Protective Effect of Water against Toxicity of Pyroiysis and Combustion Products of Wood and Polyvinyl Chloride)

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ABSTRACT

The toxicity by inhalation of combustion and pyrolysis products of a PVC and a wood (Douglas Fir) has been studied by the physiogram method, in the rabbit in controlled ventilation and

by the mask cage method in spontaneous ventilation. The protecting effect of water was evaluated by trapping the gases and vapors with an impinger placed on the air flow just before the animals. Combustion and pyrolysis products were generated by an annular furnace moving along a strip of samples. The temperature of the furnace was 400°C for smoldering conditions and 850°C for flaming conditions. CO, CO_2, O_2, and HCl were determined in the atmospheres; pO_2, pCO_2, pH, CO, COHb in the animals' blood and EEG, EKG, and arterial pressure were recorded. The animals are very significantly protected by water against PVC fumes (HCl is very soluble in water) and in part protected against fumes from combustion of wood. In both cases, CO is not trapped and seems to be then the determinant toxicant.

INTRODUCTION

To study experimentally the toxicological problems of combustion and pyrolysis of synthetic and natural materials, one has to use two major kinds of models, one for fire the other for animals.

Among the possible fire models, we have selected the annular furnace technique of burning strips of materials, which is easily adaptable to the chosen animal models.

To intoxicate animals with atmospheres, two main procedures may be used: either to let the animals breathe freely or to impose on them controlled ventilations. The advantages and disadvantages of these different situations show that these two kinds of experiments are complementary. By all evidence, the free-breathing situation is very realistic, but it is difficult to know the exact quantity of toxicants getting to the lungs and the true anatomic level of their penetration. In controlled ventilation, the dose-effect relationship can be well studied at the alveolar level with a good reproducibility but the effect of toxicants on the regulation of ventilation cannot be estimated. Practically, both systems have to be used to obtain the maximum information.

Intoxication in spontaneous ventilation of rabbits is achieved by the way of special restraining cages called mask cages, in which only the head of the animal is exposed to the vapors (Fig. 1).

Intoxications in controlled ventilation are realized by tracheotomy on curarized, locally anesthetized rabbits [1]. Several parameters can be recorded simultaneously, biological ones (arterial blood) as
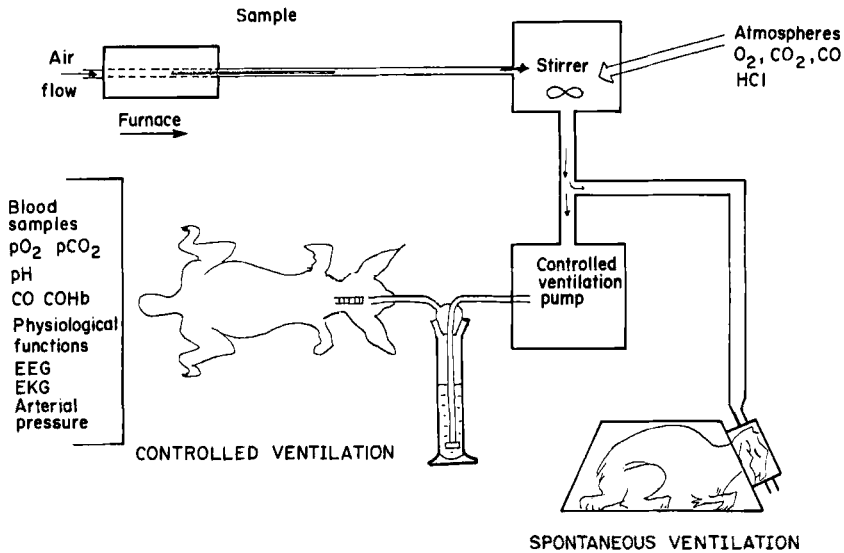
FIG. 1. Intoxication systems for rabbits.

well as physiological (activity of organs). To facilitate the interpreta-
tion of the data, diagrams called physiograms can be set up [2]
which allow easy comparisons of the events happening during the
intoxication and the immediate recovery of animals intoxicated with
various materials. By using this methodology, we have already
studied the toxicity of the combustion products of materials used
in the building industry and of the main major toxicants which may
occur [3]. As the water is one of the most frequent weapons used
to fight against fire, it seemed worth while to test its efficacy (use
of sprinklers) against the gases and vapors produced during the
combustion and pyrolysis of a rigid poly(vinyl chloride) (PVC) and of
fir, a commonly used wood.

