# Physiological and Toxicological Aspects of Combustion Products: International Symposium (1976)

Pages
257

Size
8.5 x 10

ISBN
0309025214

Committee on Fire Research; Commission on Sociotechnical Systems; National Research Council

# PHYSIOLOGICAL AND TOXICOLOGICAL ASPECTS OF COMBUSTION PRODUCTS: INTERNATIONAL SYMPOSIUM

March 18-20, 1974

Flammability Research Center

The University of Utah

Salt Lake City, Utah

Conducted by

COMMITTEE ON FIRE RESEARCH

COMMISSION ON SOCIOTECHNICAL SYSTEMS

NATIONAL RESEARCH COUNCIL

NATIONAL ACADEMY OF SCIENCES

Washington, D. C.

1976

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Financial support for the Symposium and the publication of the Proceedings was provided in part by the support of the Committee on Fire Research given by the Defense Civil Preparedness Agency of the Department of the Army, the National Science Foundation, and the National Bureau of Standards of the Department of Commerce.

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ISBN 0-309-02521-4
Library of Congress Catalog Card Number 76-024955

Copies of this report may be obtained from:

Printing and Publishing Office
National Academy of Sciences
2101 Constitution Avenue
Washington, DC 20418

Printed in the United States of America

80 79 78 77 76 10 9 8 7 6 5 4 3 2 1

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*KEYNOTE ADDRESS*

Dr. Carl W. Walter, Chairman
Committee on Fire Research
National Research Council

A previous symposium at Ft. Collins considered the impact of smoke from urban, rural and forest fires on the environment. This symposium intends to explore the effects of combustion products on the human biologic system. Smoke in confined space is the theme—excluding of course, that closed system of generating smoke entitled "Smoking."

The subject is pertinent because of society's mounting concern with the chronic disability and loss of life following exposure to smoke in contrast to injury by flame or heat. Building codes, flammability standards, fire prevention programs, prohibition of materials are examples of societal reactions to an awesome problem. Yet there seems to be a singular dearth of data on the cause of disability or death. There is controversy because advocates of simplistic solutions ignore the complexity of a problem that challenges many disciplines and a complex technology.

As a surgeon, I am interested and baffled by the impact of smoke on the patient. What causes the spectacular acute death? Why the sudden difficulty with respiration hours after exposure? What is the cumulative insult that causes pulmonary insufficiency following repeated exposure? What underlies the deterioration of the nervous system? How is the occupational hazard recognized and defined? There may be a general theme to these questions; yet some of the answers are highly specific. It may be that each material has hazards that are inherent and unique.

As a safety officer in a hospital I am concerned by the hazard created by current hospital practices. Disposable supplies result in a serious combustible load throughout the hospitals. Storage and distribution creates caches of combustibles everywhere. Daily, some 28 pounds of trash per bed must be collected, removed and disposed of. The loose trash is highly flammable and produces toxic smoke characteristic of its high content of plastic. Hospital furnishings, upholstery, bedding and carpets are highly flammable and produce dense smoke. The soil that accumulates from contact or spillage often adds to flammability. All this adds up to 34,000 fires in health-care facilities annually. The majority cause more smoke than flame. But remember, bedridden patients can't escape the exposure. Every small fire is the ignition source for a potential holocaust.

Control measures are directed toward prohibition of combustibles and the use of flame retardants. Early detection of smoke and containment of the fire and venting of the smoke are advocated. It is ironic that the smoke from materials treated with fire retardants is more profuse and toxic than the untreated material.

In this context the symposium will endeavor to provide a perspective of the physiologic and toxicologic impact of combustion products. The prospects are good because our host institution is so confident in the safety of this auditorium that it has chained the fire exit doors to ensure security. Again this is an expression of well-intended social forces that are mutually self-defeating.

*SESSION I*

# SMOKE PROBLEMS ENCOUNTERED DURING FIRES

Moderator:

William J. Christian
Underwriters' Laboratories, Inc.

# *SMOKE PROBLEMS IN URBAN FIRE CONTROL*

Leon DeKorver, Chief
Salt Lake City Fire Department

The United States is an advanced nation technologically and is increasingly urban in character. Another way of saying this is that most Americans live in an environment of concentrated man-made objects. Their homes—which are generally close to neighboring homes (and sometimes in the same building) —are complexes of building materials, finishes, chemicals, paper, foodstuffs, and utility systems, all composed of objects processed by man. At work—whether it is at a factory bench, an office desk, or a sales counter—he is usually among a concentration of people in a similarly complicated environment of man-made objects.

In this built environment, as it is called, Americans live side by side, day and night, with ignitable materials, combustible furniture and upholstery, and products and appliances which through wear or misuse may offer dangerous fire potential. Fumes from their gasoline, their paint thinner, or their cleaning fluid fill the atmosphere with combustion potential. The structures in which they live and work, through flaws in design and poor maintenance, often encourage entrapment rather than escape from fire. Few give these hazards any thought—until fire occurs.

As every urban fire fighter can attest, fire does not victimize only the poor. There is no ground for complacency about residential fires among more affluent citizens. There, too, ignorance breeds indifference. No less than in a slum, a single spark can set off a chain of events that guts a mansion and kills its inhabitants. Fire, like sin, knows no class distinctions.

The consequences of a fire depend, however, not only on how it starts, but on what happens after ignition. Human beings can intervene to lessen the consequences of a fire caused by a defective product. Products can be designed to lessen the consequences of human carelessness, as for example, with matches and cigarettes. Whatever the cause of fire, buildings can be designed and maintained to ease fire suppression and the evacuation of potential fire victims. The consequences of fire, in short, depend on man - environment interactions.

Before turning to environmental factors alone, it is appropriate to consider one aspect of the man - environment interaction that tends to be overlooked. The ways in which man acts upon the environment to cause fire come readily to mind. What is not so obvious is that the built environment influences the behavior of man in a way that aggravates the fire problem.

The modern urban environment imparts to people a false sense of security about fire. Crime may stalk the city streets, but certainly not fire, in most people's view. In part, this sense of security rests on the fact there have been no major conflagrations in American cities in more than half a century. In part, the newness of so many buildings conveys the feeling that they are invulnerable to attack by fire. Those who think only of a building's basic structure (not its contents) are satisfied, mistakenly, that the materials—concrete, steel, glass, aluminum—are indestructible by fire. Further, Americans tend to take for granted that those who design their products, in this case buildings, always do so with adequate attention to their safety. That assumption, too, is incorrect.

The American Indian lived in his tepee with the hole at the top to permit the smoke to escape to the outside as did the people who started the construction of places to live. The fire was contained in what is now called the fireplace and the hot smoke and gases were carried to the outside by the chimney.

Around the turn of the century, in the wake of many conflagrations, so-called fireproof buildings began to be constructed. They had thick walls and floors to keep fires from spreading. Like older buildings, they still had windows that could be opened to allow heat and smoke to escape. They had fire escapes or internal fire stairs, and seldom were they too tall for the topmost occupants to escape. Many of the buildings that were constructed during this period of time were remodeled in the past few years creating greater hazards to both the occupants and the fire fighters. Where windows previously existed, there are no more windows or openings thus closing up the entire building.

The built environment was created to serve the needs of the people. When a portion of that environment goes up in smoke, those needs are not being served.

A synthetic material was implicated in a fire that killed 145 teenagers. It happened in a door-locked dance hall in St. Laurent-du-Pont, France, that had been lavishly sprayed with a plastic foam to give the appearance of a cave. The fire raged furiously within seconds after it began, leaping "like a red panther in a small cage" in the words of one survivor.

By no means do synthetics stand alone as hazardous materials. A frame house can be a tinderbox. Restaurants decorated with natural materials, basements full of old newspapers, and warehouses storing lumber or paper products provide the fuel for major fires. Burning silk and wool release

deadly quantities of carbon monoxide and cyanide gas—and these and many other natural materials ignite at lower temperatures than many synthetics do. Plastics manufacturers contend that synthetics based on carbon, hydrogen, and oxygen exclusively are generally no more toxic, when burned, than natural materials.

Most people, when they think of a fire as a killer, think of flames. In a list of the five ways in which fire can kill, when arranged in declining importance, flames rank last. One of these killers is what we are discussing here today, and this is smoke. Fires burning in an open area with a production of heavy smoke are not too bothersome to firemen since operations may be ordinarily conducted to the windward or flanks of the area. Where the operation must be conducted, of necessity to the leeward, discomfort will intensify in relation to the amount of heat and smoke encountered.

When fuels reach their ignition temperature, they react with oxygen to form new compounds called products of combustion. Most common fuels contain hydrogen and carbon in varying proportions. Complete combustion due to the reaction of carbon and oxygen produces very high temperatures.

## INDICATIONS OF EXISTING CONDITIONS

The question arises, "How can men tell existing conditions when answering an alarm of fire?" The answer is, "They must be able to observe indications and interpret them." Two indications are (1) smoke conditions, and (2) heat conditions.

## SMOKE CONDITIONS

Some materials give off more smoke than others, even in free burning. Liquid fuel material or those that liquefy at above ordinary temperatures generally give off heavy black smoke. Oils, tar, paint, varnish, molasses, sugar, rubber and like materials generally give off a dense smoke in large quantities.

Fire burning in an enclosed area or room develops products of combustion progressively as the heat increases in build-up (see Figure 1).

The first stage represents an ordinary fire in an enclosed room in a free-burning stage soon after ignition. Adequate oxygen is available so that carbon dioxide is produced in quantity. Only a small amount of carbon monoxide is present. The average temperature within the structure has risen only slightly. Very little discomfort would confront fire fighters and extinguishment should be easily accomplished.

The second stage represents a reduction of oxygen content from the original 21% to approximately 17%. Operation within the area is still possible without the use of rescue breathing apparatus. The size of the flame indicates that burning has slowed and carbon monoxide production has increased though not apparently. The average temperature would be

approximately 400°F. The existing condition for fire fighters would be uncomfortable but not particularly dangerous. Hazards to consider would include the possible quick ignition of cellulose contents under the presence of sufficient oxygen.

The third stage represents a reduction of oxygen content to approximately 15%. Fire is barely visible, carbon monoxide is produced in increasing amounts, free carbon and unburned fuel form dense smoke and a state of incomplete combustion exists. Intense heat and gases are present to imperil personnel and constitute an explosion hazard. The pressure of gases has increased and the formation of explosive gases such as natural gas is present. Only the introduction of oxygen and the presence of flame are needed to establish a state of instantaneous combustion. The oxygen content of the room is barely sufficient to sustain human life. The use of rescue breathing apparatus should be confined to self-contained air or oxygen types; the use of canister types should be avoided under these conditions. It is also well to remember that some substances burn in oxygen-depleted areas as low as 8% while others create their own oxygen.

The fourth stage represents a reduction of oxygen content to approximately 13%. Fire is smoldering, carbon monoxide is produced in abundance, the area is filled with smoke and heat perhaps exceeding 1000°F. The intense heat distills some fuels from the combustible materials; the fuel gases mix with other gases present adding to the fire hazard. The use of rescue breathing apparatus must be confined to the self-contained air or oxygen type. Danger to personnel and probability of explosion are extreme.

Should a fire start in combustible material in a closed room, it will burn freely for such a time as the oxygen supply is sufficient. At first as the heated gases of combustion are produced, they will rise because of their buoyancy through expansion, and by convection will diffuse with the cool air and by the time they reach the ceiling, will be cooled below the danger point. At the same time cooler air in the room moves downward and to the fire, thus providing it with available oxygen.

Most of the common combustibles are made up of one or more of the following fuel elements: carbon, hydrogen and sulphur. So long as sufficient oxygen is available and the proper burning temperature is maintained, the carbon burns to carbon-dioxide, the hydrogen to water vapor and the sulphur to sulphur-dioxide. These gases, heated to the temperature of combustion, pass off into and mingle with the air that is providing oxygen to the fire.

Seldom, however, does "complete" combustion take place, even under ideal conditions, and as the fire continues to burn, the air moving to the fire contains less and less oxygen. Complete combustion no longer is taking place, and carbon monoxide, methane and free hydrogen are reaching higher concentrations, until they are within their explosive range and above their ignition temperature, or at least above the ignition temperature of the lighter hydrocarbons.

Three things are happening: (1) the atmosphere in the room is being heated; (2) the oxygen is being exhausted; and (3) flammable and toxic gases are building up in concentration.

Once the room is filled with hot gases and smoke, they will find their way into adjoining rooms, thus filling much of the structure and making the calculation as to the location of fire more difficult.

If the building has several stories, many things may happen to complicate matters. The gases and smoke will follow the line of least resistance and seemingly seek out openings to floors above. Such openings as elevator shafts, stairwells, air conditioning shafts and others are common and natural courses of travel. Through these openings a draft is set up, and the hot gases and smoke rise to the highest possible level and there spread out, filling each succeeding space. As hot gases find their way upward, the heavier cool air finds its way downward, to feed the fire below with all available oxygen. Once a floor space is filled, the hot smoke finds its way to the outside through cracks and crevices around windows, roof, etc. and may be seen oozing through.

Serious thought should be given to what can be expected upon entering a situation just described—a building filled with smoke and hot, poisonous, flammable gases. Many a conflagration has been started by opening up a building under these conditions without considering possibilities of the situation. Thus, when approaching a closed building apparently containing a smoldering fire, it behooves the officers and men of the companies answering the alarm to "Stop, Look and Consider." A hurried attack to reach the seat of the fire may further the chance and so spread the fire that a major conflagration will be the result.

The process of combustion may also include the combination of some other elements with oxygen to form poisonous and flammable gases. For example, burning sulphur produces sulphur dioxide, a very toxic and irritating gas. This gas is not flammable but produces a suffocating effect. Burning nitrates produce nitrous fumes which are nonflammable but have an anesthetic effect.

Having a knowledge of the kinds of gases which may be encountered in a burning building is beneficial. However it is also essential to be familiar with their physical properties, particularly under fire conditions. When air or any other gas is heated, there is an increase in either its volume, its pressure, or both. If the heated air or gas is not confined, the volume is increased, conversely, if confined, the pressure is increased. In a closed room where fire is burning, the air expands as it is heated and rises to the ceiling; cooler air moves in to replace it. The cooler air is heated in the process and rises when it becomes lighter than the heated air above. It is now evident how an entire floor of a building may be heated with heat and smoke traveling from room to room and continuing as long as there is movement in the atmosphere.

When burning occurs, fuel combines with oxygen, depleting the oxygen in the air. If the supply of air is limited, as when the fire burns in an enclosed space, or a building, the fire may use up the available oxygen to such an extent that burning with flame ceases. In such a situation the fire fighter without his own supply of air cannot breathe. When he tries to work and breathe in such an atmosphere, the cells in his body quickly use up their supply of oxygen. The blood going through the lungs cannot pick up a new supply of oxygen. The blood is no longer red but blue, because it lacks oxygen. Thinking ceases because the brain lacks oxygen. Soon the victim becomes unconscious, heart action weakens because the heart muscle requires a plentiful supply of oxygen, and after a time the heart fails and death ensues.

When wood burns without adequate oxygen, the products of its combustion include generous amounts of carbon monoxide, carbon dioxide, formaldehyde, formic acid, carbolic acid, methyl alcohol, acetic acid, and other compounds. If the oxygen is poor, smoke will be dense and contain many organic irritants. If the oxygen supply is good, most of the irritants burn and the smoke contains carbon dioxide, some carbon monoxide, and only a small amount of organic compounds.

The smoke from burning plastics is very similar to that from the combustion of ordinary materials, carbon monoxide, carbon dioxide, hydrogen chloride, aldehydes, ammonia, cyanides, and oxides of nitrogen being produced. Of these, carbon monoxide and hydrogen chloride are of greatest toxicologic significance.

It doesn't make much difference what burns, the smoke will probably be poisonous. In an actual fire, the smoke is a mixture of gases, and evidence indicates that the total toxicity is greater than the sum of the toxicity of the components of the mixture.

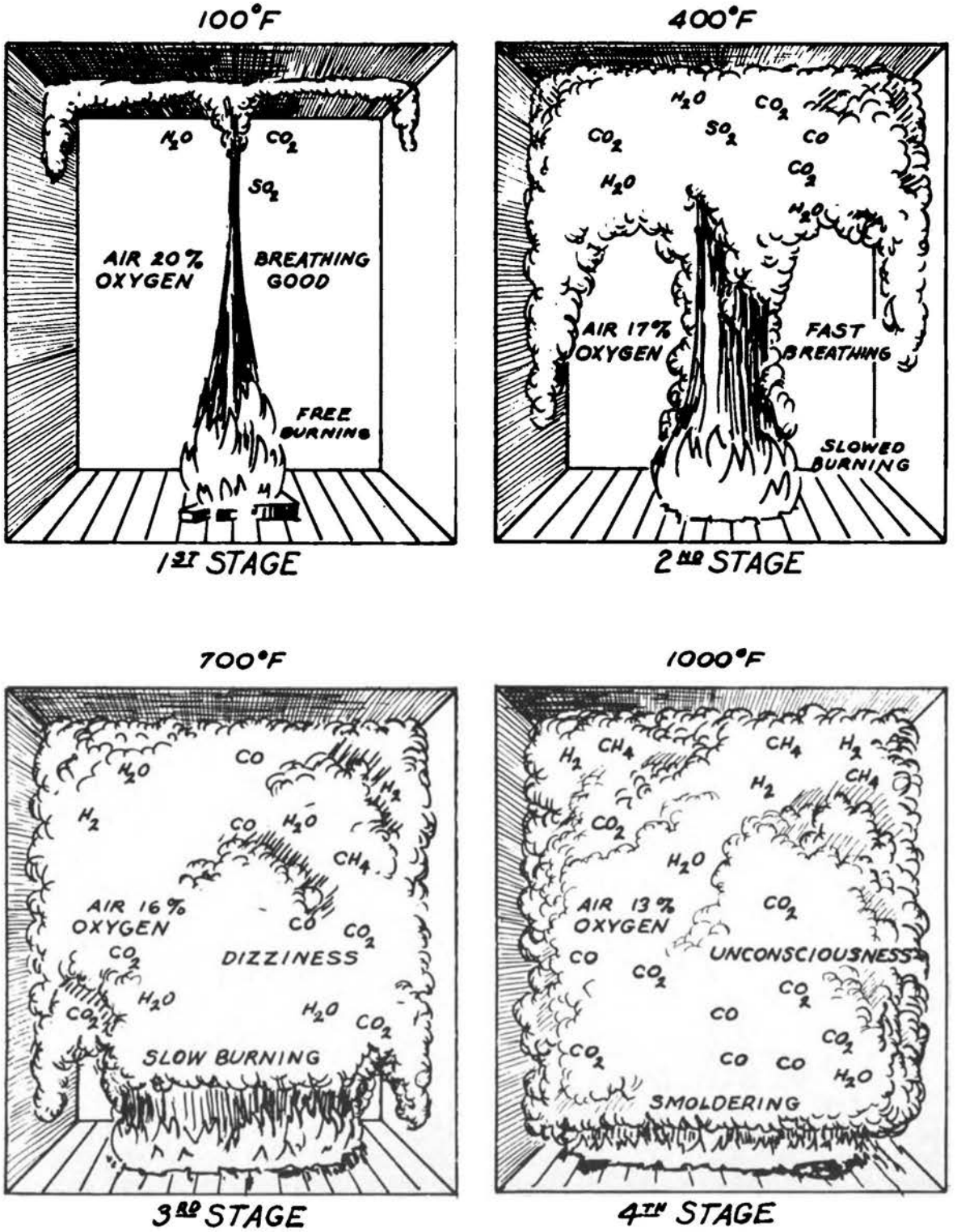


FIGURE 1. Progression of Fire in a Closed Room.

# *SMOKE CONTROL DURING FIRES IN HIGH-RISE BUILDINGS*

Sidney Ifshin, Deputy Chief
New York City Fire Department

Much has been written and said about smoke, or rather products of combustion (inasmuch as visible smoke consisting of particulate matter is only part of the products of combustion). I will use the term "Smoke" in the context of meaning products of combustion.

The fact that smoke hinders life rescue operation, fire fighting procedures, produces panic among occupants, and causes injury and at times death, has been known for many years. Nevertheless, in order to properly detail the reasons for the measures we took to attempt to control smoke (contained in the legislation mandating certain controls), I will briefly go into certain background material.

Several widely-publicized fires in modern, high-rise office buildings in New York City in 1970, namely, the One New York Plaza fire and the 919 Third Avenue fire, highlighted the awesome multiple fire death potential and raised public concern about the safety of those who work in such buildings.

<u>One New York Plaza Fire - August 5th, 1970</u>

50-story building - $10,000,000 damage

33rd and 34th floors fire involved

At One New York Plaza, the fire occurred after 5:00 p.m. and most of the occupants were gone. A most fortunate thing. The fire originated on the 33rd floor of the building. At approximately 5:45 p.m. the odor of smoke was detected on the 32nd floor. At approximately 5:50 p.m., the lobby began to fill with smoke. At about the same time, the 32nd floor (the floor below the fire floor) and other floors above the fire floor were becoming contaminated. An executive on the 43rd floor noticed a smarting and irritation of his eyes and then saw smoke in the vicinity of the peripheral air conditioning units. The building had one air conditioning zone for the entire building. The fact that the return fans on the air conditioning system kept going for quite some time was a factor in the contamination throughout the building. Because of the heat involved, we could use men for only 3 to 5 minutes each. We used 50 companies (300 men).

## 919 Third Avenue Fire - December 4, 1970

47-story building - $2,000,000 damage

At 919 Third Avenue the fire occurred at approximately 9:50 a.m. on the 5th floor. The building had two independent air conditioning zones, one supplying the 2nd through the 21st floor and the other, the 22nd through the 47th floor. The building engineer, when he learned of the fire, immediately shut down the supply and return fans for the zone for the fire floor but later started return fans for 100% exhaust. Employees on upper floors above the fire floor became panic-stricken when smoke seeped into their offices and they were unable to open windows for fresh air. Smoke issued from wall and ceiling air conditioners on the 11th floor. Occupants of the lower floors were subjected to severe smoke conditions and stairways filled with smoke. Smoke was also carried through elevator shafts and supply air ducts. Even though above the 22nd floor conditions were better than the floors below, employees who tried to use the stairs to descend, found them untenable at about the 33rd floor and turned around and went up to the roof. Fortunately, the fire was on the lower floor within reach of exterior Fire Department heavy deluge streams enabling quick extinction. In addition, fire separations through the suspended ceiling permitted containment of the fire itself within the area of involvement.

New York City reacted quickly to the potential described in this brief background picture. The Mayor appointed an "Advisory Committee To Improve Fire Safety in High-Rise Buildings" in response to the recommendations contained in a report prepared after intensive investigation of the previously-mentioned two high-rise fires by the Fire Department and the Building Department. The Mayor's Committee consisted of representatives of the Fire Department, Building Department, City Administration, Building Industry, Real Estate Industry, New York Board of Fire Underwriters, Labor Unions, and City Council. Charles E. Schaffner, Vice President of Syska and Hennessy (Consulting Engineers), was Executive Director and William Correale of Polytechnic Institute of Brooklyn was the Technical Director.

As a result of the report of the Mayor's Advisory Committee, a comprehensive Fire Safety System, including provisions for smoke control, was enacted into law for both new and existing buildings. My talk will be primarily on the Smoke Control portion of the legislation, as follows:

Smoke Exhaust Shafts are required for new office buildings over 100 ft in height that are not sprinklered. Highlights of the requirements for these shafts are as follows:

1. Provide at least 60 air changes per hour for the largest compartment served.

2. Smoke shaft to exhaust heat and smoke to the outdoors.

3. Maximum shaft velocity of 4,000 fpm and minimum 1600 fpm.

4. Maximum air velocity of 3,000 fpm at openings into smoke exhaust shaft at each floor.

5. Exhaust fan at roof, electrically serviced from emergency power source or a takeoff ahead of main switch.

6. System to be activated automatically by various smoke detectors required by Local 5 (1973), also can be activated manually at the mechanical control and at the Fire Command Station required by legislation.

