PNEUMONITIS FOLLOWING ASPIRATION OF CRUDE OIL AND ITS TREATMENT BY STEROID HORMONES

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This is a report of a patient who aspirated large amounts of crude oil into his lungs and his treatment by steroid hormones. The patient was 39 years old. On August 9, 1954, he and a fellow-worker approached a 13,000 gallon tank containing 8 inches of crude oil #5 at its bottom to clean it. The fellow-worker entered the tank and was seen to fall unconscious into the oil. My patient went to his rescue, turned him on his back, and then collapsed himself, falling on his right side with his mouth open and one nostril under the oil and one above the level of the oil: He remained there 15 minutes until rescued by firemen wearing special masks.

On the left of Chart I are listed the products which emanate from a petroleum fractionating column in the order of their volatility.[1] Thus, at the top are the fuel gases, the alcohols, gasolines, kerosenes, heating or crude oils, which are subdivided into 10 degrees of viscosity and of which the oil in our tank was #5—a thick, black, heavy oil. Then come the lubricating oils, waxes, residual oils and asphalt. The more volatile the product, the more acute and extensive is its damaging action on pulmonary tissue and the quicker is it removed from the body. Conversely, the less volatile products cause less acute reaction in the lung, but remain in the lung for longer periods of time. The more volatile products are known to cause central nervous system depression and undoubtedly the fumes breathed by these men accounted for their coma. The oil aspirated by my patient is believed to have had some kerosene in it which is known to cause severe pulmonary damage in a matter of minutes.[2-8] The large bulk of it was of a cruder mixture responsible for the prolonged irritant effect to the lungs which will be observed in this case and which proved to be correspondingly slow in its evacuation from the body.

The patient was taken to the Boston City Hospital where he was revived from coma and shock after several hours and treated with oxygen and penicillin. He was extremely sick, had constant cough productive of blood-tinged oil-flecked sputum, was cyanotic and had a high fever. The X-rays here shown reveal the pneumonitis on the right (he had been lying on his right side) which was observed at that hospital.

Nine days later, on August 17, he was transferred to the Faulkner Hospital where his wife worked as a technician. On entry he appeared extremely ill, was wracked by a constant cough productive of oil and some

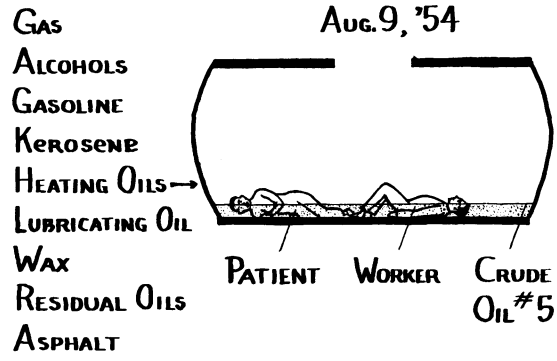
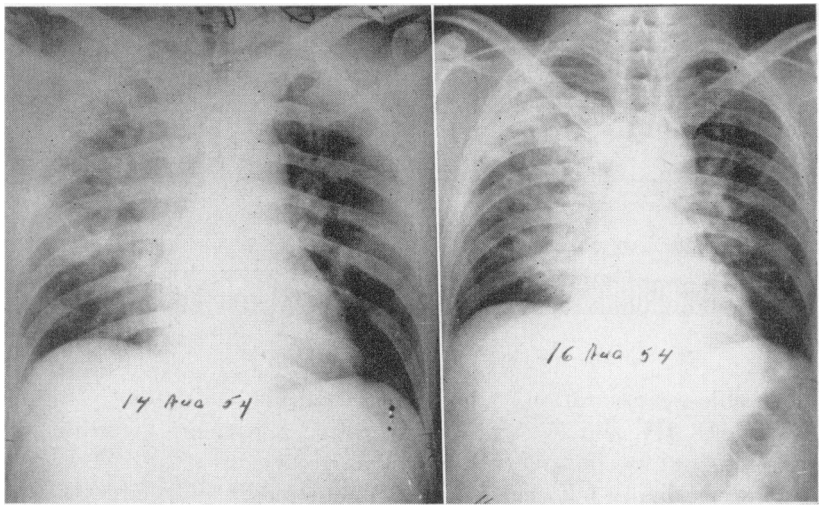


Chart I.



blood, was cyanotic, dyspneic, and had a temperature of 102° and pulse of 120.