METHODOLOGY

Fire Model

The sample, a strip of the material to be tested, is placed in a
quartz boat in a quartz tube. The air flow goes through that tube.

Combustion and pyrolysis are generated by an annular furnace (maximum temperature 1000°C) moving along the tube in the same way as the air flow (concurrent condition).

Of all the possible conditions, we have fixed an air flow of 120 liter/hr, two temperatures, 400°C for smoldering combustion and 850°C for flaming condition; the speed of the furnace was 30 cm/hr, allowing 30 min of intoxication for the animals.

Water Trap

As a sprinkler-like action was very difficult to realize in these experimental conditions, we simply put an impinger containing 200 cm^3 of water in the air track just before the animal.

Animal Model

We used burgundy fallow rabbits of 2.5 kg mean weight. The duration of the experiments was 30 min, when possible. This length of time is generally accepted by firemen as the maximum period of exposure permitting efficacious treatment of fire victims.

For intoxications in free-breathing conditions, the mask cage was used. For intoxications in controlled ventilation, the vapors were delivered to the animals by a Bird ventilation pump through the trachea.

The rabbit must remain conscious in order to retain a good central nervous system (CNS) activity. So, all the surgery necessary to set the canulas, catheters, and probes, was carried out under local anesthesia with procaine. The animals were then curarized with the gallamine triiodomethylate (Flaxedil). Proper ventilation was assured by monitoring the arterial pO_2, pCO_2, and pH values.

Several physiological parameters were recorded, corresponding to the activity of the most important organs (except ventilation) subject to acute failure: the CNS which is followed by electroencephalography (EEG) in frontal and occipital location, and the cardiovascular system (CVS) by electrocardiography (EKG) in D_1 and precordial leads and continuous recording of the arterial pressure. The toxicants are thus systematically administered to the alveolar level in the lung. The intoxication lasts 30 min maximally and the immediate recovery period lasts 4 hr afterwards. This total observation period is limited to that time because control animals remain in good physiological shape during that period but cannot afford more time in such experimental conditions.
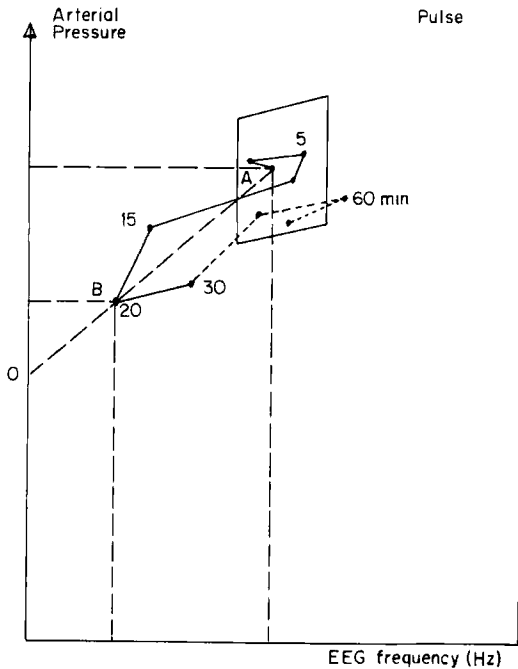
FIG. 2. Method of setting up physiogram and determination of the overall index of intoxication: (—) intoxication; (--) recuperation. AB/AO = 3.8 cm/5.8 cm = -65.5% at 20 min of intoxication.