7. Closures cover the exhaust shaft openings on each floor. Such closure for the individual compartment involved, opens automatically when the detector at the return shaft of the compartment or any other detector in the compartment is activated.

In lieu of a smoke shaft, existing buildings may provide a system of stair pressurization for fire emergency use. Fire towers are not required to have stairs pressurized. Highlights of stair pressurization requirements are as follows (not required for sprinklered buildings):

1. Supply fans to maintain a pressure differential between stair shaft and fire floor of at least .05 in. of water column. At all nonfire floors, the differential shall be at least .02 in. Net flow to be upward.

2. Maximum force to open a door shall be limited to 25 lbs at the knob. Mechanical devices may be used as required.

3. Maximum velocity through a single open door shall not exceed 2,000 fpm with all other doors closed.

4. Design to be based on 3 stair doors being open; i.e., fire floor door, generally a door above and a door below the fire floor.

5. Supply fan shall be electrically serviced from emergency power source or a takeoff ahead of main switch.

6. System to be activated automatically by various smoke detectors required by legislation. Can also be activated manually at the mechanical control center and at the Fire Command Station required by the legislation.

7. Existing buildings to comply within 3½ years of enactment of legislation (legislation enacted January 18, 1973).

Note: Prior to enactment of legislation containing stair pressurization requirements, a number of questions concerning the feasibility of pressurizing

stairwells were unresolved. Luckily, a 22-story office building at 30 Church Street was scheduled for demolition. The building, owned by the New York Port Authority, was made available to the Mayor's Committee for testing. A number of preliminary tests, using cold smoke, was made. Finally, four tests were run at three different locations on the 7th and 10th floors using smoke and heat generated by large-scale actual fires using office contents (desks, chairs, paper, typical office). Temperature, pressure, oxygen, carbon monoxide and smoke measurements were made. The report of these tests concluded that:

1. Pressurization of stairwells is feasible.

2. Smoke exhaust systems can remove smoke and heat from fire areas without excess temperatures in exhaust shaft. (We used stairway as smoke shaft.)

3. Maximum temperatures and pressures can be reached within four minutes of ignition.

4. Maintenance of the air supply and the air return of air conditioning systems can increase temperature, pressure, rate and extent of fire growth, as well as spread of smoke, gases, and heat.

5. Fire fighter controlled means of ventilation is essential.

6. Rapid spreading of toxic concentrations of gases can take place through ducts and ceiling plenums.

7. Attention must be given to fire stopping of plenum and elimination of combustibles from them.

As a result of these tests, the Smoke Control legislation in the bill was passed.

I would like to point out that during the drafting of the provisions for fire safety in high-rise office buildings by the Mayor's Advisory Committee, what had to be considered was smoke control for both existing buildings and new buildings.

For new buildings, the objective was removal of smoke by means of smoke exhaust shafts. For existing buildings, inasmuch as configuration of existing buildings is a decisive factor, the objective was a choice of either smoke removal shafts or pressurized stairways. The aim in both cases is to enable the occupant to leave the contaminated area in a reasonable time and manner. The difference between the two, of course, is that a smoke shaft directly removes the smoke from a contaminated space and thus limits contamination of other spaces and vertical arteries such as stairway and elevators, while pressurization of stairways limits contamination of stairway by increasing the air pressure of the stairway so that smoke from a contaminated area will not

enter, or will enter in lesser quantities in the event many stairway doors are opened at the same time.

The factors that were considered included the stack effect resulting from the combination of the height of the structure and the temperature differential between the inside of the buildings and the outside, the effect of wind velocities on smoke movement patterns, air conditioning systems and related items. The committee, after extensive investigation of many concepts and sources all over this country and other countries, opted for the smoke control system now in the legislation. If, in the future, the state-of-the-art and technological data indicates that refinements and changes are necessary, we have made provisions in the law for such changes with a minimum of complications.

Another provision in the law for smoke control, is that activation of smoke detectors at air return shaft at each floor, elevator landing, lobby, and in compartmented space, will do the following:

1. Stop the supply and return air of fire floor only, by activation of approved remote control reversible fire shutters or as an alternative, by automatically shutting down supply and return air fans serving fire floor. The first affects air supply and return of contaminated floors only and the other floors are unaffected. The other alternative generally affects a number of floors served by the related air conditioning zone, even though the other floors may not be contaminated.

2. Smoke detector activation will also trigger the fans and dampers for smoke shafts or pressurized stairs.

The compartmentation requirement in the law contains the fire in a limited area and also provides an area of refuge for the occupants giving them additional time to leave the floor via the stairway, as follows:

1. If compartment or space is more than 7,500 sq ft, then fire separation is required. Fire separation penetrates the hanging ceiling to the floor slab above.

2. If compartment is more than 10,000 sq ft, then fire separation has to create two areas of refuge for either portion, with a stairway in both areas of refuge.

3. However, if space or compartment has complete area smoke detection, then undivided compartment can be up to 15,000 sq ft in area.

4. If compartment is more than 15,000 sq ft, then entire floor has to be sprinklered.

It is apparent that even though the Fire Code provisions for immediate implementation of Fire Drills, Fire Safety Director, and Fire Safety Plans for existing buildings are an improvement, the potential for multifire death incidents is still very evident and will be until the Building Code legislation (which has a time schedule for existing buildings) has been implemented. An example of some of the items in the fire reports that were flagged (with respect to smoke contamination) are:

37-story office building

Fire after office hours -
Stairway filled with smoke - woman at 31st floor could not use stairs because of contamination.

46-story office building

Fire at 6:40 p.m.
Smoke from 6th to 40th floor -
Origin of fire - 6th floor.

45-story office building

Fire at 7:00 p.m.
Smoke from 2nd to 45th floor
Origin of fire - 2nd floor.

19-story building

Fire at 10:45 p.m.
Smoke did not become heavy until 9th floor and was heaviest at upper floors. (Stairway smoke not too heavy.)
Origin of fire - 1st floor elevator shaft.

50-story office building

Fire after working hours
Smoke filled hallways and upper portion of stairway.
Origin of fire - 5th floor elevator landing lobby.

34-story office building (Hurok Fire)

Fire before 10:00 a.m.
Smoke carried to upper floors by means of mail chutes, stairs and elevator shafts. Rendered stairs unusable almost immediately.
Floor of origin - 20th floor.

I wish to caution that the above items are not an analysis of the fires because in many, there were varying circumstances. I just point them out as an indication of smoke travel.

In conclusion, I emphasize that smoke control is only a part of the total building fire safety system that is in the legislation, such as compartmentation, alarm and communication system, elevator controls, detectors, fire command station and fire safety plan, etc., and is to be considered in such context.

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# *METHODS FOR COMBATING SMOKE*

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The necessity of combating smoke is a problem which the fire service has been confronted with since the first bucket brigade attempted to deal with an unfriendly fire. As our extinguishing techniques developed and became more scientific, the need for smoke control or ventilation has become more obvious. In conjunction with our other basic training considerations, such as fire streams, hose handling, pumper operation, ladder handling, forcible entry, salvage and overhaul, etc., ventilation is dealt with as a necessary basic knowledge for fire fighters.

The goal of the fire service is to protect life and property. On the scene of a fire this goal transforms into rescue of trapped victims, locating the fire, and extinguishing the fire. In most cases, if we are successful at fulfilling these objectives, fire fighters must enter the confines of the structure involved with fire which subjects the fireman, not only to heat produced by the fire, but to a further reaching product of fire - Smoke. Therefore, combating smoke is often of greater priority to the fire officer, at least in the first few minutes at a fire, than our primary objectives of rescue and extinguishment. Ventilation, with all the consideration it must be given in the overall picture of fire fighting, is the most complex, unpredictible and least used of all our skills. Traditionally, the International Fire Service Training Association has defined ventilation as "The planned and systematic release and removal of heated air, smoke, and gases from a structure and the replacement of these products of combustion with a supply of cooler air."[1]

How to achieve this objective has stimulated many all night discussions in fire stations across this country. Since no two fires are alike, most seasoned fire fighters have witnessed the unpredictible results of improper ventilation such as a back draft or smoke explosion. Smoke explosion is a term used to define a type of explosion which may occur when unburned gases ignite and burn rapidly. Situations which create a smoke explosion are confinement and intense buildup of heated gases in an atmosphere being depleted of oxygen. To a fire fighter ventilation is not the answer—ventilation is the question. The question of when to ventilate, where to ventilate, with what to ventilate, and how to ventilate must be carefully considered prior to ventilation. My subject, "Methods for Combating Smoke" as it implies, deals with how smoke is dealt with.

First, the fire service is concerned with providing features in building which can aid them in smoke control, or better yet, prohibit or at least reduce smoke migration. This approach is best satisfied through a building code. As pointed out in a study printed by the Council of American Building Officials, "the fire protection profession has generally relied on construction regulations to achieve an adequate level of safety. These regulations have emphasized fire resistive construction, slow burning exterior finish and adequate enclosed exitways."[2] In other words, our average code has approached the problem of fire safety by developing requirements which deal directly with fire itself. Fire rated walls and doors, fire dampers in the heating, ventilating, and air conditioning systems, flame spread requirements for interior finishes, etc. The necessity of this type of requirement is obvious, but as stated in Fire Protection of High Rise Buildings, "smoke spread is unquestionably the most significant life hazard problem existing at the time of a fire in a high rise building."[3] Smoke kills the majority of fire victims. "Seventy percent of fire victims are killed, not by heat or flame, but rather asphyxiated."[4] This disturbing percentage will hopefully emphasize that attention to code requirements should be away from property considerations which are fire hazards, toward life considerations which are smoke hazards. This position is supported in the report of the National Commission on Fire Prevention and Control, "The hazards of flames have been studied and regulated to some extent, but recognition of the hazards of smoke and toxic gases has come belatedly. Ironically, efforts to make materials fire-retardant may have increased the life hazard, since incomplete combustion of these materials often results in heavy smoke and toxic gases."[5] Even if the treatment of smoke problems has come belatedly, much research and work has been done in recent years. Rather than deal with deficiencies in our codes, I choose to review, in a general manner, some of the solutions to the smoke problem.

Compartmentation is a concept which divides a building into separate areas. The intent is to provide areas of refuge within the building to which people can go in order to escape fire or smoke which is being generated in another compartment. Compartmentation can be accomplished vertically by creating a fire rated and smoke barrier separation between floors or can be established horizontally by dividing a given floor into two or more separate compartments by means of fire walls and smoke barriers. Ideally, the fire and smoke will be confined in a compartment while other compartments in the building will be maintained at a normal level.

Products of Combustion Detectors are early warning devices which sample the air and activate alarms when minute products of combustion are detected. These devices function within 20 seconds after a fire starts and their primary purpose is to alert a building's occupants and allow for evacuation or control of the fire during its incipient phase.

Pressurization of the corridors, smoke towers and elevator lobbies is a concept which is gaining support as a smoke control method. In the proceedings of the Chicago Committee on High Rise Buildings, Mr. Harold Nelson states, "I believe pressurizing is going to be one of the most useful

methodologies of developing smoke control without spending a fortune on new systems and devices."[6]

The pressurization concept is based on providing a positive pressure in exitways and a negative condition in rooms. The higher pressure forces the fire and smoke to remain in the negative area and, therefore, provides smoke-free areas for escape and evacuation from the building.

In the greater Miami area, the South Florida Building Code is the standard for all construction. This past summer, the fire service was instrumental in the adoption of a Fire Suppression-Life Safety System as a chapter in the code. The primary feature of the chapter is the requirement of an automatic sprinkler system. The International Association of Fire Chiefs have long advocated and supported sprinkler systems as the best approach to life and fire safety protection. Chief Volkamer, recently retired Fire Chief from Chicago, puts it this way, "Automatic sprinkler systems are the only complete answer; again, we realize this is the epitome of fire protection."[7]

Our new fire suppression—life safety system ordinance—requires buildings exceeding 75 feet in height to have the following features:

1. The entire building shall be protected by an Automatic Sprinkler System.

2. A manual operated Fire Alarm System shall be provided.

3. An approved system which will provide automatic detection of products of combustion other than heat, in mechanical equipment rooms, return portion of the air-conditioning and ventilating systems. These detectors will activate a Voice Alarm System, announce to a central control station and shut off all ducts not a part of the smoke control system.

4. The Fire Detection System and the Fire Alarm System shall activate a Voice Alarm System. The Voice Alarm shall be designed to be heard by all occupants within the building.

5. There shall be two separate Voice Communication Systems; one two-way system for Fire Department use and the other for a public Voice Communication (Public Address) System.

6. A Central Control Station for Fire Department operation shall be provided near the street level building entrance. It shall include the voice communication system panel, fire detection and alarm system panel, status indicators and controls for elevators, air handling systems, a

public telephone, sprinkler valve and water-flow detector indicators and standby power controls.

7. A Smoke Control System to remove products of combustion shall consist of one of the following:

   a. Panels or windows in the exterior wall which can be opened from an approved location, other than the fire floor.

   b. The mechanical air handling equipment may be designed to assist removal of products of combustion.

8. Standby Power and Lights shall be provided within the building.

9. Elevators shall be arranged for Fire Department use.

10. The above requirements are in addition to other exit and fire protection requirements existing in the code.

In buildings more than 36 feet or more than three stories, the following are required:

1. Automatic Sprinkler System.

2. Smoke Control System.

3. Elevators provided for Fire Department use.

4. Other exit and fire protection requirements existing in the code.

The Miami Fire Department is confident that this new code will greatly enhance our capabilities to combat smoke effectively in new buildings. We are now turning our thoughts toward developing a life safety system for existing buildings.

The preceding cursory overview of smoke control features, hopefully points out how smoke can be combated using a building code as a vehicle.

A second method of combating smoke is the implementation stage of codes; plans must be examined and construction must be inspected. Traditionally building departments have been responsible for plans, examination and new construction inspection. Fire departments have concerned themselves with enforcing fire prevention codes, which are in essence concerned with maintenance of existing conditions. In *America Burning*, The National Commission on Fire Prevention and Control considers this subject, "The effectiveness of codes is compromised by lack of coordination among inspection

programs. The building department generally has responsibility for enforcing building codes, the Fire Department for enforcing fire prevention codes. Because fire prevention bureaus are responsible for fire safety throughout the life of a building, they ought to be consulted by building departments during the design and construction phases."[8] Transforming building plans to finished building leaves room for error in code requirements. Integrity of rated fire walls can be violated by improper installation of a standpipe hose cabinet, chases running vertically through the building can be left unprotected, poke throughs are left unsealed, compartmentation is violated by not continuing walls from slab to slab. Recently I had the reality of code intent and code fulfillment forcefully brought to my attention when a fire occurred on the ninth floor of a 10-story apartment building that was nearing the final stages of construction. The fire was started by a plumber using a torch and originated in a combustible fiber board material being used for sound attenuation. This fiber board was installed on metal studs and covered with sheet rock and plaster. The fire smoldered and was finally discovered four hours after it had started. Our fire suppression forces responded and extinguished the fire. A fire inspector investigated the fire that night and on reporting to work the next morning researched the records and found a similar fire had occurred in the same building a few weeks prior. When the matter was brought to my attention, I directed him to return to the scene and photograph the fire area. After being back on the scene for approximately an hour, a smoldering fire was discovered still existing and slowly creeping through the fiber board. This discovery was made some 14 hours after the Fire Department had extinguished the fire. In a closed building the smoke created by such a smoldering fire would have created a severe life hazard. My point here is code requirements as compared to code fulfillment. Our building code calls for Type I or incombustible fire-resistive construction in this type building. The plans of this structure so indicated, but the building in its final stages was insulated to provide sound proofing and, therefore, a combustible and potentially deadly product was incorporated into the structure. Inspection of this building during construction by a life safety-oriented inspector could have prevented this condition. We in the fire service are oriented toward life safety where building inspectors, while concerned with life safety, must concern themselves with many other features including the prime concern for structural stability. The occupants of a building are entitled to all these concerns and the time has come for the fire service to assume its duty in providing life safety features in buildings as the code requires. This can only be done by on-site inspections during construction.

A fireman who has experienced the effects of a smoke-filled building, while extinguishing a fire, certainly has a first-hand understanding of the need for life safety code requirements.

Fortunately, the City of Miami Fire Department has had a competent plans examining section for several years. This section has much expertise in life safety requirements as provided in our code and has complete responsibility and authority for approving this portion of the plans. In conjunction with our plans examiners, we have recently entered the area of new construction

inspection and our Fire Prevention Bureau will be the responsible agency regarding fire and life safety enforcement. This approach is certainly a noteworthy method to combat smoke.

While gathering information for this paper, I visited our Fire College and spent a morning talking to one of the lieutenants that instructs the rookies in basic training, which includes ventilation. I pointed out how I intended to treat the subject of combating smoke, emphasizing code requirements, code enforcement, and even mentioned an experiment being conducted by the Tokyo Fire Service, which concerned removal of smoke by an electrostatic smoke particle collecting system that attracted smoke particles to electrodes installed on walls and ceilings.[9] He let me ramble for some time, but interrupted and abruptly brought me back to the day-to-day reality of the fireman by saying, "That's great for these kids I'm training, but what about me tonight at 2:00 a.m. when I roll up on a smoke-filled hotel?" This sometime neglected fact of the present brings me to my final method of combating smoke. How do firemen cope with smoke problems in the majority of buildings which do not include the sophisticated systems I've mentioned thus far?

There are three basic methods of ventilation used by the fire service:

1. A common method by which smoke and heat is removed from a burning building is by opening the structure at a strategic location. This technique is accomplished by top or vertical ventilation as well as cross or horizontal ventilation. This method is often referred to as natural ventilation because it depends on the natural flow of air currents.

2. Forced ventilation is a method which utilizes mechanical blowers or fans. This type of ventilation is used often, either because a structure is unsuited for natural ventilation or because there is a need to expedite smoke removal.

3. The application of water fog to control confined fires is an established extinguishment technique. A beneficial side effect is that it materially aides ventilation. The application of water as a spray stream has the ability to attract or collect the carbon, tar, and ash particles from smoke. Also, as these water particles absorb heat from the heated area the water is converted into steam which in its expansion process, forces the heat and smoke out of the building.

Although not a method of smoke removal, the use of self-contained breathing apparatus must be mentioned as a method of combating smoke. Fire fighters, by the nature of their work, must enter contaminated areas if rescue and fire extinguishment is to be accomplished, therefore, protection must be provided for their respiratory systems.

The fire service ideally combats smoke prior to a fire by requiring and enforcing regulations which restrict and confine smoke. After a fire has started the job of smoke control is difficult at best.

The fire service supports forums such as this and it is every fireman's hope that out of these next few days more knowledge will become available and maybe we can soon say that ventilation is the answer—not the question!

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*SESSION II*

# SMOKE AND FIRE CASUALTIES

Moderator:

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# *STUDY OF FIRE DEATHS IN MARYLAND*
## *Sept. 1971 - Jan. 1974*

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This report deals with our studies on fire victims, carried out in cooperation among our three laboratories. We are also indebted to the Fire Marshals and Fire Department personnel, whose aid has been invaluable in investigating the circumstances of the fires.

One hundred seven fatalities have been investigated from 85 fires. To be included in this series, the case had to have died at the fire and had an autopsy by the Medical Examiner's Office, with carboxyhemoglobin measured in the School of Hygiene laboratory. In addition other postmortem chemical measurements including blood methemoglobin and alcohol have been done, and all cases have had detailed evaluation of the coronary circulation at autopsy. A thorough review of the circumstances at the fire scene has been carried out, of critical importance in evaluating the cause of death. Because of the necessity for an autopsy, this series cannot be considered representative of all fire deaths in this state. The cases have been drawn primarily from Baltimore City and Baltimore, Anne Arundel, and Prince George's Counties. We estimate that between one-fourth and one-third of all fire fatalities that have occurred in Maryland in this time period are included.

The causes of the fires are shown in Table I. There were 85 separate fires represented; with 15 fires where 2 cases died; 2 fires with 3 fatalities; and 1 with 4 fatalities. The overwhelming role of smoking is evident. Eight fires were caused by children playing with matches. Table II shows the time of day for these 85 fires. It is evident that there was a preponderance of fatal fires at night, in contrast to the distribution of fire calls in general, which peak in the late afternoon around 5:00 p.m.

The age distribution of the cases is shown in Table III. In this sample approximately half of the cases were 40 years of age or older, and nearly one-fourth were children under the age of 10. Thus the principal victims at fires are children and older adults.

Figure 1 shows the factors contributing to the deaths of the 107 cases. In the Figure three groups are shown depending on whether carbon monoxide was definitely, probably, or not contributory to the death. The large number of cases with coronary vascular disease present at the time of the fire is especially striking. Of these cases dying at the fire, carbon monoxide alone can account for the death in 48; in most cases carboxyhemoglobin (COHb) was greater than 65%. A blood COHb of over 65% is considered to be nearly always lethal due to depression of respiration and the circulation. Between 50 and 65% COHb, death has been known to occur, and some of the cases with blood COHb were in this range. High blood alcohol was also present with liver disease as a contributory factor in several of these cases with COHb between 50 and 65%.

It is evident from Figure 1 that carbon monoxide was a major contributor to death. Eighty-nine cases had blood COHb over 20%, and in 35 of these cases the combination of CO and coronary vascular diseases was sufficient to account for sudden death. Thus a second major contributing factor to death in this group was the presence of pre-existing cardiovascular disease.

Analysis of the data indicates that for the groups with more than 20% COHb, the incidence and severity of coronary vascular disease was inversely related to the level of COHb reached. This observation is consistent with the high blood COHb and coronary vascular disease together leading to death in 35 cases. The terminal COHb is mainly a function of the depth and duration of breathing, and a high blood COHb at death requires that the cardiopulmonary system continue to function until that concentration was reached. Those cases with lower blood COHb had cessation of either respiration of circulation with less absorption of CO. Ventricular fibrillation from acute myocardial hypoxia induced by a rapid rise of COHb in individuals with a restricted coronary circulation is a highly likely explanation of a lower terminal blood COHb the more severe was the pre-existing coronary vascular disease. The possibility that other pyrolysis products may have also contributed to ventricular fibrillation in the coronary cases cannot be ruled out, but one may conclude that unless their concentrations were comparable to CO in the fire, and their mode of action potentially as rapid as CO, they are unlikely to affect the myocardium more rapidly or severely than CO.

Another factor of importance in this series is the extent of body surface burned. In Table IV is shown the number of cases by the proportion of skin burned and the carboxyhemoglobin present at autopsy. Of the 107 cases, 72 had moderate or severe skin burning (50% or more of surface burned), but only in 18 cases is the burn the cause of death. Also shown in Table IV are 18 other cases where burns could have contributed to death. Inflammation of the respiratory tract was present in a few cases, especially in cases where clothing was the principal material burned.

Burns alone appear to be a relatively infrequent cause of death in cases that die at the fire. It is significant that 32 of 72 cases with COHb over 50% had mild or no burns of the body surface. The inference is that if these people had not been overcome by carbon monoxide they could have escaped death

in the fire. This point raises the question of why the victims did not escape. One case was a fireman who died of an acute myocardial infarction associated with severe coronary vascular disease immediately after leaving a smoky fire. He had 6% COHb, which may have been contributory, although the major cause was vascular disease. Two cases died of clothing fires outdoors. With these exceptions, all the cases that died at the fire either never escaped or were overcome after entering the burning building.

Table V shows an analysis of these cases in terms of blood COHb and blood alcohol. Of the 79 cases over age 18, 53 cases (67%) had COHb greater than 40%, an amount consistent with loss of sensorimotor function leading to collapse. In addition, 28 cases had blood alcohol above 0.15 gm/100 ml (0.15%), considered to be at or above the intoxication level for most individuals, with ataxia, slow reaction time, and impaired judgment. The highest alcohol was 0.39% in two males, 29 and 45 years old. An additional 14 cases had blood alcohol between 0.05% and 0.15% or a level which, taken with a high blood carboxyhemoglobin, also probably contributed to the loss of neuromuscular function. Of this group of 79 cases, most had enough absorption of CO to impair their ability to escape, but alcohol ingested prior to the fire also played an importance in 28. A similar pattern is present in the group of nine cases with COHb between 20 and 40%. Five of these cases had alcohol above 0.15%, and the combination of alcohol and CO can account for loss of neuromuscular function and impaired judgment.