There were signs of consolidation in his right mid-lung field, râles all over the right lung, and some on the left. Urine and stools were normal, white blood count was 32,000. Sputum showed a mixed flora of coliform, alpha streptococcal, and catarrhalis organisms. Microscopic examination of the sputum showed blood, polymorphs, and black droplets of oil surrounded by histiocytes. His vital capacity was 1.2 liters out of an expected 4.1 liters. He had a penicillin rash and was consequently switched to achromycin, streptomycin, and later chloromycetin, depending on sensitivity studies. None of these antibiotics seemed to have any effect on his condition.

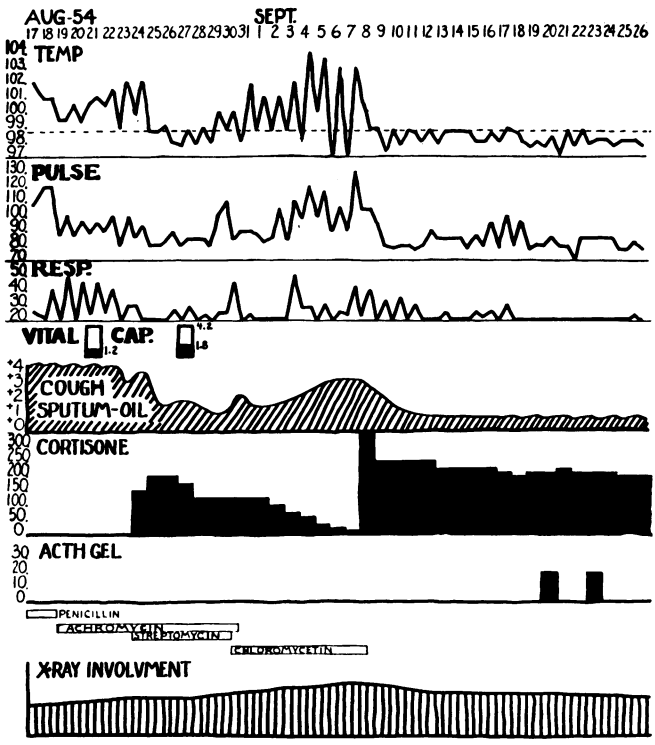


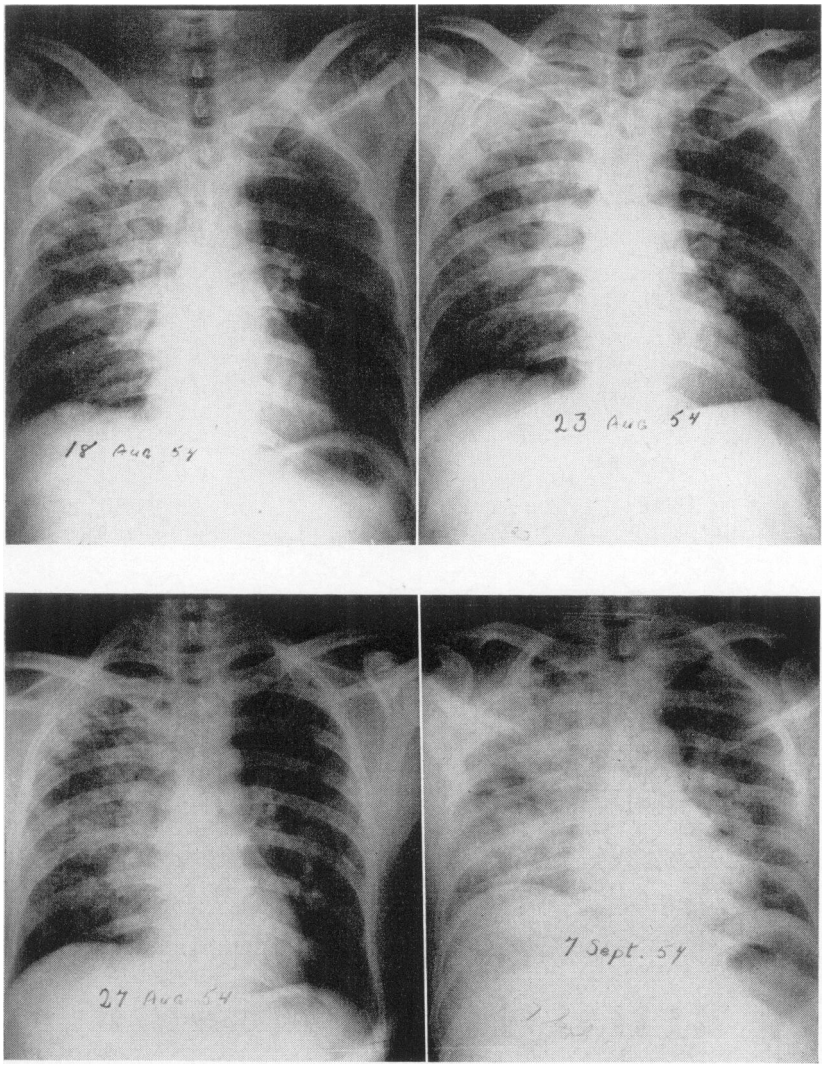
CHART II.

