deserves further clinical investigation. As stated in the title, this article merely records our clinical impressions of surital. At present a comparable pentothal series is being conducted and it is hoped at a later date to publish an accurate comparison of these two agents.

SUMMARY

A new thiobarbiturate for intravenous anæsthesia has been discussed. Its use in 490 cases involving a wide variety of techniques has been described.

Decreased respiratory depression, laryngospasm and circulatory depression, increased potency and more rapid recovery, are its apparent advantages over intravenous barbiturates in current use.

Complications and contra-indications are the same as for pentothal.

The advantages displayed by this agent entitle it to further clinical trial.

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MEDICAL PROBLEMS IN CHEMICAL WARFARE*

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IN 1943 I predicted[1] that the task of delivering a successful chemical attack against the American people was so great that our enemies would not consider it worth trying. With the possible exception of the nerve gases, it seems equally unlikely today that chemical agents offer our potential enemies effective weapons for long range attack. The problems of civil defense against chemical attack, therefore, can be reduced to consideration of a single group of chemical agents, the nerve gases, until such time as an enemy may be able to establish a base at or within our borders.

The nerve gases were first developed by the Germans[2] but are now well known to both our allies and our potential enemies. They are a family of chemicals having the common property of irreversibly inhibiting the enzyme cholinesterase. They are nearly colourless, essentially odourless liquids, which yield toxic vapours on evaporation. More toxic than formerly known chemical warfare agents, they may gain entrance to the body by inhalation of the vapour or by absorption of the liquid agent through the skin, the eyes or the gastro-intestinal tract.[3]

The symptoms induced are due largely or entirely to the inactivation of cholinesterase. This leads to the accumulation of acetylcholine in both the central and the peripheral nervous system and to acetylcholine poisoning. Most of the classic symptoms of both muscarine and nicotine poisoning develop. In severe cases the excessive accumulation of acetylcholine at the myoneural junctions causes a curare-like flaccid paralysis.[4]

Man and experimental animals exhibit a rapid progression of essentially identical symptoms.[5a] Exposure to traces of the vapour causes pinpoint constriction of the pupils in a few minutes, usually accompanied with mild paroxysmal bronchoconstriction and a watery nasal discharge. A slightly greater exposure induces ciliary spasm, pain on focusing the eyes and a drawing sensation or pain in or back of the globes, radiating frontally or to the occiput, and is often accompanied with moderate photophobia.

At these low doses the paroxysmal bronchospasm does not produce anoxia, lasts only a few days and is readily relieved by small doses of atropine sulphate. The miosis, ciliary spasm and headache are more persistent and do not yield to the usual parenteral doses of atropine. The ophthalmic administration of homatropine hydrobromide is required for relief of mild cases, or repeated instillations of atropine, for the severer cases, until good mydriasis is obtained. The headache and eye pain are usually relieved promptly with the induction of mydriasis. The ophthalmic instillations may have to be repeated several times, as miosis and ciliary spasm frequently recur.[5]

The inhalation of larger doses of vapour, or the absorption of liquid nerve gas by other routes, causes a rapid and severe bronchospasm, which obstructs both inhalation and exhalation. The subject becomes confused and cyanotic, may

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have nausea and vomiting and soon falls unconscious.[5a] Meanwhile, his blood pressure falls to shock level; severe bradycardia develops, and cardiac arrest may occur as a temporary or terminal event (Fig. 1).[6]

If the subject can be given medical assistance before the anoxia is too profound and prolonged, large intravenous or intramuscular doses of atropine may completely reverse the cardiorespiratory condition. The bronchial tree relaxes; ventilation of the lungs becomes normal; anoxæmia is rapidly overcome; the slowed heart regains its rhythm and normal rate, and the blood pressure rises above normal and quickly drops again to normal level (Fig. 1).[6]

