spasm relaxed. The recovery period is always quiet and not associated with the restlessness which often follows the use of evipan. I have yet to see a case of post-anæsthetic vomiting or headache after pentothal narcosis. The period of inco-ordination which follows the return of consciousness makes it imperative that the patient should be observed for some hours afterwards. He should never be allowed to drive a car for at least six hours, however normal he may appear to be.

A small proportion of cases develop a minor thrombosis in the vein used for the injection, but the 5 per cent solution lessens this risk considerably. Two or three cases have been reported in which a massive thrombosis has taken place (Payne, 1939).

Contra-indications.—The contra-indications to pentothal anæsthesia are the same as those applied to evipan. Of these the most important is dyspnœa, particularly if caused by partial respiratory obstruction. Others are marked hypotension, sepsis in the mouth or pharynx, and severe liver or kidney disease. I personally regard any operative procedure in the mouth or pharynx, which is likely to interfere with maintenance of a good airway, as a contra-indication, thus limiting the use of pentothal to cases such as the simple extraction of teeth, etc. Incidentally, it should not be given to patients who are being treated with sulphanilamide preparations owing to the sulphur atom contained in the molecule.

Premedication.—If the metabolic rate of the patient is lowered by a preliminary dose of morphia, the scope of pentothal anæsthesia can be considerably extended. The length of anæsthesia can be increased and more traumatic operations undertaken. Although I do not recommend it, on two occasions I have used satisfactorily this anæsthetic alone, when the peritoneum has been opened for perforated gastric ulcer. In my experience, the best form of premedication is a combination of omnopon and hyoscine, and I now use this as a routine. The dosage should be adjusted to the physical and psychic make-up of each individual patient, with particular emphasis on the latter element. In nervous patients these drugs are of the greatest value as they produce a quiet co-operative frame of mind, and, in many cases, actual sleep. Morphine derivatives should always be given at least one hour before operation, so that the full effect on the respiratory centre can be observed before the anæsthesia is begun. The added depressant effect makes it essential that respiration is watched very carefully when giving the pentothal.

The excellent muscular relaxation makes this anæsthetic very suitable for the reduction of fractures, and minor gynæcological operations. Like evipan, it may be used as a method of induction before inhalation anæsthesia for those patients who have a horror of their faces being

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NAPHTHALENE POISONING

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NAPHTHALENE is a hydrocarbon obtained from the middle oil distillate of coal tar. Occurring in large, lustrous crystalline plates, it is easily recognized by its peculiar pungent odour. It is insoluble in cold water, but soluble in hot water, alcohol, ether, chloroform, benzene, and oils. Its industrial use is in the manufacture of indigo and of certain azo dyes. It has also been popularly employed as a disinfectant in the form of moth balls. Occasionally, it has been used internally for therapeutic purposes, as an intestinal disinfectant or vermifuge, and externally, in the form of ointment for pruritus and scabies.

Naphthalene may gain entrance into the body either by inhalation or by ingestion.

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covered. The dose should be kept as small as possible or the decreased respiratory exchange will delay the intake of the inhalation anæsthetic.

No arbitrary statement can be made as to the length of anæsthesia which may be expected with doses of pentothal up to 1 gramme. This depends to a great extent on the operation and on the type of patient. I have seen a large man, who had received 1 gramme during five minutes for the extraction of some teeth, to be awake within the next five minutes. On the other hand, an amputation of the cervix and anterior and posterior colporrhaphy were carried out on a woman, who was given only 0.6 gramme in fifty minutes. Although certain indications may permit of larger doses being used, it is inadvisable to exceed 1 gramme. As a general rule, satisfactory anæsthesia for twenty minutes can be expected in the average patient. It has been pointed out above that the period and depth of anæsthesia can be greatly increased by the use of morphine premedication. For those who have been using evipan, it can be said quite definitely that, other things being equal, pentothal will always provide a better anæsthesia.

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Prolonged inhalation of naphthalene vapour is known to have given rise to symptoms of poisoning. Evers (1884) records a case where persons sleeping under bed clothing dusted over with naphthalene as a moth powder developed symptoms of poisoning. Lutz (Mann, 1922) also records three cases of chronic poisoning by naphthalene vapour. Poisoning by ingestion is usually accidental, occurring amongst children or insane persons. Suicidal cases are extremely rare.

