ROCKET PROPELLANTS: THEIR TOXIC EFFECTS, HAZARDS AND FIRST AID

BY

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"The pursuit of scientific truth in which you are trained, is a grand, exacting and splendid discipline indeed. There is the acquisition of exact and precise knowledge by observation, experiment, the inspired guess, even by accident. There is the testing of it by applying it and seeing what trial and error teach you. And then out of it come fresh problems, fresh study and more truth."*

It would be true to say that much of the work carried out in Government Establishments is of particular interest to medical officers in all three Services. In view of the large amount of scientific work which is being undertaken by the Government at the present time and the inadequate facilities for the medical profession in the fighting services and in Civil Defence to become acquainted with this work, it gives me much pleasure to accept the opportunity which your Editor has afforded me to publish this article in the columns of your Journal.

HISTORY OF ROCKETS

A few salient facts about the history of rockets will help you to assess our present state of development. Rockets are of ancient origin. History relates that the Chinese were the first to attach plain-powder rockets to feathered arrows about A.D. 1220, and about A.D. 1500, Wan Hoo, a Chinese Civil Servant, tried a manned rocket projectile and disappeared effectively in a cloud. In the Middle

* From an address given by Doctor Geoffrey Fisher, Archbishop of Canterbury, on 4th October, 1949, at the opening of the new session of the University Medical School, Birmingham.
Ages rockets became quite common in addition to primitive guns, and during the fifteenth century the French used rockets against English troops. In 1688 experiments with war rockets weighing 120 lb. were carried out in Berlin. Fresh impetus was given to the use of rocket weapons after British Colonial Forces had met with severe losses in India against organized rocket troops of Tippu Sahib. The rockets employed were hollowed bamboo sticks filled with the charge and carrying some incendiary material. About 1800 Colonel Sir W. Congreve, M.A., of Woolwich Arsenal, developed new types of iron-cased, finned-stick rockets with incendiary or shrapnel war-heads, the propellant charge being, of course, gunpowder. These rockets, launched from the ground from special rocket-throwing vessels in quick succession and in large quantities, proved of immense value in the wars against Napoleon. In 1812, at the battle of Blandersburg, American militiamen broke under the fire of British war rockets and left Washington defenceless. As a result of these successes, practically all armies and navies introduced special rocket artillery detachments. The weapons used were the Congreve model, and the United States Army alone established more than ten such batteries, and in all wars up to about 1860 rockets were common as war missiles.

In 1846 the American, W. Hale, replaced the stabilizing stick by three curved vanes at the aft end of the rocket and the first spin-stabilized rocket was thus created and proved a great success. From about 1850 until about 1870 there was a decline in rocket artillery in favour of field artillery. The war-rocket came into discredit, although in Britain a spinning Hale war-rocket was still on the equip-
ment list in 1914 and available for use with the Royal Flying Corps. So far as is known, no operational use was, however, made of this rocket for aircraft. The only use for rockets then thought practicable was for fireworks, for illumination, for conveying life-lines to ships in distress, the latter application having been originated in Prussia during the late eighteenth century. During the 1914-18 war, rockets were employed defensively against kite balloons. The rocket missiles used were rather small, were stick-stabilized and acted as incendiaries. They were carried by single-seater fighters on outboard struts and electrically fired. In one instance an allied Newport Fighter biplane equipped with incendiary rocket projectiles was able to destroy a number of German kite balloons in one operation, thus temporarily blinding the enemy. Incendiary rockets were also tentatively used against Zeppelins, but did not meet with success as their range was very limited. During the Second World War the lack of sufficient ack-ack guns compelled the War Office to use barrages of modern war rockets against enemy raiders. The Z-batteries set up met with fair success and for the first time rockets were fired operationally which burned smokeless powder propellants. This development was due to British work on explosive research and established superiority for Britain in this particular field. Rockets were also used operationally by the Germans from a chemical mortar which could throw smoke and gas missiles for relatively short ranges. In 1942 rockets were used by the Russians as armour-piercing projectiles fired by low-flying aircraft against armoured vehicles. The relief of Stalingrad was greatly aided by the use of rocket barrages. All these war rockets were unguided.

