zygotes were male, while all the abnormal metabolic ones were female. It would seem that abnormal growth is dominant in the male and recessive in the female; while abnormal metabolism may be dominant in the female and recessive in the male. But, of course, the observations are far too few to draw such general conclusions, and many more families and individuals will have to be studied to obtain sufficient data of value.

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INDUSTRIAL ANILIN POISONING IN THE UNITED STATES

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Industrial poisoning from anilin and substances closely allied to it has long been recognized in Germany, where the manufacture of anilin dyes is an enormously important industry, and the possible effects on the workmen of the various substances used or evolved during such manufacture has there been a subject of study since the sixties. More recently the growth of the dye industry in Great Britain has led to reports in that country concerning cases of anilin and of nitrobenzene poisoning.¹ As we know, there was no attempt in this country to compete with Germany in the manufacture of anilin colors or of anilin itself till the war shut off the supply and forced us to begin to make for ourselves what had heretofore been obtained from Germany. Uncertainty as to the duration of the war and lack of skill in these complicated processes delayed for many months the equipment of American factories, but once started, the movement has gained great impetus, and it is said that there are already more than a dozen factories for the production of anilin from benzene and of anilin dyes, and undoubtedly more will be constructed during the present year.

The sudden introduction of these industries brings new problems before physicians and sanitarians. Making anilin from coal tar involves exposure to poisons that are new to us in America, and because they are unfamiliar it is most improbable that the planners of these factories and the industrial chemists in charge of the processes will have given much thought to the protection of their workmen against the dangers inherent in the industry. If the experience of one town is typical, the greater number of American practitioners are not prepared to recognize the symptoms of poisoning from these bodies, or to estimate their seriousness. After the introduction, a couple of years ago, of anilin as a compound of rubber in the tire factories of Akron, Ohio, it was some time before the physicians of that city realized what was the cause of the peculiar illness that had appeared among rubber workers; many patients were treated for chronic cardiac disease, and others for epilepsy. Therefore it becomes a very important matter in the interests of public health that the medical profession should know the nature of these substances, their mode of entrance into the body, and the means which must be adopted to protect those exposed in the course of their work.

POISONS USED IN THE MANUFACTURE OF ANILIN AND ANILIN DYES

A short summary of the processes used in making anilin colors will show that the workmen employed in this industry are exposed to highly poisonous volatile compounds. The process starts with coal tar, which is a mixture of benzene (C_6H_6), toluene, xylene, etc., all of them volatile poisons responsible for a fairly large number of fatal industrial cases in the literature.² The benzene is treated with nitric and sulphuric acids to form nitrobenzene ($C_6H_5NO_2$), a poison which, according to Lewin,³ has a mortality of 39.3 per cent., and according to von Jaksch⁴, of 20 per cent. Then the nitrobenzene is reduced by means of iron filings and hydrochloric acid to anilin ($C_6H_5NH_2$). The most important colors are made by the action of nitrous acid on anilin and the allied aromatic amines. There is opportunity, therefore, for poisoning from benzene, nitrobenzene and anilin, to say nothing of the multitude of similar bodies used or produced in the various processes, such as the nitrophenols, the nitrochlorbenzenes and the naphthylamines. Methyl alcohol is one of the reagents used.

Grandhomme,⁵ who was for many years attached to the great dye works at Höchst, reported 128 cases of industrial poisoning there, 109 of which were caused by anilin. Leymann,⁵ who does not name the factory with which he was connected, gives a record of 171 cases of anilin and toluidin poisoning, and 101 from nitrobenzene and its allied bodies. German rubber works also have had their share of anilin poisoning. The factory inspectors' report for 1908 tells of the occurrence of wholesale poisoning in the extraction of rubber from crude gum. In this instance the workmen, seventeen in all, were working in twelve hour shifts in an atmosphere contaminated with anilin fumes. Eleven were poisoned, two being rendered unconscious and requiring hospital care. The following year two instances are reported of anilin poisoning in the recovery of rubber from scrap. Four acute cases developed in one factory, and on examination fifteen other men were found to be suffering from chronic poisoning. In the second place, where twenty-five men were employed, no less than fourteen were overcome by the fumes, and ten of them were ill for more than ten days.