Most of the fire victims made some attempt to escape, and Table VI summarizes the results of an analysis of why the victims did not escape from the fires. In four cases it was not possible to determine whether an attempt to escape was made, but in the rest of the cases nearly two-thirds showed evidence of having made escape attempts, or had re-entered the fire for rescue or other purposes.

In Table VI, it is clear that carbon monoxide, with or without previous ingestion of alcohol, accounts for the failure to escape in more than 60% of the cases. Many of the infants (age 4 or less) that were killed, also died of CO inhalation. In many fires the victim was found at some distance from the burned material, and thus if instructions on how to avoid CO exposure had been followed, these victims need not have died.

In summary, four factors have been identified as contributing to deaths of individuals who die at the fire itself. Of these factors two relate to conditions existing prior to the start of the fire. First is alcohol ingestion, a well-known problem in fire victims. Excessive consumption may be related to the cause of the fire as well as why the victim does not escape. Blood alcohol was over 0.15% in 28 (35%) of 79 cases over the age of 17. Second, and less well appreciated, is moderate to severe coronary vascular disease, found to be present in 33 (45%) of 73 cases over age 20. This pre-existing disease is particularly important when carbon monoxide may be inhaled. CO interferes with oxygen delivery to the heart and in these individuals their inability to compensate by increasing blood flow may suffice to cause ventricular fibrillation and rapid death.

Two other factors resulting from the fire are also of major importance as causes of death. First, of course, is extensive burning of the body, including in some cases the upper airway, but of the 107 cases dying at the fire, only two-thirds were moderately or extensively burned. In general we consider most of the burns to be secondary, in many cases occurring after death. The location of the body in relation to the origin of the fire is the principal basis of this conclusion. The final factor is carbon monoxide, present in significant amounts in the blood and definitely or probably contributing to the death in 84% of the cases dying at the fire. Clearly carbon monoxide exposure is a major problem in fires, and many cases die with no other cause present (unburned, found dead remote from the fire).

The contribution of smoke or other pyrolysis products to death or acute symptoms cannot easily be assessed from postmortem examinations alone. Most of the 107 cases had obviously inhaled smoke, and evidence of pulmonary edema was present in a few of the cases. Soot was usually present in the trachea and bronchi, and often there was inflammation of the respiratory tract. In many of the 18 cases designated as burn deaths (Table IV), there is a definite likelihood that inhalation of smoke or other fire products was an important factor in causing the death of these individuals although whether glottal spasm or laryngospasm was involved is not obvious from the postmortem results. Despite the importance of carbon monoxide as a lethal factor in fires, these other fire products are also important, especially in casualties surviving the fire. It is clear that in some of our cases acute effects of smoke products on the respiratory tract were significant.

TABLE I

ORIGIN OF FIRE
(85 fires with fatality at fire)

| | | |
|---|---|---|
| Smoking | 47 | 55.3% |
| Matches or candles | 10 | 11.8% |
| Heater or flammable material next to heater | 7 | 8.2% |
| Arson | 5 | 5.9% |
| Suicide | 4 | 4.7% |
| Clothing on fire | 3 | 3.5% |
| Electrical | 3 | 3.5% |
| Other (gasoline, fireman, auto accident) | 3 | 3.5% |
| Unknown | 3 | 3.5% |
| Totals | 85 | 100.0% |

TABLE II

TIME OF FIRE
(85 fires with fatality at fire)

| Time | Number | % |
|---|---|---|
| 12:00 - 1:59 a.m. | 10 | 11.8 |
| 2:00 - 3:59 a.m. | 17 | 20.0 |
| 4:00 - 5:59 a.m. | 8 | 9.4 |
| 6:00 - 7:59 a.m. | 6 | 7.1 |
| 8:00 - 9:59 a.m. | 8 | 9.4 |
| 10:00 - 11:59 a.m. | 2 | 2.4 |
| 12:00 - 1:59 p.m. | 5 | 5.9 |
| 2:00 - 3:59 p.m. | 8 | 9.4 |
| 4:00 - 5:59 p.m. | 3 | 3.5 |
| 6:00 - 7:59 p.m. | 4 | 4.7 |
| 8:00 - 9:59 p.m. | 7 | 8.2 |
| 10:00 - 11:59 p.m. | 7 | 8.2 |
| Totals | 85 | 100.0 |

TABLE III

AGE DISTRIBUTION
(107 fire deaths)

| Age | Number | % |
|---|---|---|
| 0 - 4 | 15[a] | 14.0 |
| 5 - 9 | 10[a] | 9.3 |
| 10 - 19 | 7 | 6.5 |
| 20 - 29 | 10 | 9.3 |
| 30 - 39 | 11 | 10.2 |
| 40 - 49 | 15 | 14.0 |
| 50 - 59 | 15 | 14.0 |
| 60 - 69 | 12 | 11.2 |
| 70 - 79 | 9 | 8.4 |
| 80+ | 2 | 1.8 |
| Unknown (adult) | 1 | 0.9 |
| Totals | 107 | 100.0 |

[a]Five fires with more than one child: 3 with 2 children, 1 with 3 children, and 1 with 4. Thus, total fires involving children under 10 = 17.

TABLE IV

DEGREE OF BODY BURNED
(107 fatal cases)

| Degree of Burn | Blood Carboxyhemoglobin | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | >65% | 50-65% | 35-49% | 20-34% | 5-19% | <5% | Total | % |
| No or slight burn (<25% of body surface) | 23 | 9 | 0 | 2 | 1 | 0 | 35 | 32.7 |
| Moderate burn (25-50% of body surface) | 4 | 3 | 1 | 0 | 0 | 1 | 9 | 8.4 |
| Severe burn (>50% of body surface) | 21 | 12 | 8 | 6 | 11 | 5 | 63 | 58.9 |
| Total Cases | 48 | 24 | 9 | 8 | 12 | 6 | 107 | 100.0 |
| Burn probably cause of death (including respiratory) | 0 | 0 | 4 | 3 | 8 | 3 | 18 | 16.8 |
| Burn possibly contributory to death | 2 | 7 | 3 | 3 | 2 | 1 | 18 | 16.8 |

TABLE V

CARBOXYHEMOGLOBIN AND BLOOD ALCOHOL

| Blood Alcohol gm/100 ml | COHb% >40 | COHb% 20-40 | COHb% <20 | Total | % |
|---|---|---|---|---|---|
| | | Cases Age 18 and Over | | | |
| None | 22 | 3 | 10 | 35 | ( 44) |
| <0.05 | 0 | 1 | 1 | 2 | ( 3) |
| 0.05-0.15 | 13 | 0 | 1 | 14 | ( 18) |
| 0.16-0.25 | 11 | 4 | 4 | 19 | ( 24) |
| >0.25 | 7 | 1 | 1 | 9 | ( 11) |
| | 53 | 9 | 17 | 79 | (100) |
| | | Cases Under Age 18 (No alcohol present) | | | |
| | 23 | 2 | 3 | 28 | |
| Total All Cases | 76 | 11 | 20 | 107 | |

TABLE VI

FACTORS RESPONSIBLE FOR FAILURE TO ESCAPE FROM FIRES

| Reason for Failure to Escape | Attempt to Escape | | | Total | % |
|---|---|---|---|---|---|
| | Yes | No | Undetermined | | |
| Carbon Monoxide Alone | 23 | 6 | 3 | 32 | 29.9 |
| Carbon Monoxide + Alcohol | 31 | 6 | 0 | 37 | 34.6 |
| Alcohol Alone | 3 | 0 | 0 | 3 | 2.8 |
| Burn (including Respiratory) | 5 | 0 | 0 | 5 | 4.7 |
| Coronary Occlusion | 2 | 1 | 0 | 3 | 2.8 |
| Infant | 1 | 12 | 0 | 13 | 12.1 |
| Invalid | 1 | 3 | 0 | 4 | 3.7 |
| Explosion | 0 | 3 | 0 | 3 | 2.8 |
| Clothing Fires (generally suicides) | 2 | 3 | 0 | 5 | 4.7 |
| Suicide | 0 | 1 | 0 | 1 | 0.9 |
| Car Accident | 0 | 0 | 1 | 1 | 0.9 |
| Totals | 68 | 35 | 4 | 107 | |
| | (63.6%) | (32.7%) | (3.7%) | | |

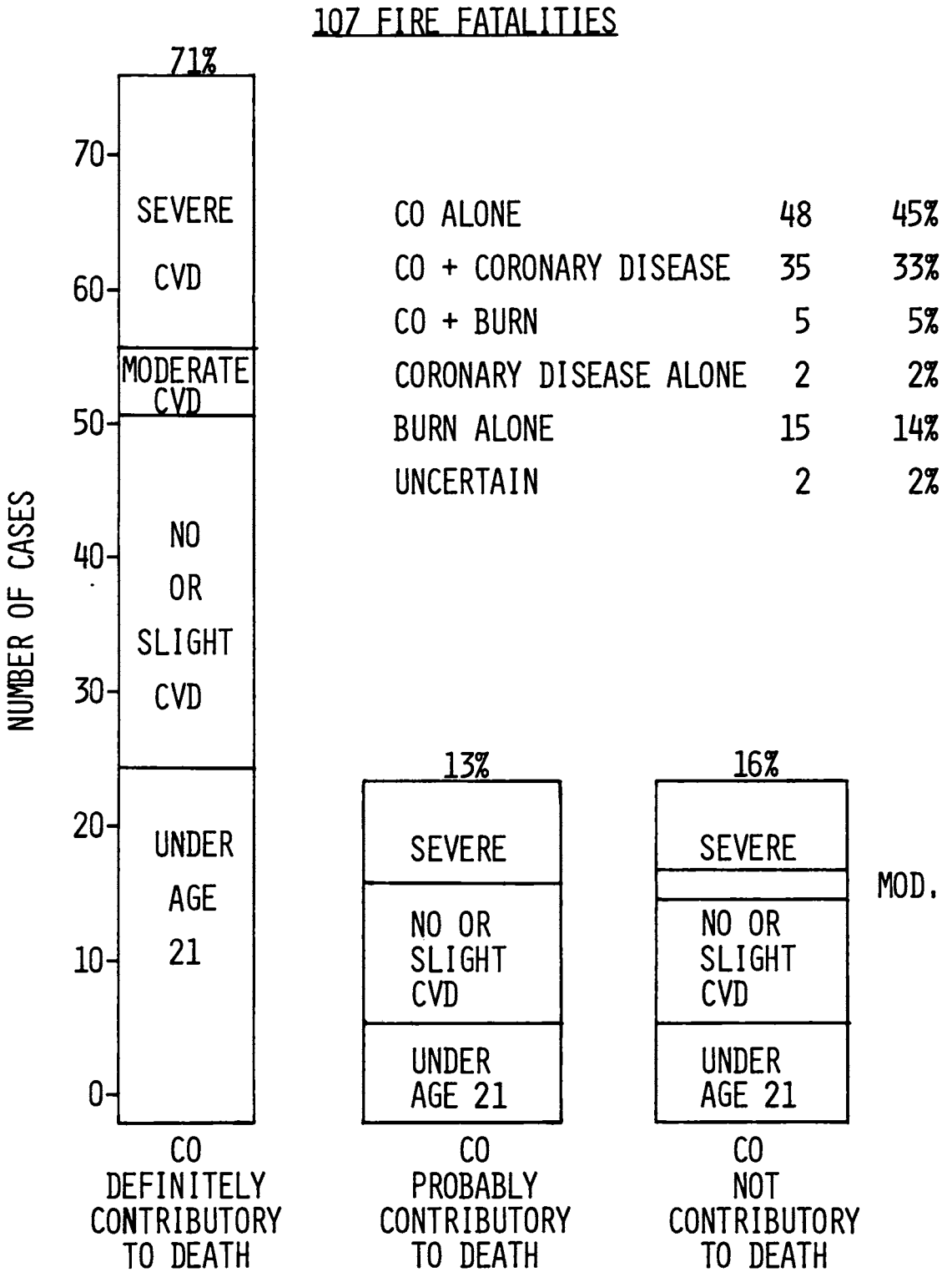


FIGURE 1. CARBON MONOXIDE AND CORONARY VASCULAR DISEASE AS CAUSES OF DEATH.

# *A CLINICAL VIEW OF "SMOKE POISONING"*

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The scientific definition of smoke and the medical meaning of "smoke poisoning" and "smoke inhalation" have not been well understood. The purpose of the present investigation is to define more specifically what "clinical smoke poisoning" is as we understand it at the present time. "Respiratory burn" pathophysiology has been described previously.[1]

In 1962 Phillips and Cope[2] labeled respiratory tract damage as a "principal killer" in burn victims. In 1967, Stone reported "respiratory burns" in 15% of 197 patients some of whom may have had concomitant smoke poisoning.[3] Because of recent recognition and difficulty in differentiation between respiratory burn and smoke poisoning, there have been no available clinical statistics concerning incidences of the two conditions or the incidence of their coexistence.

In 1971 our analysis of the cause-of-death among fire fatalities in New York City during the years 1966 and 1967 revealed that 50-60% of these victims died at the site of the fire or on the way to a hospital. Smoke poisoning and/or asphyxia was listed as the primary cause of death in 30% of all fire fatalities. Respiratory tract involvement was found in 70% of deaths occurring within 12 hours and in 46% of victims who survived more than 12 hours. Of 185 victims who survived less than 12 hours, 53.5% had smoke poisoning and/or asphyxia as their only significant acute pathology. Significantly, 79% of those having primary diagnosis of smoke poisoning and/or asphyxia had carbon monoxide poisoning. Also 24% of these patients had lethal levels of CO (above 50% carboxyhemoglobin saturation).[4]

Since almost one-third of all fire fatalities are due to "smoke poisoning" it behooves us to understand this syndrome better which may help our understanding of the subtle chronic effects of air pollution and cigarette smoking so that we may find effective and expeditious treatments and methods for their prevention.

## MATERIALS AND METHODS

In the 13-year period from 1960 through 1972, 51 patients were admitted to Columbia-Presbyterian Medical Center with the primary diagnoses of "smoke inhalation," "CO poisoning" or "respiratory burns." Patients whose main problem was their body surface burns were not included in this study. Thirty of these patients were admitted during 1960-1970 and 21 patients in the two years thereafter. Their records were evaluated critically with respect to admission diagnoses, laboratory analyses, hospital course and treatment.

## RESULTS

Forty (78%) had "smoke poisoning" as their sole diagnosis on the basis of having been exposed to the smoke of a fire with minor or no body burns. There were six primary clinical diagnoses of carbon monoxide poisoning and five patients with the primary diagnosis of respiratory burns. The ages of patients ranged from 6 to 76 years, and there were 37 males and 14 females (Figure 1).

During the decade of 1960 to 1970 there were no blood carbon monoxide determinations because emergency room equipment for the test was not available (Figure 2). With the availability of a quick laboratory determination in the two-year period that followed (1971-1972) all 21 patients admitted for possible inhalation injuries from fire were tested for carbon monoxide poisoning (Figure 3).

Clinical diagnosis of CO poisoning without the help of blood CO determination was made in five patients during 1960-1970 primarily on the basis of history and neurologic sequelae (Figure 2). It is interesting to note that clinical diagnosis of acute CO poisoning was made on the basis of cherry-red skin in only one patient; this patient was subsequently found to have a blood CO saturation of 51%. Seventeen out of 21 (80%) who had acute CO poisoning as manifested by blood CO determination were subsequently shown to have carboxyhemoglobin saturations >15% (Figure 3).

This study also shows that where clinical diagnosis of "smoke inhalation" or "smoke poisoning" is made, if blood CO level is determined 16/19 (84%) will reveal some degree of carbon monoxide poisoning (CO saturation above 15%). Nine patients had carboxyhemoglobin saturation of 29% or more.

Ten patients (20%) had sustained burns of face, upper trunk and extremities. Their burns were mostly 1st and 2nd degree burns. Patients with the primary diagnosis of smoke poisoning had an average of 0.97% body burns whereas respiratory burn patients had an average of 12.4% body surface burns. Only one of these patients had a third degree burn necessitating a small skin graft (Figure 1).

Ten patients (25%) with the primary diagnosis of smoke poisoning and all five patients with the diagnosis of respiratory burns had some visible evidence of respiratory tract injury ranging from erythema and blisters of the

oropharynx to congestion and edema of the tracheobronchial tree as observed by direct or indirect laryngoscopy or bronchoscopy. In only three patients did the initial chest x-ray reveal pulmonary edema (Figures 2 and 3). Although only three chest x-rays showed initial pulmonary changes, a total of eleven patients (28%) developed either pulmonary edema or x-ray evidence of pneumonitis during their hospitalization. The rest of the patients were treated for atelectasis, probable penumonitis or bronchitis on the basis of fever and their chest physical findings.

Since 1970, the mode of initial respiratory resuscitation has been influenced greatly by the admission arterial blood gases and CO determinations. Six required mechanical ventilation, three via tracheostomy, and in three patients, nasotracheal or orotracheal tubes were used. Seven patients were treated with IPPB, five of whom required tracheostomy. The remaining cases received humidified oxygen by nasal catheter or oxygen tent for varying periods of time.

Fourteen patients were placed on prophylactic antibiotics, usually Penicillin, though Ampicillin, Keflin and Tetracycline were also utilized. An additional six patients developed significant pulmonary infection requiring specific antibiotic therapy.

Ten patients were treated with bronchodilators and three elderly patients required digitalis and/or diuretics for delayed pulmonary edema and congestive heart failure. Eight patients out of the 40 with smoke poisoning and one patient out of five with respiratory burns received corticosteroids; none of the patients with CO poisoning only received steroids. The steroids were given in 2-4 divided doses within the first 24-48 hours only.

Five patients died: three cardiorespiratory arrests and two with intractable pulmonary edema, 4 days post injury, and overwhelming pulmonary infection, 12 days post injury, respectively (Figure 1). There were two deaths among 40 smoke poisoning patients, one death among six carbon monoxide poisoning patients and two deaths among the five with respiratory burns. Thus the overall mortality was 9.8%.

Carbon monoxide clearance from the blood was followed with serial determinations of carboxyhemoglobin levels. The two patients who had the highest CO levels, one 82% and the other 51% both on MA-1 respirators with 100% $O_2$, had rapid clearance of CO from the blood within four hours. Those patients who had CO levels below 30% were treated fairly effectively with nasal oxygen and ventimask, bringing their blood CO levels to 10-15% within four hours (Figure 4).

It is interesting to note that all patients who were hospitalized for longer than two days had initial CO levels about 15%. As is shown in Figure 5, there is a definite relationship between the initial blood CO level and the patient's length of hospitalization. The patient with 82% carboxyhemoglobin who died seven hours after admission with fatal CO poisoning and another patient whose hospital stay was prolonged because of skin grafting were

excluded in the calculation of correlation coefficient, since patient's length of hospital stay was primarily determined by the degree of pulmonary complications. The 0.87 correlation coefficient between initial blood CO levels and hospital days, indicated to us a fairly good correlation between initial blood CO levels and pulmonary damage as well. Since CO is not noxious and as we have shown in dog experiments, does not cause pulmonary edema, the reason for this relationship may very well be nothing more than CO acting as an indicator of the presence of other substances which are injurious to the lungs but are not detected in blood or other tissues. Therefore, it is pertinent to use carboxyhemoglobin levels as an index of pulmonary injury by other injurious gases which coexist with carbon monoxide in smokes of common structural fires. Smoke poisoning patients who have clinical evidence of tracheobronchial tree or pulmonary injury and those patients without clinical evidence of such injury but who have carboxyhemoglobin levels of 20% or more are usually hospitalized for observation for at least 24 hours. It must be pointed out that in many smoke poisoning patients evidence of pulmonary injury may not become clinically evident until 12-48 hours. A chart has been construed on the basis of clinical blood CO clearance curves and the experimental data. From such a chart one can interpolate the approximate blood CO levels at the time of exposure if the delay time and emergency blood CO determination are known (Figure 6).

## DISCUSSION

Clinical: We believe that the significance and diagnosis of inhalation injuries in fire victims, by and large, have been underestimated and overlooked. The smoke poisoning or chemical injury to tracheobronchial tree and lung parenchyma may occur without significant body surface area burns. Such injury can extend to the peripheral respiratory tissues and rapidly emerge clinically as dyspnea with wheezes and rales. In "respiratory burns," however, which occur in patients with significant burns of anterior chest, neck, face, mouth and/or nose with singed vibrissae, thermal energy rather than chemicals seems to inflict the primary injury. This injury usually involves more proximal respiratory tract structures (i.e., larynx, pharynx, trachea) than chemical injuries. Blood carboxyhemoglobin level may have no relevance in such primary injury. Pathophysiology of this type of injury has been described in another publication.[1] No doubt there are some patients who have a combination of chemical gaseous and thermal injuries to the respiratory tissues.

From our studies it appears that the clinical smoke poisoning or "smoke inhalation" is a chemical respiratory injury with concomitant carbon monoxide poisoning. Our studies show that visible respiratory tract damage is seen in about 25% of smoke poisoning cases and in five out of five (100%) respiratory burn patients. Because of low specific heat of gases the thermal injury does not seem to proceed beyond the major bronchi,[5] whereas chemical gas injury easily reaches the alveolar level. Note that most of the tracheostomies were performed in respiratory burns for upper airway obstruction. Occasional tracheostomy was necessitated in smoke poisoning patients for prolonged

intubation as a result of excessive secretions and unabating patchy atelectasis.[6]

The ability to ascertain CO saturation levels has made possible the diagnosis CO poisoning earlier thus enabling faster treatment. The treatment is directed to improve the alveolar ventilation and increase the alveolar oxygen tension so that by mass action the oxygen will replace the CO on the hemoglobin molecule.[7,8,9,10,11,12] No doubt the vigor of treatment will vary with the extent of carbon monoxide poisoning. However, in our institution, in smoke poisoning for patients who had very high carboxyhemoglobin saturation (82% and 51%) with interstitial edema, intubation (naso or orotracheal) and respirators with PEEP were used for both clearance of carbon monoxide and the treatment of hypoxia and interstitial edema. The patient with 82% carboxy-hemoglobin saturation died seven hours after admission in spite of vigorous resuscitation as a result of massive CNS and myocardial damage. Possibly hyperbaric (2-2½ atmosphere) oxygen chambers may hold promise for such cases.[13,14]

The evaluation of late sequelae following inhalation injury was difficult to accomplish since many of the patients admitted were not seen in the hospital prior to their emergency admission. In follow-up clinic visits, six of forty smoke poisoning patients were felt to have some pulmonary disease such as bronchitis, asthma, and emphysema. Two of the five respiratory burn patients similarly demonstrated some mild degree of pulmonary symptoms. One out of forty smoke poisoning patients and three out of six carbon monoxide poisoning patients had neurologic sequelae such as residual encepholopathy (poor mentation, choreoathetosis, hemiparesis) and/or peripheral neuropathy. Four of these patients were electively admitted primarily for neurologic evaluation of previous furnace and stove carbon monoxide poisoning.[15]

In spite of controversy with regard to the use of steroids in inhalation injuries, it is our impression that patients with smoke poisoning who sustain chemical respiratory injury may benefit from two to four doses of steroids within the first 24 to 48 hours. These doses are likely to help reduce interstitial edema and more significantly to release bronchial and bronchiolar spasm. In respiratory burns, on the other hand, the injury is proximal with inevitable break of respiratory mucosal barrier against invasive infection. Steroids may be harmful to these patients since infection plays a major role in such injury. In respiratory burns, we have recommended use of penicillin against β-hemolytic streptococcus for the first 3-4 days and then the use of proper antibiotics according to culture and sensitivity. In the case of smoke poisoning mucosal break is not common initially, and the problem is peripheral with bronchospasm, edema, atelectasis and increased secretions. In such cases, therefore, proper antibiotics are only used on the basis of culture and sensitivity in established infections.