**CLASS OF MISSILE**

Rocket-propelled units can generally be classified according to the following headings:

(a) Type of application—Aeroplane power plant; rocket projectile; guided missile power plant; assisted take-off unit.

(b) Type of propellant used—Liquid propellant; solid propellant; gaseous propellant or combination of liquid, solid and/or gaseous propellant.

**USES**

There are essentially two types of jet propulsion: rocket propulsion, wherein the matter to be ejected is stored within the device and ducted; and propulsion wherein the surrounding fluid is ducted through the device and accelerated to greater momentum by mechanical or thermal means prior to ejection.

**Examples of Mechanical Compression Units**

* Turbo Jets. — The fluid is compressed by mechanical means. It is then burned and expanded in a nozzle. This compression is usually accomplished by a mechanical compressor which is driven by a turbine (turbo jet) or other means. The momentum of the ducted fluid is increased and this produces a propulsive force.

* Rocket Projectiles: (a) Unguided Missiles. — An explosive charge, a smoke
charge or some other military payload is propelled the first part of its flight by a suitable simple rocket system. During the remainder of the flight the projectile is a freely flying body, not as accurate as artillery and small arms. Because of simplicity of rocket projectiles and light weight of launching mechanism, they can more readily be fired in greater numbers.

(b) Guided Missiles.—Similar to unguided missiles, but they are usually larger and their flight path is controlled by an automatic mechanism or pilot. Although the main application of guided missiles is a military one (German V1), they are also used as vehicles of scientific instruction. The art of warfare is now being revolutionized by the manifold application of this weapon. The weapon may be subdivided into several classes depending on their launching method and type of target, including ground-to-ground, ground-to-air, air-to-air, air-to-ground, ship-to-ship and ship-to-air missiles. The payload would be explosives, smoke or poison gases charge. It is the aim of many rocket experimenters to design a guided missile which will leave the earth. Advanced space missiles are intended for inter-planetary travel and research. It will probably take several years before this field will be accomplished. These missiles may be guided or unguided.

Many other applications of rockets can be mentioned; such as rocket-powered racing cars, rocket-driven small boats, rocket to assist heavy trucks out of mud ruts and even rocket-fired railroad cars and water rockets for torpedoes and submarines.

Having described the types, application and uses of rocket devices, the underlying principle is the same throughout—the liberation of the largest amount of heat in the shortest possible time by means of a chemical reaction, this heat being converted into momentum of the exhaust gases. This is done by propellants—oxidants and fuels. It would be quite impossible in this paper to cover all the systems which have been used in the past and are being tested at the present time, but it is proposed to deal with a few substances—oxidants and fuels—which are in constant use in this country. These have been selected by virtue of their somewhat differing toxic properties. These systems include: Nitric acid (oxidant) and kerosene (fuel); hydrogen peroxide (oxidant) and C-fuel (fuel); hydrogen peroxide (oxidant) and kerosene (fuel); liquid oxygen (oxidant) and methanol (fuel); liquid oxygen (oxidant) and kerosene (fuel).

NITRIC ACID

A good deal is already known about the toxicity of nitric acid, but this substance is included since it serves as a good comparison with the toxicity of other substances mentioned above.

General Properties

Concentrated nitric acid (HNO₃) is a fuming liquid which is heavier than water. Its specific gravity is 1.51. The commercial acid is more or less brown in colour on account of dissolved nitrogen tetroxide, but the pure acid is colourless. For the purpose of this paper nitric acid is referred to as a concentrated nitric
acid of 90 per cent. concentration or over. It is generally a highly fuming liquid and is a corrosive.