A graphic representation of the clinical course of this patient is recorded in Charts II, III, and IV, and representative X-rays are herewith reproduced, taken at significant points in his progress.

Having originally felt that his pneumonitis might be in large part bacterial, but having been disappointed in his response to antibiotics, Dr. Samuel Cohen, who had been called in consultation, and I decided to attempt to allay the chemical pneumonitis with cortisone. Dr. Albert Seeler and Dr. Harriet Hardy, specialists in industrial medicine, although they had not met this particular problem before, felt this was worth a trial.

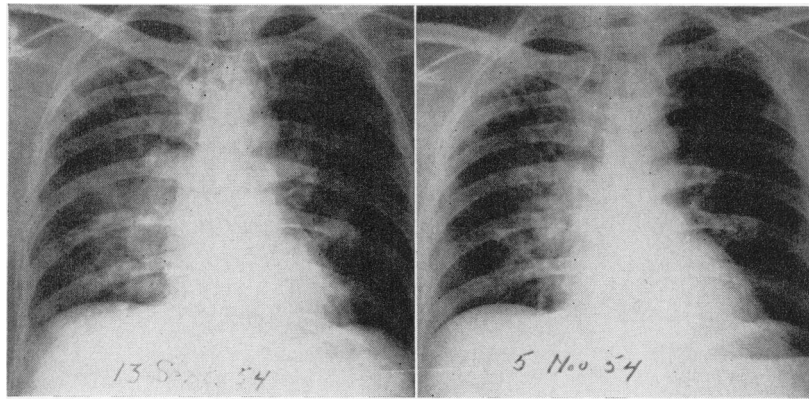
You will see by Chart II that the overt clinical manifestations of his illness were immediately benefited. His temperature, pulse, and respiration became normal and his cough and sputum production decreased markedly.

His X-ray picture, however, did not show improvement, and fearing that bacterial spread during cortisone therapy might be fatal, we reduced and finally discontinued the cortisone. As this was done, the patient became clinically sicker and sicker. The X-ray dated September 7 shows the state of his lung as the cortisone was removed.



At this point, Dr. Maxwell Finland was consulted and it was decided to reinstitute cortisone therapy and omit antibiotics, keeping a sharp watch the while on his sputum and blood cultures. You will note the immediate clinical response which was again obtained. The concept then developed that we should give just enough steroid to keep the patient clinically well, but still coughing, so that he might eventually get rid of the oil.

This policy was pursued with good effect. ACTH was added twice a week to keep his adrenal cortex stimulated since we did not know how long



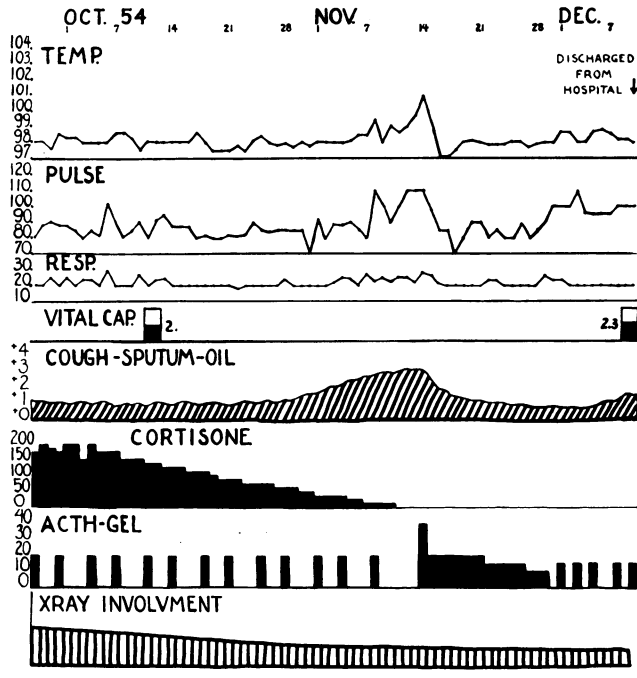
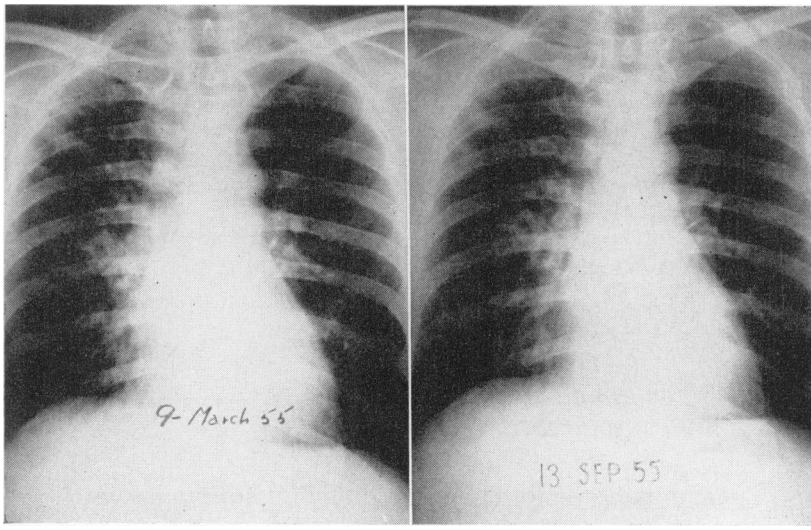