## EXPERIMENTAL

In order to learn more as to what smoke poisoning was etiologically as well as pathophysiologically, we had conducted several sets of experiments.

In one set of experiments we exposed anesthetized mongrel dogs to standarized smokes of wood and kerosene. To our surprise, kerosene smoke neither killed nor produced pulmonary edema whereas wood smoke killed 50-60% of the animals and caused heavy lungs with pulmonary edema and congestion.[16] It was also found that neither carbon monoxide nor soot per se caused pulmonary edema or death in animals following resuscitation. Therefore, it was concluded that an invisible noxious gas or gases were responsible for pulmonary damage and death of animals. When wood and kerosene smokes were analyzed by mass spectrometry, gas chromatography and chemical analyses, there were significant quantitative differences in only two types of gases; carbon monoxide and low-molecular-weight aldehydes. There was 10 times more carbon monoxide and 15 times more of these aldehydes in wood smoke than in kerosene smoke. Since carbon monoxide was known and was shown in the above-mentioned experiments as non-irritating, aldehydes were considered as the likely etiologic gases in the production of exudative pulmonary edema. Short chain aldehydes are well known irritants of mucus membranes in industrial toxicology. In experimental animals these aldehydes have caused pulmonary edema and death. For example, acrolein, a three carbon aldehyde compound in a concentration of 5.5 ppm (parts per million) has been shown to cause irritation of the upper respiratory tract and death with pulmonary edema occurring in a few minutes at a concentration of 10 ppm.[17] In human volunteers, inhalation of acrolein at concentration of 0.805 ppm cause lacrimation and irritation of all exposed mucous membranes.[18] The industrial maximal allowable (MAC) concentration of acrolein is 0.1 ppm.[19] Acrolein was present in wood smoke as 50 ppm and in kerosene smoke as 1 ppm. It is likely that the agents causing tracheo-bronchial and pulmonary parenchymal damage of smoke poisoning in man are also the aldehydes which are found in large quantities in smoke and combustion of wood, cotton, furniture and nonsynthetic structural materials.[20]

The reaction occurring between these aldehydes and nucleoproteins is said to be one of denaturation followed by reaction with freed nucleotide amino groups, especially in the case of adenine and cystosine residues with the reaction most likely occurring with the nitrogen in position 6.[21,22,23] The denaturation of amino acids or RNA by aldehydes appears to be partial or complete according to the extent of involvement of hydrogen binding sites. It is reasonable to believe that such denaturation by aldehydes is cumulative during long term or repeated exposures. As a result, the possible role of aldehydes in lung damage by cigarette smoking is presently under consideration.

Although our experiments identified carbon monoxide and aldehydes as the poisonous gases from wood, kerosene, cotton and furniture smokes, there is no doubt that other noxious gases such as sulfur oxides, hydrochlorides, hydrocyanic acids, chlorine, fluorine, lead oxides, ammonia and possibly some other gases can damage respiratory tissues when produced in significant concentrations from combustion of other materials. Further work is needed to elucidate the role of these factors in fire damage via the respiratory system.

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| PRIMARY CLINICAL DIAGNOSIS | CASES | BSA BURNS | | CLINICAL EVIDENCE OF LATE SEQUELAE | | LABORATORY DIAGNOSIS OF CO POISONING | DEATHS |
|---|---|---|---|---|---|---|---|
| | | Subjects % burn | Average % burn | Neurologic | Pulmonary | | |
| SMOKE POISONING | 40 | 3%<br>1%<br>10%<br>10%<br>15% | 0.97 | 1 | 6 | 16 | 2 [2 days and 12 days] |
| CARBON MONOXIDE POISONING | 6 | 0 | 0 | 3 | 0 | 1 | 1 [7 hours] |
| RESPIRATORY BURN | 5 | 30%<br>20%<br>5%<br>4%<br>3% | 12·4% | 0 | 2 [no follow up in 1] | 0 | 2 [4 days and 8 days] |
| TOTAL | 51 | | 2% | 4 | 8 | 17 | 5 |

FIGURE 1. Columbia-Presbyterian Medical Center Admissions - 1960 through 1972.

| PRIMARY DIAGNOSES | NUMBER OF CASES | COHb (carbon monoxide) DETERMINATIONS | ROENTGENOGRAPHIC PULM. EDEMA | GROSS RESP. TRACT DAMAGE |
|---|---|---|---|---|
| SMOKE POISONING | 21 | 0 | 1 | 5 |
| CO POISONING | 5 | 0 | 0 | 0 |
| RESPIRATORY BURNS | 4 | 0 | 1 | 4 |
| TOTAL | 30 | 0 | 2 | 9 |

FIGURE 2. Columbia-Presbyterian Medical Center Admissions - 1960-1970.

| PRIMARY DIAGNOSES | NUMBER OF CASES | COHb (carbon monoxide) DETERMINATIONS | ROENTGENOGRAPHIC PULM. EDEMA | GROSS RESP. TRACT DAMAGE |
|---|---|---|---|---|
| SMOKE POISONING | 19 | 19 | 1 | 5 |
| CO POISONING | 1 | 1 | 0 | 0 |
| RESPIRATORY BURNS | 1 | 1 | 0 | 1 |
| TOTAL | 21 | 21 | 1 | 6 |

FIGURE 3. Columbia-Presbyterian Medical Center Admissions - 1971-1972.

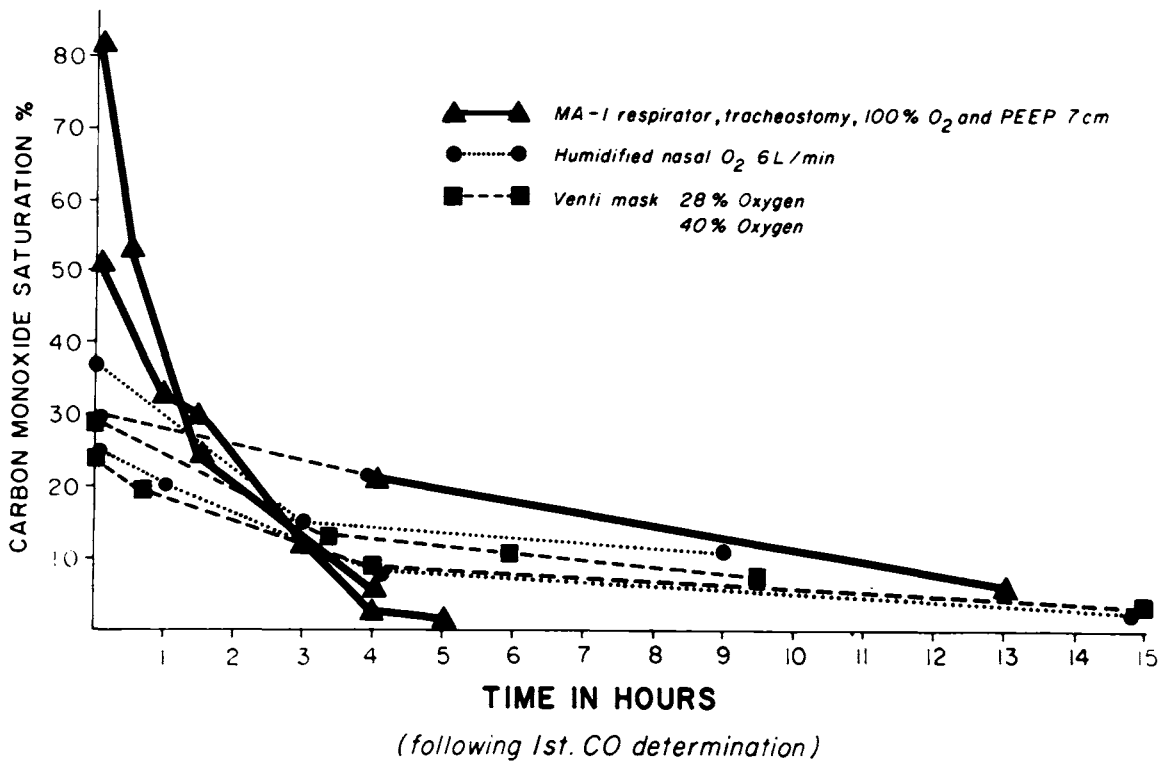


FIGURE 4. Carbon Monoxide Disappearance in Blood of Patients with Smoke Poisoning.

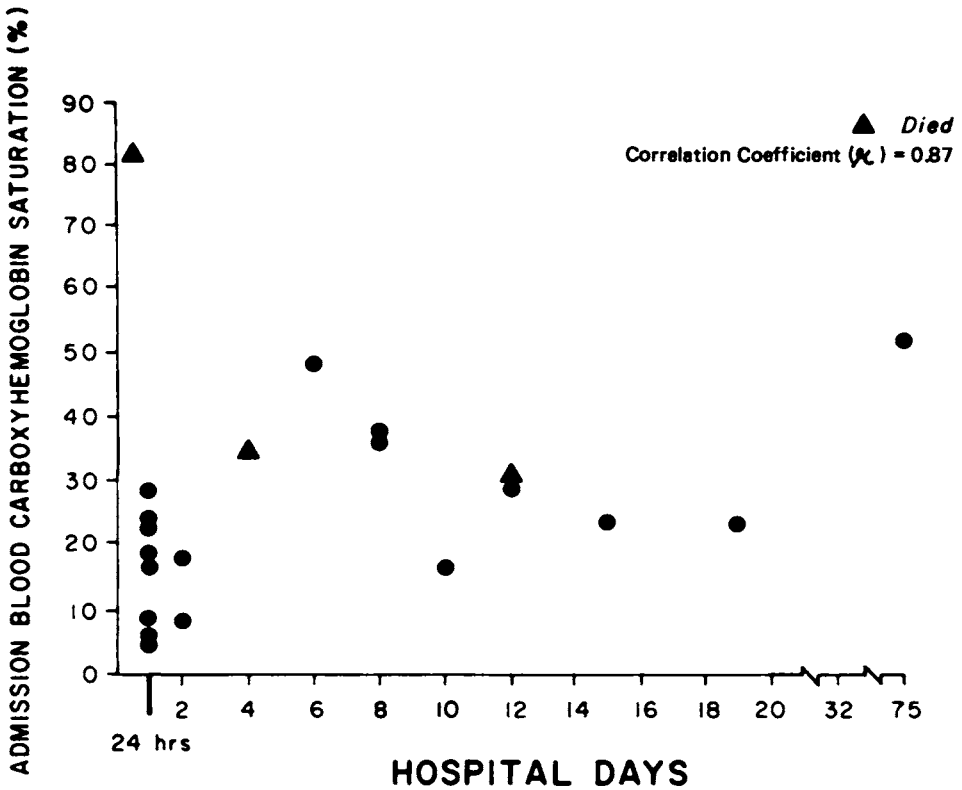


FIGURE 5. Relationship of Blood CO Level with Pulmonary Complications.

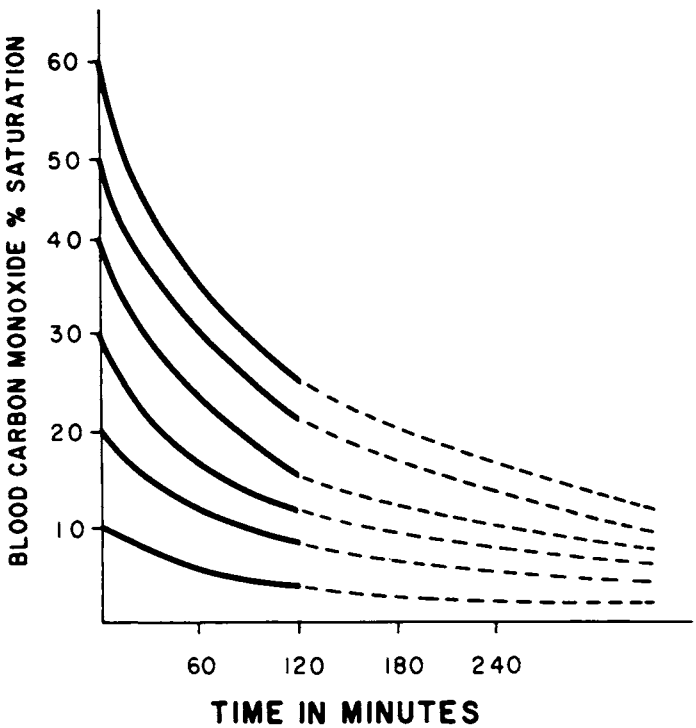


FIGURE 6. Clinical Blood CO Clearance Curves.

# *MEDICAL ASPECTS OF TOXICITY RESULTING FROM FIRE EXPOSURE*

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## INTRODUCTION

Statistics on fire deaths and injuries in this country present a gloomy picture. For example, each year an estimated 12,000 persons die as a result of fires while several million persons are injured. Even though a number of books and articles are available on treatment of burned patients, much less information is available on the injurious or potentially injurious effect from exposure to the thermodegradation and combustion products generated from uncontrolled fires. As is now well known by those dealing with fires, the death or injury of a person is often precipitated by the inhalation of toxic agents generated from the fire, while in other cases the obscuring of vision due to smoke prevents the person from escaping the fire zone. In fact, it is now estimated that over half of the persons who die in a fire die because of the inhalation of toxic agents such as carbon monoxide and other toxic gases.

As the title of my paper indicates, I will direct attention to some of the present medical problems associated with the toxicity of thermodegradation and combustion products generated in a fire. Additionally, I will also discuss some other potential clinical problems which can arise from repeated exposures to thermodegradation products. This latter topic is generally missing when research workers discuss fire hazards, probably because the interest of the group is upon acute exposures which have immediate life-threatening effects in a fire situation. Since many more man-made polymeric materials are being used, some with flame retardants and others without, the potential dangers to the population from heated or burned synthetic materials might be expected to increase in the vicinity of fires or in areas where high temperatures are encountered, such as a working environment.

## PROBLEMS FROM ACUTE EXPOSURES TO FIRE SITUATIONS

Throughout this symposium a number of factors will be examined which the speakers feel contribute to the death or injury of fire victims. In the past, I have summarized seven factors which I felt could play a role leading to death or injury. In my mind these factors are still sufficiently important to repeat. They include the following:[1]

1. Direct consumption by the fire (whole or portions of the body)

2. High temperatures

3. Absence of oxygen

4. Presence of carbon monoxide

5. Presence of other toxic gases

6. Presence of smoke, obscuring visibility and/or leading to particulate inhalation or ingestion

7. Development of fear, altering judgment and behavior.

It should be apparent that a number of these factors would most likely be operative in a real fire situation, and thus, often a combination of these factors can be associated with deaths or injuries of fire victims. Even though other speakers on this program will present detailed accounts of several of the factors I have mentioned, it may serve some purpose to touch upon several of these factors at this time. For this presentation "direct consumption by the fire" will not be discussed.

Temperature

One of the major consequences from exposure to the heat of fires is the resulting thermal burns, the severity of which is dependent at least upon how close the person is to the burning portion of the fire. If the person is not properly protected, high temperatures, for even very short time periods, will cause thermal burns, and in turn, can lead to fluid loss and a fall in blood pressure. If the fluid loss continues the individual can go into shock and die. If the body has been exposed to extremely high temperatures, the tissue in the burned area will upset the electrolyte balance, in particular, in the distribution of potassium ions between the cells and the extra-cellular fluids. Escaping potassium ions from blood cells into plasma could precipitate cardiac arrest.

It is interesting to note that hot, dry air, for example at 300°F or perhaps even higher, does not seem to have much effect upon the lower respiratory tract when inhaled. Inhalation of this hot air, however, may lead to tissue damage in the larynx, followed by a "closing-off" of the larynx and suffocation. In the presence of water, however (for example steam), thermal damage to the lung tissues can occur since steam has a much higher heat capacity than dry air.

In the presence of smoke and heat, there is a general tendency for the person to hold his breath and then to breathe rapidly leading to hyperventilation and a decrease in the normal concentration of carbon dioxide. Shifting of the pH in the blood stream can then lead to fainting. If the

person is not removed from the environment of the fire, death can follow.

Depletion of Oxygen

As Table I indicates, a decrease of oxygen content of ambient air can lead to signs and symptoms of toxicity. In a fire situation the oxygen level can vary from normal or 20%, down to nearly zero. As an example, a fire in a closed room may consume most of the oxygen causing the oxygen level to fall to nearly zero, while in the area of a fire which is well ventilated, the oxygen level may be almost normal. In any case, levels of less than 10% can be considered life-threatening if the person is not removed from the environment.

Presence of Carbon Monoxide

Evidence up to the present time suggests that most fire deaths have been the direct result of carbon monoxide inhalation. As with any toxic agent, its concentration at the time of exposure will play an important role in leading to death or injury of the fire victim. When inhaled, carbon monoxide is absorbed through the lungs into the blood stream and there combines reversibly with hemoglobin forming carboxyhemoglobin (COHb). The oxygen-carrying ability of the blood is then markedly decreased leading to hypoxia and varying degrees of toxicity including death. A summary of signs and symptoms of carbon monoxide as related to the % COHb is shown in Table II. In general, it can be stated that levels of COHb above 70% can be considered as life-threatening. It has already been mentioned that not only is the level of carbon monoxide in the ambient air important in regard to toxicity, but that the exposure time should also be considered. For example, empirical equations can be used to estimate the time and concentration effects upon toxic responses. One example is shown here:[2]

Hours x ppm (in ambient air) = 300 (no perceptible effect)
Hours x ppm (in ambient air) = 600 (just perceptible effect)
Hours x ppm (in ambient air) = 900 (headache and nausea)
Hours x ppm (in ambient air) = 1,500 (dangerous to life)

Of course in real fire situations, an exact accounting of the levels of CO as well as other factors such as heat and additional toxic gases generally are not known, and thus, the relationships shown most likely are inappropriate.

Since another speaker on the program will deal with the toxicity of carbon monoxide in detail, I will not elaborate further on this important subject.

Presence of Other Gases

Even though heat, absence of oxygen and presence of carbon monoxide are considered by many investigators and fire-fighting groups as perhaps the three most important factors leading to loss of life from fires, it is becoming increasingly apparent that consideration must also be given to the

generation of other gases from thermodegradation of the vast number of man-made polymeric materials. In certain experimental situations, animal deaths were attributed to causes other than carbon monoxide toxicity since COHb levels in the blood were below that considered as lethal. Evidence is now emerging to suggest that certain fire victims may have died or been injured from gases other than carbon monoxide. Thus, one should expect that polymers containing carbon, nitrogen, and oxygen when exposed to heat, could release a number of effluent gases such as HCN, various nitrogen oxides, phosgene and perhaps other toxic gases. The presence of Cl or F in a polymer could lead to HCl and HF as well as other products. If the polymer contains sulfur, one could expect $SO_2$ formation. Each of the gases produced would present a toxic liability for fire victims, the gravity of which would depend upon the concentration in the ambient air as well as duration of exposure. Unfortunately, there is generally a combination of these gases generated in a fire situation, and thus the exact toxic profiles for these combinations are not known. It has been shown, however, that combinations of these agents often have synergistic effects leading to a higher degree of toxicity than can be predicted by knowledge of the toxicity of the individual agents.

Since in most cases physicians, nurses and others attending the fire victims are not sufficiently knowledgeable in regard to the variety of toxic agents produced when materials are heated or burned, the actual causes of deaths or injuries may be attributed to the more accepted causes, heat, suffocation or carbon monoxide.

### Presence of Smoke

If toxic symptoms are observed on the inhalation of smoke, it most likely is not due to the actual particulate matter of the smoke. Fortunately the respiratory tract can deny entrance into the lungs of the larger particles. Even small particles which can pass into the lungs may not present a toxic liability to the fire victim. If toxicity occurs, it is most likely due to the presence of a toxic agent or agents such as aldehyde and acids which are adsorbed on the particulates. Some experimental data also suggests that HCl and fluorinated compounds can be adsorbed on smoke particles and thereby be carried to the lungs from which the toxic substances are absorbed into the blood stream. If the agents are corrosive, such as HCl, HF and lower-molecular-weight aldehydes, local lung damage and edema will occur leading to a decrease in the ventilating capacity of the lungs. It is also possible that particulates which are inhaled may contain agents which are still burning, and thus, as they travel through the respiratory tract can cause thermal burns to the trachea and alveolar tissues even though the ambient temperature is relatively low.

## POTENTIAL PROBLEMS FROM REPEATED EXPOSURES

Up to the present time most of the medical problems dealing with fire victims who have been exposed to thermodegradation and combustion products generated in a fire have been directed toward acute toxic effects or one-time exposure to the toxic gases. In general, also, the exposure time is

relatively short, from a few minutes to perhaps 15 to 30 minutes depending upon the location of the victim with respect to the fire. Toxic effects from these gases develop because of relatively high levels of these agents in the ambient air surrounding the victim. If the concentration is sufficiently high the victim might die because of the lethal doses of the toxic gases. If less than lethal concentrations are inhaled, the victim might become incapacitated and unable to exit from the scene of the fire. For fire victims, emergency medical treatment certainly becomes the first priority in order to render life-saving assistance.

Less explored are the possible latent effects in the victims from exposure to thermodegradation and combustion products. Fire victims are often discharged from emergency care and permitted to return to their homes without a careful follow-up to detect if the patient has, in fact, completely recovered.

Newer medical problems should be anticipated when individuals are exposed to sublethal levels of thermodegradation and combustion products from the many man-made materials. The victims in this case could be fire-fighting personnel not adequately trained or protected, as well as industrial workers exposed to low concentrations of toxic vapors resulting from locally high temperatures which could thermally degrade a plastic or elastomeric material. Repeated inhalation of these gases at sublethal concentrations could bring about a number of medical problems which often might be sufficiently masked to prevent either the victim or the attending physician from associating the ailment to the exposure. To illustrate some of these newer medical problems, I would like to use three examples which have appeared in the literature.

The first example deals with a report in 1951, by Harris[3] which describes the symptoms produced by the heating of polytetrafluorethylene to above 300°C. The victims in this case were workers, who in conduct of their duties, were exposed to thermodegradation products of the halogenated polymer. The symptoms often did not become apparent until several hours after the persons had been exposed to the gases. At first the workers felt that they might be developing a cold and often the workers felt that they had recovered sufficiently to return to work the next day. On further inquiry, the workers reported that they developed a mild chest pain, in particular, when they took a deep breath. Medical examination of these workers brought to light the symptoms in more detail. These consisted of a gradual increase in temperature and pulse rate which generally was followed by attacks of shivering and sweating. Interestingly enough all of the symptoms seemed to disappear when the worker was removed from his working environment and rested, such as overnight at home. Harris related the cause of these toxic symptoms to fumes developed when the polyfluorinated material was heated above 300°C and called the disease "polymer-fume fever" analogous to "metal-fume fever" produced by the inhalation of metal fumes.

A second case illustrates a medical problem which developed in an industrial environment in which a specific polyurethane was used as a coating material for wires. The report was issued by Dr. D. P. G. Paisley,[4] Ministry

of Health and Social Services, Belfast, Northern Ireland. In this report Dr. Paisley recounted that in March, 1967, an outbreak of respiratory distress occurred among workers in an electrical manufacturing plant. Complaints included shortness of breath and spasmodic coughing. The symptoms occurred not only during the working period but at night with additional symptoms of fever and sweating. Approximately 40 workers were affected over a period of months. An investigation of the plant revealed that only workers adjacent to and using soldering irons dipped in pots containing solder heated to 360°C developed symptoms. In these cases, as the worker heated the wire covered by a polyurethane coating, the polymer degraded releasing small concentrations of toxic gases which were then inhaled by the worker. These symptoms occurred only when the coating was of this specific polyurethane.