Toxic Effects and Hazards

Associated with this acid are three important dangers:

(a) Oxides of nitrogen poisoning.
(b) Acid burns.
(c) Corrosive action on ingestion.

(a) Oxides of Nitrogen Poisoning.—The gaseous oxides of nitrogen are as follows:
- Nitrous oxide (N₂O)—known as laughing gas, the well-known anaesthetic.
- Nitric oxide (NO)—a colourless gas, non-irritant. Combines with Hb to form methaemoglobin and has been found to produce paralysis and convulsions in animals.
- Nitrogen dioxide (NO₂)—dark chocolate brown gas, an irritant dangerously toxic.
- Nitrogen tetroxide (N₂O₄)—colourless, an irritant and dangerously toxic.

It matters little whether these oxides of nitrogen enter the air as nitric oxide, nitrogen dioxide or nitrogen tetroxide, since the nitric oxide in contact with the air and oxygen at once turns brown and changes to nitrogen dioxide, and thus the nitrogen dioxide/nitrogen tetroxide balance comes into play. It is, however, intended to emphasize particularly the effects of the nitrogen dioxide/nitrogen tetroxide gaseous mixture since separately or together they are highly toxic. These gases are always present as a mixture and the relative proportions of these gases vary considerably according to the circumstances—i.e. temperature and humidity. The colour of these gaseous oxides varies from colourless to chocolate brown, depending upon the composition of the mixture, though it is practically always yellow to light brown. The intensity of the colour is, however, no indication of the degree of toxicity. These gaseous oxides are formed when nitric acid comes into contact with certain heavy metals (copper, brass, zinc) or with any organic material (wood, sawdust, paper). These gases are among the most treacherous because of the insidious onset of severe and sometimes fatal pulmonary oedema.

The effects of exposure to and inhalation of this mixture may be conveniently divided into (1) acute and (2) chronic, depending to some extent on the concentration.

(1) Acute.—On exposure, above 50 p.p.m. moderately irritating to the eyes and upper respiratory tract. Higher concentrations—150 p.p.m.—not painfully irritant, but causes an acid taste in the mouth. On inhalation, very high concentrations give rise to the following signs and symptoms: Weakness; shivering; dyspnœa; tightness across the chest; tachycardia; abundant expectoration of frothy serum; cyanosis; abdominal pain; pulmonary oedema; convulsions; collapse and death within five to eight hours.

Acute cases have been recorded where no signs or symptoms or pulmonary oedema have occurred and the victims have recovered completely.
Also there is a type of acute case which gives rise to asphyxiation, convulsions, respiratory arrest.

(2) Chronic.—Inhalation: The signs and symptoms are—chronic cough; headache; inflammation of the mouth, nose and eyes; corrosion of the teeth; loss of appetite; constipation.

These cases occur after prolonged exposure to gaseous oxides of variable concentrations under very different temperature and humidity conditions. They depend also to some extent upon the susceptibility of the individual. Inhalation of this mixture throughout an eight-hour period of as little as 25 p.p.m. (American Standard Association) may cause pulmonary signs and symptoms referred to under the acute stage after five hours to as many as forty-eight hours. Delayed pulmonary oedema may follow exposure to high concentrations of 100-150 p.p.m. for only half an hour to an hour. While the American Standard Association have fixed this safe limit for an eight-hour daily exposure at 25 p.p.m., cases have been recorded which have developed toxic symptoms under this low limit. Recent experiments in America, where rats were repeatedly exposed to an average concentration of 25 p.p.m. of nitrogen dioxide, resulted in pulmonary oedema. On the basis of experiments at present in progress of exposure to 5 p.p.m., it is suggested that the safe limit be reduced to 5 p.p.m. of gaseous oxide.

Since the gaseous oxides may cause little or no discomfort at the time of inhalation, the exposed worker may continue his work feeling quite well though severe lung damage has been caused, the signs and symptoms of which become distressingly obvious several hours later.