CHART III.

cortisone therapy was to be continued. Over a period of six weeks cortisone was reduced gradually. Before it was eliminated a febrile episode, and a return of chest signs, cough and more oil production, recurred. These were quickly suppressed by more constant use of ACTH. The patient appeared so well that he was allowed to go home on December 10. His vital capacity was now 2.3 liters.



Over seven more months his ACTH was continued. A flare-up of cough and sputum occasionally demanded a short increase in ACTH. Cough, sputum, and fever could be titrated against ACTH dosage in inverse proportion throughout his course. In February 1955 he returned to activities on a part time basis. (He was studying to be a teacher at Boston University.) After examinations were over and vacation had begun, we gradually eliminated ACTH. He no longer brought up oil with his slight cough. Gradually his cough itself stopped. He has now been off ACTH for four months and is well, carrying a full schedule at Boston University. His vital capacity is now 3.2 liters, and his X-ray dated September 13, 1955 reveals some residual fibrosis in his right lung, but represents a marked improvement over films taken a year previously.

This patient raises several points for consideration. Selye[9] has shown that inflammation localizes the action of an irritant or toxin. If the toxin is one which by spreading may be lethal to large areas of neighboring tissue, inflammation serves a useful purpose by putting up a barrier. On the other hand, if the irritant is not terribly damaging but stirs up a lot of inflammatory response, especially in the lung, the inflammation itself may be more threatening to life than the irritant.

It would seem that in the case of crude oil #5, the latter condition prevails. It may also prevail for other hydrocarbons lower on the scale of toxicity, such as mineral oil. In the case of the more acutely damaging hydrocarbons, such as gasoline and kerosene, a question remains as to whether inflammation serves a useful or detrimental rôle and as to whether its suppression by steroids is indicated or not. This is a question which probably

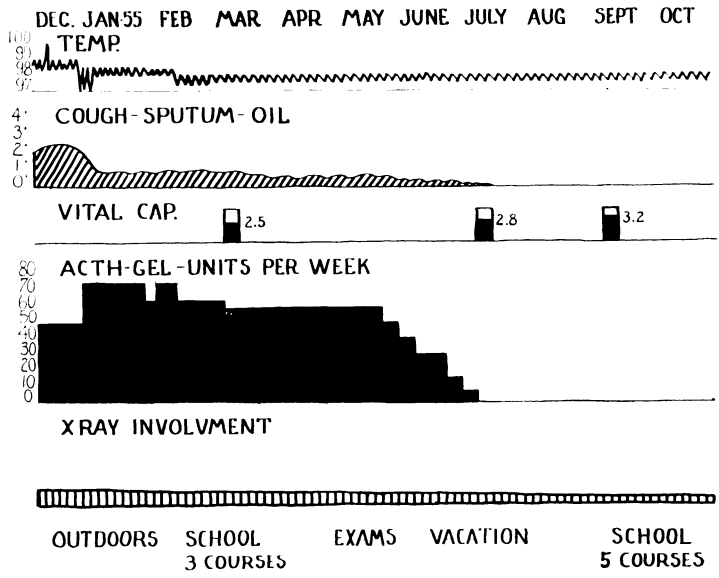


CHART IV.

can be solved by animal experimentation and possibly by the use of cortisone in some desperate human clinical example of kerosene poisoning—of which there are still far too many occurring, especially in rural areas, among small children.[2-8]

This case suggests the probable usefulness of steroids in treating other industrial accidents involving similar products, and also their possible usefulness in merchant seamen and sailors who are subject to aspirating crude oils in sea disasters and who have been thought hitherto to die of exposure.