The minimum fatal dose of naphthalene taken by mouth is not definitely known. It has been recorded (Mann, 1922) that in a particular case even seven grains of naphthalene produced severe symptoms of poisoning. A boy (Prochownik, 1911), 6 years old, died in two days after taking 1.75 grammes of naphthalene in seven doses as an anthelmintic. Vyas (Modi, 1936) reported a case in which a boy of 2 years died on the third day after he had swallowed a naphthalene ball weighing about 40 grains. A boy (Heine, 1913), 12 years old, who had eaten two naphthalene-camphor tablets ('bon-bons') each containing 2 grammes of pure naphthalene suffered from severe symptoms of poisoning, resembling those of alcohol but he ultimately recovered.

When naphthalene is taken by mouth, it may irritate the stomach and cause nausea and vomiting. This may get the poison out of the body, in which case further development of symptoms does not take place. Mild irritation of the stomach also occurs when the poison has been inhaled. This occurred in the cases cited by Evers and Lutz. If the ingested naphthalene is not vomited, it passes into the small intestine where it becomes absorbed into the portal circulation. Naphthalene is usually taken in an insoluble form, so that in order that it may be absorbed there must occur in the intestine some physicochemical process, which, at present, is little understood. Wieland and Sorge (1916) have shown that naphthalene forms a co-ordination complex with desoxycholeic acid. This complex, naphthalene choleic acid, has been shown to be easily soluble and absorbable. Recent experimental observations of Stekol and Mann (1937), however, do not confirm the rôle played by desoxycholeic acid, as naphthalene may be absorbed even without the presence of bile in the intestine.

The first vital organ which naphthalene meets after its absorption from the intestine is the liver. This organ, vested with the powers of detoxication, attempts to get rid of the poison partly by excreting it in the bile and partly by oxidizing it to β-naphthol and then combining it with glycuronic, sulphuric and mercapturic acids (Stekol, 1935). These products after detoxication pass into the general circulation from where they are filtered out in the kidneys. The liver itself does not remain unhurt during the process; its glutatheone content diminishes markedly (Nakashima, 1934) and acute necrosis of the polygonal cells takes place here and there. This in its turn produces the clinical manifestations of jaundice and cholæmia. A part of the poison which has not been successfully detoxicated enters the general circulation and exerts its harmful effects on various other important structures. Red blood corpuscles may be hæmolysed, the myocardium may be enfeebled, the higher cerebral centres may suffer functional depression, and the kidneys also may share the general damage. Thus will arise the clinical manifestations of rapidly developing anæmia and even hæmoglobinuria, enfeebled circulation and weakened heart sounds, confusion and coma, albuminuria, cylindruria and hæmaturia. Optic neuritis, cataractous changes in the eye lens and opacities in the cornea may also occur in the chronic forms of poisoning (Bouchard and Charrin, 1886; Leschke, 1934).

We came across some of the above deleterious effects of naphthalene in a fatal case of poisoning, the notes of which we append below. So far as our knowledge goes, this is the second case of fatal poisoning due to naphthalene reported in India, the first report being that of Vyas to which reference has already been made.

Case note

L., Mohammedan, aged 24 years, was admitted into the Medical College Hospital, Calcutta, in a semi-conscious state on 4th June, 1939. The attendant said that the patient took some naphthalene (exact amount was not known) by mistake in place of an Indian sweet on 2nd June.

On examination.—The patient was semi-conscious; axillary temperature 99°F.; pulse rate 120 per minute; respiration rate 28 per minute. He looked pale and severely jaundiced. No signs of trauma were found on the head, neck or body. There was nothing in the breath suggestive of poisoning. Liver and spleen were not palpable. Heart sounds were feeble, lungs clear, pupils equal and reacting well to light. The neck was soft but Kernig's sign was positive. All the tendon reflexes were sluggish. Superficial abdominal reflex was absent. Babinski's sign was positive on both sides.

Twelve hours later, the coma deepened, pallor became more marked, heart sounds became feebler and the rectal temperature rose to 102°F. Systolic blood pressure was 95 and diastolic 48 mm. of Hg. At this stage a lumbar puncture was done. Ten cubic centimetres of clear cerebro-spinal fluid came out under slightly increased pressure.

After another twelve hours, the patient's condition became worse. There was evidence of right-sided hemiplegia. The rectal temperature rose to 103°F. The pulse rate was 142, and respiration 44 per minute. Patient ultimately expired—three days after swallowing the naphthalene.

Laboratory findings.—Blood examination report:—Hæmoglobin 30 per cent; red blood corpuscles—2,410,000 per c.mm.; white blood corpuscles—31,200 per c.mm.; polymorphonuclear cells—90 per cent; lymphocytes—8 per cent; monocytes—2 per cent. No malarial parasites were found. Plenty of normoblasts and marked anisocytosis were noticed.