The third illustration of a clinical toxic event due to thermodegradation products of a polymer was reported last year by Sokol, Aelony and Beall.[5] This problem did not occur in the usual industrial environment but in a supermarket in which meat was being wrapped in a polyvinyl chloride film. Briefly, it was found that those employees wrapping the meat and sealing the film by the use of an electrical wire were complaining of difficult breathing, coughing and wheezing after four to five hours of work. The authors concluded that more than a casual relationship existed between the symptoms and the degrading polymer.

The several clinical problems I have reviewed have been attributed to the thermodegradation of three different polymers. For the most part, the levels of the degradation products to which the workers were exposed were low, and based upon animal toxicological data would be considered, in most cases, as falling below a threshold concentration necessary to cause a toxic response. Yet, clinical symptoms of toxicity were noted in man. Since in each of the cases the actual chemical agent or agents causing the symptoms are still not known, one can argue that the people affected might have been more sensitive than normal to the effluent gases, or perhaps even developed a sensitivity to the agents. In any case, no matter what the actual agent might be, the illustrations do demonstrate that, in certain situations with certain workers, unforeseen toxic events may occur which can be attributed to thermodegradation products of polymeric materials.

To date, there is no animal or human evidence to indicate that thermodegradation or combustion products of new man-made materials will present any carcinogenic hazards to persons repeatedly exposed to the effluent gases. However, a mild alert might be echoed in regard to polyvinyl chloride, since one of its degradation products is vinyl chloride. As I believe all of you are aware, this chemical has now been linked to human liver cancer in workers employed in the manufacture of polyvinyl chloride.[6] Cancer in these workers became evident only after a long-term exposure averaging approximately 20 years. I do not suggest that the working environment for the manufacture of the polyvinyl chloride is the same as that found in fire situations or in other industrial situations in which polyvinyl chloride might be exposed to high temperatures. No doubt the levels of vinyl chloride generated by a fire or high temperatures are not the same as those encountered in the plastic

industry, nor are the exposure periods comparable. My point in mentioning this problem is to convey that often months and years may pass before a potential toxic hazard becomes a clinical reality; thus, all steps should be taken to ensure that the population at risk is adequately protected.

## SUMMARY AND CONCLUSION

In this presentation I have attempted to point out a number of medical problems which can occur when people are exposed to thermodegradation and combustion products generated by a fire. From an acute or single exposure to these gases, the most prominent agent leading to deaths or injuries is carbon monoxide. With the introduction of more man-made polymeric materials, with or without flame retardants, other toxic gases should now also become suspect as playing an important role in the toxic characteristics of the effluent gases generated when these materials are exposed to fires or high temperatures. Even though individual gases may be identified, the toxic aspects of thermodegradation and combustion products of materials are probably greater than the sum of the individual components.

I have also included in this presentation another feature of medical problems which has not received much attention in the past. This deals with the incidence of toxic responses in persons repeatedly exposed to fumes of certain types of polymeric materials, when these materials are accidentally or purposely heated. Finally, I suggested that some thought might also be given to possible carcinogenic effects from inhaling such thermodegradation products, if the products generated include potential carcinogenic agents such as have now been recognized for vinyl chloride.

It is clear to me that there is an urgent need to expand research efforts on the toxicity of thermodegradation and combustion products of materials if more medically related problems are to be avoided in the future. Unfortunately, this phase of research is still receiving very little support in relationship to the magnitude of the problem. Preclinical testing should not only include acute toxicity studies of thermodegradation and combustion products but consideration should also be given to subacute and chronic effects as well as studies for carcinogenic activity. Efforts should also be made to encourage local municipalities to set up programs for collecting clinical toxicity data on fire victims as well as workers in industrial environments who may have been exposed to effluent thermodegradation products from polymeric materials. A few cities in this country in fact are starting this type of program. It would seem that preclinical research and clinical toxicity information on fire victims would be an important step in helping to reduce the hazards of exposure to thermodegradation and combustion products of the materials used in our society.

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TABLE I

SIGNS AND SYMPTOMS OF TOXICITY OF REDUCED LEVELS OF OXYGEN DUE TO FIRE CONDITIONS

| Percent of Oxygen in Air | Sign or Symptom |
|---|---|
| 20 (or above) | Normal |
| 12 to 15 | Muscular coordination for skilled movements lost |
| 10 to 14 | Consciousness continues but judgment is faulty and muscular effort leads to rapid fatigue |
| 6 to 8 | Collapse occurs quickly but rapid treatment would prevent fatal outcome |
| 6 (or below) | Death in 6 to 8 minutes |

From Underwriters Laboratories Inc., *Bulletin of Research*, No. 53, July, 1963, p. 49. Information abstracted from *National Fire Protection Association Quarterly*, "Fire Gas Research Report," Vol. 45, No. 3:280-306 (1952).

TABLE II

SIGNS AND SYMPTOMS AT VARIOUS CONCENTRATIONS OF CARBOXYHEMOGLOBIN

| % COHb | Signs and Symptoms |
|---|---|
| 0 - 10 | No signs or symptoms |
| 10 - 20 | Tightness across forehead, possible slight headache dilation of the cutaneous blood vessels |
| 20 - 30 | Headache and throbbing in the temples |
| 30 - 40 | Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting and collapse |
| 40 - 50 | Same as above, greater possibility of collapse, syncope and increased pulse and respiratory rates |
| 50 - 60 | Syncope, increased respiratory and pulse rates, coma, intermittent convulsions and Cheyne-Stokes respiration |
| 60 - 70 | Coma, intermittent convulsions, depressed heart action and respiratory rate, and possible death |
| 70 - 80 | Weak pulse, slow respiration leading to death within hours |
| 80 - 90 | Death in less than an hour |
| 90 + | Death in a few minutes |

From: Schulte, J. H., *Arch. Environ. Health*, *7*:524 (1963).

*SESSION III*

# PHYSIOLOGICAL AND TOXICOLOGICAL ASPECTS OF FIRE EXPOSURE

Moderator:

Irving N. Einhorn
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# *FIRES, TOXICITY AND PLASTICS*

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Fires have occurred since the beginning of time, and when man came on the scene, he must have learned quite early that fire was both his friend and foe. As the old proverb states: "Fire is a good servant but a bad master."

Fire burns both wood and flesh. This kind of damage is very obvious. Sometimes, however, fatalities occur in fire situations and there is no visible evidence of burning of the flesh. In this case, death was attributed to "smoke inhalation," although it is more accurate to attribute the fatalities to the inhalation of fire gases rather than to the particulates which produce visible smoke. It is in these cases also that the toxicologist plays a role.

The third aspect of my talk concerns plastics. Some plastics when ignited burn like other flammable substances such as wood, paper, or coal. Others when exposed to intense heat will decompose and char but will not continue to burn if the intense heat source is withdrawn. The significance of all this is that plastics are finding increased use in homes, other buildings and transportation vehicles, either as materials of construction or in furnishings. Hence their flammability characteristics as well as the toxicity of their pyrolysis products become important. Even their capacity to produce smoke, i.e., particulates, becomes important because smoke obscures vision and may interfere with the ability to see, find and use escape routes.

My own interest in the toxicology of fire began during World War II when I was an officer in the U. S. Public Health Service assigned to work with the Medical Division of the Army's Chemical Corps.

By mid-1944 it had been observed that it was very difficult to neutralize Japanese bunkers on the Pacific islands by the traditional explosives. It was also observed that the bunkers were effectively neutralized if the flame thrower could be fired into an embrasure. The occupants of the bunker were often found dead without visible burns and the question then arose as to the cause of death. Was it heat or fire gases—or both? The Chemical Corps established a Flame Attack Section to work in cooperation with the National

Defense Research Committee and the Canadian Chemical Warfare Laboratories and to undertake an investigation into the casualty-producing factors in flame attack. I was assigned to that Flame Attack Section.

In order to get some preliminary data it was decided to do three things:

1. A team of pathologists from the Canadian Mobile Chemical Warfare Laboratories and an American medical officer were sent to the Pacific theater to try to establish the cause of death in operational flame attack casualties. The attempt failed because of the chaotic conditions of the battlefield and was abandoned after two of the Canadian officers were wounded.

2. An NDRC project to study the effects of fire in a chamber which could be either well ventilated or poorly ventilated was initiated under the direction of the eminent pathologist, Dr. Alan R. Moritz of Harvard University.

3. The Canadian military initiated a series of experiments in a model Japanese bunker at their Suffield (Alberta) Experiment Station.

The combustible material used in all of these approaches was flame thrower fuel, i.e., gasoline either unthickened or thickened with Napalm®. Chemically, then, it was essentially pure hydrocarbon.

Dr. Moritz' work was initiated in a 600 $ft^3$ concrete chamber located near Boston. Gasoline was placed in shallow pans which completely covered the floor of the room and was ignited electrically. Air temperatures were recorded in several locations by means of sensitive continuously recording thermocouples, while gas samples were taken through long tubes inserted through the wall of the chamber.

The pilot experiments indicated that when the conflagration chamber was poorly ventilated, as were the Japanese bunkers, the gasoline fire was quickly extinguished by lack of oxygen, but that in no case did the oxygen concentration drop below 14% or the carbon monoxide concentration exceed 1%, or the carbon dioxide concentration exceed 4%. Peak air temperatures of 900°C were recorded but were not maintained above 400°C for more than 20 seconds. In conflagrations where the chamber was well ventilated, the gasoline burned completely and there was no evidence of significant oxygen depletion or development of significant amounts of carbon monoxide or carbon dioxide in the atmosphere.

On the basis of these experiments, Moritz and his co-workers concluded that heat was probably the most important casualty-producing factor in gasoline conflagrations. They accordingly directed their efforts to an investigation of the production of casualties by exposure to heat alone. Much of this work was published shortly after the war.[1,2,3,4,5,6,7,8,9] Figure 1 shows a plot of time: temperature data for local injury to pig skin and human

skin. Figure 2 shows similar data for whole-pig exposure and effects from local burning to acute hyperthermic death. Toxic gases played no part in these experiments.

In the Canadian experiments, goats were placed in the model Japanese bunker, shielded from contact with flame, and the bunkers were then subjected to flame thrower attack. Deaths occurred in some of the goats and, in the majority of those succumbing, a lethal level of carbon monoxide hemoglobin was found in the blood. The Canadians, therefore, concluded that toxic gases might be more decisive than heat in producing flame thrower casualties in poorly ventilated bunkers.

It developed later that the discrepancy between the Moritz and Canadian conclusions was due to the difference in combustion characteristics of gasoline ignited statically in a poorly ventilated space and burning gasoline injected into such a space.

Because of unfavorable climatic conditions at Suffield, Alberta, it was decided that further investigations of the effects of flame thrower attack would be carried out in the U. S. in model Japanese and German bunkers. The objective was to characterize the physical and chemical changes occurring inside a bunker subjected to flame attack. The measurements considered most important were:

1. Air temperature and caloric bombardment at various locations in the bunker.

2. Oxygen, carbon monoxide and carbon dioxide content of air within the bunker.

3. The concentration of unburned hydrocarbons and other gases developed within the bunker.

The results of these experiments can, perhaps, be best summarized in a series of four graphs. Figure 3 shows what happened to air temperature.

Considering that air temperatures as high as 100°C can be tolerated only under very special conditions (i.e., still air) for more than a few minutes and that some people are incapacitated by breathing air at 65°C, it is seen that heat might well have been casualty-producing in the majority of the experiments but not necessarily lethal.

Figure 4 shows a similar graph for carbon monoxide concentration. Since Dr. Smith will be discussing the effects of exposure to carbon monoxide, I will only comment that the observed levels might be expected to produce lethal levels of carbon monoxide hemoglobin within the time frame of the experiments.

Figure 5 shows the graph for oxygen concentration. Note that in some experiments the oxygen concentration was exhausted to zero for periods

exceeding one minute. The mean lowering of $O_2$ concentration might well have been lethal in itself.

Finally, the graph for combustibles concentration is shown in Figure 6. A mass spectrometer analysis of the combustibles showed essentially unburned aliphatic and aromatic hydrocarbons and some acetaldehyde. It did not appear that the unburned gases were a very significant casualty factor although the phenomenon of cardiac sensitization to epinephrine was considered and investigated separately.

The basic lessons to be drawn from the experiments described so far seem to be these:

1. Statically ignited fuel in a poorly ventilated space is self-extinguishing when the oxygen concentration falls to about 14%, a concentration compatible with life. The heat generated in getting to the self-extinguishing point may not be compatible with life.

2. When thickened or unthickened fuel is injected into a poorly ventilated space, the oxygen concentration can be forced to very low levels, even zero, for a short time. The carbon monoxide concentration can reach easily lethal levels. The unburned hydrocarbons in the air seem to be a minor casualty hazard in comparison with heat, oxygen depletion, and carbon monoxide.

We did not, at the time, have good instrumentation for continuous measurement of carbon dioxide concentrations, but the maximum peak of $CO_2$ concentration observed was approximately 10% and the mean peak was approximately 7%.

It was the impression of the Flame Attack Section that, in these poorly ventilated fortifications under flame attack, the most important lethal factors were high carbon monoxide together with low oxygen concentrations which would, of course, act additively. Heat contributed its own effect in accelerating death, but it was very difficult to assess the individual importance of each factor since all of the factors were highly correlated with each other.

The Flame Attack Section also investigated the potential casualty-producing factors in well-ventilated conflagrations. As might be expected, toxic gases could not accumulate under such conditions and heat was the decisive casualty-producing factor.

Briefly, heat can cause burns of the skin of various degrees. If the damage is extensive, fluid loss occurs by leakage from capillaries in the burned area, there is a fall in blood pressure and the victim may go into shock. And, if the temperature gets high enough, there is a release of

potassium from the blood cells into the plasma, and this hyperpotassemia can cause cardiac arrhythmias and cardiac arrest.

Another type of damage that can occur in the heat situation is that caused by breathing hot air. If the air is heated to about 150°C, the shock to the larynx may cause it to close off the trachea so that no air can get through to the lung. Death can then occur through suffocation. Incidentally, if the larynx is bypassed by having an anesthetized dog breathe very hot dry air through a tracheal cannula, there is no damage to the deep structures of the lung because of the low heat capacity of air. If, however, there is enough water around to produce steam, and the steam is inhaled, there is damage deep into the lung because steam has a much higher heat capacity than dry air.

The experiments which I have described briefly were carried out with current military objectives of mid-1944 in mind. They are reported in detail in a Chemical Corps Report, "The Toxicology of Fire," issued in April 1951.[10]

The combustible material was essentially hydrocarbon. What relevance does these findings have to civilian fire situations?

Personally, I believe that they have a lot of relevance, since most of the combustible materials involved in ordinary conflagrations contain an abundance of carbon and hydrogen. These two elements when burned completely produce the end products $CO_2$ and $H_2O$, just like gasoline. When burned incompletely, the products are $CO_2$, CO, and some intermediate aldehydes and ketones. Incomplete burning takes place when the oxygen supply is limited, so we would also have oxygen deficiency, just as in the model bunkers.

But how about plastics? We have all read that the increasing use of plastics as materials of construction or in home furnishings has added a new dimension to the hazards of fire situations. Some produce hydrogen cyanide or hydrogen chloride or other "deadly gases." We have read that in certain fires occurring in aircraft cabins that fatalities were caused by inhalation of hydrogen cyanide generated from the burning of seat cushions made of polyurethane foams.

For example, on November 11, 1965, a Boeing 727 made a hard landing at Salt Lake City. A fire broke out in the cabin. Forty-three passengers died and 42 escaped. Hydrogen cyanide was mentioned in the press, but the official FAA investigation concluded:

> "Our preliminary findings indicate that none of these fatalities were due to traumatic injuries, but all died from suffocation during the resultant fire. This is evidenced by the elevated carboxyhemoglobin concentrations in the victims and the lack of trauma."

This does not mean that hydrogen cyanide could not have been present in the fire gases or in the victims. Table I shows data from an Underwriters' Laboratory report[11] which reports the amount of HCN found in the pyrolysis products of certain materials, natural and synthetic, most of which are likely to be present in an aircraft cabin. But if they were present, they were produced by pyrolysis by a fire from a broken fuel line which produced a lethal amount of carboxyhemoglobin in the victims. Consequently, the more exotic deadly gases may not be very important.

Let us, however, look at plastics from a broader perspective. They are being increasingly used as substitutes for wood and metals in construction. Synthetic plastics are replacing cotton, silk and wool in furnishings. What is the net effect on the hazard from fire and toxic fire gases?

Plastics are polymers, molecules composed of long chains of fundamental starting units called monomers. Most of the links in the chain are carbon atoms, but other links may be inserted, such as oxygen, which produces polyethers. In addition to polyethers we have polyamides, polyesters, polyamines, polyimines, polycarbonates, polysulfides, polyurethanes, polyacrylates, polyacrylonitriles, etc. Some polymers are built up from aliphatic monomers like the polyolefins. Others are built up of aromatic monomer units, like Nomex® nylon.

If polymer chains become cross-linked to one another, the polymers tend to become rigid. In the absence of cross-linking, the polymer chain can move against each other, so that the polymer is flexible.

One generalization that can be made is that all polymers based on carbon chains will break up when sufficient heat is applied. The temperature at which the chains begin to break, however, will vary by as much as 200°C - 300°C between various kinds of polymer. Note also that natural polymers like wood and wool show the same kind of decomposition when sufficient heat is applied as do the synthetic polymers. I mention this because I believe it important to realize that the hazards from plastics—synthetic polymers—should always be considered in relation to the same kind of hazards from familiar natural materials such as wood, silk and wool (Table II).

When some polymers break up under the influence of applied heat, they produce fragments which are combustible, and if the amount of applied heat is sufficient to bring these fragments to their ignition point, they will burn. The heat of combustion of these fragments may be sufficient to cause adjacent parts of the polymer molecule to break off and ignite and the combustion of the polymer continues even if the initial source of applied heat is withdrawn. Such polymers burn in the sense that a piece of wood burns.

Consider a log and a fireplace. If we want to burn that log in the fireplace we must first apply a source of heat. It may be burning paper, or kindling wood, hydrocarbon lighter fluid (e.g., kerosene) or an electric heating element. At first the log chars on the outside, then tongues of flame

can be seen above the log. Finally, the log bursts into flame and continues to burn after the initial "kindling" process is burned out or withdrawn.

It may be, however, that another kind of polymer will break up under the influence of applied heat, but the fragments produced are not flammable, or if flammable do not have sufficient heat of combustion to trigger a self-sustaining decomposition of the remaining polymer. In this case, pyrolysis of the polymer will cease when the applied heat source is withdrawn.

Let us consider a representative polymer of each class.

Polyethylene is composed of chains of $CH_2$ groups. When heated to about 250°C, it melts, sublimes and to a degree decomposes without burning. The decomposition products include low-molecular-weight hydrocarbons and some partial oxidation products such as aldehydes and ketones. If enough heat is applied to reach the ignition temperature of the hydrocarbon gases, they will burn, and the heat of combustion is sufficient to make the combustion process self-sustaining. In other words, polyethylene is a flammable plastic.

Consider also polytetrafluoroethylene (PTFE). This polymer consists of chains of $CF_2$ groups. When heated to above 399°C, - $CF_2$ - radicals break off from the chain and recombine to form tetrafluoroethylene, and to a lesser extent, hexafluoropropylene and the perfluorobutenes. If the external heat source applied to the PTFE is very high, say above 800°C, the tetrafluoroethylene gas will burn with a visible flame to produce $CF_4$ (nonflammable) and $CO_2$. The heat of combustion of tetrafluoroethylene is not sufficient to make the process self-sustaining. When the external heat source is withdrawn, the further production of tetrafluoroethylene ceases. PTFE, therefore, does not continue to burn under its own power. PTFE is not a flammable material like wood or polyethylene. But because it is an organic polymer it can be completely decomposed—burned up—if sufficient external heat is applied. Scrap PTFE can be destroyed <u>in</u> an incinerator but does not become fuel <u>for</u> the incinerator.

Both polyethylene and PTFE can be completely destroyed by applied heat without any visible evidence of burning. The polyethylene, if held at temperatures slightly above 250°C, will break up in the presence of air into hydrocarbons, aldehydes, ketones and acids. If inhaled, this mixture can produce pulmonary irritation and pulmonary edema. With PTFE, the situation is more complex. Between 300°C and 500°C in the presence of air, it yields only the perfluorocarbon gases of 4, 6 and 8 carbon atoms and a particulate which appears to be low-molecular PTFE chains with acid end groups. We believe, but cannot prove, that this particulate is responsible for the syndrome known as "polymer fume fever" in man, which closely resembles metal fume fever. As the particulate cools it agglomerates, again like metal fume, and loses its power to produce the fume fever syndrome. One of the best ways to get "polymer fume fever" is to smoke a cigarette loaded with about 0.5 mg or more of PTFE because the travel path to the lungs is short and direct.

At about 500°C in air, the perfluorocarbon fragments become unstable and are oxidized to $COF_2$. Between 500°C and 800°C, $COF_2$ is the major pyrolysis product of PTFE. Above about 800°C, $COF_2$ is unstable and is oxidized to $CF_4$, $CO_2$ and C (black smoke). If moisture is present, HF is also found.

Polyethylene is not the only flammable plastic and PTFE is not the only nonflammable plastic. I have used them only as examples. In fire situations, it is obviously important to distinguish between plastics which can become fuel and those which cannot. It would seem important in locations like aircraft cabins, buildings and other places where people might gather to make as much use of nonflammable plastics as possible.

Synthetic plastics are also used for furnishings, draperies, carpets, and clothing, and it is not always possible to select nonflammable synthetics for these purposes. Hence there has arisen a great interest in fire retardants which can be applied to or mixed internally with the polymers and which make them nonflammable. Such fire-retarded polymers do not burn but, like PTFE, can still be completely decomposed by sufficient applied heat. Indeed, the fire retardants themselves decompose and add their decomposition products to those coming from the polymer molecules. The pyrolysis products from a fire-retarded polymer may be more toxic than those from an unretarded polymer.

This situation seems to illustrate the general law that you never get any advantage at no cost. The late Judge Learned Hand summed it up by saying: "There is no value to be attained without some sacrifice to be assumed." Someone else expressed the same thought in the words: "There is no such thing as a free lunch."

There is no time to get into a detailed discussion of the pros and cons of fire-retarded polymers, except to express my personal opinion that I would rather trade off some increased toxicity of pyrolysis products under applied external heat for the advantage of having a garment, carpet or furnishing which would not continue to burn under its own power after the applied heat source is withdrawn.

I hope that these remarks may provide some background for the more detailed discussions which follow.

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*Obtainable from Library of Congress, Photo-Duplication Service, Washington, D.C. 20540; Accession No. PB 143732; Cost $18.30.

TABLE I

DETERMINATION OF HYDROGEN CYANIDE
IN PYROLYSIS PRODUCTS

| | Hydrogen Cyanide (Micrograms/Gm Sample) | |
|---|---|---|
| Material | Air | Nitrogen |
| Paper | 1100 | 182 |
| Cotton | 93<br>130 | 85 |
| Wool | 6500 | 5900 |
| Nylon | 780 | 280 |
| Polyurethane foam | 1200 | 134 |

From Underwriters' Laboratories, Inc., Bulletin of Research, No. 53, July 19, 1963, page 30. Information abstracted from unpublished report, "Thermal Degradation of Polyurethane to Hydrogen Cyanide," by W. H. McDermott and F. E. Critchfield, Union Carbide Plastics Co. (February 24, 1961).