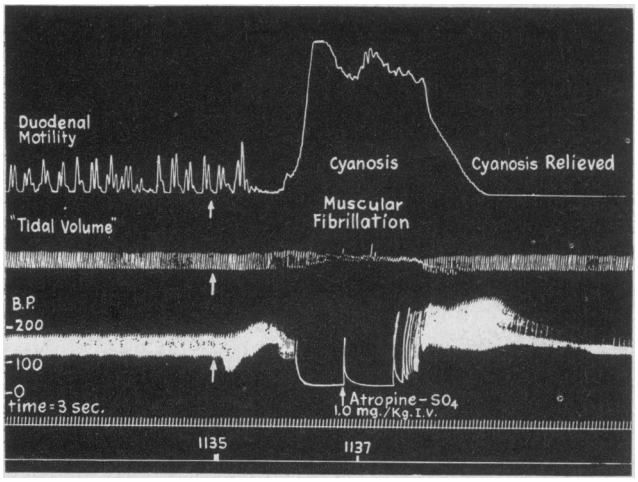

Fig. 1.—The effects of nerve gas and atropine on a dog. Nerve gas was injected intravenously into dog at first marker (arrow) and atropine sulphate at second marker. Upper, the effects on intestine (duodenum); middle, the effects on tidal air (under artificial respiration), and lower, the effects on blood pressure and heart rate.

Relatively large doses of atropine are required for the severe cases, and the principal danger lies in undertreatment. It is essential that the atropine be given by a route by which it reaches the circulation rapidly. Intravenous administration is preferable, from the standpoint both of rapidity of action and of ease of control of dosage. The intramuscular route may be used if the patient is not cold or in shock. Absorption from the subcutaneous and oral routes is too slow for the initial treatment.

Doses of 2 mgm. (1/30 grain) of atropine sulphate should be repeated every few minutes until the cardiorespiratory symptoms are relieved and some dryness of the mouth appears. The amount of atropine some patients can take without the development of atropinization is amazing. Thereafter, smaller oral or parenteral doses of atropine must be administered every few hours for at least several days, since the poisoning is far more persistent than the duration of atropine effects.[7]

Some of these patients will show nicotinic and central nervous system effects, which persist or appear after the muscarinic effects have been controlled with atropine. These effects range from muscular fasciculations and spasmodic twitchings possibly to grand mal seizures of clonic and tonic convulsions. The convulsions may be controlled, to the point that they do not threaten life, with thiopental sodium, trimethadione (tridione) or ether anæsthesia (Fig. 2).

Overdosage of thiopental (or any barbiturate) must be avoided, as it acts synergistically with the nerve gases in depressing respiration. A 20% solution of trimethadione, given intravenously in 1 gm. doses every 15 minutes, with a maximum dose of 5 gm., has the advantage of depressing cortical activity effectively without depressing respiration.[8]

If the severely affected patient cannot be treated promptly, profuse salivation, intestinal hypermotility and spasm and incontinence of urine and fæces will develop. The profound anoxia and increasing accumulation of acetylcholine in the nervous system lead to intermittent then almost continuous grand mal convulsions, until flaccid paralysis supervenes.[5a]

The use of atropine is dangerous in severe cases with profound and prolonged anoxia. In experimental animals the sudden release of the heart from vagal control, with the attendant increase in work by the cardiac muscle, in the presence of severe anoxia, leads immediately to ventricular fibrillation and death in a high percentage of the animals.[9] The administration of atropine in these cases should be delayed until the lungs have been ventilated and the heart has made some recovery from anoxia. The convulsive seizures can be controlled, or largely prevented from reappearing, by intravenously administered trimethadione or thiopental sodium, but the urgent problem in these cases is the paralysis of respiration.