The physiogram is a three-coordinate diagram (Fig. 2). On the abscissa is plotted the activity of the CNS as measured by the frequency of the EEG waves (hertz). Pulse and arterial pressure are plotted as ordinates, and the resulting line shows the activity of the CVS. At a given time, the overall activity of the animal is represented by a point. The displacements of that point with time are followed all along the experiment. Vertical moves are related to the CVS work, horizontal moves to the CNS activity. The "shapes" are easy to follow in this way.

An overall intoxication index can be calculated, measuring the distance of the representative point of the animal, at given times, from the control point. The complete recordings of EEG and EKG allow classical interpretations of these parameters.

From time to time, blood samples are taken to appreciate the

quality of the ventilation (blood gases) and the amounts of major toxi-
cants (CO essentially) present.

Balance Sheet

All the parameters recorded can be divided into three categories,
in order to get dose-effects relationships according to time: (1)
atmospheric composition including O_2 (paramagnetic effect), CO_2
(infrared spectrophotometry), CO (infrared spectrophotometry) and
HCl (potentiometry); (2) blood composition from pO_2, pCO_2, pH
(Radiometer apparatus), CO by the method of Boudene et al. [4], and
COHb (spectrophotometry); (3) organ activity: EEG, EKG, arterial
pressure (Beckman apparatus); ventilation is evaluated in free-
breathing experiments.

RESULTS

Intoxications in Spontaneous Ventilation

The 50% lethal concentration (LC_{50}) of the major toxicants alone
have already been determined for the rabbit [2] and evaluated by
using the Reed and Huench calculation as 10500 ppm for CO and
12500 ppm for HCl for 30-min exposures.

Animals exposed to CO died with respiratory failure. The tissues
had the classical reddish color due to COHb but had no acute pul-
monary edema (APE), and the main organs were macroscopically
normal. All the deaths occurred during the intoxication period,
never delayed.

Animals exposed to HCl had violent reactions. Dyspnea appeared
very rapidly and lasted throughout the intoxication period, with
marked hypoventilation. The animals rarely died during the intoxi-
cation period but generally failed afterwards, with very long delays,
sometimes as long as a month. In any case, a spectacular loss of
weight was noted. APE was frequent for medium concentrations
but systematic for the high HCl levels. At autopsy, infarcts and
pulmonary congestion were quite common but the trachea was very
frequently normal in appearance.

The 50% lethal dose (LD_{50}) obtained with the materials submitted
to combustion are reported in Table 1; two temperatures were used
and in some experiments the water traps were put in the air stream.

PROTECTIVE EFFECT OF WATER 1535

TABLE 1. Comparison of LC₅₀ for Combustion of Wood and PVC, with and without Water Traps, with Their Major Toxicants

Furnace temperature (°C)	Parameter	Fir		PVC			
		With water	Without water	With water	Without water	CO	HCl
400	LC_{50} (g/m³)	37.5	97	33.3	166		
	CO (ppm)	6000	13000	775	9000	10500	
	HCl (ppm)	–	–	10000	–		12500
850	LC_{50} (g/m³)	41.6	83.3	25	145		
	CO (ppm)	5000	11000	1150	8700	10500	
	HCl (ppm)	–	–	7500	–	–	12500

If the intoxication with products of combustion of wood looks like CO intoxication, delayed deaths can be observed (after 48 hr). Pulmonary infarcts appear in the lungs. When the water trap is used, these symptoms are not seen, and the animals are significantly protected; the death can be due to CO alone (11000-13000 ppm). In the exposure to products of combustion of wood, CO is consequently not alone, and other toxicants are stopped by the water, which, by the way, is acidic at the end of the experiment (ca. pH 5).

The intoxication syndrome due to combustion of PVC looks like the result of exposure to HCl. The animals die during the recovery period, sometimes after 15 days. The loss of weight is important; burns and pulmonary infarcts are systematic. The acute pulmonary edema (APO) is more or less severe. The CO present seems not to be important.

The toxicity of PVC seems to be slightly greater at 850° C than at 400°C and less related to the HCl concentration (7500 instead of 10000 ppm). In that case, other toxicants are involved in the toxicity.

