CARBON MONOXIDE AND AUTOMOBILE EXHAUST GASES*  

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There are many problems involved in the prevention of poisoning by carbon monoxide and automobile gases, one of the most important of which is the problem of diagnosis of carbon monoxide poisoning. We are not concerned about the cases where exposure is evident and severe symptoms or death follows. However, when exposure to limited amounts is possible we are much concerned whether such symptoms as the common ones of headache, weariness, weakness, dizziness, nausea, vomiting, loss of strength and muscular control, increased pulse and respiratory rates, loss of reflexes, and even coma with intermittent convulsions, cessation of respiration, and death are due to carbon monoxide poisoning or any of the many other possible factors connected with the environment or the condition of the victim.

The presence of pallor and symptoms of anemia, but with a high hemoglobin reading, associated with polycythemia is said to constitute a characteristic diagnostic combination. Direct blood tests for carbon monoxide may or may not have significance. Norris and Gettler find that 64 per cent of post-mortem tests of "normal cases" so far as carbon monoxide may or may not have significance. Norris and Gettler find that 64 per cent of post-mortem cases show CO present to as much as 5 per cent saturation in the blood, in New York City, upon delicate tests such as that of Van Slyke as modified by O'Brien and Parker. They found this test sensitive to within about 1 per cent. In deaths due to carbon monoxide, in victims found after varying periods after exposure, the CO present ranged from 10 to 92 per cent. This raises the question of the utility of delicate tests. Sayers and Yant have perfected the pyrotannic acid method for practical utility purposes with a delicacy which appears to be sufficient, they consider, for all practical purposes.

One of the most characteristic post-mortem findings, when present, is the loosening of the epidermis and perhaps blisters, located usually on the extremities and about the face and neck. Death from no other cause appears to show such phenomena. Associated with this is the occasional appearance, in those who survive a serious exposure, of gangrenous areas which may later take life through depletion or secondary infection.

We have been accustomed to lay great stress upon red discolorations of the body and blood just preceding and following death, but recently Banham, Haldane and Savage have pointed out the possibility of confusion with nitric-oxide hemoglobin, not from the administration of this compound as nitrites, etc., but from the presence of a nitrifying organism which may cause the body to have a red color similar to that often found in carbon monoxide poisoning. Such symptoms and causative organism were found in a case dying of broncho-pneumonia. These authors insist that CO-hemoglobin may be differentiated from NO-hemoglobin by the fact that on great dilution with water the former stays pink while the latter is

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not so pink, while boiling gives a grey
coagulum with CO-hemoglobin but a
pink coagulum with NO-hemoglobin.
They regard this as the simplest and
most delicate means of detecting the
presence of CO in the blood and of dif-
ferentiating it from other colored blood
solutions.

**DELAYED TYPE**

Our second problem concerns the ques-
tion of serious or fatal consequences of
delayed type following soon after an
alleged exposure in which the victim has
been able to extricate himself from the
CO environment of his own volition.
For example, he leaves the mine, furnace
room, garage or similar place experienc-
ing a shortness of breath; goes home,
utilizing a period of 20 minutes to an
hour; gets gradually more dyspneic and
dies with evidences of acute cardiac
dilation within 1 to 4 days; or develops a
severe form of broncho-pneumonia which
is usually fatal. A typical case is pre-
sented by Dr. J. M. Thorne. Does this
constitute delayed carbon monoxide
poisoning? If other workers under simi-
lar circumstances experience no such
symptoms, or allowing that they have
symptoms of headache and a little slow-
ing up of activity, may the fatality still
be due to carbon monoxide poisoning?
Are we justified in labeling a death as due
to carbon monoxide poisoning when the
individual is not prostrated or rendered
unconscious at the place of exposure?
These are all important practical ques-
tions in compensation matters regarding
exposures to carbon monoxide.

**POSSIBLE LATE SEQUELAE**

Four prominent afflictions alleged to be
due to breathing carbon monoxide in
amounts producing semi-consciousness to
just short of death, and either in one or
multiple exposures, present themselves.
Usually there has remained a degree of
ill health perhaps not affecting work
ability in the interim before the alleged
sequelae develop. These sequelae are
(a) the development of serious pul-
monary complications like pneumonia
some weeks or months afterwards; (b) the development of multiple sclerosis
usually some years afterwards; (c) the
question of pernicious anemia, recently
reëmphasized by Beck and Fort, follow-
ing in due course of time and usually
after repeated exposures; and (d) the
question of psychoses or, simply, psych-
asthenia and neurasthenia developing
months or years after exposure. May
these afflictions really occur as late
sequelae of carbon monoxide inhalations?
Remember, we are not considering im-
mediate or early sequelae here following
exposures, nor situations in which vari-
ous disabling complications continue to
exist after rescue until one or another of
these sequelae eventuates.