ANILIN IN INDUSTRY IN THE UNITED STATES

Aside from the production of anilin colors, the oil is used in this country in the compounding of rubber, especially for tires, and in reclaiming rubber from scrap, for which purposes it has come into increasing demand during the last few years. When the war suddenly shut off the supply from Germany, some rubber manufacturers gave up using it, but others set themselves to provide it by home manufacture, so that now we must add to the men exposed to anilism in rubber manufacture those who are engaged in producing the anilin. Industrial anilin poisoning, which a few years ago was negligible in this country, has suddenly assumed decided importance. So far the making of dyes is too new to have furnished any reported cases; those which are on record come from the rubber industry and from the use of a wash for printers' ink which has anilin as one of its ingredients.

1. The earliest report quoted by Kobert (*Die Intoxikationen*, Stuttgart, 1906) is English: Letheby: *Proc. Roy. Soc.*, 1863.

2. Rambousek: *Gewerbliche Vergiftungen*, Leipzig, 1911, p. 142.

3. Lewin: *Virchows Arch. f. path. Anat.*, 1879, lxxvi, 443.

4. Von Jaksch: *Die Vergiftungen*, Vienna, 1912, p. 325.

5. In Rambousek (Footnote 2).

It is impossible to say how much of this substance is used in the United States or even to say how many plants use it, for the making of rubber, and even more the reclaiming of rubber, is an industry fairly hedged about with trade secrets. That is why it was so difficult for Akron physicians to trace this kind of poisoning to its source. It is, however, well known in Akron now, and common enough for the victims to be familiarly known as "blue boys," from the cyanosis which is so prominent a symptom. Since the discovery of the cause of the trouble, the rubber manufacturers have taken measures for the protection of their men, either by installing exhaust ventilators, or simply by warning them to seek the open air as soon as flushing of the face and violent headache come on. It is said that there is now far less serious poisoning in Akron rubber works.

THE TOXICITY OF ANILIN

Pure anilin has never been known to cause poisoning; but pure anilin is not used in industry. What we know under this name is a mixture of anilin (amido-benzene) with metatoluidin, paratoluidin and orthotoluidin and xyloidin in varying proportions. Every medical student is familiar with the anilin oil which he uses as a clearing fluid for stained specimens, that golden brown oil with its pleasant nutlike odor. Unlike most volatile poisons, it gives no warning of its character, for it does not irritate the eyes and throat, and is apparently as harmless as cedar oil. Yet according to the researches of K. B. Lehmann,⁶ anilin is toxic in even smaller doses than are benzene, chloroform or carbon disulphid. Toxic symptoms follow the inhaling of from 0.1 to 0.25 gm. of anilin, while it takes from 1 to 1.1 gm. of carbon disulphid to produce symptoms of poisoning. Anilin volatilizes at room temperature; the boiling point is high, 182 C.

Krause⁷ has reported two instances of poisoning from insignificant quantities of anilin. The men were working in the Zeiss Optical Works in Jena, testing the clarity of rock crystals by dipping them in small receptacles containing anilin, and then holding them up to the light. One man worked alone in a small room, and he felt the effects at the end of his second two hour shift, complaining of nervousness and exhibiting a marked cyanosis of the skin and mucous membranes. The second was in a larger room in which the fumes were more diluted, and he worked almost four hours before becoming cyanosed. The blue color increased in intensity after a second day's work.

Still more striking in this connection is a non-industrial case related by Trespe, of a little boy who breathed what must have been a very slight quantity of fume and developed marked symptoms. He slept in the same bed with an older brother who just before going to bed had rubbed some anilin on his frost bitten fingers, with the result that he himself was severely poisoned and the little fellow moderately poisoned.

SYMPTOMS OF ANILIN POISONING

These are common to the whole group, including nitrobenzene and its derivatives, though there are minor variations which serve to distinguish anilin poisoning in a typical case. The first symptom noted is weariness, or sleepiness, flushing of the face, a sense of fulness in the head and even of slight mental con-

fusion, dryness of the throat and difficulty in swallowing. The color of the face changes from red to livid blue, which may increase to a deep purple. The pulse is rapid and weak, the temperature subnormal. Headache comes on early and is often violent, accompanied by dizziness and nausea, and in the more serious cases there is decided dyspnea, increasing mental confusion, and finally loss of consciousness. Sometimes this occurs suddenly, sometimes not till some hours after the man has left the poisoned atmosphere. The urine at first is usually normal; later it becomes smoky in appearance.