One further word about the causation of pulmonary oedema. While it is believed that a high concentration of the gaseous oxide of nitrogen acts by irritation, it is by no means certain that this is so at low concentrations and the following explanation has been suggested. Nitrogen dioxide hydrolyses slowly in water or humid air to form nitrous acids. The theory is that during inhalation the relatively dry gas/air mixture reacts little with the slightly moist surfaces of the respiratory passages, whereas after reaching the alveoli and the interstitial tissues of the lung, the humid air and moist surfaces promote almost complete hydrolysis with the alveolar tissues. When patients survive pulmonary oedema, pneumonia is often a sequela, which sometimes proves fatal in later weeks.

In connection with the cyanosis, while methaemoglobin may be found in the blood, it is probable that the exudate oedema is the main factor in preventing oxygen penetrating to the pulmonary capillaries, and the cyanosis results from anoxæmia which is neither cardiac nor intrinsically pneumonic. The precyanotic stage reveals flushing, drooping eyelids but no blueness. Within a short time, the patient changes colour and develops heliotropic cyanosis. The patient is not generally in physical distress, but the prognosis is almost hopeless. In the terminal stage the lips and ears arrest notice by their deep purple hue, the face is less heliotropic and the patient may live for another twelve to twenty-four hours.

(b) Acid Burns.—Nitric acid burns, in common with other burns, are associated with varying degrees of shock, but they are not associated with toxic absorption. They generally occur on the hands and face and tend not to char the
tissues. They are intensely painful. Splashes of nitric acid may involve the eyes, giving rise to excruciating pain followed by corneal ulceration with consequent impaired or permanent loss of sight.

(c) Corrosive Action on Ingestion.—Nitric acid is similar to sulphuric acid when ingested and the symptoms are immediate with severe burning of the mouth, throat and stomach, spreading over the whole abdomen. The acid has no tendency to char the tissues and therefore perforation occurs less frequently than with sulphuric acid. Gaseous irritations which are very distressing to the patient often occur associated with vomiting, the vomit being dark brown or black in colour mixed with altered blood. The respiration is difficult and noisy. The voice is hoarse and speech may be impossible. Acute œdema of the larynx may occur and cause rapid death from asphyxia. Death usually occurs within twelve to twenty-four hours from the local action, or partial recovery takes place and death occurs after a few days with gastric inflammation. The patient may recover from the acute symptoms and die after two or three weeks from weakness and exhaustion, or he may live for years with stricture of the œsophagus or other sequelæ. Should stricture not occur, the destruction of the gastric mucosa cause a loss of digestive juices and the patient emaciates from chronic malnutrition. In addition, there may be symptoms due to inhalation of gaseous oxides with the subsequent onset of pulmonary œdema.

CONCENTRATED HYDROGEN PEROXIDE—H₂O₂

General Properties

It is generally referred to as H.T.P., which means High Test Peroxide, and it is a concentrated solution of hydrogen peroxide in water of 80 per cent. or higher concentration of high purity. It is a colourless or nearly colourless liquid of density of about 1.35. H.T.P. is not itself inflammable, but due to its powerful oxidizing properties it can give rise to fires in contact with combustible materials. In contact with many materials—iron and certain other metals—H.T.P. decomposes, evolving heat and forming steam and oxygen. It is most important that H.T.P. should be kept free of dust and dirt generally and only come in contact with inert materials, such as glass, earthenware, stainless steel, certain plastic materials and natural rubber, not as a container, only as protection for personnel. H.T.P. is rendered relatively harmless when diluted with four parts of water.