The protecting effect of water is very efficient and with the water trap animals are able to resist fivefold greater amounts of material toxicant. HCl does not reach the lungs and CO becomes the major toxicant. This is due to the fire model we are using however; on increasing the amounts of material in the quartz tube so greatly, the air flow is not sufficient to bring enough oxygen to the combustion area, and pyrolysis produces a lot of CO. At autopsy, burns and pulmonary infarcts are not apparent, but the reddish color of COHb is present.

A slight difference in toxicity can again be noted at the two combustion temperatures.

Intoxications in Controlled Ventilation

During these intoxications, where the toxicants are directly driven to the pulmonary alveolae, a concentration of 41.5 g/m^3 of PVC is the upper tolerance limit of rabbits during a 30 min intoxication period and 4 hr of recovery. Greater concentrations produced death rapidly. When the water trap was used, the animals exposed to 140 g/m^3 of PVC were able to survive. Consequently, we tested the efficiency of water by burning such amounts of materials at 400°C, in concurrent conditions (the furnace and the air flow going the same way) with an air flow of 120 liter/hr. We used a rigid PVC and an untreated fir, both materials being used for the same purpose in buildings. For comparisons, some rabbits were intoxicated by comparable amounts of each major toxicant, that is 15000 ppm of CO and 42500 ppm of HCl.

TABLE 2. Concentration of Major Toxicants in the Combustion Products of Wood and PVC, with and without Water Traps

| System | | | | | |
Combustible material	Water trap	CO (ppm)	CO_2 (ppm)	O_2 (%)	HCl (ppm)
Fir	None	17,000	45,500	14.75	-
(140 g/m^3)	Present	15,000	55,000	14	-
PVC	None	15,000	44,000	15.1	42,500
(140 g/m^3)	Present	11,000	43,000	15.4	0

Combustion

Table 2 shows that the amounts of CO_2 and CO are almost the same for the two materials, according to the fire model we used.

The water in the trap does not stop CO, and this is very logical in view of the solubility of this gas. As this liquid is very rapidly acidified, it also does not stop CO_2. On the contrary, it fixes HCl very efficiently during the 30 min PVC combustion period.

Global Index of Intoxication

The determination of this index with time shows the overall disturbances occurring in the rabbits and how far the sum of various parameters lies from the norm. It is a first approach. Animals exposed to products of combustion of wood (140 g/m^3) are disturbed for the first 15 min as if they were intoxicated by CO alone (Fig. 3). After 20 min, however, a very rapid decay of the vital functions occurs, and the animals die. A white liquid invades the trachea, the APE is established. This does not occur with CO alone, the animals being able to recover to some extent.

When the water trap is used, the animals do not die during the intoxication and their intoxication indices are very similar to those measured with CO intoxication alone. But, 3 or 4 hr after the intoxication, APE appears, which was not noted with CO. This finding indicates that additional toxicants are involved which are not stopped by water.

With PVC (140 g/m^3), just as with HCl, the animals fail in about 8 min, the death occurring with a severe APE. The trachea is most often burned and locally destroyed. The thorax is frequently distended,
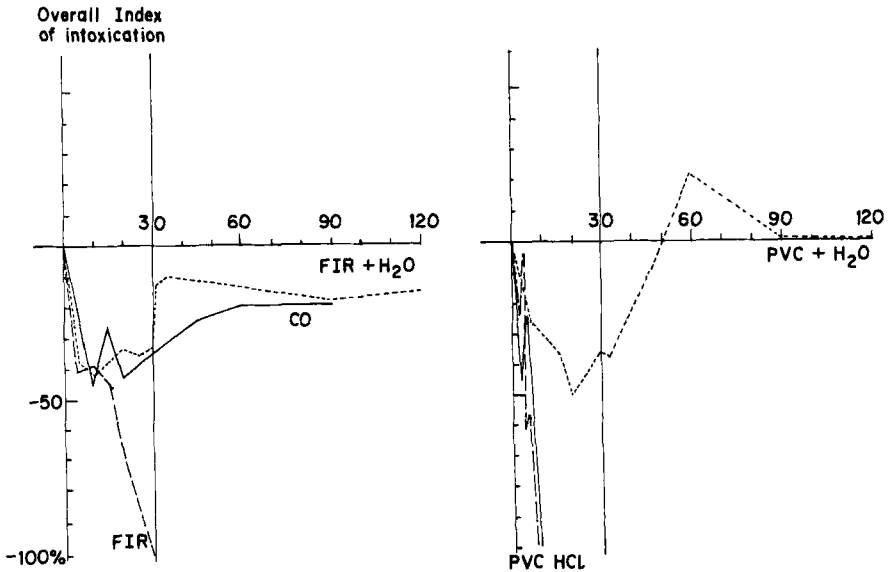
FIG. 3. Overall index of intoxication of rabbits with various toxic substances: (left) Fir + H_2O; (right) PVC + H_2O.