In some cases the subjective symptoms are slight and the man seeks advice because of his livid or blue color, which is alarming to his family. This was true of two cases reported by G. Apfelbach and described farther on. Usually industrial cases do not exhibit the severest symptoms, but we have records of instances of epileptoid convulsions and of protracted coma which sometimes have proved very puzzling to the attending physician. One of these is interesting, illustrating as it does the vasomotor depression which is a feature of this poison.

The patient, who was chief chemist in one of the large rubber companies, was found by his physician suffering from violent headache and nausea. He was profoundly cyanotic. Soon after the physician arrived, a convulsion, epileptoid in character came on—the man never had a convulsion before this—and after it he was greatly prostrated. The physician, not knowing that it was a case of anilin poisoning, administered inhalations of amyl nitrite which very much aggravated the symptoms, and for a while after that the patient's condition was most precarious.

Recovery in the milder cases is prompt, the men returning to work on the following day as a usual thing, though the cyanosis often persists for several days more. Severer cases may incapacitate for work during several weeks. Some men are said to establish a tolerance to the poison, and do not suffer a second attack; but it is more usual to find an increased susceptibility following an acute poisoning. Thus in one of the Akron plants in which anilin is being manufactured, a foreign workman, who incidentally is said to have been a heavy drinker, was told to fill a drum with anilin, and while doing this he lost consciousness and had to be taken home. The next day he came back to work, but as soon as he entered the room where the oil was being poured into drums, he again fainted away.

Chronic anilism is described by von Jaksch as a condition resulting from repeated doses of the poison so small as not to set up symptoms of acute poisoning. There are more or less headache, nausea, vertigo, muscular twitchings, disturbances of vision, sense of exhaustion and loss of strength. The skin is usually livid, or it may be jaundiced. In one instance of chronic poisoning which was reported to us in Akron, the man had been treated for chronic heart disease because of his cyanotic appearance and his dyspnea on exertion. He was found to be profoundly anemic, but there was no heart lesion, and he recovered after changing to outdoor work.

We have as yet no record in this country of the occurrence of tumors of the bladder in anilin workers, such as has been described by German observers. These tumors are adenomatous or carcinomatous and are supposedly caused by the irritation of some of the products of anilin decomposition excreted by the kidneys.

6. Lehmann, K. B., quoted in Kobert: *Lehrbuch der Intoxikationen*, Stuttgart, ii, 1109.

7. Krause: *Med. Klin.*, 1908, iv, 10.

MODE OF ENTRANCE OF ANILIN

The poison may enter the skin or the lungs, more often by both ways. Birge's⁸ cases are instances of pure skin absorption.

Two painters were using anilin-black paint, applying it with a brush and then washing the surface with hot water and soap suds. There were no fumes, but both men were seized with nausea, general weakness, palpitation of the heart, then violent headache with vomiting in one case and diarrhea in the other. Both passed dark colored urine. The skin was very pale, the lips blue. The next day they were able to go back to work, and by wearing rubber gloves they avoided a repetition of the trouble.

On the other hand, von Jaksch⁴ reports a case of pure respiratory origin.

A boy of 17 years had been set to work for the first time mixing anilin with other compounds in an open receptacle. Headache came on almost at once, and after four hours' work he was forced to seek the outer air. He went home, and a physician, summoned some hours later, found him in coma, deeply cyanosed, with a weak, rapid pulse and dyspnea. Edema of the lungs developed and did not clear up for four or five days. The coma lasted several hours. In this case anilin could be detected in the urine.

The greater number of industrial cases follow the spilling or splashing of anilin over clothes or skin, and doubtless both skin absorption and lung absorption play their part.