**ASPHYXIA**

Does asphyxia explain all of the phe-
omena of carbon monoxide poisoning?
If carbon monoxide poisoning simulates
the gradual withdrawal of oxygen from
the blood, or the tenuity of the atmos-
phere as one ascends to great heights
in aviation or in mountain climbing,
as is claimed by most authorities, how
do we explain the fact that in acute
cases of poisoning, such as occur within
a few minutes' exposure in a closed
garage or a few hours' exposure to
the fumes of a maladjusted gas heating
stove, nervous symptoms such as weak-
ness of the knees, faintness, and loss of
consciousness invariably occur before
respiratory symptoms such as shortness
of breath, rapid breathing, cyanosis, and
similar phenomena usually associated
with asphyxia or air hunger?

In this connection we are much inter-
ested in a recent paper by Barcroft to the
effect that in death by carbon monoxide
in animals, although the blood in the
general circulation contains a very high
percentage, that in the spleen contains
practically none. When carbon monoxide
eventually reaches the spleen it is re-
tained much longer than in the general
circulation. (Note here the value of
examining the spleen for CO in post-mortemss.) Barcroft states that unless CO reaches more than 20 per cent saturation in the resting animal it may not penetrate the spleen pulp, which remains entirely free from the gas for as much as 4 hours. This discovery has offered one of the best explanations for the function of this mysterious organ—that of a storehouse for surplus blood corpuscles which are called into use only on extra demands. In active animals the CO-hemoglobin penetrates at once into the spleen pulp so as to be noticeable within 5 minutes. Splenectomized animals die markedly sooner under carbon monoxide than do controls. Thus the spleen appears to be a reservoir for blood corpuscles to be put into the circulation as required, and the old saying "to vent one's spleen" has an intelligible meaning.  

A DISEASE OR AN ACCIDENT

Is carbon monoxide poisoning a disease or an accident? Do small amounts produce any disease? If acclimation takes place in those who do not show very noticeable symptoms, may gradual disease nevertheless take place? (Obviously where complications or sequelae immediately follow a given exposure, the case is an accident, that is, an event fixed in time as well as in place.) Our general belief is that any disability consequent upon the breathing of carbon monoxide has followed an exposure producing complete inertia if not unconsciousness and therefore can be fixed in time of occurrence. Hence such a disability does not constitute a disease but the result of an accident. Therefore, chronic poisoning from a single exposure to carbon monoxide does not exist; persons who have not been noticeably damaged by a single exposure (unconsciousness, paralysis, acute cardiac dilation, etc.) recover from all symptoms within a few moments and no disease is known to follow. We may be wrong in this, but if not, then carbon monoxide poisoning should be removed from occupational disease schedules and put into accident schedules. The same should be said for caisson disease (compressed air illness) which is apparently always an injury and not a disease. Is our nomenclature garbled?

CARBON MONOXIDE-HEMOGLOBIN DURING SMOKING

Numerous investigators have found CO-hemoglobin present in one's blood during the smoking of an ordinary cigarette, pipe, or cigar. Incomplete combustion, i.e., carbon monoxide, results from pulling air down through the fuel bed of the burning tobacco, because of poor aeration there. Several per cent of CO may occur in undiluted smoke from cigars. Naturally, those who inhale show the most in the blood. Quantities ranging from 5 per cent to 22 per cent CO-hemoglobin have been reported in smokers. The question arises whether this produces any chronic disease, whether acclimatization does not take place in the habitual smoker, and whether, in the end, smoking is not a form of exercise similar to walking at 3 to 4 miles per hour, so far as the CO factor in burning tobacco is concerned. If it is true that these rather significant amounts which occur in smokers do no harm, do similar amounts which occur in garage workers or perhaps traffic policemen, etc.?

A PROBLEM IN MINES

We find that many are under a misapprehension as to carbon monoxide as a mine problem. It should be emphasized that carbon monoxide is not a normal or natural gas in subterranean spaces. It is always the more or less immediate result of a fire, and that fire has burned without sufficient oxygen; so that whenever carbon monoxide occurs in a mine, it is necessarily the result of blasting in poorly ventilated quarters, or of fires, usually of smoldering type located about the passageways. Carbon monoxide does not occur naturally anywhere, volcanoes and fumaroles excepted. For this reason it should be pointed out that some other gas,
usually carbon dioxide, or a plain want of oxygen, has been the "gas" responsible for the mishap in a sudden asphyxiation in a closed or deep space, rather than carbon monoxide. This item should not be closed without calling attention to the perfection of the "carbon monoxide self rescuer" devised by Fieldner, Katz and Reynolds which may be held in the rescuer's teeth and provides protection from this gas for at least one-half hour while helping miners to escape from places in which fires are burning or explosions have occurred.