At this late stage a very considerable relaxation of the bronchial tree occurs spontaneously, but respiratory paralysis prevents effective respiration. The paralysis is both central, due

to anoxia, and peripheral, due to muscle fatigue and the curare-like blocking of the myoneural junctions of the diaphragm and accessory muscles of respiration by excessive amounts of acetylcholine. The chest is flaccid and collapsed. The usual methods of artificial respiration, such as the Schaefer prone pressure method or the Eve tilt table method are ineffective or impractical.[5b] A new method suggested by Emerson[10] may be worth a trial in emergency, but there has not yet been sufficient work to assess its effectiveness properly. This method consists in placing the patient in a prone position, grasping his thighs at the level of the pubis and alternately lifting and lowering his hips 10 to 12 inches. Preliminary trials are said to indicate that this method may effectively ventilate the lungs in cases of flaccid paralysis of the respiratory muscles.[11]

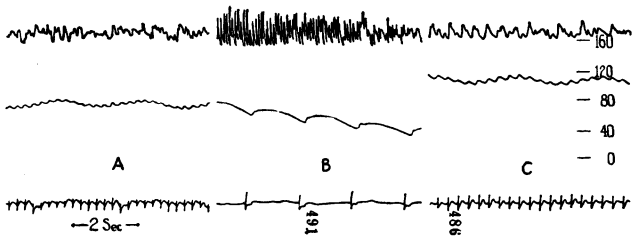

Fig. 2.—The effects of nerve gas and trasentine(R) methiodide on brain waves of a rabbit. Upper, electroencephalogram (motor cortex); middle, blood pressure, and lower, electrocardiogram. *A*, normal tracings; *B*, three minutes after the intracarotid injection of nerve gas (convulsive pattern, falling blood pressure, pronounced bradycardia), and *C*, thirty seconds after the intravenous injection of 2.5 mgm. per kilogram of body weight of trasentine(R) methiodide (restoration of normal electroencephalographic findings and circulation).

The use of an efficient mechanical resuscitator is probably the most practical and reliable method of giving artificial respiration in these cases, provided that the device can be got to the patient, or the patient to it, before irreversible anoxic damage occurs. A light, portable, hand-powered, bellows type resuscitator may be the most practical for emergency rescue work. Animal experiments indicate that 45 minutes of artificial respiration may be required to restore natural breathing after two or three lethal doses of nerve gas.[12]

If the skin should be splashed with liquid nerve gas, it is important to remove the contamination as soon as possible. The safest and most effective method is to swab the skin immediately with an alkaline fluid. Ammonia water, a 5 to 10% solution of sodium carbonate or a 1 to 2% solution of sodium hydroxide is suitable for this purpose. If none of these is available immediately, any available absorbent material may be damped with water and the area swabbed with this. Swabbing or rubbing the contaminated skin with dry materials must be avoided, as this greatly increases absorption and toxicity. If only dry absorbent material is available, the excess liquid may be gently blotted from the skin, provided wiping and rubbing are carefully avoided, but the contaminated area must be washed with soap and water or swabbed with an alkaline fluid as soon thereafter as possible.[13]

Clothing which is splashed with liquid nerve gas should be removed promptly and left outdoors. Patients should not be admitted to hospitals or other enclosed spaces until all liquid nerve gas contamination of skin and clothing has been eliminated, because the vapours from such contamination will endanger other patients and hospital personnel.

This information may be useful in dealing with poisoning by some of the newer insecticides, notably parathion and tetraethyl pyrophosphate (TEPP), which are also powerful though less dangerous anticholinesterases.[14]