PATHOLOGY OF ANILIN POISONING

Rambousek² places anilin and all the nitro and amido derivatives of the aromatic series among the true blood poisons, the principle action of which is to cause the formation of methemoglobin. He attributes all the resulting symptoms to the fact that in place of oxyhemoglobin with its easily released oxygen, there is methemoglobin with its firmly bound oxygen, and consequently a condition of "internal suffocation" from lack of oxygen in the tissues. The headache, feeling of anxiety, oppression, weakness, dizziness, dyspnea and finally loss of consciousness are all caused by the deprivation of oxygen. The later symptoms, icterus, methemoglobinuria, bladder irritation and bronchitis, are caused by the elimination of the poison. Kobert¹ lays almost equal stress on the importance of the blood changes; but, unlike Rambousek, he believes that there is also a direct effect on the central nervous system. Curschmann⁹ agrees with the latter that anilin and the anilin group are pure blood poisons, and that all the symptoms are referable to blood changes.

Experimental studies of these blood changes were made by Price-Jones and Boycott,¹⁰ using anilin hydrochlorid on rabbits. This compound, which is used in rubber manufacture under the name of "anilin salt," is essentially anilin dissolved in hydrochloric acid. These authors succeeded in producing severe poisoning, the hemoglobin falling to 50 per cent. The blood was turbid and brownish, and the spectroscope showed a band almost, but not quite, corresponding to that of methemoglobin. There was a transient leukocytosis, from 30,000 to 40,000, and both the circulating blood and the bone marrow showed evidence of efforts at regeneration of the red corpuscles in the form of megaloblasts, nucleated reds, and basophilic granules.

The destruction of cellular elements in the blood was sometimes accompanied by an increase in the total volume.

Malden¹¹ examined the blood of thirteen men employed in an English factory where anilin dyes and nitrobenzene were made. Six of the thirteen had a high red count with a low color index and many imperfectly developed reds. The spectrum of methemoglobin could be detected only when it was present in the proportion of one part to ten of oxyhemoglobin. Loss of hemoglobin ran from 5 to 50 per cent. Stained specimens showed great variations in the size of the red corpuscles, from 5 to 11 microns, the large predominating, but there were no nucleated reds. It is noteworthy that basophilic granules were found in six of the thirteen men, and Malden believes that this is often the earliest sign of anilin poisoning, as it is of lead poisoning. In pronounced cases there may be ten or twelve stippled cells in every field. A differential count of whites in nine cases showed a diminution of polymorphonuclears—marked in five—with a corresponding increase in small mononuclears. Eosinophils were increased in three, mast cells in six.

Malden summarizes the effect on the blood of small repeated doses of anilin thus: reds increased in number with loss of hemoglobin; low color index; degeneration and imperfect regeneration of red corpuscles; decrease in polymorphous leukocytes, increase in lymphocytes.

DIAGNOSIS OF ANILIN POISONING

The importance of the blood examination in the diagnosis is very evident. Curschmann advises in all doubtful chronic cases an estimation of the hemoglobin, and if this has fallen 15 or 20 per cent., the man should be kept under observation, for anemia is the earliest symptom. As we have seen, Malden would look for stippled cells first. When toxic symptoms have developed, the presence of hemoglobin in the urine should be looked for. The physician who is not equipped with an apparatus for making blood examinations will depend on the cyanosis, the dark colored urine, the odor of anilin in the breath, the rapid pulse of low tension, the dyspnea, and the nervous symptoms, together with the history of employment in a factory in which this group of compounds is handled.

CASES OF INDUSTRIAL ANILIN POISONING
IN THE UNITED STATES

The first cases of this sort in the literature that we have been able to discover were reported in 1913 by G. L. Apfelbach:¹²

Two men were referred by factory inspectors to Dr. Apfelbach on account of the marked cyanosis of skin and mucous membranes. The first was a press-feeder in a printing establishment who had been using a new sort of roller wash to remove the ink from the press rollers. It was a black oily fluid, which on analysis in the state laboratory was found to contain anilin. On the day when he was seen by the inspector, he had cleaned more rollers than usual, but he had been cyanosed before, though never so profoundly. The symptoms elicited by questioning were headache, mostly occipital, dizziness, pain in the epigastrium, dryness in the throat, and slight difficulty in swallowing, but none of these was severe enough to make him seek medical aid. It was the blue color which alarmed him. His pulse was of high tension, his blood pressure 170 mm. Hg (the man was 47 years old), but no other abnormality was observed with the exception of the pro-