the gases pushed by the ventilation pump passing through the holes in the lungs. When the water trap is used, nothing of this kind is noted. The CO effect is evident, but the recovery is generally excellent without any macroscopical pulmonary alteration.

Physiograms

Diagrams

The physiograms recorded with CO alone show typical variations in the CNS: the EEG activity decreases, the characteristic point of the rabbits moving towards the left. Modifications of the CVS are not very large, and a recovery is possible after the intoxication.

When animals are exposed to fumes from combustion of wood, the shape of the diagram looks like the preceding one up to 15 min, but just after, a dramatic decay of the CVS occurs, and the EEG slows down. Death follows at about the end of the intoxication period.

With the water trap, the effect of CO on the CNS is evident but
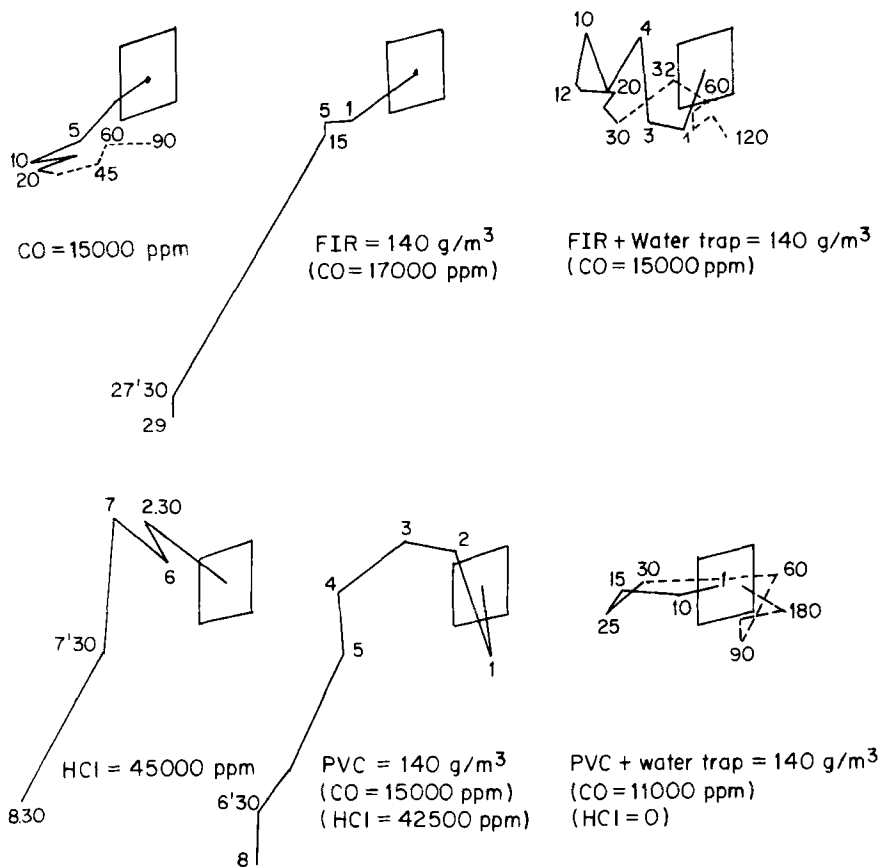
FIG. 4. Physiograms of rabbits intoxicated in controlled ventilation.

accompanied by great variations in the CVS. The most important fact is the appearance of an APE 2 hr later, although the immediate recovery seems good.