ABSTRACT OF DISCUSSION

Dr. David Grob, Baltimore: The nerve gases are organic phosphate compounds whose pharmacologic and toxic effects arise from their ability to inhibit the cholinesterase enzymes present in all animal tissues. Two compounds in this group are useful therapeutic agents: di-isopropyl fluorophasphate (DFP), in the management of abdominal distension, urinary retention and glaucoma, and tetraethyl pyrophosphate (TEPP), in the management of myasthenia gravis. The toxic effects of the organic phosphate compounds in animals of all species has led to the use of some of them as agricultural insecticides, including parathion (O,O-diethyl O-pananitrophenyl thiophosphate tetraethyl pyrophosphate) and hexaethyl tetraphosphate (HETP). At least six deaths have followed accidental exposure to lethal amounts of parathion, and a few deaths have been due to di-isopropyl fluorophosphate, tetraethyl pyrophosphate and the nerve gases. Although the toxic and lethal doses of parathion, di-isopropyl fluorophosphate and tetraethyl pyrophosphate are larger than those of the nerve gases, and their volatility less, the experience gained with the former compounds has proved in general applicable to the nerve gases and may serve to acquaint the civilian population with the hazards of this group of compounds and to introduce physicians to the problem of recognition and management of their toxic effects. The effects of the nerve gases include muscarine-like, nicotine-like and central nervous system signs and symptoms. Absorption of the organic phosphate compounds may be by any route, and, since they do not produce local inflammatory changes, absorption may be undetected until symptoms begin, unless the cholinesterase activity of the plasma or red blood cells is determined. The danger of these compounds lies not only in the small doses that are lethal but also in the persistent effects of sublethal doses. After absorption of these compounds the cholinesterase enzymes are restored slowly over a period of many days, ap-

parently by the regeneration of new enzyme protein. For several days after exposure and until these enzymes are restored to normal activity, there is increased susceptibility to any repeated exposure. This cumulative action is particularly dangerous because there is only a moderate margin between the doses of these compounds that produce symptoms and the doses that are lethal, so that little or no warning may be given by impending serious effects. It is probable that the cholinesterase activity of the tissues may be considerably reduced before the appearance of warning symptoms, while a further reduction below the level compatible with normal function may result in pronounced symptoms and death. The precise cause of death from the organic phosphate compounds is not yet known, but contributing factors are believed to be depression of the respiratory and circulatory centres in the brain, weakness of the muscles of respiration and, particularly if the respiratory tract is one of the routes of absorption, bronchoconstriction and pulmonary œdema. The treatment of poisoning by these compounds relies chiefly on atropine, and on artificial respiration if respiration fails. Atropine has a moderate inhibitory effect on the muscarine-like manifestations of the organic phosphate compounds and a less striking effect on the central nervous system manifestations. In the presence of moderately severe symptoms due to these compounds, there is an increased tolerance for atropine, so that fairly large doses may be given. Studies are in progress to develop an agent which will block the muscarine-like, the nicotine-like and the central nervous system effects of the organic phosphate compounds more effectively than atropine and to develop a reliable skin decontaminating agent and improved methods of artificial respiration. The group of organic phosphate anticholinesterase compounds is being continually expanded by the synthesis of new, and in some instances, more potent compounds. Some members of this group will be increasingly useful in medicine and in agriculture, while others may someday be used as chemical warfare agents. Appreciation of the properties and hazards of these compounds not only will prevent the harmful results of their careless or indiscriminate use in peacetime pursuits but will also provide a background of preparedness in the eventuality of their use as chemical warfare agents.

Dr. George M. Lyon, Washington, D.C.: Colonel Wood's description of the physiological effects of the new nerve gases is useful because information regarding these effects has not been generally available. I agree with Colonel Wood that chemical attacks on our civil communities are unlikely and that, with the possible exception of the nerve gases, chemical agents do not offer potential enemies effective weapons for long range attack. However, an enemy might decide to use chemicals against combatant forces. Medical personnel must therefore be prepared to meet the problems which may be encountered in such a situation. Chemical warfare medicine has come to be an important phase of military medicine. It has no exact counterpart in civil medicine. In my opinion it is doubtful that the technological developments of modern warfare have in any way altered the basic responsibilities of the medical departments of the armed forces in this respect. The nerve gases, in addition to being highly toxic, pose complicated toxicological problems. These new agents present difficulties of treatment for all but the relatively mild cases. Furthermore, they add to the difficulties of medical logistics. I can visualize the problems which may be encountered in interpreting these new gases to non-medical personnel and in explaining the first aid and therapeutic indications to medical personnel. Medical leadership, well qualified and confidence inspiring, will be essential. During World War II, medical officers were required to discharge a variety of duties in connection with the medical aspects of chemical warfare. Perhaps the most important of these was in connection with medical and toxicological research. Closely allied to this was the assistance the medical officers gave to chemical officers in the interpretation of the medicomilitary significance of various chemical agents and in the preparation of training material relating to the medical aspects of chemical warfare. Medical officers were required in connection with industrial hygiene and care of accidental chemical casualties in plants were toxic chemicals were manufactured and processed, in training medical and non-medical personnel in the medical aspects of chemical warfare and not infrequently in instructing personnel of the line in chemical defence. They served in connection with matters of medical intelligence and chemical warfare intelligence and as specialist advisers in chemical warfare medicine on the staffs of major commands. How to prepare medical officers to meet these varied responsibilities poses a difficult problem. All medical officers should have a broad general knowledge of the medical aspects of chemical warfare, and a limited number should be trained to carry out more advanced or specialized duties of the nature described.