Toxic Effects and Hazards

H.T.P. itself is not poisonous but is damaging to the tissues. The vapours given off by H.T.P. or in contact with the ground, etc., are not toxic and, although they do irritate the eyes and the nose, they are practically harmless. Splashes in the eye are very painful. The mild irritant effect on the eyes tends to cause lachrymation associated with conjunctivitis and a mild coryza associated with an injected mucosa. When completely decomposed H.T.P. gives free oxygen and is thus not toxic for use in an enclosed space. Exposure to a heavy mist of H.T.P. droplets has an irritant effect on the lungs, and for this reason asthmatics,
chronic bronchitics, bronchiectasis and other chronic lung conditions should not be exposed to it.

Recent experiments in America using animals (rats) have been carried out with 90 per cent. hydrogen peroxide, the results of which are preliminary and subject to revision.

**Inhalation.**—On exposure of rats to a concentration of 2,800 p.p.m. at 75°–80° F. the animals were observed for fourteen days. No symptoms other than excitement at the beginning of exposures were noted—the animals remained calm and appeared normal. Intravenous injection of this substance into rabbits—the rabbits died from gas embolism. Skin absorption tests on rabbits with this substance resulted in absorption of hydrogen peroxide, causing death by gas embolism. There was apparently a marked species variation by this method; cats, pigs, guinea-pigs, rats and dogs revealing far less general systemic toxicity and a far greater local skin reaction. Rabbits are known to be susceptible to air embolism. Skin absorption tests, subcutaneous and intravenous injections of this substance have led to the conclusion that the greater the local reaction the lower the toxicity. Similarly, intravenous injection led to the conclusion that the toxicity was increased with the dilution.

Applied to the skin of rats, there was local swelling and blanching with subsequent sloughing and ultimate partial regeneration or scarring. Applied to the corneæ of rabbits, minor effects were caused which disappeared without residual injury. On the other hand, permanent opacities occurred.

**Conclusions.**—Hydrogen peroxide inhalation involves no major respiratory hazards. Skin hazards may be severe and liquid splashes should be avoided. Liquid splashes in the eyes must also be avoided. Severe corneal damage giving rise to impaired visual acuity or permanent blindness may be caused by relatively small amounts of the liquid.

**LIQUID OXYGEN—O₂**

*General Properties*

Liquid oxygen boils at −183° C. and its density is 1.14 at this temperature. It is non-inflammable.

*Toxic Effects and Hazards*

Inhalation of 100 per cent. oxygen at atmospheric pressure for long periods has caused no observed injury to man. At higher pressures (three atmospheres) for three hours produces no distressing symptoms. Convulsions have occurred in man after oxygen has been breathed for forty-five minutes at four atmospheres pressure, while after one to three hours at one atmosphere pressure, concentration and co-ordination become impaired or increased effort was necessary to maintain them. Healthy young men can breathe oxygen at three atmospheres pressure for three hours without ill effect, but during the fourth hour the pupils dilate and the visual fields become distracted and some impairment of central vision—all of which are criteria of oxygen toxicity. Circulatory changes include a peripheral vascular constriction associated with visual impairment and an abrupt
rise in systolic and diastolic blood-pressure, techycardia and extreme pallor. At this stage the patient experiences dizziness and a feeling of impending collapse. Rapid and complete recovery associated with a feeling of alertness and stimulation results within an hour when air is substituted for oxygen.

In animals (dogs) it has been found that there was a fall in oxygen saturation of the blood, a rise in hæmoglobin associated with lung congestion, œdema, right heart failure and congestion of the liver. Gaseous oxygen may be harmful to persons with pulmonary tuberculosis, chronic bronchitis, asthma and bronchiectasis.

Liquid oxygen is not toxic, but owing to its low temperature the liquid can cause serious burns if in prolonged contact with the skin.

Moreover, fingers and hands when directly exposed to it may develop "frostbite" and gangrene. But small degrees of skin contamination are not dangerous owing to evaporation.

Liquid oxygen may cause severe burns of the cornea associated with scarring and impaired or total loss of sight.

**Hydrazine and Hydrazine Hydrate**

*General Properties*

Hydrazine (N₂H₄) is a colourless liquid or a white solid.