Physiograms with PVC and HCl intoxication look very much alike; there is a great decay of the CVS, lowering of the EEG, and rapid appearance of the APE. When the water trap is present with PVC combusion, only the CO effect is noted and the recovery is good (Fig. 4).

CO = 15,000 ppm

    control   30 min   90 min

FIR 140 g/m³

    control   16 min   29 min

FIR + water
    trap

    control   15 min   210 min

HCl = 42,500 ppm

    control   7 min 30 sec

PVC 140 g/m³

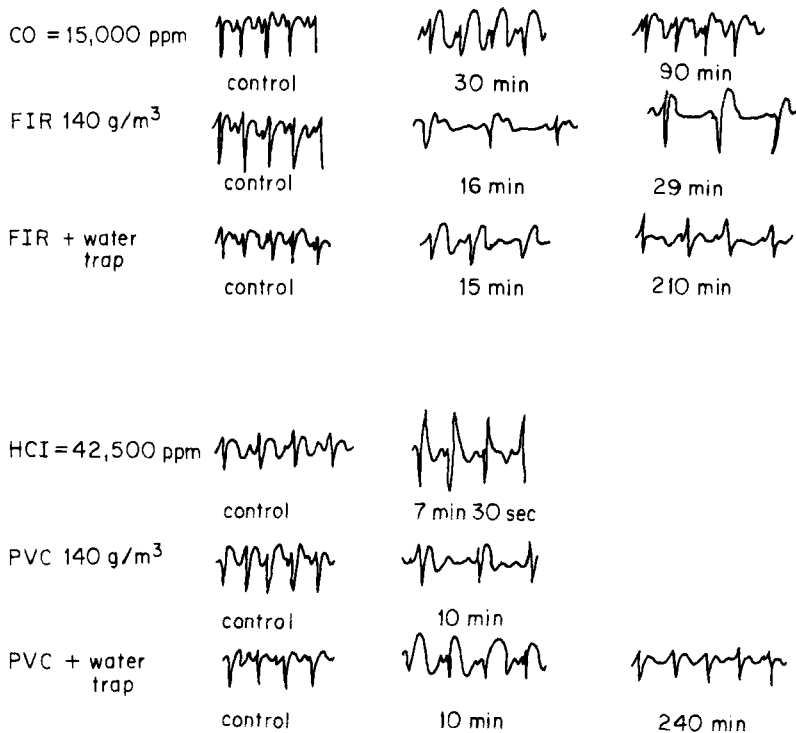   control   10 min

PVC + water
    trap

    control   10 min   240 min

FIG. 5. Changes in electrocardiographs (D₁ lead) in intoxicated rabbits.

Cardiovascular System (CVS)

With combustion of wood, the bradycardia is intense, and much more than with CO intoxication alone, especially when the water trap is not present. This is accompanied with marked, prolonged hypertension, never seen with CO.

On the EKG, the major symptom due to CO is an elevated T wave voltage which may also be biphasic; it appears irregularly with fir, whether or not the water trap is present, but we know that other toxicants may interfere. Fir fumes provoke mainly blocks and modifications of the RS segment, the T wave being sometimes flat, even negative. The water trap does not prevent these changes (Fig. 5).