TABLE II

COMBUSTION PRODUCTS OF PLASTICS AND OTHER COMMON SOLIDS

| | Poly-styrene | Ethyl Cellulose | "Saran" | PVC | Nylon | Rayon | Wool | Silk | Wood | Paper |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | *Condition No. 1 - Free Burning, grams/gram of sample* | | | | | | |
| Carbon dioxide | 2.192 | 2.294 | 1.047 | 0.433 | 1.226 | 1.836 | 1.541 | 1.352 | 1.626 | 1.202 |
| Carbon monoxide | 0.174 | 0.440 | 0.022 | 0.229 | 0.304 | 0.116 | 0.446 | 0.634 | 0.270 | 0.135 |
| Aldehydes† | — | — | — | — | 0.0064 | — | — | 0.0024 | Trace | — |
| Phosgene | — | — | — | 0.0001 | — | — | — | — | — | — |
| HCN and RCN | — | — | — | — | 0.0076 | — | 0.007 | 0.036 | — | — |
| Ammonia | — | — | — | — | 0.032 | — | — | 0.053 | — | — |
| Chlorine-HCL | — | — | 0.621 | 0.496 | — | — | Trace | — | — | — |
| Acidity†† | — | — | — | — | — | — | — | — | — | 0.0009 |
| | | | | *Condition No. 2 - Smoldering, grams/gram of sample* | | | | | | |
| Carbon dioxide | 1.698 | 0.202 | 0.416 | 0.743 | 0.907 | 1.130 | 0.650 | 1.033 | 0.934 | 1.001 |
| Carbon monoxide | 0.540 | 0.172 | 0.221 | 0.086 | 0.355 | 0.225 | 0.138 | 0.141 | 0.366 | 0.273 |
| Aldehydes† | 0.003 | 0.012 | — | — | 0.0065 | — | ††† | 0.0012 | Trace | Trace |
| Phosgene | — | — | — | 0.00008 | — | — | — | — | — | — |
| HCN and RCN | — | — | — | — | 0.0098 | — | 0.008 | 0.007 | †††† | — |
| Ammonia | — | — | — | — | 0.210 | — | 0.035 | 0.308 | — | — |
| Chlorine-HCL | — | — | 0.774 | 0.473 | — | — | Trace | — | Trace | — |
| Acidity†† | — | — | — | — | — | 0.042 | — | — | 0.009 | — |

† — As formaldehyde.
†† — As acetic acid.
††† — Positive, but interference.
†††† — Interference.

From Underwriters' Laboratories, Inc. Bulletin of Research, No. 53, July 19, 1963, page 22. Information abstracted from unpublished report, "Products of Combustion of Plastics and Other Common Solids," by A. P. Hobbs and G. A. Patten, Dow Chemical Co.(March 1, 1962).

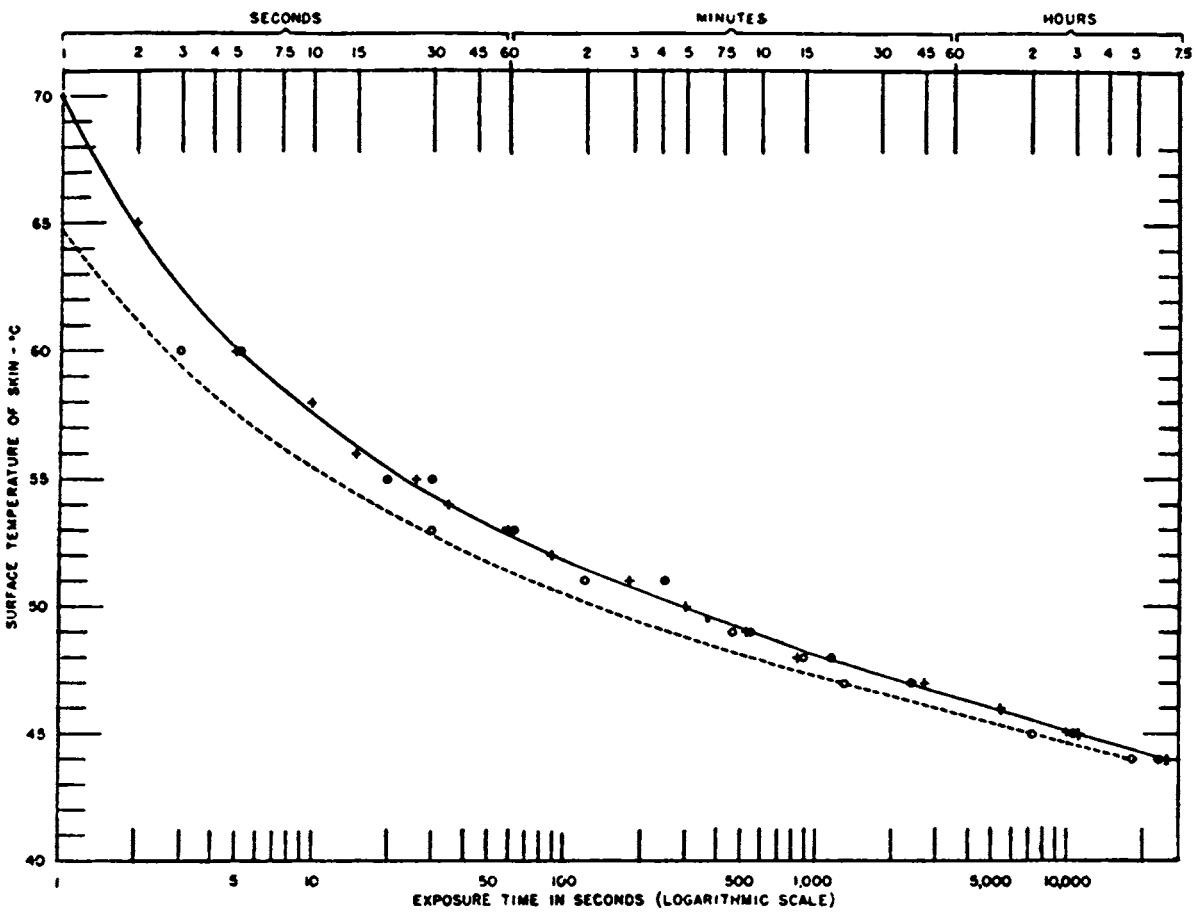



FIGURE 1. Time-surface temperature thresholds at which cutaneous burning occurs. The broken line indicates the threshold at which irreversible epidermal injury of porcine skin is first sustained. The solid line indicates the threshold at which epidermal necrosis of porcine skin occurs. Critical exposures of porcine skin are represented by crosses. Each cross denotes the shortest exposure time at the temperature indicated which resulted in trans-epidermal necrosis. The results of critical experimental exposures of human skin are indicated by circles. The open circles represent the longest exposure at the temperature indicated that failed to destroy the epidermis, and the solid circles represent the shortest exposure at the temperature indicated that resulted in trans-epidermal necrosis.

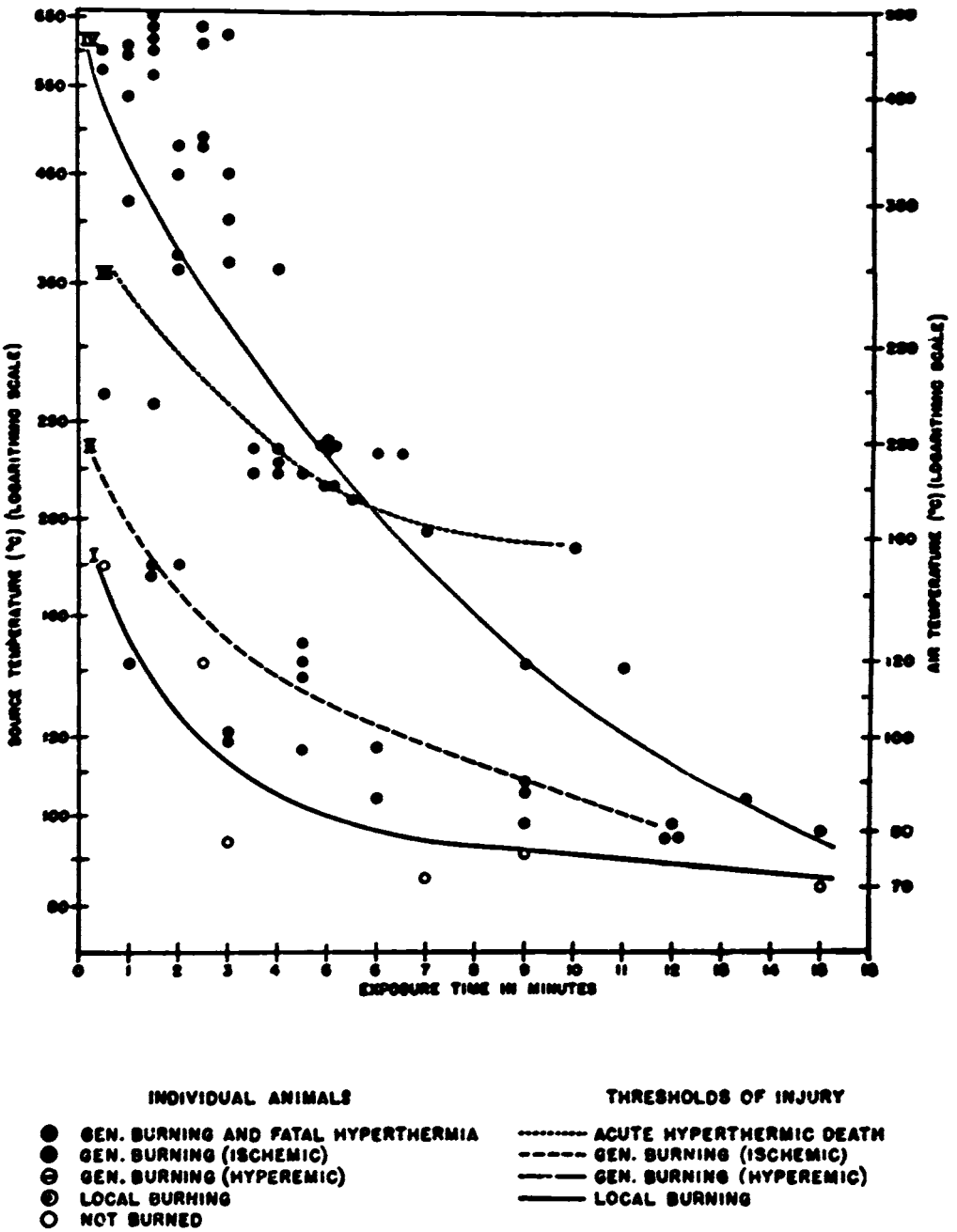
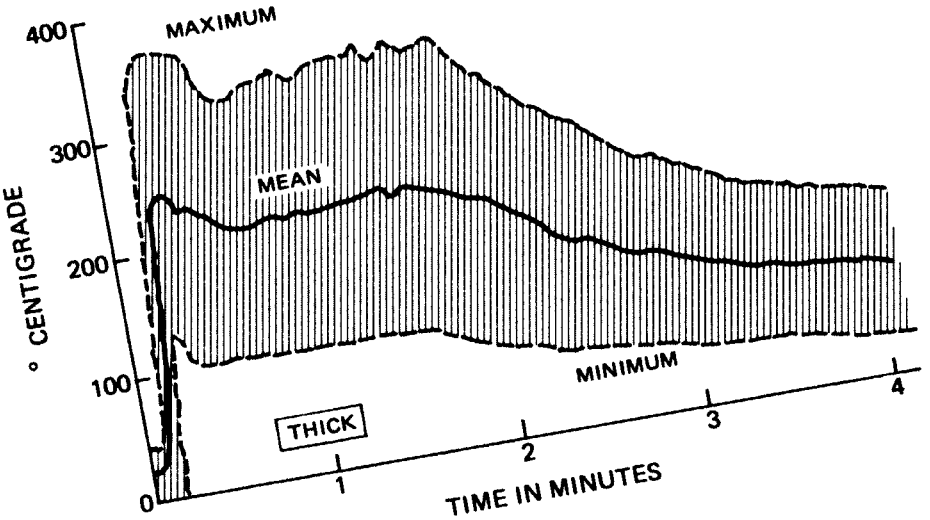



FIGURE 2. Graph showing results of 71 experiments in which pigs had their general cutaneous surface exposed to ambient and radiant heat in an oven. Each experiment is depicted by a circle. The duration and the temperature of the exposure are indicated by the position of the circle in the grid. The effect of the exposure on the pig is shown by the character of the circle. The curved lines traversing the grid depict the approximate thresholds at which varying degrees of cutaneous and systemic injury occurred.



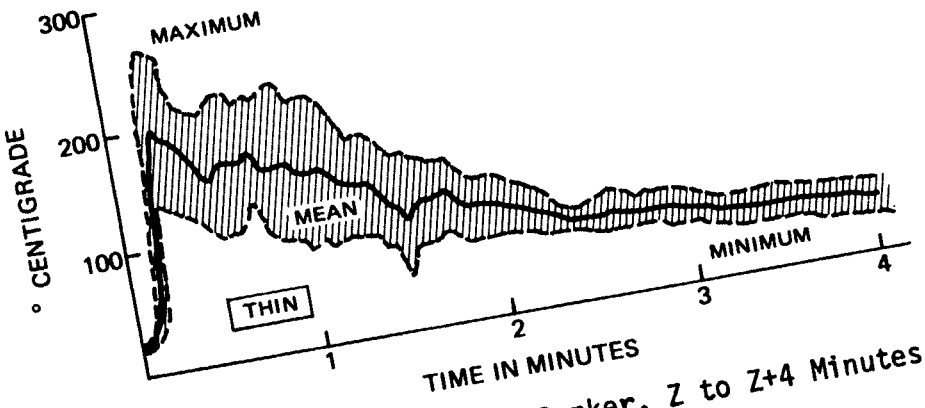




FIGURE 3. Air Temperature in Bunker, Z to Z+4 Minutes.

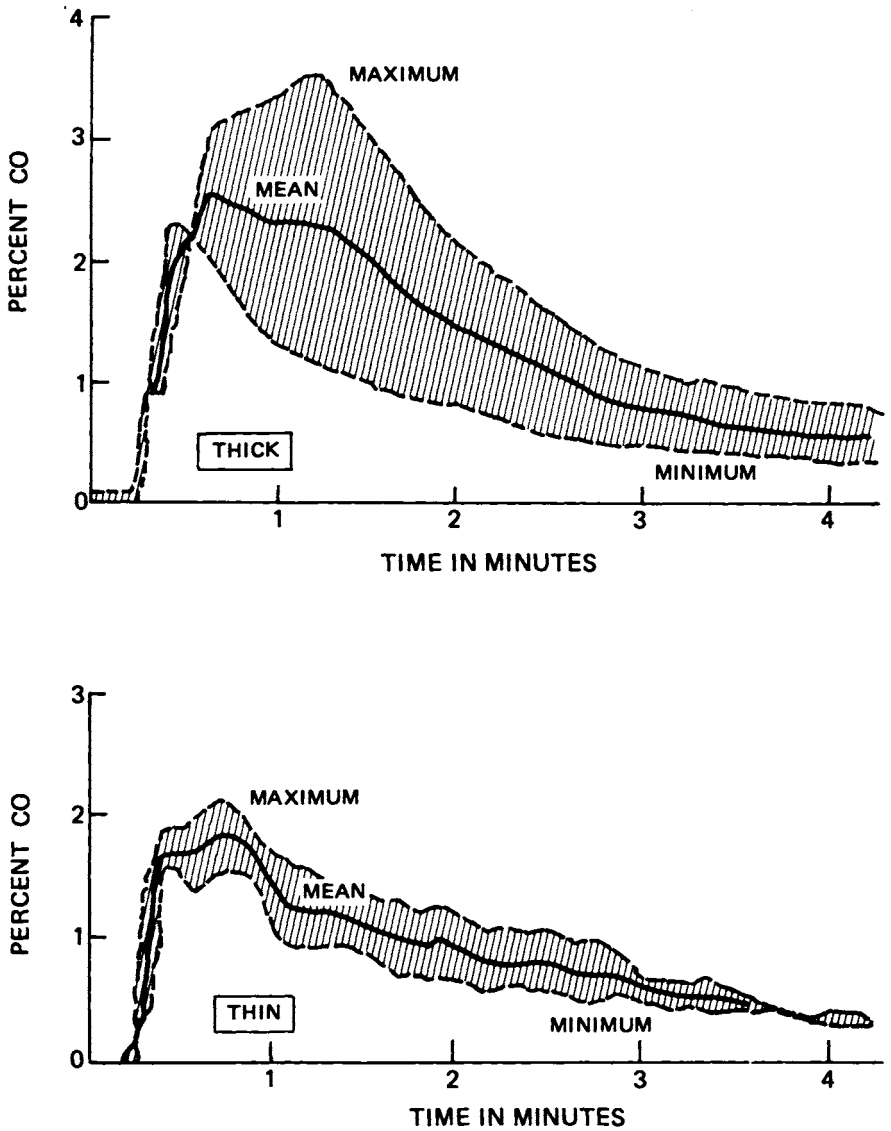



FIGURE 4. Carbon Monoxide Concentration in Bunker, Z to Z+4 Minutes.

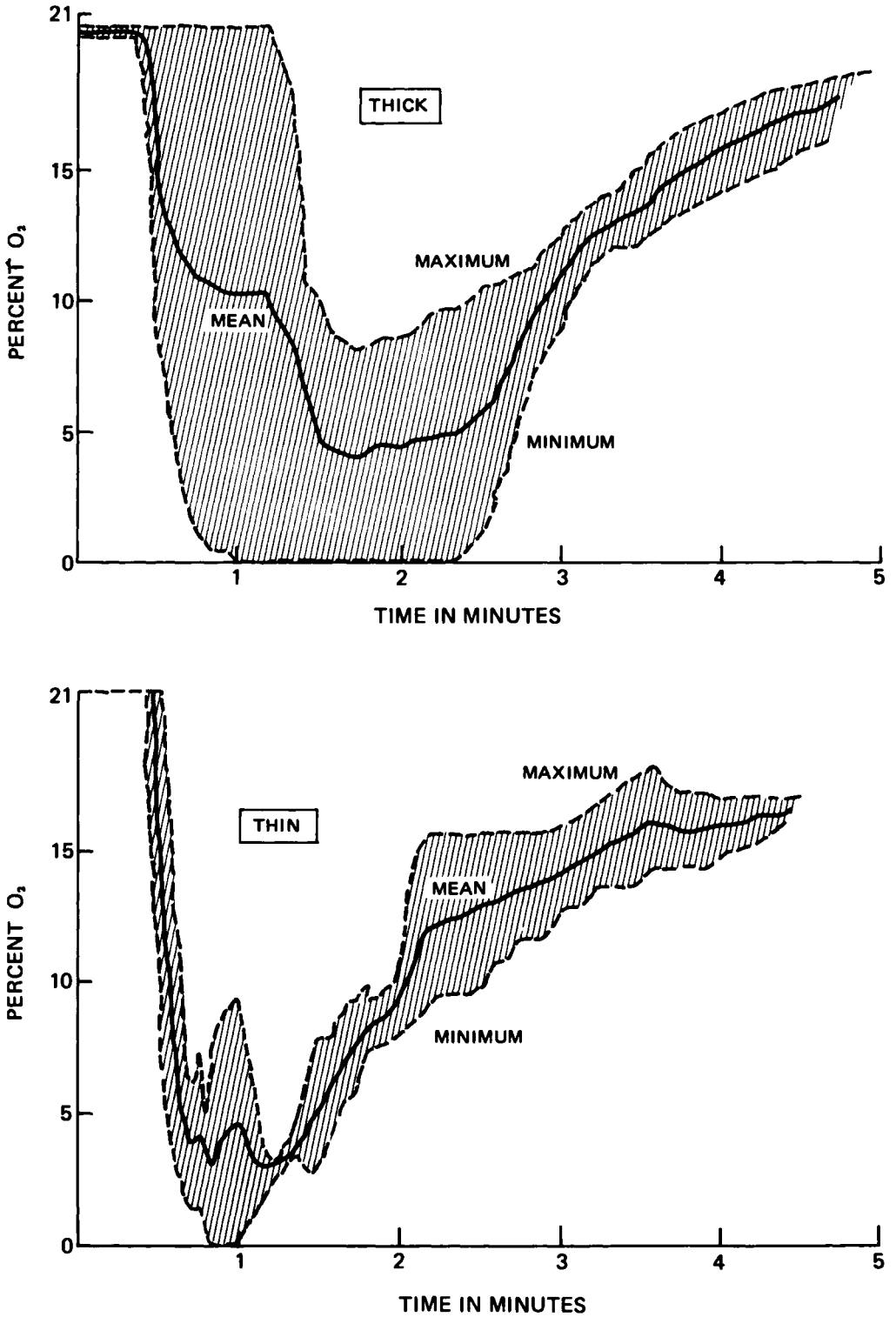
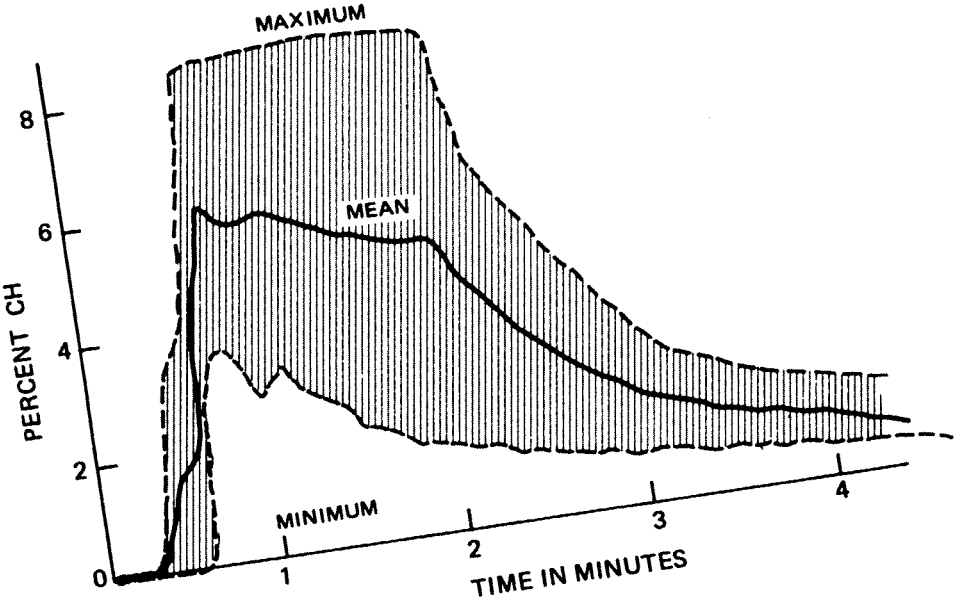



FIGURE 5. Oxygen Concentration in Bunker, Z to Z+4 Minutes.



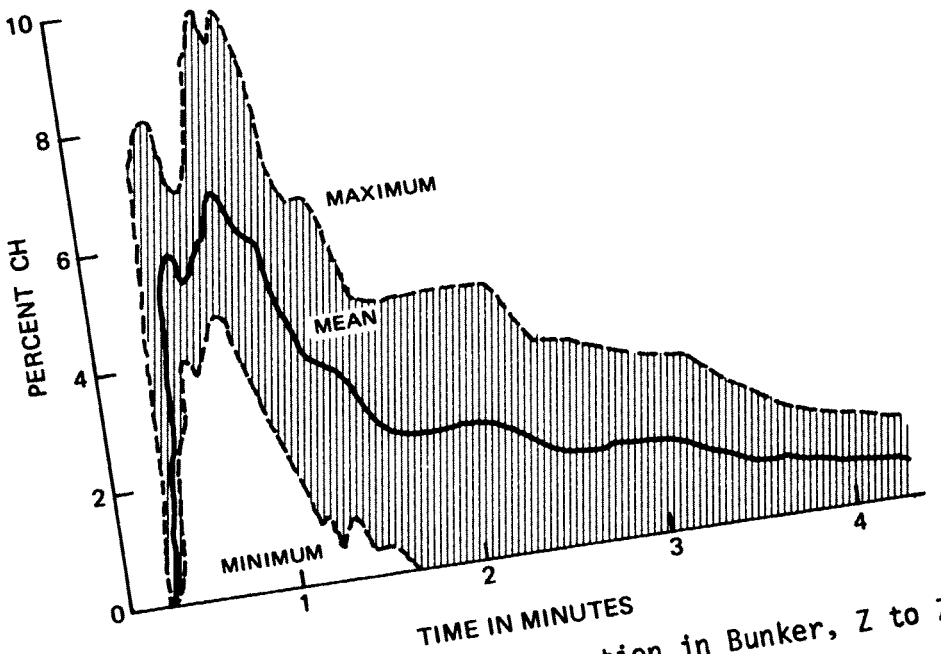




FIGURE 6. Combustibles Concentration in Bunker, Z to Z+4 Minutes.