Hydrazine hydrate (NH₂NH₂H₂O) is a colourless or pale yellow alkaline liquid with faint ammoniacal odour. Since hydrazine in contact with air or water forms hydrazine hydrate, the toxic risks are the same for both, excepting that the symptoms are considerably reduced and the survival time longer in the case of hydrazine hydrate. Both liquid forms fume visibly in air. It is, therefore, proposed to deal with these substances as one.

*Toxic Effects and Hazards*

**Hydrazine.**—This is chemically allied to ammonia, which it is well known has a very pungent smell and irritates the upper respiratory tract and the eyes. The presence of ammonia in the air can easily be detected owing to the strength of smell at concentrations too low to be dangerous. If, however, an individual is exposed to high concentrations of ammonia vapour they may be overcome. Signs and symptoms include—restlessness, vertigo, gastric pain, nausea, vomiting. Contact of ammonia with the cornea of the eye may lead to permanent or serious damage.

Various reports have been received from medical authorities in Germany about workers exposed to the fume of hydrazine during the recent war. The signs and symptoms include—headache, gastritis, diarrhœa, restlessness, nausea, vomiting, loss of power of concentration, respiratory embarrassment, depression, bradycardia, coma and death.

**Skin Irritation.**—At Pennemunde it is reported a small proportion of workers were highly susceptible and developed a contact dermatitis. These workers had been handling mixtures of hydrazine hydrate, ethyl alcohol and traces of potassium cupro-cyanide. There was a tendency for cuts to become septic.
Some workers at Farnborough handling C-stuff experienced dermatitis of the hands and forearms which was associated with severe itching. Calamine lotion cleared it up and previous application of vaseline and of lanolin gave some protection. One man whose face was sprayed with hydrazine hydrate developed blisters in spite of washing. These cleared up with vaseline within two days. Recent skin tests with this substance revealed no marked skin irritation.

**Inhalation.**—In America experiments on rats reveal that inhalation of saturated hydrazine vapour for half an hour resulted in fatalities in about 17 per cent. of the animals exposed. Restlessness was clearly evident at the beginning, followed by epistaxis, and there was pronounced salivation. Neurological disturbances occurred, terminating with convulsions. Death was delayed about two days after exposure.

**Skin absorption.**—Tests have also been carried out with undiluted hydrazine on the skin of rabbits. A prompt local reaction occurred with a delayed systemic effect. The local effect consisted of a purplish discoloration which appeared in two to five minutes, rising to a maximum and gradually disappearing in forty-eight hours. It was thought that the discoloration was due to subcutaneous haemorrhage, which sometimes resulted in sloughing of the skin and subsequent scar formation. The systemic effect consisted of the development of extensor rigidity followed shortly by intermittent clonic convulsions. There was also congestion of the lungs, congestion of the kidneys and tubular casts. Applied to the cornea of the rabbit's eye, hæmorrhages occurred.

**Conclusions.**—Inhalation of hydrazine is moderately severe so that respiratory protection is indicated. Hydrazine skin toxicity is of a high order of magnitude. Splashes on the skin should be removed as soon as possible by washing freely with water. Eye protection is necessary for both compounds. Investigations of subacute and chronic effects of this substance are still in progress.

**Methanol—Methyl Alcohol—CH₃OH**

(Other names—wood alcohol, wood spirit, carbinol, columbian spirit)

**General Properties.**
A colourless volatile liquid, miscible with water, boiling well below the temperature of water and having a specific gravity of about 0.79.