Urine examination report:—Colour—brown; reaction—acid; specific gravity—1020; turbidity—slight; albumin—trace; sugar—*nil;* acetone—*nil;* bile—present. Occult blood test—negative; indican—*nil.* Microscopically occasional pus cells, few epithelial cells and mucus.

Biochemistry of blood:—Urea—53 mgm. per cent; non-protein nitrogen—46 mgm. per cent; cholesterol—

106 mgm. per cent; chloride—468 mgm. per cent. Van den Bergh reaction—immediate direct positive; bilirubin contents—20 units. Wassermann reaction of blood was negative.

Cerebro-spinal fluid:—Clear fluid, microscopically no pus cells or organisms seen. Culture—no growth.

Autopsy findings.—Rigor mortis all over the body. Pupils dilated. Conjunctiva—markedly icteric. Scalp layer stained yellow. Brain and spinal cord stained yellow. Larynx and trachea—mucus stained yellow and contained froth. Lungs—yellow frothy exudations from sections on pressure. Heart—thin fluid blood on both sides. Peritoneum—stained yellow. Gastro-intestinal tract—mucous membrane yellow. Kidneys—pale. Spleen—congested. Gall-bladder full of bile. Liver—soft, pale.

Histology.—Patchy necrosis of the liver, chiefly in the central zone. Naphthalene was detected in the tissues, in the stomach contents and in the urine by the chemical examiner.

Comments

The remarkable features of the above case were deepening coma, severe jaundice, rapidly developing anæmia, hyperthermia, and patchy hepatic necrosis, as revealed by the post-mortem examination. Such features of naphthalene poisoning have not been frequently noticed by the previous reporters. Indeed, there is no mention of hyperthermia, hemiplegia and marked anæmia in the previously recorded cases reviewed by us. The marked degree of anæmia which developed so rapidly was possibly due to acute hæmolysis, although, in this case, there was no hæmoglobinuria. This hæmolysis enhanced the jaundice primarily caused by hepatic necrosis. Hyperthermia might be explained by the cholæmia consequent to liver necrosis, like the hyperthermia met with in cases of acute yellow atrophy of the liver. It is also possible that, in the metabolism of naphthalene, naphthylamine (an amino derivative) was formed and was responsible for the fever. Naphthylamine has been used by laboratory workers for causing experimental hyperthermia.

Summary

1. A brief review of cases of naphthalene poisoning has been made.

2. Metabolism of naphthalene and its effects on the body have been discussed.

3. A case of fatal poisoning has been described, including its autopsy findings.

4. The possible pathogenesis of the unusual features has been suggested.

We are grateful to Colonel J. C. De, I.M.S., Superintendent, Medical College Hospitals, Calcutta, for the permission to publish the case, to Dr. K. N. Bagchi, Rai Bahadur for the report of the chemical examination of the viscera, to Major D. Ahmed for the post-mortem reports, to Capt. P. De for the bio-chemical reports and to Prof. B. P. Tribedi for his permission to undertake the histological examinations.

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CARDIO-VASCULAR SYPHILIS AND CEREBRAL SYMPTOMS

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THE clinical results of a syphilitic infection have long been described in more or less definite three stages: primary, secondary and tertiary. This division into stages has been possible apparently because of a hypothetical tissue susceptibility of the various types of body tissues at different periods of the infection. We are taught that after the primary sore on the genitals or elsewhere—the lesion at the site of entry of the organism associated with trauma—the skin and mucous membranes follow with a rash, etc., in a regular sequence, and later, after a variable period, the so-called tertiary symptoms appear in the internal viscera.

Anyone with a long clinical experience in a general hospital will have been struck by the number of syphilitics (as proved by the Wassermann reaction) who do not show or give any history of the disease going through the different stages. Often, syphilis in a patient is discovered when he develops signs or symptoms of an aortic aneurysm or of coronary artery damage with thrombosis. Ordinarily, however, a syphilitic does not die of his infection in the earlier stages, or even in the later stages, unless one of the vital organs is involved. A more severe secondary infection, such as tuberculosis, sets in, from the lowered vitality of the syphilitic infection, which kills him off.

The writer still remembers the quaint aphorism that used to be dinned into him during his student days 'Syphilis is the bed on which tubercle is born'. In a general hospital, if a patient comes with symptoms of any disease that cannot be directly referable to a syphilitic infection, these symptoms get diagnosed and treated; and it is only when this treatment becomes unsuccessful, partially or

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