# *EFFECTS OF EXPOSURE TO CARBON MONOXIDE AND HYDROGEN CYANIDE*

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## INTRODUCTION

Two commercial aircraft accidents in the United States during the 1960's (Denver, Colorado, 1961; Salt Lake City, Utah, 1965) contributed greatly to the initiation of the present concern over the toxic hazard of the gases generated in aircraft fires. These accidents were of special significance because careful analysis indicated that few if any of the occupants could have suffered significant physical injury from the relatively mild impacts involved,[1] yet a total of 60 persons perished as the result of thermal and chemical injuries sustained in the ensuing fires.

Carboxyhemoglobin measurements on 16 victims of the Denver crash revealed CO saturations ranging from 30 to 85 with a mean of 63.3%. Similar analyses on 36 victims of the Salt Lake City accident yielded CO saturations ranging from 13 to 82%, the mean being 36.9. The lower carboxyhemoglobin values found in the second accident have been attributed to the fact that fire was present within the aircraft before evacuation could be attempted, and that the survival time of many victims must have been shortened by direct thermal effects.

It has also been assumed that gases other than carbon monoxide must have contributed to the toxicity of the cabin environment, but there is no supporting evidence for this assumption.

Our own interest in the toxicity of pyrolysis-combustion products was enhanced when, in the late 1960's, we were asked to evaluate the results of a large-scale materials test program from the toxicological point of view.[2] The potential toxicity of the gas mixtures evolved in certain of the aircraft fire simulations conducted in this study was clear.

Later, in 1970, blood samples from victims of an aircraft crash followed by fire (Anchorage, Alaska, November, 1970) were analyzed in our laboratory for the presence of cyanide; the first time, to the best of our knowledge,

that such analyses had been made on victims of an aircraft fire. Cyanide was estimated by the method of Feldstein and Klendshoj.[3] Measurable amounts of cyanide were found in 18 of the 19 specimens submitted, accompanying carbon monoxide saturations ranging from 17 to 70%. In the one sample in which cyanide could not be detected, the carboxyhemoglobin concentration, 4.9%, did not exceed that which could result from smoking, indicating the probability of death on impact. Blood cyanide levels in these victims corresponded closely with those reported in the literature for victims of structural and vehicular fires,[4] ranging from the lower detection limit (circa 0.01 µg/ml) up to 2.26 µg/ml. The relationship between cyanide levels and carboxyhemoglobin content varied in random fashion, perhaps representing relative proximity of the victims to cyanide-producing materials.

Nothing in these findings permitted speculation concerning the relative contribution of the two gases to lethality, and a further search of the literature revealed nothing helpful. In addition, there was no way of assessing the possible contribution of other gases which must have been present in the pyrolysis mixture to which these victims were exposed.

In mid-1970, a team of CAMI scientists initiated a joint research effort with the Toxic Hazards Division, 6570th Aerospace Medical Research Laboratory, Wright-Patterson AFB, Ohio, to study the short-term toxicity of certain potential components of pyrolysis mixtures, singly and in combination with carbon monoxide. Gases selected for study were hydrogen fluoride, hydrogen chloride, nitrogen dioxide and hydrogen cyanide.

The results of the study showed the toxicity ranking of the four gases to be HCN, $NO_2$, HF and HCl, in decreasing order. The addition of CO at concentrations which alone are not hazardous to life did not contribute additional toxicity.[5]

This study left certain practical points unexplored. Of the four gases selected, only HCN and (less frequently) HCl have risen to potentially-lethal concentrations in the early minutes of simulated aircraft fires. At these times CO is usually present at or above its short-term $LC_{50}$ concentration. Therefore, we do not know the manner or degree of participation of the "secondary" gases in the toxicity of "real-life" pyrolysis mixtures. Their significance is further clouded by the observation that certain irritants and the suffocant, $CO_2$, can actually delay incapacitation in an environment containing a high concentration of carbon monoxide.[6] The experiments also did not preclude the possibility that the effects of HCN and CO could be additive at higher CO concentrations.

It seemed clear that we needed additional information on the combined toxicity of CO and HCN before we could hope to assess the roles of other constituents of complex pyrolysis mixtures. We also needed to know blood carboxyhemoglobin content and blood and tissue cyanide levels in experimental animals at the time of death after exposure to varied concentrations of the two gases to assist us in interpreting the findings in victims of aircraft fires.

We therefore conducted such a study restricted to the two gases, again in cooperation with the Toxic Hazards Division, Wright-Patterson AFB, in 1972. The results of this study[7] were highly satisfactory in some respects and fell short of our goals in others.

All exposures and the measurement of blood carboxyhemoglobin and cyanide levels were carried out at Wright-Patterson AFB. In those areas of greatest interest to us, the findings were: (1) carboxyhemoglobin content of the blood at death spanned a narrow range, from 72 to 76% CO saturation of the hemoglobin; (2) blood cyanide levels at death covered a broad range, from 4.95 μg/ml to 12.2 μg/ml; (3) when both gases were present at the highest concentrations used in these exposures, there was no clear-cut evidence of additive toxicity.

Tissues (brain, heart, lung and liver) from a limited number of exposed animals were collected immediately after death and were transported to CAMI for analysis. The specimens were collected in tared tubes containing measured amounts of 1.0 molar NaOH, which was calculated to trap cyanide and prevent enzymatic and direct chemical transformation.

When analysis of these tissues was attempted at CAMI, as soon as possible after their arrival, we encountered difficulties.

The tissues were homogenized in the collection tubes by means of a Polytron® instrument. Aliquots of homogenate were subjected to Conway diffusion and analyzed colorimetrically,[3] and by the cyanide electrode (Orion®). We could not achieve satisfactory precision on replicate analyses of the homogenates by either method, and the means of analyses by the two methods were not in agreement. Even more disturbing was the fact that the indicated cyanide levels in all tissues changed over a period of a few days in unpredictable direction and quantity. Thus, our best analytical efforts seemed incapable of yielding true values at the time of death in view of the delay involved in transporting specimens from Wright-Patterson to CAMI.

At this point we decided that we must establish an animal exposure capability on premises in order to make tissue analyses promptly; a move which we had been reluctant to make because our facility is not well-adapted to handling toxic gases. We also decided to defer further tissue analyses until we had done additional methodological research.

Prior to 1970 we had begun a systematic investigation of the methods available for use in our growing program of Forensic Toxicology in aircraft accident investigation. When it became clear that cyanide analysis would become routine in accidents involving fire, we turned our attention to this substance. To date, we have evaluated fluorimetric, colorimetric, selective-ion electrode and gas-chromatographic methods of analysis. In the course of research on the latter, we have developed a highly-sensitive method based on the nitrogen-detection capability of the alkali-flame detector. It is this method, certain improvements in the collection and transfer of cyanide-

containing atmospheres for analysis and the results of cyanide-CO exposures in our own facility which we shall describe.

## PROCEDURE

The exposure chamber used in these experiments was designed and constructed locally from 1/4-inch thick plexiglas in the form of an inverted box (75 cm long, 61 cm wide, and 45 cm high) which rested on a rubber-tubing gasket set in a phenolic base. Flanges on the box allowed it to be clamped to the base at each corner, effecting a gas-tight seal. Four self-closing ports were built into one side to receive cylindrical insert cages (3 1/4-inch diameter x 11 inches long) constructed from perforated plastic for introducing experimental animals into an established atmosphere.

Rotary cages (3-1/4 inches wide x 11 inches diameter) were constructed with perforated 1/4-inch thick plastic sides and 1/4-inch steel wire mesh rims. Four of these cages were spaced on a 1/2-inch steel shaft suspended at the ends from uprights mounted on the floor of the chamber. A steel shaft with two right-angle drive units allowed the rotary cage assembly to be driven by a power unit mounted on a shelf beneath the chamber base. The power unit consisted of a 1/8-horsepower electric motor equipped with a continuously-variable fluid speed control. A gas-tight packing assembly around the vertical power shaft provided an adequate seal where the shaft passed through the floor of the chamber.

Two 4-inch "muffin" fans (Rotron Mfg. Co., Woodstock, New York, model SP2A2) were mounted inside the chamber at opposite ends, using 1-1/2 inch spacers to provide adequate distance from the chamber wall. By placing one fan at the bottom-front position and the other at the top-rear position, a circular movement of air was obtained which provided a very rapid equilibration of the gas mixture. Four Luer-Lok syringe fittings were mounted in a rectangular pattern in the top of the chamber and equipped with varying lengths of weighted tubing to allow sampling of the chamber atmosphere at different levels and locations. Large-bore inlets (1/4-inch) at the top and sides provided for the rapid introduction of gases into the chamber and for their subsequent removal.

To provide for the introduction of relatively large volumes of gas (i.e., 0.5 to 1.5% of chamber capacity) without creating positive pressure or losing gas due to venting, a rubber bladder inside the chamber was connected to an outside vent and a pinch-clamp was placed on the outside vent tube. Prior to transferring a known volume of gas to the chamber, an equal volume of air was pumped into the bladder and the pinch clamp was closed. As gas was introduced into the chamber, the pinch-clamp was opened and the equal volume of air was allowed to escape, thus introducing the gas without dilution or change in internal pressure.

Hydrogen cyanide was generated *in situ* by the introduction of sodium cyanide solution into a constantly-stirred solution of 10% sulfuric acid inside the chamber. A 3 1/2-inch Petri dish containing the sulfuric acid and

a stirring magnet was placed on the floor of the chamber opposite the lower fan and a small magnetic stirrer was mounted beneath the floor of the chamber directly below the Petri dish. The sodium cyanide solution was transferred at a controlled rate by an infusion pump (Harvard Apparatus Co., model 1100) via a 10 cc glass syringe connected to a length of plastic tubing which passed through the wall of the chamber, its delivery tip positioned directly over the dish containing the sulfuric acid.

In a typical chamber experiment where both carbon monoxide and hydrogen cyanide were used, four rats were weighed and placed in the rotary cages, 10 ml of 10% $H_2SO_4$ and a small stirring bar were placed in the Petri dish, and the chamber was lowered over the cages onto its rubber gasket and locked into position. Inert cage ports were opened and the internal fans were started to provide adequate ventilation during the remainder of the chamber preparations. A glass Luer-Lok syringe containing the NaCN solution (concentration adjusted to provide a theoretical 500 ppm HCN in the 205-liter chamber/ml of NaCN solution) was connected to the tubing leading to the $H_2SO_4$ dish and then placed in the infusion pump frame. Using the motor drive on the infusion pump, the NaCN solution was pumped into the tubing connector to a point about 2 mm from the outlet. The rubber bladder was inflated and the bladder outlet was closed. The large plastic syringe used to inflate the bladder was then filled directly from a tank of pure carbon monoxide by allowing the internal tank pressure to push the syringe plunger back to the desired volume (2.84 liters for this study). After removal from the tank outlet, the gas in the syringe was allowed to come to ambient pressure, the syringe outlet was closed and connected to the inlet in the top of the chamber. The inert cage ports were then closed, the magnetic stirrer and rotary cage motor were started, and the cage rotation was adjusted to 8-10 rpm.

After a short countdown, the following actions were initiated simultaneously:

1. The outlet from the air bladder was opened.

2. Five timing devices were actuated (one for each rotary cage and a master timer for the entire experiment for timing the withdrawal of gas samples.

3. The infusion pump was started, beginning the addition of the NaCN solution to the sulfuric acid.

4. The carbon monoxide inlet valve was opened and the gas was delivered by manual operation of the syringe at a rate corresponding to the addition of the NaCN solution (i.e., a comparable percentage of final volume/unit time).

After the addition of exactly 1.0 ml of the NaCN solution (0.75 minutes), the infusion pump was turned off. Sampling of the chamber atmosphere was begun and the animals in the rotary cages were observed for signs of incapacitation. Time to incapacitation (TI) was recorded when the

individual animal was no longer capable of maintaining the coordinated movement of walking in the rotating cage. When TI had been reached for all four animals, the cage rotation was stopped and the animals were observed for cessation of respiration. Time to death (TD) was recorded as "tentative" when the test animals appeared to have stopped breathing and was considered final if no movement of any kind was observed in 20 additional seconds.

To determine the TD for animals in a pre-established stable atmosphere, four rats were loaded into the insert cages. The individual cage timers were reset and the cages were inserted into the chamber, each timer being started at the instant of insertion. TD was recorded in the same manner described for the rotary cage studies. After all animals were dead, inlets were opened and a vacuum was applied to the chamber outlet. Cyanide in the exhaust gas was trapped in a series of sodium hydroxide absorbers and the residual carbon monoxide was vented to an outlet on the building roof. Design and construction details of the chamber are shown in Figs. 1 and 2.

CO concentration was monitored by gas chromatographic analysis. The instrument used was the Carle Model 8000 equipped with 8-10 Kohm thermistors as the detector. Other characteristics were: Column, 3' x 1/8" stainless steel packed with 40-60 mesh silica gel followed by 5' x 1/8" stainless steel packed with 40-50 mesh Linde Molecular Sieve 5A. Column temperature, 52°C. Carrier gas, helium 20 ml/min. Recorder, Honeywell Electronik 19 operated at 0.5 mv full scale.

Samples were taken in 20 ml plastic syringes, the first at one minute and four or more others thereafter at intervals of three to four minutes. Replicate 4 ml aliquots of the sample were flushed through a 117 µl loop for introduction into the instrument. Quantitation was by comparison of peak heights with those of standards prepared by dilution of pure CO with air.

The method of collecting chamber atmosphere for HCN measurement depended upon the analytical method to be employed.

For colorimetric analysis, 17 to 20 ml of the atmosphere were drawn into acid-washed, dry, 30 ml glass syringes. After detachment of the syringe from the sampling port, the gas volume was quickly adjusted to 15 ml and the syringe tip was immediately placed below the surface of 0.1 molar NaOH in a beaker. NaOH was drawn in rapidly so that all glass surfaces were coated by the entering jet of liquid. When NaOH had been taken in to the 15 ml graduation, the tip of the syringe was capped and the syringe was placed on a mixer (Ames Model 4651, Ames Co., Elkhart, Indiana) and rocked endwise for two hours to ensure complete HCN absorption. Analysis followed the method as described[3] except that optical density was recorded and values were read at peak density. Standards were prepared from commercial NaCN (Reagent Grade, Fisher Scientific Co.) analyzed potentiometrically in our laboratory.

Other collection techniques have been tested and have resulted in erratic variations between replicate samples. By this method, multiple, successive samples from a compressed $N_2$-HCN mixture have been analyzed with a

coefficient of variation of less than 3%. It should be pointed out that relative volumes of gas and absorbent can be adjusted to suit known or predicted atmospheric concentration so that no dilutions are necessary and losses due to volatilization during such processing can be avoided. It is our hope that mixtures of reactive gases may be satisfactorily collected in this way if it is compatible with subsequent analytical techniques.

If monitoring was to be done by gas chromatography, samples were drawn in 5 ml glass syringes which had been siliconized to remove surface adsorptive sites. Each sample was immediately transferred to and flushed through a 104 µl sampling loop at the inlet of the gas chromatograph. Quantitation of resulting peak heights was by use of a regression equation derived from chromatography under identical conditions of gaseous HCN standards. These standards were prepared from a compressed HCN-in-$N_2$ mixture purchased from Matheson Gas Products, Houston, Texas, at a nominal concentration of 1000 ppm (V/V). The gas mixture was analyzed in our laboratory and found to be 1044 ppm (V/V).

The gas chromatograph used in these analyses was the Tracor Model MT 220. The operating parameters were:

| | |
|---|---|
| Column: | 6' x 1/4" glass U-tube. |
| Packing: | Porapack Q, 80/100 mesh. |
| Temperatures: | Oven, 110°C; Injector, 135°C; Detector, 150°C. |
| Gas flow rates: | Carrier, $N_2$, 40-60 ml/min.; Detector $H_2$, 10-35 ml/min; Detector air, 250-450 ml/min. |

The detector is of the alkali flame ionization (AFID) type. It is not available as such for the MT 220 chromatograph and was therefore fabricated in our laboratory. It consists of a pellet of an alkali metal salt (Rubidium is in use currently—anion appears not to be critical) pressed at 10,000 psig into a 1/8" Swagelok front ferrule. A hole, 0.025" diameter, is drilled through the pellet and the ferrule is mounted by press-fitting onto the top of the regular FID flame tip which has been previously shortened by 4 mm.

This modified detector increases the sensitivity of the GC for HCN by a factor of at least 50, and simultaneously exhibits an enhanced selectivity for HCN over hydrocarbons. The lower limit of detectability at a signal/noise = 2 is at least as low as 200 picograms HCN. The magnitude of the response and the stability of the detector is critically dependent on precise control of flow rates of the three gases and on the condition of the pellet. The method will be described in greater detail elsewhere.

Hydrogen cyanide generated *in situ* as in our experiments does not immediately rise to a uniform, final concentration in the chamber atmosphere due to the high solubility of the gas in aqueous solution, even at low pH, and its origin from a "point" source. Therefore, whatever the method of collection, samples were taken as frequently as possible during the first few minutes of each run to determine the concentration profile in the vicinity of

the exposed animals, and at convenient intervals thereafter to test the maintenance of the final concentration in the chamber.

Figure 3 is a composite presentation of all HCN and CO measurements in all exposure experiments thus far.

## RESULTS

The concentrations of CO and HCN selected for use in this preliminary study, whether employed singly or in combination, were slightly below the five-minute $LC_{50}$ values found in the second Wright-Patterson study,[7] which were 14,200 ppm (V/V) and 480 ppm (V/V), respectively. Although high, these concentrations are not unrealistic in comparison with values which have been found to occur within minutes after source ignition in simulated aircraft fires.

The generation of hydrogen cyanide gas within the chamber was a device adopted in order to avoid bringing liquid HCN into the CAMI building. The rate of rise of HCN to its final concentration can be controlled by adjusting the NaCN concentration and the rate of delivery. The rate chosen represents a typical rise-time in test fires.

Although CO can be introduced at any desired rate, it was added to the chamber rapidly in both the single and combined exposures of this study because it usually reaches dangerous levels first in real-life situations.

Table I summarizes the results of all exposures conducted thus far. It represents mean values and S.D. for 12 animals under each of six exposure situations; e.g., in HCN alone, 12 animals were observed for time to physical incapacitation and time to death in rotating cages in the developing toxic atmosphere. Twelve others were observed for time to death only at maximum HCN concentration. Three consecutive experiments were required to test this total of 24 animals.

Physical incapacitation is an end point which can be determined with considerable precision. Under all exposure conditions the animals have collapsed and become inert, sliding or tumbling in their cages within the time required for one cage rotation. There is also surprisingly little variation in time between the TI of individual animals in the same exposure situation. These facts are evident from the low S.D. of the means of these measurements. They are also emphasized by the fact that the difference between the TI in CO alone (2.4) and in CO-HCN (2.0) is statistically highly significant ($p<.001$).

It should be remembered that in all instances, time to incapacitation and time to death in the rotating cages include the rise-time of the toxic gases, slow in the case of HCN, rapid for CO.

Time to death cannot be judged with equal precision. The complete cessation of respiration, the only objective sign available to us, is

difficult to assess and the decision is slightly arbitrary. Also, such movements may persist in only one animal of a group of four long after the others are obviously dead. In addition, we have noted that terminal behavior differs in the three exposure atmospheres. Although there may be a terminal convulsive episode in all three situations, respiratory movements tend to remain fairly ample, though infrequent, then cease abruptly in CO. In HCN, on the other hand, respiratory efforts gradually became more feeble and less frequent, and may persist for a minute or longer as barely perceptible movements of the chest wall. In the mixed-gas atmosphere, respiration ceases abruptly.

The most striking result of these exposures is the markedly shortened survival time in CO-HCN; 3.7 minutes in the rotating cages, which includes rise-time of the two gases, and 3.2 in the pre-established atmosphere.

## SUMMARY AND CONCLUSIONS

We have designed and constructed a facility which permits exposure of animals to developing or static toxic atmospheres in which rise-profiles and final concentration of the contaminants can be controlled at will with satisfactory precision.

We have developed improved methods for collecting and transferring HCN for analysis.

We have developed a highly specific and sensitive gas-chromatographic method for HCN analysis.

We have conclusively demonstrated additive toxicity of CO and HCN at the concentrations employed in this study.

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TABLE I

TIME TO PHYSICAL INCAPACITATION (TI) AND TIME TO DEATH (TD) IN CO, HCN AND CO+HCN ATMOSPHERES

| | HCN-450 PPM | CO-13,500 PPM | CO+HCN |
|---|---|---|---|
| Rotary Cages | | | |
| TI Minutes | 4.1 ± 0.7 | 2.4 ± 0.3 | 2.0 ± 0.3 |
| TD Minutes | 10.9 ± 2.0 | 5.8 ± 1.2 | 3.7 ± 0.4 |
| Insert Cages | | | |
| TD Minutes | 7.9 ± 2.0 | 6.3 ± 1.5 | 3.2 ± 0.4 |

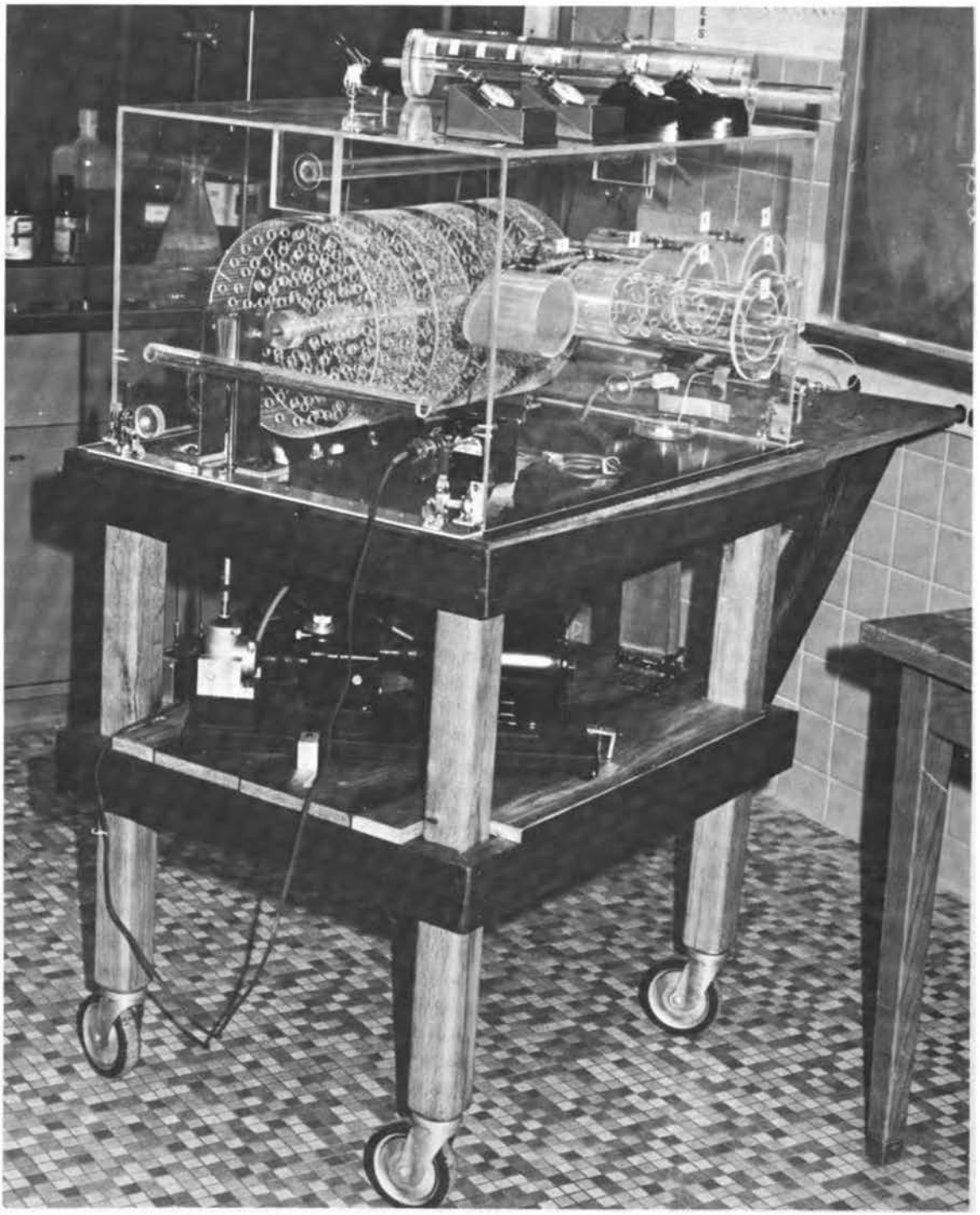

FIGURE 1. Exposure Chamber. Over-all view indicating design and construction.