GAS-FIRED HEATERS

We present herewith a table showing the carbon monoxide fatalities which have occurred in Ohio during the past three winters, due to gas-fired heating appliances used in occupied rooms, although those of the first winter, 1922-23, were not recorded systematically until January, 1923. We believe that the considerable falling off in deaths from gas-fired room heaters last winter (1924-1925) is the result of the vigorous campaign of publicity instituted, in which all were strongly advised to see that such heaters were connected to effective chimney flues. It will be noted that gas-fired water heaters, usually used in bathrooms and much more difficult to connect with flues, have taken a small but constant toll each winter. There is also a surprising increase in asphyxiations from automobile exhausts.

CARBON MONOXIDE FATALITIES

Three-Year Comparative Record in Ohio
(Compiled by Ohio Dept. of Health)

<table>
<thead>
<tr>
<th></th>
<th>Winter 1922-23</th>
<th>Winter 1923-24</th>
<th>Winter 1924-25</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gas-fired room heaters</td>
<td>41</td>
<td>43</td>
<td>23</td>
</tr>
<tr>
<td>Gas-fired cooking stoves</td>
<td>2</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>Gas-fired water heaters</td>
<td>5</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Hotplates</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Furnaces (gas and coal) and stoves (coal)</td>
<td>6</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Automobile exhaust</td>
<td>5</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
<td>64</td>
<td>58</td>
</tr>
</tbody>
</table>

As a considerable part of the gas used in Ohio is natural gas and contains no carbon monoxide, it is easy to see that carbon monoxide poisoning has occurred as the result of the burning of the gas.

This sometimes occurs from improperly constructed heaters, which are constant carbon monoxide generators from the moment they are lighted, sometimes from burning the heaters too high, and sometimes from a rise in the gas pressure, particularly over night while one is asleep and while less gas is being used in the neighborhood—which allows a certain amount of the gas to slip through the heater only partially burned (i.e., as carbon monoxide) and without reaching a temperature of 1100° F., which our metallurgists claim is the temperature necessary to burn or oxidize carbon monoxide completely to carbon dioxide. 10, 11, 12

The chief problem here is the creation of city ordinances and of state laws prohibiting the flueless heater as well as the abolition of leaky gas tubing and the control of leaky connections.

OTHER DANGEROUS COMBUSTION PRODUCTS

The question arises whether other injurious products are given off than carbon monoxide from carboniferous fuels in heating stoves. One set of chemical analyses made at Ohio State University shows that under ideal combustion, over 6 per cent carbon dioxide and less than 10 per cent of oxygen constitute the combustion products of a high class gas-stove heater. This is a chemical condition incompatible with life for the average human being. In addition to this, we strongly suspect that other sharp and offensive substances may gradually debilitate human beings, i.e., lower their resistance, especially to diseases of the respiratory tract. The other substances found are said to be formaldehyde, acrolein, ammonia, and sulphurous products. 13 All carbon fuel appliances should be effectively vented to the exterior and so constructed as not to endanger life or health. The U. S. Bureau of Standards has laid down the principles which should be followed in the construction of air-gas burners to insure complete combustion and thereby avoid carbon monoxide and other menacing by-products. 10
AUTOMOBILE EXHAUSTS

It has been pointed out that statistics on fatalities from inhaling carbon monoxide in automobile exhausts, while limited in absolute figures, show an apparent decided increase in the state of Ohio. The question has been raised by a certain motor corporation in New York City whether this may not be due to the use of wet rather than dry mixtures said to obtain in Ford, Maxwell, Chevrolet, Dodge and other makes of cars, usually the cheaper ones, and not in dry mixtures which commonly obtain in the higher priced cars. So much insistence has been made upon this point to public health officials by this corporation that we addressed a letter upon the subject to the U. S. Bureau of Mines Experiment Station, Pittsburgh, Pa., some of whose statistics are quoted by Mr. Deppé, and we received the information following:

"From the same 101 automobile tests which were made under my direction at Pittsburgh and which Mr. Deppé quotes in his letter of August 6 to the Society of Automotive Engineers we found no appreciable difference in the relative amount of CO traceable to using easily vaporized or difficultly vaporized gasoline. We found that the amount of CO was directly proportional to the richness of the mixture. If we adjusted the carbureter so as to give a lean mixture the CO was low—in the neighborhood of 1 per cent; if the carbureter was adjusted rich it was in the neighborhood of 10 per cent. We could get just as low CO in a Ford as in any other car. There seemed to be little practical advantage on this score in any one make of car or any one time of mixture." Whether wet or dry, "if the mixture is rich it is bound to contain large quantities of CO."

