but the picture was complicated by the unusually early age of onset and by the ready relief of symptoms on administration of a diuretic mixture. It is generally accepted that the retention of menstrual blood in cases of haematocolpos is almost invariably due to a membrane situated above the true hymen (Bourne, 1955). Spontaneous rupture of this membrane must be excessively rare, and the only previous reference to this occurrence appears to be an article by Puech (1878) entitled "Imperforation de l'hymen; accidents dépendant de la rétention des menstrues se terminant au bout de dix-huit mois par la rupture spontanée de l'obstacle." In the present case there had been no history of trauma, and it seems likely that rupture was due simply to the effect of increasing intravaginal tension on an abnormally thin area of the obstructing membrane.

RONALD P. CUMMING, M.B., F.R.C.S.Ed.,
Late Senior Surgical Registrar, Aberdeen General Hospitals.

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