Dr. A. C. Ivy, Chicago: For our recent comparison of the various manual and mechanical methods for resuscitation, my associates and I obtained the bodies of patients without pulmonary disease, and we used the warm, non-rigid corpse to determine the pulmonary ventilation obtained by the various manual and mechanical methods. Normally, inspiration is an active process, and expiration is passive. But, in the corpse or in the apnœic person, force has to be applied to cause either inspiration or expiration; that is, air must be pulled into the lungs or out of the lungs, or both. Some of the various manual methods use the principle of pulling the air into the lungs. In the familiar Schaefer prone pressure method, the air is pushed out, and, as a result of the recoil of the chest wall, air is pulled in. In the Nielsen method, the patient is lying prone, and the operator lifts the arms, thus pulling the air into the lungs; then he drops the arms and presses down lightly on the scapulas, pushing the air out. If a combination of the Schaefer, Nielson and Drinker methods is used, with two operators, one operator lifts the arms to pull air in, and the other uses the Schaefer prone pressure method and pushes air out. If a combination of the Schaefer and the Emerson method is used, the operator pushes on the chest and then lifts or rolls the hips, which pulls air into the lungs. This is a combination of push and pull. When all the data on the manual methods were averaged and tabulated, it was found that those methods which use either a push or a pull procedure yield only one-half the pulmonary exchange obtained by a procedure which involves both push and pull. Otherwise, no statistically significant difference was found between the amounts of ventilation obtained by the various manual methods. The Emerson procedure is not a new one. It was suggested a number of years ago by Guy Thompson and several others; nevertheless, it was rediscovered by Emerson. The patient lies prone, and the operator assumes the position of the Schaefer prone pressure method and then lifts or rolls the hips of the patient. This procedure has been modified so that the operator assumes the Schaefer position (demonstrating position) and places his left knee next to the patient's left hip and his right foot next to the patient's right hip with the right leg flexed on the thigh; then he grasps the patient's hip bone on each side. In this position it is possible to lift or to roll the hips by lifting the right hip of the patient. We have found that is is necessary to lift the hips only 4 inches. In order to avoid fatigue due to lifting the hips, the right hip should be lifted in a rolling manner. Here the roll is caused by lifting with the right arm. When the right arm becomes tired, the left arm can be used. Lifting or rolling the hips yields a tidal air of around 225 c.c. But, if this procedure is used and then pressure is applied to the loin or the lower thorax, as in the Schaefer prone pressure method, a tidal air of about 500 c.c. will be obtained. The important point is that with a free airway any manual method which combines a push and pull procedure yields a tidal air of about 500 c.c. Mechanical respirators, when operated at the recommended rate and the recommended pressure, will yield a minimum volume per minute of about 5,000 or 6,000 c.c., or the same amount which can be obtained with a combination of the manual push and pull procedures. Ventilation of 5,000

or 6,000 c.c. per minute is sufficient; greater amounts will produce the undesirable effects of hyperventilation.

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CORTISONE IN THE TREATMENT OF LEUKÆMIA

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SINCE the introduction of cortisone by Hench and his co-workers, some evidence has been produced to show that the drug may act to inhibit the lymphocytic elements in the body. With this in mind, two cases of lymphatic leukæmia in children and a case of acute lymphatic leukæmia in an adult were treated with cortisone. A case of acute myelogenous leukæmia in a child was also treated.