**Toxic Effects and Hazards**
As a vapour by inhalation methanol is toxic. Signs and symptoms are the result of injury to central nervous system with particular emphasis on optic atrophy. It also causes degenerative damage to kidneys, liver, heart and other organs. Symptoms are generally delayed from nine to thirty-six hours, during which time all individuals may continue to carry on. Suddenly weakness, headache, nausea, vomiting, abdominal pain, dimness of vision and even unconsciousness may develop. Less concentrated amounts give rise to symptoms of local irritation of eyes, headache, fatigue, drowsiness and sweating. Blindness affects both eyes and may set in within a few hours or it may be delayed several days.
Toxic absorption may be slow and it is accumulative and slowly secreted. The accepted maximum allowable concentration of vapour is 200 p.p.m. for an eight-hour work day.

_Ingestion._—Symptoms are quite prompt after swallowing: redness and blueness of the face and mucous membranes associated with depression, weakness, headache, nausea, abdominal pain, shortness of breath, delirium and cold sweats. Many cases develop blindness, temporary or permanent, due to optic atrophy.

_Skin absorption._—Absorbed through the skin, this substance gives rise to the general symptoms already referred to. In addition, after repeated or prolonged contact, dermatitis occurs. The skin becomes rough, red and dry and cracks easily and is very susceptible to infection.

**Kerosene**

*General Properties*

Many varieties have been considered as gas turbine fuels for aircraft use. The current fuel boils between 160° C. and 275° C. and a saturated vapour is too weak to ignite at normal temperatures. Kerosene consists of a mixture of hydrocarbons.

*Toxic Effects and Hazards*

On account of the low volatility and composition, kerosene does not give rise to toxic effects at normal temperatures. Toxic risks may arise, however, from additions such as lead tetraethyl. Toxic gases, especially carbon monoxide, are given off after combustion. The general symptoms of poisoning of this gas are well known—lassitude, headache, giddiness, faintness, nausea, muscular weakness. Pulse respirations are increased and concentration becomes impaired. Later there is mental confusion, diminution of sight and hearing, palpitations and dyspnœa with ultimate complete paralysis, coma and finally painless death. Death is due to deprivation of oxygen and there is no specific toxicity in the gas, although this is not necessarily true of coal gas and similar mixtures of gases. The patient may show reddish patches on the skin and occasionally blisters may occur. Albumin and sugar may be found in the urine. Sequelæ—amnesia and nervous disorders.

**First Aid**

It is not intended in a paper of this kind to discuss the comprehensive treatment of the toxic effects of these substances, but a few words about first aid will be helpful.

In cases of ingestion of acid, emetics should not be given nor should a stomach tube be used. The first thing to do is to attempt to neutralize the acid with copious draughts of water or milk, adding two teaspoonfuls of magnesia, chalk or whitewash. Carbonate or bicarbonates, such as washing or baking soda, should not be administered if other substances can be obtained, by virtue of the liberation of carbonate dioxide, which may cause gastric perforation from the dilation of the stomach or may precipitate heart failure.
Workers who inhale the oxides of nitrogen must be kept under observation for at least twenty-four hours, because of the delayed effect of nitrogen dioxide. The individual who has inhaled the mixture should not be allowed to undertake any exertion, but should be made to rest for twenty-four hours.

In the more serious cases the patient should be removed at once into a clear atmosphere and kept warm and the doctor sent for immediately. Quiet and rest is essential. Hot tea or coffee may be given but not alcohol. If there is difficulty in breathing and coughing continues, pure oxygen is given. Only if respiration has ceased should artificial respiration be conducted. Morphia should not be given since it depresses the respiratory centre.

The most important treatment of burns is to wash the acid off as quickly as possible—within seconds—with copious supplies of water. For this purpose baths must be provided for containing clean water, preferably heated, at convenient places so that the whole body if necessary can be totally immersed without delay. Acid-soaked clothes should be stripped off. Splashes in the eyes should be treated immediately by irrigation with water, followed by irrigation in 2 per cent. solution of sodium bicarbonate, an eye-pad applied and the doctor sent for at once. Time is an essential factor in these cases. Shock from acid burns is treated in the accepted way.

