which belongs particularly to chronic appendicitis, I call the "cider barrel sign." If we percuss the left side of the abdomen in a case of chronic appendicitis, we shall obtain a normal resonance note suggesting percussion of the cider barrel in October. If we percuss the right side of the abdomen, we obtain a percussion note suggesting that of the cider barrel in January.

These two points, then, the "cider barrel sign" and the point of tenderness on pressure to the right of the navel, appear to be important in distinguishing between chronic appendicitis and pelvic disorders of the sort that interest the gynecologist.

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A METHOD FOR FILING NECROPSY SECTIONS

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The ordinary methods of storing microscopic slides of necropsy sections in drawers or in boxes has always seemed to me not only an awkward method, but one which renders the sections very inaccessible. I wish, therefore, to submit the following means of adapting the ordinary filing cabinet

Adaptation of the ordinary filing cabinet to the filing of microscopic specimens of necropsy sections.

to the filing of microscopic specimens of necropsy sections in a convenient and readily accessible form.

Our filing cabinet takes a card $9\frac{1}{4}$ by $7\frac{1}{2}$ inches. One of the local publishing houses furnished us with folders of this size. The paper used is 140 pound tag manilla, cut to a size $9\frac{1}{4}$ inches square. One margin is then turned over for a distance of $1\frac{3}{4}$ inches, and wire staples eleven-sixteenths of an inch long put in so as to divide the folded strip into six equal compartments. In this way a folder which is $9\frac{1}{4}$ inches wide by $7\frac{1}{2}$ inches high is made. A strip of the same material $1\frac{3}{4}$ inches wide, and of the same length as the width of the folder, is stapled in position so that the lower border is $3\frac{3}{4}$ inches above the folded lower border of the folder. This strip not only has the seven staples which divide it off into equal compartments, but also a staple at the lower part of each compartment, thus making space for the filing of twelve sections, and leaving room at the top for the number, name, date, and also a description of the principal pathologic lesion that has been revealed by the necropsy. There is sufficient room on the back of the folder for writing the complete anatomic diagnosis.

These folders are filed away in rotation, and can be cross indexed according to name and pathologic lesion. A cabinet 22 inches deep will hold about 250 of these folders when filled with microscopic slides.

Military Medicine and Surgery

INDUSTRIAL POISONING IN AIRCRAFT MANUFACTURE

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In the latter part of 1914, Jungfer¹ reported a new industrial disease that had appeared during the preceding year in an aeroplane factory in Johannisthal and had affected four out of a force of eight men. These men were employed in painting and spraying a solution of cellulose acetate over the linen that covers the aeroplane. All of them developed a severe acute hepatitis, and one died. Later in another factory in the same place ten men were poisoned and one died. Grimm,² writing shortly after, was able to give details of fifteen cases of poisoning among aircraft employees engaged in such work. The solutions used were sent to the pharmacologic institute of the University of Berlin for analysis by Heffter and Joachimoglu,³ who reported that the toxic constituent was tetrachlorethane, of which some specimens contained as much as eighty-four in a hundred parts. They experimented on dogs, rubbing the solutions into the skin and also exposing the animals to the fumes. From the commercial mixtures and from pure tetrachlorethane they obtained the same results, namely, loss of weight, vomiting, presence of bile and blood in the urine, death with fatty degeneration of the liver, and hemorrhages into serous and mucous surfaces and in the heart muscle. In inhalation experiments narcosis came on, but was slower in appearance and less deep and lasting as the days went on, showing a gradually increasing tolerance on the part of the animals. The symptoms described above, however, put in their appearance and the course of the poisoning was the same, no matter how the compound was administered.

Tetrachlorethane had already been tested by Lehmann⁴ of Wurzburg, the authority on industrial gases and vapors. As always, his experiments were for the purpose of determining the minimum dangerous amount of the vapor in air, and he found that air containing no more than 1 or 2 mg. of tetrachlorethane to a liter could be breathed for a relatively long period—six or seven hours a day for eighteen days spaced over four weeks—with no more serious result than loss of weight and drowsiness. If the fumes were stronger, narcosis came on, and after repeated exposures, serious disturbances of metabolism. In fatal poisoning he found solution of red corpuscles, basophilia, fatty liver, the heart muscle yellowish and the kidneys congested. He found it the most poisonous of the chlorin derivatives of the hydrocarbons, four times as poisonous as chloroform and nine times as much so as tetrachlormethane (carbon tetrachlorid).