CASE 1

O.P., aged 3½ years. This male patient was referred to the Saskatoon Cancer Clinic on June 10, 1950. The mother gave a history of noting a lump on the right side of the baby's neck, which increased slowly in size for two months. For two weeks prior to admission she had noticed a listlessness and loss of appetite. Physical examination showed a normally developed child with a sallow complexion who cried a great deal. There were three large nodes in the right posterior cervical chain, the largest being 3 cm. in diameter. These were discrete and rubbery. There were numerous pea-sized nodes in both supraclavicular triangles and in the left posterior cervical chain as well as in both axillæ and groins. The liver was enlarged one finger below the costal margin and the spleen was down two fingers. The remainder of the examination was negative.

Laboratory findings showed: hæmoglobin, 24%; red blood cells, 1,330,000; white blood cells, 12,500; lymphocytes, 81%; prolymphocytes, 9%; polymorphonuclears, 1%; rhabdocytes, 6%; platelets, 360,000. A sternal puncture showed: lymphocytes, 94%; prolymphocytes, 5%. No blast forms were seen.

A diagnosis of lymphatic leukæmia was made and 900 mgm. of cortisone were administered in seventeen days as shown in Table I. At no time was any reduction in size of spleen, liver or nodes observed.

COMMENT

(1) No evidence of increase in erythroblastic activity was shown. The red blood count continued to fall and reticulocyte counts remained low. (2) No significant change was noted in the white blood cell pattern, but there was a drop in the total white blood count from 15,000 to 1,600. (3) No significant clinical improvement was observed. (4) No evidence of cortisone toxicity was observed.

CASE 2

L.M., aged 2½ years, male. This patient was admitted to the Regina Cancer Clinic with a history of irritability, paleness, anorexia and increasing size of the abdomen, all of two weeks' duration.

Examination showed the skin and mucous membranes to be quite pale. The spleen was enlarged medially to the midline and descended three fingers below the costal margin. Numerous discrete rubbery nodes ranging in size up to 1.5 cm. were noted in both posterior cervical triangles as well as both axillæ and groins. Hæmatological examination showed: hæmoglobin, 32%; red blood cells, 1,057,000; white blood cells, 76,350; polymorphonuclears, 1%; lymphocytes, 92%. No blast forms were seen. The patient was given 500 c.c. of blood and transferred to the Saskatoon Cancer Clinic for treatment. A total dose of 900 mgm. of cortisone was given over a period of nineteen days with daily dosages and blood changes shown in Table II.

Tibial punctures were done before and after treatment.

	Before treatment	*After treatment*
Hæmoglobin	40%	67%
Red blood cells	2.1	3.4
White blood cells	36,000	10,450
Colour index	0.93	0.99
Lymphocytes	91.0	62.4
Prolymphocytes	7.0	4.7

PROGRESS NOTES

May 18.—The general good condition remained until sixteen days after completion of treatment when the patient developed a fever of 105, became restless, taking food poorly and developing pain in the right knee which was held in a position of flexion and felt quite warm. X-ray films revealed no evidence of osteomyelitis or periosteal reaction as seen in leukæmia. The liver and spleen were unchanged, but the lymph nodes which had disappeared were again enlarged. A course of penicillin was started, giving 500,000 units daily for six days.

May 22.—The patient is able to stand on both legs; the liver and spleen show an increase in size.

June 2.—The abdomen is markedly distended due to an increase in the size of the liver and spleen. His general condition appears to be deteriorating.

June 7.—Hæmoglobin, 70%; white blood count, 18,500; lymphocytes, 42%; prolymphocytes, 8%.

June 13.—Hæmoglobin, 41%; white blood count, 61,000.

June 21.—Hæmoglobin, 23%; white blood count, 93,000. A second course of cortisone was started giving 100 mgm. daily in divided doses. This was given for three days at which time the patient died.