A final question is raised as to whether these hydrocarbons will eventually prove to have a carcinogenic effect on the pulmonary tissue to which they were so long exposed. I would welcome opinion on this point.

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DISCUSSION

DR. THEODORE L. BADGER (Boston): There is probably no parallel; but it is very interesting to observe that, in bailing out a severe asthmatic, 200 mg. of cortisone per day are necessary, for an average of five or six days, in order to get a diminution and disappearance of symptoms. It is interesting to note here that, while this patient was treated with smaller doses of the steroids, he made some improvement and, although the type of disease is entirely different, it is important here that, when he was started with big doses of first one and then the other, he showed his first real improvement.

DR. RICHARD A. KERN (Philadelphia): I should like to ask whether bronchoscopic aspiration was attempted during the early days of his illness.

DR. WALTER PALMER (Chicago): I want to add a note to Dr. Badger's comment with respect to "bailing out" and dosage.

In terms of the amount of steroids we have found it necessary to use in extreme situations in ulcerative colitis, 300 mg. of cortisone is not a big dose.

DR. HENRY M. THOMAS, JR. (Baltimore): I would like to comment on just one small point. Dr. Graham mentioned pneumonia produced by other oils, bland ones such as mineral oil.

We have encountered, I think, five cases now. We reported the first one of an inhalation-oil pneumonia, which was a very benign disease; in fact, asymptomatic, and discovered only through accidental x-ray, then thought to be a tumor of the lung, and removed surgically, at which time it was found to be an oil-aspiration granuloma. A lipoid-cell pneumonia, if you like, which is somewhat different, and in which the oil is not coughed up except in small amounts, mostly remaining and producing a chronic inflammatory disease; but nothing similar to this case of yours.

Then, also, I was fascinated by this very good first slide which you showed; and I wondered how the one nostril happened to stay out of water. That was a very fortunate thing.

DR. JOHN R. GRAHAM (Closing): Dr. Thomas, we had to reconstruct the story of what happened to this man, from a description as given by the firemen who rescued him. All I can say is that this was their description. It was extremely fortunate, and gave him a 50 per cent chance.

I am also interested in your mentioning the pneumonia resulting from the inhalation of a more benign type of oil.

It is difficult to find too much about this sort of thing in the literature; but one does get the concept that the more volatile the product of the fractionating petroleum column is, the more toxic it is when applied to pulmonary tissue.

I was particularly interested to find out how prevalent kerosene poisoning is in the rural areas. Apparently, in some areas, it is the most common cause of death from accidental poisoning among children. Quite a large series of cases have been collected in some hospitals over the course of two or three years, with a mortality ranging from 2 to 9 per cent. There are some other series in which there are no cases which died.

Kerosene poisoning, especially if the kerosene is aspirated into the lungs, can be a very acute and even fatal disease. There is interesting animal experimentation which has been done, which more or less goes to show that the toxicity of the kerosene comes when it gets into the lungs by direct aspiration or by aspiration of vomitus containing kerosene; and that the coma and central nervous-system involvement result from absorption from kerosene that gets into the intestinal tract. A baby may take as much as a pint by mouth and, if he does not cough or get it into his lungs, may suffer no serious consequences except drowsiness, while as much as a teaspoonful getting into the bronchi may prove to be fatal.

I would like to make one point because, actually, it is kerosene poisoning I find myself most interested in, in studying this case. One of the features which has come out in that disease is that, contrary to the usual procedure in treating a case of poisoning, it would become apparent that perhaps it is better to leave the kerosene in the stomach of the child who has ingested some, rather than try to wash it out, because about 50 per cent of children vomit in the process of gastric lavage and then get the kerosene into their bronchi, where it causes possibly fatal damage to pulmonary tissue. I have even wondered whether thorazine might be used in small quantities to prevent spontaneous vomiting.

In response to Dr. Kern's question, the patient was not bronchoscoped. I would gather that, in the first week at the Boston City Hospital, he was just too sick to do almost anything with. He was very critically sick, more so than the chart would suggest.

When he was brought to our hospital, we felt that perhaps the time had gone by. We began to change our minds about that, and worry about whether we should or should not bronchoscope him. We ended up by not doing so. We tried all sorts of other measures: inhalation of isoprel and administration of mists with pancreatic enzymes, and all that sort of thing. But we felt that none of those measures really produced any significant effect.

As for Dr. Badger's and Dr. Palmer's comments on the dosages of cortisone, I am sure we should have given the patient more the first time we tried it. It was quite obvious that he was not getting enough; and, as we reduced it, his symptoms all returned with increasing vigor. It was only when we used considerably larger amounts that we got the desirable clinical effect.