The fatty degeneration of the liver as found by these German experimenters is more marked than that produced by any known substance except phosphorus, and as in phosphorus poisoning, it is associated with a yellow, flabby heart muscle, and with acute degenerative

1. Jungfer: Zentralbl. f. Gewerbehyg., 1914, 2, 222.

2. Grimm, Heffter and Joachimoglu: Vrtljschr. f. gerichtl. Med., Series 3, 1914, 48, supplement, p. 192.

3. Heffter and Joachimoglu: Vrtljschr. f. gerichtl. Med., Series 3, 1914, 48, supplement.

4. Lehmann, K. B.: Arch. f. Hyg., 1911, 74.

changes in the kidney epithelium. It also resembles delayed chloroform poisoning with its hematogenous jaundice. Tetrachlorethane is seven times as hemolytic for mammalian blood as is chloroform.

POISONING ABROAD

The first case of tetrachlorethane poisoning reported from England was in October, 1914, when a man died of acute jaundice after working in an aeroplane factory in Hendon. He had worked eleven weeks when jaundice developed, and he died after three weeks' illness. The liver was shrunken and green. Legge, chief medical inspector of factories, went there to investigate, and found that the man had been applying to aeroplanes what is called in English and American factories "dope." On examining the other dopers, Legge found ten with evidence of inflammation of the liver. They had worked from three to sixteen weeks before they were taken sick. All had gastric symptoms and jaundice. The next three deaths from what was now called "dope poisoning" were in women, two girls under 20 and a woman over 30. By February, 1916, the Home Office had had reports of forty-three cases, with seven deaths, five in women. The amount of tetrachlorethane in the dopes varied, but British factory inspectors considered even 10 per cent. dangerous.

The dopes were subjected to analysis and animal tests by Wilcox,⁵ who also examined the organs of some of the victims of dope poisoning. His results were exactly the same as those of the Germans. Though the English dopes contained benzene, methyl alcohol and acetone as well as tetrachlorethane, Wilcox proved that the last was the cause of dope poisoning. He described, as the distinctive features of this form of hematogenous jaundice in man, a slow, insidious onset; long duration of the disease after the onset of jaundice, longer than in acute yellow atrophy of the liver; absence of marked fever, which distinguishes it from infectious jaundice, Weil's disease; and absence of the degree of anemia that would be found in arsenical jaundice. The jaundice is deeper than that of delayed chloroform poisoning.

The last paper on the subject of dope poisoning in Germany appeared early in 1915. Koelsch,⁶ a *Landes-gewerbearzt* of Bavaria, made a study of industrial poisoning from tetrachlorethane, and divided the cases into two classes: (a) those with pronounced gastrointestinal symptoms, nausea, vomiting, cramplike pain, enlarged liver and jaundice; and (b) those with slight or no abdominal disturbance, but with various nervous symptoms, headache, paresthesias, tremors of the hands, disordered reflexes and slight paralyses. Two cases belonging to the first group had been diagnosed as lead colic, though there had been no exposure to lead. Other symptoms noted by Koelsch were increased craving for food coincident with loss of weight, and great drowsiness, persisting all of the working hours.

After this, reports from Germany ceased to come, and we have no way of knowing whether or not the advice of the factory inspectors was followed and tetrachlorethane dope forbidden. In England this has been virtually effected. Toxic jaundice from aeroplane dope was made a reportable disease in February, 1916, and brought under the workmen's compensation act. By August, 1916, the Home Office was able to state

that dopes free from tetrachlorethane were now obtainable and approved by the War Office and Admiralty. Letters from Sir Thomas Oliver and Dr. T. M. Legge, written in January of this year, assured me that tetrachlorethane dopes were no longer used in British aeroplane manufacture.