Hence we have not considered it worth while to collect statistics on the types of automobiles associated with CO fatalities in Ohio.

We have been struck, of late, by the evidently successful attempts at suicide reported to us by means of the automobile exhaust method. It is only necessary to mention furthermore the rather frequent occurrence of narrow escapes and fatalities due to exhaust gases permeating enclosed cars and, in some instances, automobiles, so that ordinances have already been passed by a number of cities covering the proper ventilation of automobile vehicles used by the public.

TETRA-ETHYL LEAD POISONING FROM AUTOMOBILE EXHAUSTS

The title of this paper does not permit a discussion of the subject of poisoning by ethyl gasoline in its manufacture or distribution. The public is quite familiar with the mishaps which have occurred in New Jersey and Ohio in connection with the manufacture and blending, respectively, of tetra-ethyl lead. Contrary to popular opinion, there have been no reports of fatalities and, so far as we have been able to learn, of mishaps of moment which have occurred, outside of the laboratory, due to the handling of this substance dissolved in gasoline, i.e., in handling "ethyl gasoline."

While a possible hazard may still exist for auto filling station employes in dispensing ethyl gasoline, although it is no longer made by them, but at the bulk distributing centers—and perhaps to individuals careless in its use—the big problem of interest to the public is the question of the possibility of chronic lead poisoning from the inhalation of automobile exhaust fumes from burning "ethyl gasoline."

As you well know a general conference was called on this subject by the Surgeon General of the Public Health Service on May 20, 1925, where it was decided that not enough evidence was presented on either side of the question and that a special Committee of Seven should be appointed by the Surgeon General to investigate the subject further, a report to be made during January, 1926. In the meantime the Ethyl Gasoline Corporation voluntarily stopped the sale of the substance, which has later been modified in that the Surgeon General's Committee has authorized its sale under careful supervision by the Public Health Service in order to gain experience, as we under-
stand it, through the observation of gasoline filling station employees in certain states and limited territories. This supervised investigation is now going on in Ohio and perhaps elsewhere.

At the Washington conference it was pointed out by prominent public health leaders that authorities could not proceed to ban a substance alleged to be dangerous without sufficient evidence of danger to the public and that the results of animal experiments alone would never satisfy them; also that the feasibility of close observation on exposed human beings was entirely practical.

At this date it is truly remarkable, when we consider all of the complaints which come to the notice of health departments, particularly following the publicity of an alleged hazard to the public, that not a single one concerning gasoline treated with tetra-ethyl lead or alleged lead poisoning from motor exhausts using the same has been reported in Ohio. An invitation to the public to make such reports began immediately upon the occurrence of the mishaps at the Bayway plant of the Standard Oil Corporation in New Jersey approximately a year ago.

In view of the extensive experiments with automobile exhausts carried out upon animals by Dr. Sayers and his associates at the U. S. Bureau of Mines Experiment Station in Pittsburgh for approximately a year and a half, i.e., ending May 20, 1925, it is not likely that lead poisoning may be feared by the public. Dr. Sayers reported at the Surgeon General's conference that where the ethyl gasoline contained the regular or normal amount of tetra-ethyl lead, animals exposed 188 times to exhaust gases from an engine during a period of about 8 months and for 3-hour and 6-hour periods each day, no symptoms usually associated with lead poisoning occurred and that chemical analyses of animals gave no evidence of lead storage in their bodies. Where the amounts of gasoline contained 5 times the normal or commercial amount of ethyl fluid, a large group of animals, many of which had been used in the previous 188 days' experiment, still showed no symptoms usually associated with lead poisoning, but some of them showed storage in their tissues. Thus this situation stands until more information appears. We know of no other experiments upon this phase of the subject. Final reports of experiments conducted by the Bureau of Mines Experiment Station have not yet come to hand. We understand that the question of possible lowered resistance from the inhalation of such fumes in motor exhausts may be a part of the final report.

It is safe to say that none of us prefer to hazard lead in our systems if it is possible to prevent same. If, however, there is a certain negotiable amount which can be handled without accumulation and subsequent ill effects on health, and such an amount is absolutely indispensable if the economics of automotive transportation is to progress, we may have to tolerate the situation very much the same as we have to tolerate the automobile situation in general, and in spite of its manifold evils. The rational attitude of the present day is complete publicity and education regarding our manifold hazards so that legislative and official bodies may perfect and administer regulations for safety.

REFERENCES
12. Bull. 102, Part 8, Smithsonian Institution.