8. Birge, E. G.: A Probable Case of Anilin Poisoning, *THE JOURNAL A. M. A.*, Jan. 24, 1914, p. 314.

9. Curschmann: *Tr. Internat. Cong. Indust. Hyg.*, Brussels, 1910.

10. Price-Jones and Boycott: *Guy's Hosp. Rep.*, 1901, lxiii, 309.

11. Malden: *Jour. Hyg.*, 1907, vii, 672.

12. Apfelbach, G. L.: *Bull., Dept. Factory Inspection, State of Illinois*, 1913.

nounced cyanosis and a slight difficulty in swallowing and in speech. The reflexes were normal. Examination of the blood revealed: hemoglobin, 100 + per cent.; red cells, 5,734,600; while count, 8,200 differential: polymorphonuclears, 72.8 per cent.; large mononuclears, 0.5 per cent.; small mononuclears, 20.2 per cent. The spectroscope showed methemoglobin lines. The urine was dark, smoky color, specific gravity 1.028, no albumin, no sugar, casts, etc. Employees in this printing shop had noticed the same change of complexion in other men engaged in this work, but never so severe as in this case.

Dr. Apfelbach's second patient was employed in a large dry color factory, mixing colors in an open chaser, work which he had done for several years. Occasionally he would notice that the mixture in the chaser would give off "steam" which made him sick, and this had been true just before he applied to Dr. Apfelbach for advice. The other men in the shop had alarmed him about his blue color, and he himself complained of headache, vertigo and difficulty in swallowing. The examination in this case was negative except for the presence of methemoglobin in the blood.

Birge's cases, already quoted, were next reported in 1914; and in 1915 E. R. Hayhurst,¹³ in the course of his investigations of Ohio industries, found anilin poisoning in printing shops and rubber works in that state. Three cases had occurred among men employed at rubber mixing mills. The inspectors were also told of more serious poisoning among printing pressmen who used a roller wash rich in anilin. In several instances the use of this had caused loss of consciousness lasting several hours and accompanied with cyanosis.

In the course of an investigation for the federal Bureau of Labor Statistics of poisonous substances used in the rubber industry, we¹⁴ collected some evidence concerning anilin poisoning in the chief rubber city of the country, Akron, Ohio. Anilin is used as an aid and an accelerator of vulcanization in heat curing of rubber, chiefly in tire works. Five plants in Akron were known to be using it and probably several others did. "Anilin substitute"—perhaps thiocarbanilid—and "anilin salt"—the hydrochlorid—are used in other plants. The danger is found in the room in which the compounds are measured out, on the mills on which the compounds are warmed and mixed together, and even on the calendars, on which the rubber is sheeted. The odor is plainly recognizable in all these places, unless special precautions are taken to prevent fumes by providing tightly covered receptacles and by good exhaust ventilation over the mills.

As rubber reclaiming is a process carefully guarded from the public because of trade secrets, it was not possible for us to visit any of these plants; but we found reason to believe that most of the cases of anilin poisoning in Akron—as is true in Germany—come from this branch of the industry. Not only the secrecy of the processes made it difficult to collect evidence of anilin poisoning, but also the fact that foremen and superintendents are naturally unwilling to admit that there is any danger from a substance which is in use in the factory. If, however, they are no longer using it, they may be quite willing to tell of its disadvantages. In a tire factory when no more anilin could be procured, the foreman said frankly that he was thankful not to have to handle it any more, for he had had so much trouble from the fumes among the men at the mills.

An interesting case indirectly connected with the rubber industry was described by an Akron physician. An old boiler was sold by one of the rubber factories, and the boiler repairer who undertook to clean it and put it in order had a typical attack of anilin poisoning.

The manufacture of anilin from benzene had begun only a short time before our investigation in Akron, but already one factory had a daily output of about 2,500 pounds and employed about fourteen men in two twelve hour shifts. In spite of a good system of exhaust ventilation which is said to have been installed, the men suffered from the fumes, and we were told that during the first four months of operation the factory lost no less than fifteen men from this cause, though it was not easy at that time to find work in Akron. The regular force was from twelve to fourteen men working in two shifts.