For burns by concentrated hydrogen peroxide, dab away (not wipe) as much of the peroxide as possible. Remove or cut away contaminated clothing and thoroughly flood the affected part with water. Then apply a sterile dressing and treat as an ordinary burn. Splashes in the eyes should be treated as above.

In the event of air embolism the patient should be kept quiet and warm. Stimulants may be administered hypodermically and amyl nitrite inhaled. Death may be due to arrest of pulmonary circulation or to cerebral anaemia.

Burns caused by liquid oxygen should be treated in the same way as corrosives and treatment for the eyes is identical. Cases should be referred to a doctor as soon as possible. In cases of "frostbite" the affected part should be protected from cold and very gradually warmed by slow thawing. The temperature must not be brought above that of a cold room—the part being left outside the bedclothes and simply wrapped in cotton wool. Do not give massage—this causes damage. Treat for shock and give warm drinks. Cases of gangrene should be seen at once by the doctor.

In cases of skin contamination by hydrazine and hydrazine hydrate, thoroughly flood the affected skin with water as soon as possible and apply calamine lotion. Splashes in the eyes should be treated as above.

In cases of inhalation of methanol vapour, remove patients into the fresh air, keep quiet and warm with blankets and hot water, give oxygen, and if breathing fails apply artificial respiration until patient breathes again or until a doctor instructs otherwise. In cases of ingestion, an emetic of salt in water may be given. Splashes in the eyes should be treated by irrigation immediately and continued for at least thirty minutes. Irrigation may be followed by the installation of liquid paraffin or castor oil. The patient should then be referred to a doctor or hospital for further treatment.
The chief toxic hazard associated with kerosene is carbon monoxide. Remove the person from the cause and keep the patient warm. Clothes should be loosened at the neck and waist and artificial respiration applied. Give oxygen inhalations and treat for shock.

**PREVENTION**

From what has been said about the toxic effects of rocket propellants, it will be fully appreciated that under no circumstances should the oxidant and fuel be in close proximity. It will be understood that there are always potential risks of fire, explosion, spills, crashes, enemy action, and adequate provision must be made for them. This is more the responsibility of the Safety Officer.

Protective clothing is an essential part of the organization and is recommended as follows:

**Nitric Acid**
- White lasting cloth (flax material)—jacket and trousers.
- Lasting cloth cap with back flap.
- Eye-shields, rubber gloves, rubber boots.
- Masks should be worn. Those suitable for oxides of nitrogen have a short life of twenty to thirty minutes and therefore need changing on the spot.

**Hydrogen Peroxide**
- Polyvinyl chloride (plastic) jacket and trousers or long polyvinyl chloride smock and hood.
- Eye-shields, rubber gloves, rubber boots.

**C-Fuel**
- Clothing should be worn with all fastenings properly closed.
- Eye-shields, rubber gloves, rubber boots.

**Some maximum allowable concentrations in air**

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<tr>
<th>Substance</th>
<th>Concentration</th>
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<tr>
<td>Nitrous fumes</td>
<td>5 p.p.m.</td>
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<tr>
<td>Ammonia</td>
<td>100 p.p.m.</td>
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<tr>
<td>Methanol</td>
<td>200 p.p.m.</td>
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<tr>
<td>Carbon monoxide</td>
<td>100 p.p.m.</td>
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Finally, if the contents of this paper in connection with rocket propellants has brought home to you as fellow doctors the significance of the trend of the future, it will be reassuring to know that we are preparing to meet our ever-increasing responsibilities and problems. In this connection it is felt that there is a need for Service Medical Officers to do short spells of duty at our Research Establishments.

Acknowledgment is made to the Ministry of Supply for permission to publish this paper.

"Enthusiasm is the element of success in everything. It is the light that leads and the strength that lifts men on and up in the great struggle of scientific pursuits and of professional labour. It robs endurance of difficulty and makes a pleasure of duty."—The Right Reverend W. Croswell Doane (1832-1913), Bishop of Albany, New York, U.S.A.