With PVC, the bradycardia and the alteration of the EKG complex, resembles the effect of HCl. With the water trap these changes do not appear, but the CO effect is obvious. The recovery is good.
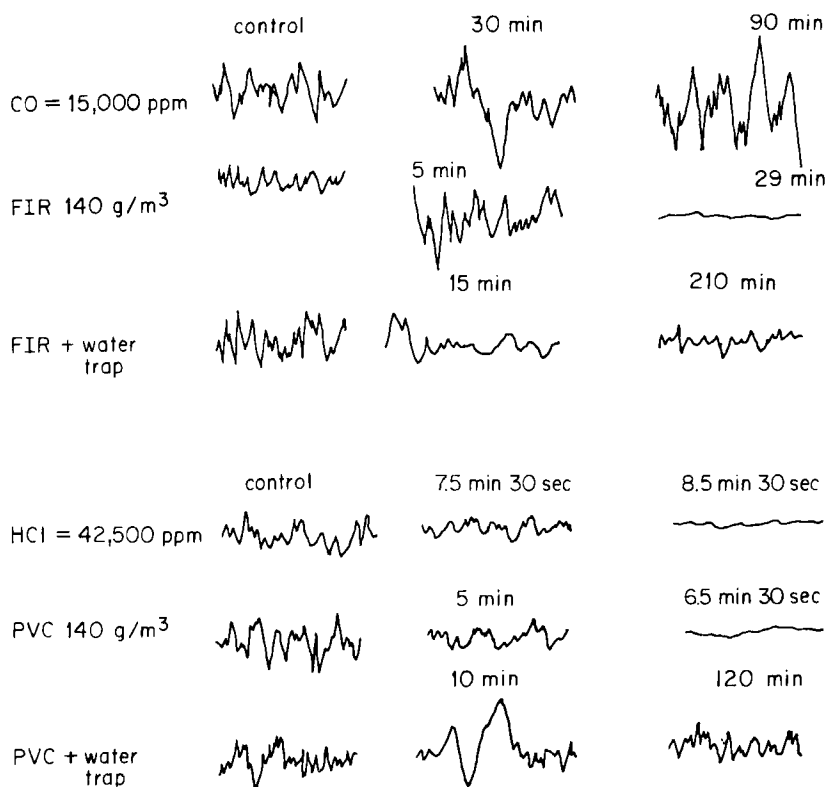
FIG. 6. Changes in EEG tracings (bioccipital lead) in intoxicated rabbits.

Central Nervous System (CNS)

CO induces large waves on the EEG which may remain during a part of the recovery period. These patterns are not so evident with fumes from fir combustion, and the CNS death comes along with the arterial pressure failure. The water trap has some protecting effect.

HCl decreases the frequency of the EEG waves and less markedly their amplitude before death. The water trap protects against it but the CO effect appears. The recovery is good nevertheless (Fig. 6).

Blood Samples

With the high concentrations used, CO and HCl induce a very severe acidosis but their effects on the blood gases are opposite. With CO,

TABLE 3. Modifications of Arterial Blood Gases of Rabbits Intoxicated in Various Conditions

	Control	Fir		PVC		CO (1500 ppm)	HCl (42,500 ppm)
		Without water	With water	Without water	With water		
Intoxication (30 min)							
pO$_2$	66.6	36.2	20.3	35.8	49.8	91.3	49.5
pCO$_2$	32.4	51.8	49.4	44.3	46.5	17.9	60.6
pH	7.42	6.96	6.98	7.11	6.95	7.10	7.02
COHb (%)	–	53	60.5	20	75	58	–
CO (cm^3 %)	–	22	24.3	22	14	15.7	–
CO (atm %)	–	1.7	1.5	1.5	1.1	1.5	–
CO blood/CO atm	–	13.0	16.2	14.7	12.7	10.5	–
Recuperation (90 min)							
pO$_2$		Death (29 min)	64.4	Death (8 min)	67	90.2	Death (8.5 min)
pCO$_2$			37.8		24	28	
pH			7.18		7.29	7.11	
COHb (%)			14.0		17.0	17.0	
CO (cm^3 %)			4.4		2.2	3.8	

the arterial pO_2 is high and the pCO_2 low; it is the contrary with HCl (Table 3).

With fir, as with PVC, with or without the water trap, the acidosis are always very strong.

The pCO_2 is generally high, and this may be mainly related to the large amounts of CO_2 present in the fumes. This gas may also accentuate the acidosis.