Shortly before the investigation referred to above, a severe case of anilism had occurred in one of the rubber factories. In this instance the poison seems to have entered both by the skin and by the lungs.

The patient was admitted to the City Hospital of Akron in a state of coma that lasted nine hours. He was deeply cyanosed, his temperature was 97.6, and pulse 116, fluttering and weak. The reflexes were normal save for a slight sluggishness of the pupils. Urine examination was negative. The patient was put to bed and given inhalations of oxygen and heart stimulants, aromatic spirits of ammonia, camphorated oil, strychnin and digalen. The temperature remained subnormal for six hours. After nine hours he regained consciousness and was discharged the following day, feeling a little weak and slightly nauseated, but otherwise normal.

His history was as follows: He was working in one of the rubber factories, and at about 1 p. m. he spilled a can of liquid over his clothes. He said it had a peculiar alcoholic odor, and when it came in contact with his skin it felt burning. The fumes were very noticeable, but he kept on at his work for about two hours more, though conscious that something was wrong. Then he began to have severe palpitation of the heart, he noticed that his face was flushed, he grew dizzy, and a violent headache came on and presently nausea, and he vomited several times. He was taken to his home, where the cyanosis kept increasing in intensity, and at about 6 in the evening he lost consciousness and was brought to the hospital. Consciousness returned at about 3 in the morning. Investigation showed that the fluid he had spilled was anilin.

Since the publication of the government report, several additional cases have been observed by one of us in connection with the rubber industry in Akron. One of them is of special interest in that we have accurate data as to the exact time of exposure to the fumes before the onset of very typical symptoms of the more intense, acute form of poisoning.

J. E. was first seen July 24, 1915. He was in profound coma, his breathing was stertorous and his pulse was irregular and of very poor quality. He was so cyanosed that the skin over his whole body was a deep plum color. That morning at 7 he had gone to work feeling perfectly well in every way and, according to the history obtained, he was exposed to fumes of anilin for an hour and forty minutes before he began to notice any symptoms of depression. First came throbbing in the head and increasing nausea, which he thought were due to the hot weather and the poorly ventilated room. He next noticed palpitation of the heart, and then a violent headache came on, increasing in intensity and accompanied by vertigo. As he said, "I felt as if I had been standing on my head for a long time and that every ounce of blood in my body had rushed to my brain." The dizziness increased and he lost consciousness completely about forty-

13. Hayhurst, E. R.: Industrial Health Hazards and Occupational Diseases in Ohio, Ohio State Board of Health, 1915.

14. Luce, R. V., and Hamilton, Alice: Bull. 179, U. S. Dept. of Labor, Bureau of Labor Statistics.

five minutes after the onset of the first symptoms. He was picked up by the other workmen and rushed to the hospital, where oxygen was administered as well as heart stimulants but apparently with little effect. The cyanosis persisted, the heart action was very feeble and remained so for over sixteen hours, and he did not regain consciousness till the morning of July 25, a period of about twenty-two hours.

A catheterized specimen of urine obtained on admission showed nothing of note, but a second examination was made eighteen hours later, when it was found to be smoky, specific gravity 1.022, there was a trace of albumin, no sugar, and the presence of hemoglobin was detected by the Heller test and by the Schönbein-Almén turpentine-guaiac test, which is even more delicate than the spectroscopic. This hemoglobi-nuria persisted for five days.

A blood examination made on entrance gave normal findings in all respects except for a slight eosinophilia, but four days later there were stippled red cells and some irregularity in the size and shape of the reds. The hemoglobin then was 75 per cent. (Sahli).

The patient suffered from severe headache for five days and complained of weakness and exhaustion for at least two weeks longer, after which he improved slowly. He was warned not to expose himself to this danger again.

The second case is cited because it is representative of the slower and much less typical forms of anilin poisoning: H. M., a chemist employed in the research laboratories of one of the large rubber companies of Akron, was seen Nov. 1, 1915. He complained of palpitation of the heart, dizziness, marked muscular weakness and occasional nausea and violent headaches. On close questioning it became evident that the palpitation of the heart was worse and headache was more apt to come on during active work, and that this work often consisted in the handling of a compound containing anilin. Most of the work was carried on under a hood, but he admitted that the odor of anilin was at times quite noticeable. He had been doing this sort of work for months, and said that he had never been cyanosed and never had had to lose a day from the laboratory.