THE SITUATION IN THIS COUNTRY

That is the history of dope poisoning in the older countries. In our country, despite the fact that since the war our aeroplane manufacture has increased rapidly, no case of dope poisoning has come to public knowledge. The Bureau of Labor Statistics, knowing the serious trouble that England had experienced, and wishing if possible to forestall it here, asked me to make an investigation of our aeroplane works during the spring and summer of this year. At that time the output of these factories was largely training planes, not war planes. I visited eighteen factories, in the Middle West, Florida, New York State, New England and northern New Jersey. Everything was in a state of experiment and of rapid expansion. Many of the plants were small, temporary structures, and all the doping was done outdoors. Others had doping sheds, open to all the winds. Few were large enough to employ more than one man in doping, and even he would not spend all his time at the work. It was easy to see why, even with the dangerous tetrachlorethane dope, which was still in use in some plants and had been used in several more, no case of toxic jaundice had developed. The exposure had not been long enough or severe enough. Dopers are a shifting body of men. Of fifty-three men interviewed, more than half had worked less than three months.

All these features, however, were already changing. The new factories had doping rooms and were preparing for work on a large scale. It was evident that in the future many more dopers would be employed, and that they would work continuously and in an atmosphere full of dope fumes from the drying wings. The men in charge told me that they knew doping was attended with danger, but for the most part they did not know which were the dangerous compounds in the dope or which variety was the more dangerous. I felt convinced that with conditions as they were in some of the new factories, this winter would see several cases of dope poisoning if tetrachlorethane dopes were to be used; for instead of installing down-draft suction to remove the heavy fumes, several managers were trusting to window ventilation only.⁷

Though I did not find evidence of toxic jaundice among dopers, men who worked with tetrachlorethane dopes had felt the effects of the fumes somewhat. Drowsiness was oftenest complained of, most troublesome early in the morning, and passing off as the day wore on; worse after a holiday, as if the immunity acquired had been lost, and much worse in hot, heavy weather. Some men said they were even sleepier after leaving work than while in the fumes.

There were instances of men being actually overcome by the dope fumes, but these were under exceptional circumstances, such as very hot weather and an unusual rush of work; or the doping was carried on in a greenhouse with the sun beating on the glass roof; or the

5. Wilcox: *Lancet*, London, 1915, **1**, 544.

6. Koelsch: *München. med. Wchnschr.*, 1915, **62**, 1567.

7. An investigation of aeroplane factories in New York State was undertaken by the State Industrial Commission and entrusted to Drs. Lester Roos and Rosalie Bell. They found one man using tetrachlorethane who showed a slight jaundice, but whose symptoms were chiefly nervous: dizziness, sleepiness, and attacks like alcoholic intoxication.

man had lain on his back to dope the under side of a wing and had breathed the fumes that fell directly in his face. Two dopers were rather seriously "knocked out" when a pail of dope was spilled over the clothing of one, and the other had gone to his assistance and tried to drag him outdoors. Both lost consciousness, and the one with the soaked clothing had an alarming respiratory failure requiring artificial respiration for some minutes. Neither had any lasting effect from the accident. Nausea and headache usually follow such an attack of faintness, but no symptoms of abdominal disturbance of any seriousness were reported to me. Constipation was complained of only occasionally. Two men spoke of an increased craving for food at the same time that they were losing weight.

At the time I made my inquiry, between April and September, 1917, two kinds of dope were in use, cellulose acetate and cellulose nitrate. The former is relatively noninflammable, and is used on war planes. Its best solvent is tetrachlorethane, to which is usually added some acetone, benzene and alcohol. Cellulose nitrate is inflammable and must be covered with a noninflammable varnish; but even with this it is not fit to be used on war planes, though it is all right for planes used in training. The majority of the factories at that time were making training planes and doping with nitrate dope. A few used acetate dope with tetrachlorethane. Now we are beginning to make war planes, and the dope advocated by our Signal Corps, the dope that will probably be generally adopted for war planes, is made of cellulose acetate dissolved in acetone, alcohol and benzene, or the acetone may be replaced by a mixture of methyl alcohol, methyl acetate and acetone. This means that the dangerous tetrachlorethane is gradually disappearing from our airplane works, and that the solvents encountered by dopers from now on will be benzene, methyl alcohol, acetone and other ketones, amyl acetate, and perhaps amyl alcohol. All these are volatile substances, capable of setting up industrial poisoning because they can be absorbed through the respiratory tract as well as by the intestinal tract; but their toxicity differs greatly in degree.

