The cellular reaction to peripheral injury was markedly depressed for several days, and there was a delay in the appearance of lymphocytes with an absence of eosinophils. Terminal bacteremia in a severely burned patient was detected at a time when blood bactericidal capacity began to decrease, a marked leukopenia occurred and the cell response to peripheral injury was depressed. Transient bacteremia sometimes occurred when there was a decreased cell response to periphery injury when the wound bacterial count was high in spite of an adequate blood bactericidal capacity. The balance between bacterial invasion and host resistance was continuously shifting. Blood stream invasion by Staph. aureus (phage type 80/81) was more likely when the initial high post-traumatic bactericidal effect had returned to normal. The primary cause of bacteremia in burned patients probably is the presence of a high bacteria/cell ratio in the wound, especially if the micro-organisms are pathogenic. -- Can. Med. Assn. J. Absts.

CHEMICAL HAZARDS

430 Nitrogen Tetroxide Exposure in the Missile Industry.
P. J. Clancy, S. L. Watson and J. B. Reardan. J. Occ. Med. 4, 691-693 (Nov. 1962).

Sixteen men were exposed to nitrogen tetroxide for 1-2 minutes. Most of the men subsequently experienced paroxysms of cough; they were given oxygen inhalation and recovered completely after 24 hours. One man became seriously ill. He was apneic and cyanotic on arrival at the aid station, where he was given oxygen and artificial respiration. His pulse was 102 and thready, his blood pressure 92/60, and his respirations feeble. Chest roentgenogram and laboratory tests showed no abnormality. Two hours after exposure the patient was complaining of chest pain and dizziness and had a dry, hacking cough. Five hours after the incident he was hospitalized. The chest film was again negative. The x-ray taken 18 hours after the exposure showed diffuse disseminated, nodular densities throughout the lung parenchyma. The second day after exposure there was some extension of the process. On the third day the symptoms were improved. The patient was afebrile and returned home. One month after exposure, the patient complained of a 10.9 kg. (24 lb.) weight loss, shortness of breath, chest pain, and occasional coughing-up of blood-tinged sputum. He was rehospitalized. X-rays showed definite fine nodular densities. He was given 80 units of corticotropin (ACTH) intravenously, and dexamethasone in a dose of 2.5 mg. 3 times /day; an ulcer diet was begun. Forty-eight hours after therapy was started, the roentgenogram showed a decrease in the size of the nodules. The patient was discharged on a reduced dosage of dexamethasone. Three weeks later he was deemed able to perform light work. About 3 months after exposure to nitrogen tetroxide the patient had no work intolerance and his lung function was within normal limits.
-- Cond. from J. Am. Med. Assn. References & Reviews

431 Polyoxyethylene(8)-Stearate. Carcinogenic Studies. W. C. Hueper and W. W. Payne. Arch. Environmental Health 6, 484-494 (Apr. 1963).

The experimental evidence presented and interpreted in the light of available competent human and experimental experience indicates that bladder stones, irrespective of their chemical composition, are not primary causes of cancer of the bladder. The observations reported confirm the previously reported finding that rats fed polyoxyethylene(8)-stearate at a 25% level develop benign and malignant tumors of the bladder. They demonstrate, moreover, that this chemical directly introduced into the bladder of rats has a like effect and that mice fed this emulsifier also develop papillomas and carcinomas of the bladder mucosa. Polyoxyethylene(8)-stearate must be considered to be a primary carcinogen which owes its carcinogenic property either due to a direct action of one or several ingredients present in this chemical mixture when excreted with the urine or due to a carcinogenic metabolite produced from one of the various chemicals composing this mixture. Different batches of polyoxyethylene(8)-stearate vary in their relative carcinogenic potency. Such fluctuations apparently are attributable to a lack of uniformity in the chemical composition of this product. Because of this insufficient standardization of the

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