An examination of the urine showed nothing abnormal: The red cell count was 5,400,000, but the hemoglobin only 68 per cent., and a stained smear showed pale, unevenly staining reds. Physical examination revealed nothing of note except a rapid heart action; the pulse was 94.

On this scanty evidence the patient was advised to give up his work and find some outdoor occupation, which he did, and as a result the symptoms disappeared entirely in six weeks.

The third patient, a lad of 18 years, was also employed in the research laboratories of one of the rubber companies. He was given a special problem to work on, which involved the grinding and mixing of certain compounds together with anilin. He did it in a corner of the room where the fumes could not spread and expose the other workmen. After working in this way for a few weeks he began to suffer from a dull headache which persisted all day. Then one day he became conscious of a severe throbbing in his head, and his face became cyanotic. He stopped work for the day and came back the next morning feeling fairly well, but it so happened that the ventilation was unusually poor that morning and the fumes very noticeable. After just one hour's work he lost consciousness and remained in a deep coma for over twenty-four hours. He was very cyanotic and his heart action was weak; at times he was almost pulseless. Camphorated oil administered hypodermically seemed to exert a better effect than did the oxygen which was given continuously for twelve hours. In this case no blood examination was made; the urine was at first bright red, later a smoky black.

Examination of the histories of a large number of cases of anilin poisoning reveals interesting facts as to the varying susceptibility of men to this poison. We have already noted the increased sensitiveness of heavy drinkers. It seems also that men of dark hair and complexion are less susceptible than blonds, as

has been observed in one of the large rubber works where records have been kept of over fifty cases of poisoning. Furthermore, it is found that young men are more susceptible than are the middle-aged, especially to the acute, violent form of anilism.

CONCLUSIONS

Commercial anilin is a well known industrial poison in Germany and in Great Britain. It is just beginning to be known in the United States, where it has already been the cause of numerous cases of poisoning among men engaged in the manufacture of rubber goods, in reclaiming rubber from scrap, in making anilin from benzene, and in using certain washes for press rollers.

It is a blood poison, causing the formation of methemoglobin with the consequent "internal suffocation."

Poisoning may take place through the skin or the lungs. Usually in industrial cases both portals of entry play a part.

Exposure to the fumes need not be excessive or long-continued to bring about serious symptoms in the susceptible.

Young men are more susceptible than the old or middle-aged, blonds than dark-haired men, heavy drinkers than the temperate.

Hot, humid weather, heated rooms and poor ventilation are important factors in the production of acute anilin poisoning.

Early recognition of anilin poisoning is of prime importance so that the sufferer may be withdrawn from the danger of further exposure to the poison.

Men working in anilin constantly seem to acquire a certain amount of tolerance to it; nevertheless, if the exposure is increased beyond the point of tolerance, there is apparently a cumulative effect, and symptoms of chronic poisoning result. After symptoms of poisoning have once manifested themselves, the patient is usually hypersensitive to the fumes.

The treatment consists in fresh air, oxygen and heart stimulants, especially camphorated oil. Prevention of subsequent exposure is imperative.

AN OUTBREAK OF TYPHOID ATTRIBUTED TO INFECTED OYSTERS

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An outbreak of typhoid fever, comprising fifty cases, which presented itself during November and December, 1916, in the city of Binghamton and in certain nearby municipalities in Broome and Chenango counties, offered an unusual opportunity for epidemiologic study, and proved particularly interesting since it was demonstrated with reasonable certainty that it was due to infected oysters.

Typhoid fever had prevailed to some extent in Binghamton and vicinity prior to the recent outbreak. Table 1 indicates the municipalities involved in this outbreak, the population of each, and the number of cases which had occurred during 1915, prior to November 1.

During the recent outbreak, cases appeared in each of these municipalities as follows: Binghamton, 25; Johnson City, 10; Windsor, 6; Norwich, 5; Oxford, 3; Greene, 1.