Benzene is much the most toxic, but there is not more than 12 or 15 per cent. of benzene in most dope, and from that amount one would look not for severe acute poisoning, but rather for the lighter forms, as dizziness, headache and transient excitement like drunkenness, followed by depression and nausea—the range of symptoms which rubber workers describe under the term "naphtha jag," and which dopers also describe fairly frequently. Probably chronic benzene poisoning may begin to appear now, for there will be much longer exposure to the effects of the vapors than heretofore.

There is a decided prejudice against amyl acetate among workmen, and many of them told me that it was much more poisonous than tetrachlorethane. Physicians sometimes believe this, and think that a change for the better is made when the foreman gives up the disagreeable "banana oil" and substitutes acetate dope with tetrachlorethane. Amyl acetate does cause smarting of the eyes, and sometimes conjunctivitis, and there may be an irritating cough; but so far we have little evidence of its danger as an industrial poison. Koelsch⁸ went carefully into the question of amyl acetate poisoning among men using it in varnishes and

lacquers, and was convinced of its slight harmfulness. He then experimented on himself, vaporizing it and breathing the fumes. He had an inclination to cough, a feeling of heat in the head, and a slightly rapid pulse. Later he became dizzy, drowsy and confused and tired, with quick, deep respirations. He found it hard to climb the stairs after he had gone home.

Koelsch also tested amyl acetate on animals, and though the symptoms caused by it were transient, yet after a long exposure (237 and 340 days) the animals lost weight and died, with fatty liver, and edema of the lungs with areas of pneumonia. Lehmann's⁹ animals also reacted to large doses of the vapors, but showed no permanent lesions. He did not, however, expose them as long as Koelsch did his.

It would seem, then, that workers exposed to amyl acetate may suffer from smarting eyes, dry throat, obstinate cough, dizziness, drowsiness and weariness, and it is possible that long continued exposure may produce changes in organs, especially in the liver.

According to our present knowledge, acetone is harmless; but very little work has been done, and the matter is far from settled. Amyl alcohol (fusel oil), according to Salant,¹⁰ is seven times as toxic for frogs as ethyl alcohol, and four times as much so for rabbits. In human beings its effects are like those of ethyl alcohol; but it is more rapid, severer, and accompanied by a more decided fall of blood pressure.

Among these solvents for dope, there remains to be considered methyl alcohol. The toxicity of this compound is very much a matter of controversy. We know, from Reid Hunt's¹¹ experiments, that pure methyl alcohol produces all the characteristic symptoms of wood alcohol poisoning, and that they are not to be attributed to the impurities only; but we do not know what proportion of it is dangerous. Our denatured alcohol usually contains about 4 per cent., and it is assumed that this is well within the danger limits; but German denatured spirits contain only 2 per cent., and yet there has been so much complaint of sickness among workmen caused by denatured alcohol that, according to Goldschmidt,¹² petitions have been sent to both the Reichstag and the Bundesrath for an inquiry into its harmfulness. Apparently individual susceptibility to wood alcohol varies enormously. The serious action on the eye makes industrial poisoning from methyl alcohol specially to be dreaded.

SUMMARY

It seems that we need not dread the "toxic jaundice" that attracted so much attention in England and Germany, for we shall probably not use tetrachlorethane dope to any great extent. Our dopers will be exposed to the fumes of benzene, methyl alcohol and other alcohols, acetone (dimethyl ketone) and other ketones, and amyl acetate. Of these we know that the first two are industrial poisons of distinct danger; the others are less known and probably much less dangerous. We ought to insist, however, on abundant ventilation for doping rooms, as the English do, and it would be an excellent thing if we also followed this example in insisting on regular medical examination of dopers, for then we should be able to gather the much needed data concerning the less well known of these dope solvents.

Hull-House.

9. Lehmann: Arch. f. Hyg., 1913, **78-79**, 260.

10. Salant: Proc. Soc. Exper. Biol. and Med., 1909-1910, **7**, 134.

11. Hunt, Reid: Bull. Johns Hopkins Hosp., 1902, **13**, 137.

12. Goldschmidt: Weyl's Handbuch der Hygiene, **8**.

8. Koelsch: Concordia, 1912, **19**, 246.