The pO_2 values are always low, especially with combustion of wood and curiously when the water trap is present.

Blood levels of CO and COHb are increased, in good agreement with the presence of CO. The ratio of blood CO% to atmospheric CO% is always higher with fumes from combustion of materials than with CO alone, as if that gas was better fixed when other toxicants are present. On the other hand, the amounts of COHb are not always as expected.

In this respect, it is important to note that the spectrophotometric method does not work well when PVC fumes or HCl are present. As a matter of fact, it is well known that HCl transforms a part of the hemoglobin into hematin, which is not able to transport oxygen.

During the recoveries, in the animals protected by the water traps pO_2 and pCO_2 return to normal; the acidosis diminishes but does not disappear. The best recovery is noted for animals exposed to PVC, as we know that the animals exposed to wood will show APE.

CONCLUSIONS

The goal was to compare the aggressiveness of gases and vapors emanating from burning chlorinated polymers like a PVC to those produced in combustion of wood, and to evaluate the potential efficacy of water in that respect.

Significant results were obtained in burning high amounts of materials (140 g/m^3) in the selected fire model.

Intoxications of free-breathing animals in mask cages showed that the LD_{50} for fir and PVC were quite close (about 35 g/m^3) in smoldering conditions ($400°$C); in flaming conditions, toxicity of PVC increases, the toxicity of fir decreases slightly. For both materials, the toxicity is not entirely and directly related to the amount of major toxicant being evolved. Additional toxicants are present; this phenomenon is particularly obvious for fir.

When water traps are used in the air flow, good protection of the animals is obtained, especially in the case of PVC. A fivefold increase in weight of burned material is possible without marked damage. The

limiting factor in our experimental conditions then becomes the CO
level. This gas, insoluble in water, is not stopped by water; for HCl
the contrary is true.

In controlled ventilation experiments we had the opportunity to
follow closely the succession of events during the intoxications and
the immediate recoveries. When the toxicants are delivered directly
to the pulmonary alveolae, they act differently and more or less
specifically. CO induces mainly a decrease of the activity of the
central nervous system through cellular hypoxia resulting from the
blockage of the hemoglobin. HCl is aggressive, burns the tissues,
and destroys hemoglobin also, but the immediate consequences of
its action appear principally on the cardiovascular system.

In both cases, acidosis is severe, but the effects on the blood
gases are opposite. With the high levels of CO used, the pO_2 in-
creases as if the oxygen consumption by the tissues was very low;
the pCO_2 is also low. With HCl, on the contrary, the pO_2 is low
and the pCO_2 high, as if the injured pulmonary alveolae had great
difficulties in bringing about the gas exchange between the atmos-
phere and the blood.

If the controlled ventilation protects against intoxications by CO,
it increases dramatically the toxicity of HCl, the animal being
unable to limit the input of this toxicant by decreasing its ventilation.

With the materials fumes, the acidosis is systematically obtained,
probably increased by the presence of CO_2 in the gases; the increase
in blood pCO_2 is a confirmation of it.

It is noteworthy that the animals intoxicated with PVC and HCl
die in the same conditions. But a consistent difference exists between
CO and fir combustion products. In the latter case, several toxicants
are present which are able to injure badly the pulmonary tissue;
acute pulmonary edema (APE) appears at the end of the intoxication.
Formaldehyde, furfuraldehyde and acrolein have been characterized
in the vapors and could be responsible for this effect.

The efficiency of water traps is particularly remarkable against
the PVC combustion products (especially HCl). The activity of the
acid gases is no longer evident, and CO could become the limiting
toxicant. Protection against fir fumes by water traps is substantial
but the risk of acute pulmonary edema is not suppressed; the toxicants
responsible for this are not stopped by water.

Water seems to be effective in combatting the action of certain
toxic compounds evolved from materials during combustion, especially
HCl. Sprinkler systems are of interest, but we must still test them
in large-scale experiments, even real fire situations, in order to
verify their efficacy.

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