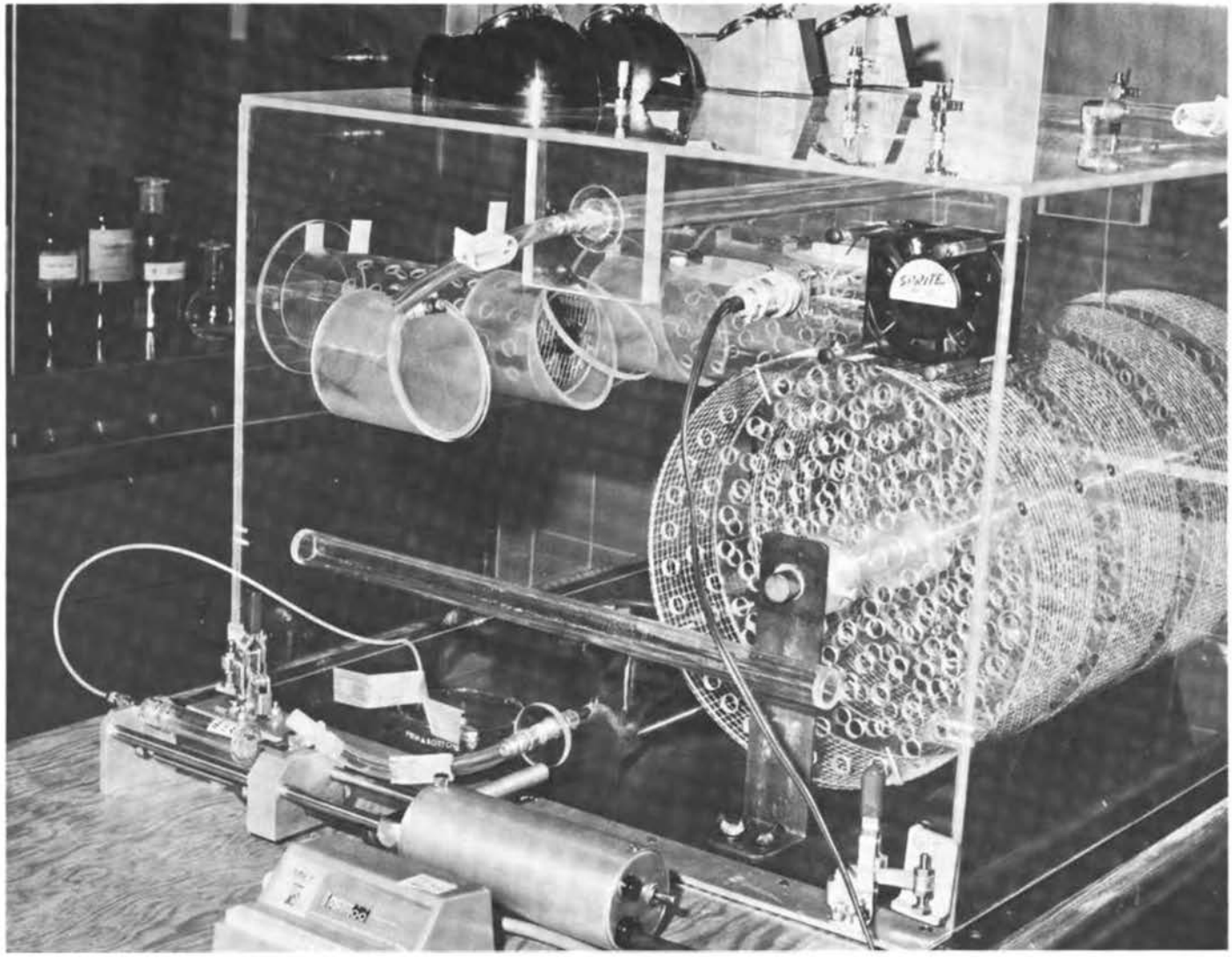


FIGURE 2. Exposure Chamber. Detail view showing HCN generator and ports for insert cages with hinged closures.

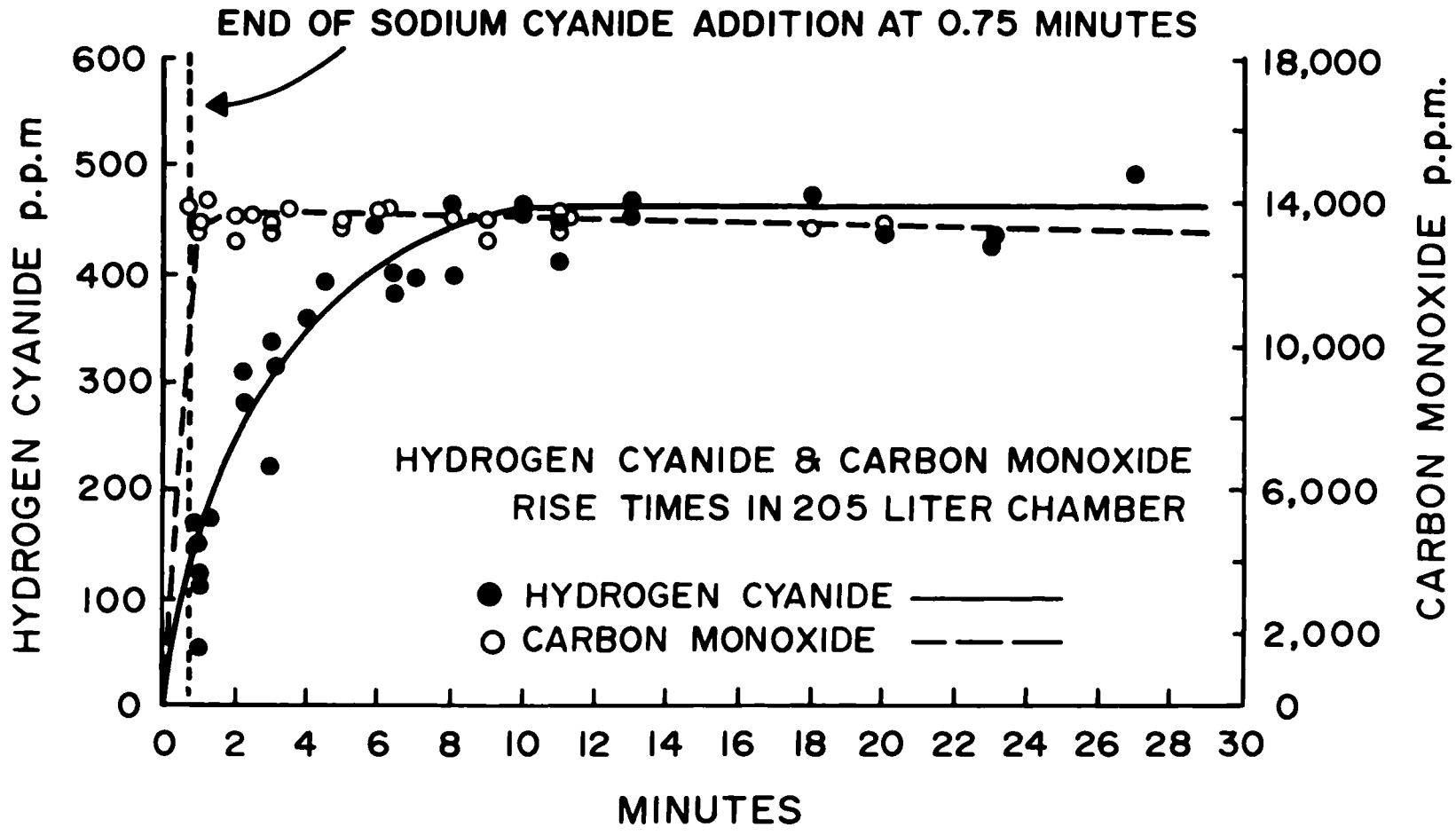


FIGURE 3. Concentration-time profiles of CO and HCN used in all exposures, single and combined.

# *SYNERGISTIC EFFECTS OF COMBUSTION PRODUCTS*

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## WHAT ARE WE DISCUSSING?

Before any intelligent discussion of the subject of synergisms can be undertaken, one must clearly establish the meaning of the terms involved. Webster defines synergism as the "cooperative action of discrete elements such that the total effects are greater than the sum of the individual effects taken independently." Combustion Products I will define as any factor found in the thermal or oxidative decomposition of materials exposed to a source of energy.

As one looks at these definitions he cannot help being appalled by the magnitude of the task confronting him—considering the possible effects of "n" variables to give an overall physiological response. One does not however, learn to walk without taking the first step. Insofar as the synergistic effect of combustion products is concerned we are still in the infant stage; we have learned to crawl but we have not yet learned to walk.

In preparation for that first step we must first stand up and look around us to see what we already know. If we do this we find that our balance is very precarious, our base of understanding is very narrow and our first efforts at walking have been unsure and halting.

## WHAT DO WE KNOW?

The suggestion of possible synergisms is not new. One of the first statements concerning this possibility dates back to 1933.[1] Since that time only a limited number of workers have dealt with the problem of synergisms and where work has been done it has invariably been in conjunction with carbon monoxide. This concentration of effort on the possible synergism of carbon monoxide and another compound is logical, since 60-80% of all fire-related deaths, other than direct burns, generally have been attributed to carbon monoxide poisoning or suffocation. Autopsy records on fire deaths have been fragmentary and even where performed have not in general been able to pinpoint

the true cause of death. Recent studies of fire deaths by The Applied Physics Laboratory of Johns Hopkins University and the Flammability Research Center here at the University of Utah, have discovered a significant number for which carboxyhemoglobin levels alone should not have caused death. In many of these cases high blood alcohol levels were also found. Does this indicate a possible synergism between alcohol and carbon monoxide, or does it suggest the presence of some other still unidentified component? One study published in 1959[2] with dogs as the subjects found "no significant summation effect of alcohol and carbon monoxide." Here we have one of the conflicts of available information. It does not, however, stand alone. In 1961 a work[3] suggests evidence of a possible synergism between nitrogen oxides and carbon monoxide, more recent works in 1966[4] and 1972[5] appear to indicate that no synergistic effects exist between nitrogen oxides and carbon monoxide. The same conflict can be seen with hydrogen sulfide and carbon monoxide.[6,7]

A list of components and the probability of synergistic effects with carbon monoxide is given in Table I. No claims as to the completeness of the list nor the validity of the conclusions drawn by the original workers is made. It is merely a summary of some of the work in the area of synergisms, and points up the confusion that can be created in the minds of those surveying the available information. In all probability the conclusions drawn were logical based upon the conditions of the experiments and the data obtained. One cannot but be concerned, however, with the lack of uniformity between test conditions and the wide variety of test animals used with the possibility of completely different physiological responses. To me this Table only indicates that we are really no further along in our efforts to define synergisms in mixtures of gases than we were in 1950 when Schrenk wrote "The toxicology of the single substances is fairly well known but the physiological action of mixed gases and the influence of aerosols is an almost unexplored field."[15] Cornish, during a symposium conducted during 1973, emphasized further: "A number of research areas still require considerable study. These include the simultaneous effects of hydrogen cyanide and carbon monoxide, decreased oxygen tension and elevated carbon dioxide levels—all of which may affect oxygen transport and utilization."

Overriding any deficiencies that may exist in published literature, is the gut-feeling that most workers in this field have—when complex mixtures such as those found in fire gases exist, one and one may not always equal two. Until more substantial evidence is presented, caution must be exercised not to underestimate the situation. Ahead of us lies the opportunity to create even greater confusion or to progress to a greater understanding of the interaction of complex gas mixtures.

## WHAT DO WE NEED TO KNOW?

Before we can successfully attack the problem of possible synergisms in combustion products from fires, a more complete understanding of the composition of such gases and the time of appearance of each of the components must be obtained. Better definition of experimental conditions leading to a standardization of exposure techniques used with test animals and better

methods for evaluating the physiological effects of single and multicomponent gas mixtures are needed. As one progresses through the relatively small volume of literature dealing with the subject he finds that virtually every investigator has his own way of defining proper procedures. Test animals have varied from mice through rats, rabbits and dogs, etc. Exposure conditions are almost as varied as the investigators and are often hard to glean from the literature. Until agreement can be achieved upon acceptable standard exposure conditions and evaluation procedures, it may be impossible to resolve apparent discrepancies in the results obtained, or for that matter ever to be sure that synergisms are a reality.

Studies on binary mixtures can never be a completely adequate explanation of the physiological effects encountered in real fire situations. All fires, regardless of the material involved, are complex mechanisms involving thermal degradation and high temperature free radical reactions. It is unlikely that two situations will ever exist in which the identical set of combustion products will be obtained. To assign synergistic properties to a given pair combination under synthetic mixture condition may never do more than further confuse the issue, since it ignores the possible presence of unidentified components in the fire gases whose lethality may be several orders of magnitude greater than either or both in the synthetic mixture. It also ignores the possible neutralizing effect of the two separately toxic components by addition or substitution reactions in the fire environment to form relatively innocuous products.

Before any realistic assessment of synergisms meaningful to the real fire situation can be made, it is essential that a great deal more knowledge must be obtained in these several areas.

## HOW CAN THE NECESSARY INFORMATION BE OBTAINED?

Toxicologist, physiologist and fire chemists will probably never agree entirely upon the importance of their individual researches. What is outlined in the following paragraphs probably falls in the same category as "which came first, the chicken or the egg?". But real progress in defining significant synergistic effects in fire situation can only be achieved from effective and continuing dialogue between the various disciplines. It will be counterproductive for any group to put its personal goals as paramount. The work of each group must direct and fortify that of the others.

With this in mind I will present one person's view of areas needing effective research.

1. Continued study and data gathering on fires and all fire deaths not directly attributable to burns must be undertaken to provide a data base from which to work. Autopsies on all such deaths must become a routine part of all fire investigations. Understanding the true causes of death will necessitate vast improvement in analytical techniques as they are applied to biological systems. The organ affected by the potential toxicants found in fire situations, the mode of attack, and the extent of damage must all be known to

define physiological effects. We have barely scratched the surface in this area, but resolution of these problems must be a precursor to determination of synergisms. Studies such as those underway at APL of Johns Hopkins University and Flammability Research Center here at Utah must be advanced as rapidly as possible, with prime emphasis on the relevance to real life situations.

2. Definition of the products of combustion to be obtained from realistic fires involving all materials must be completed. The time of occurrence may be of equal importance to the quantities produced. The ultimate resolution of the toxicologist's problem will necessitate time/concentration information. At the same time if we are to progress at other than a snail's pace, the analyst must have continuing feedback from the toxicologist if he is to avoid unnecessary effort. It would appear to be academic whether two compounds are synergistic if they are not found together in the fire environment or if one occurs only after the victim is already dead from other causes.

3. If we are to save lives in fires, it appears logical that we must concentrate our total effort first upon obtaining information on what occurs in the early stages of a fire. We must first get a victim out of the environment before we can treat him. Heat, carbon monoxide, oxygen depletion and carbon dioxide are all increasing rapidly during this period and are common to all fires. This is an oversimplification of the fire gas problem, but the interreaction of these four elements must first be understood if we are to answer the synergistic question with other elements of the fire. What happens with different rates of increase of each of these components acting together? How do variation of materials exposed in the fire effect the rate/concentration ratios? Although Pryor and his coworkers[11] answered a portion of these questions, the ultimate resolution is still needed. With this solution in hand we can then proceed to adequately evaluate the effects of other components as they occur in the time frame of the burning of a specific material and in concentrations meaningful to the life hazard cycle of a fire. We must then concern ourselves with the possible long-term effects of the exposures we have created in order to effectively treat exposure victims to reduce the delayed deaths.

4. Concurrent with the definition of the above research areas must be efforts to apply the knowledge to the design, use and regulation of all consumer products. This must be done by a process of logical consideration of all facets, to create the safest environment possible and the least possible socio-economic disruption. If we find the answers to questions raised above we can have both safety and the good life to which we have become accustomed.

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TABLE I

THE POSSIBILITY OF SYNERGISM BETWEEN CARBON MONOXIDE AND OTHER AIR COMPONENTS

| Component | Synergistic | Reference |
|---|---|---|
| Ethyl alcohol | + + - | 2, 8, 9 |
| Nitrogen dioxide | + - - | 3, 4, 5 |
| Hydrogen sulfide | + - | 6, 7 |
| Hydrogen cyanide | - | 5 |
| Hydrogen chloride | - | 5 |
| Hydrogen fluoride | - | 5 |
| Sulfur dioxide | + | 10 |
| Temperature | + + | 11, 12 |
| Carbon dioxide | + + - | 1, 11, 13 |
| Oxygen (depletion) | + + - | 1, 11, 13 |
| Benzene | + | 14 |
| Ozone | - | 4 |

# *TOXICITY ASSOCIATED WITH FLAME-RETARDED PLASTICS*

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As a result of the use of fluorinated polymers in spacecraft applications calling for heat resistance and low flammability, our laboratory began investigating the potential toxicity of the pyrolysis products of some elastomers typical of this group. Samples of pure polymers, copolymers, and terpolymers from two commercial sources were obtained through the National Research Council, Advisory Center for Toxicology. They are identified as (1) polytetrafluoroethylene (PTFE), (2) copolymer of vinylidene fluoride and hexafluoropropene ($VF_2$/HFP), (3) copolymer of vinylidene fluoride and hexafluoropropene with "additives to provide improved properties" ($VF_2$/HFP-A), and (4) terpolymer of vinylidene fluoride, hexafluoropropene, and tetrafluoroethylene ($VF_2$/HFP/TFE). The data obtained from these studies will be used as baseline for comparison with similar materials compounded and processed for use.

## METHODS

### Apparatus

The exposure chamber (Fig. 1) consisted of a modified 142-liter Bethlehem chamber with Plexiglas replacing the glass viewports. One viewport was removed and replaced with an airlock so constructed that the door on the chamber end of the airlock could be opened remotely from the outside. A stainless steel wire partition confined the rats to the forward one-third of the chamber where the pyrolysate was introduced. A dial thermometer and a pressure gauge were installed to record chamber temperature and pressure. A one-fourth inch stainless steel sampling line was introduced into the forward part of the chamber from which samples for analyses were obtained by the use of a diaphragm pump to pressurize one-half liter stainless steel sample bottles.

The pyrolysis tube (Fig. 1) consisted of a one-inch stainless steel tube configured to deliver the pyrolysate directly into the exposure chamber. A

one-inch tee at the top of the pyrolysis tube was designed to permit the introduction of a one-fourth inch stainless steel tube. This tube was used as a sample holder for the insertion of the sample into the hot portion of the pyrolysis tube and as a method of allowing air flow to the sample. A thermocouple for sample temperature measurement was installed in the tip of this small tube. Four orifices directly above the sample holder permitted air flow over the sample and up through the pyrolysis tube into the exposure chamber. A Lindberg model 55031 furnace equipped with a model 59344 temperature controller was used to carry out the thermal decomposition.

Procedure

A sample of the fluoropolymer was prepared for pyrolysis by cutting a 16-mm plug of the sample, boring a 6-mm hole in the middle, and shaving the sample until the desired sample weight was obtained. The sample was then placed upon the sample carrier on the end of the sample tube and the assembly inserted into the pyrolysis tube with the sample just below the one-inch tee. All fittings were tightened and the pressure in the system reduced to 600 torr. The preheated furnace was then placed around the pyrolysis tube so that the bottom of the pyrolysis tube was located in the middle of the furnace with the sample well above the heated zone. Ten minutes were allowed to preheat the pyrolysis tube. With the furnace and the pyrolysis tube at temperature equilibrium, air flow was initiated through the sample tube from a compressed air source at the rate of 100 cc/minute. The swagelock nut holding the sample tube in place was loosened to allow the end of the sample tube containing the sample to be pushed into the bottom, or the hot zone, of the pyrolysis tube and retightened. This initiated pyrolysis while the air flow moved the pyrolysate into the exposure chamber. Thirty minutes were allowed to assure complete pyrolysis at which time the furnace was removed from the pyrolysis tube. The air flow was then increased to bring the internal system pressure up to ambient, simultaneously sweeping any residual pyrolysis products out of the combustion tube and into the exposure chamber.

As soon as the pressure in the system reached ambient, ten male albino, 225-250 g, Sprague-Dawley rats were placed in the airlock. The outside door of the airlock was closed and the inside door was opened which allowed the rats to drop into the exposure chamber. Exposures were conducted for 30 minutes at which time the chamber door was opened and the rats removed for observation. Pilot studies on these fluoropolymers had indicated that all deaths due to exposure occurred within 48 hours. Therefore, all of the acute toxicity studies in this investigation were based upon 48-hour postexposure survival. A representative sample of the rats surviving the 48-hour observation period, and where possible, those expiring during that period were necropsied for gross and histopathological examination.

Supplemental pyrolysis exposures were also conducted at the calculated maximum sublethal dose of each fluoropolymer for extended pathological examination. Rats were serially euthanized at 1, 2, 4, 8, 16, and 32 days postexposure for gross and histological examination.

Pyrolysis exposures at the $LD_{50}$ level were also conducted for carboxyhemoglobin determinations. Blood for these determinations was obtained from the rats within one minute after being removed from the chamber and carboxyhemoglobin determinations performed on an Instrumentation Laboratories model 182 CO-oximeter precalibrated for rat blood and verified by the Van Slyke manometric method.

Samples for quantitating carbon monoxide (CO) and carbon dioxide ($CO_2$) concentrations in the chamber were obtained at 0, 15, and 30 minutes during the rat exposures. Analyses for CO and $CO_2$ were carried out using standard gas chromatographic methodology. Hydrolyzable fluoride analyses were obtained by passing the pyrolysate directly from the pyrolysis tube through two traps containing 700 ml and 200 ml of 0.01 M sodium carbonate.

The trapped fluoride was determined by an Orion ion-specific electrode. Thermogravimetric analyses of the fluoropolymers were accomplished using a Perkin-Elmer TGS-1 thermobalance with a programmed temperature rate of 20°C/minute and an air flow over the sample of 200 cc/minute. A Varian model 1740 gas chromatograph with either 6-feet x 1/8-inch Chromosorb 102 or 10-feet x 1/8-inch OPN/Poracil C columns and a flame ionization detector was used for quantitative analysis of the pyrolysis products. Qualitative identification was carried out on a Varian CH5 mass spectrometer which was interfaced with the above gas chromatograph by a Watson Biemann helium separator.

Probit analysis of the data for the $LD_{50}$ determinations and 95% confidence limits were conducted by the method of Litchfield and Wilcoxon.[1] All exposures, with the exception of those for $LD_{50}$ determinations, were accompanied by sham control exposures on an equal number of rats.

## RESULTS AND DISCUSSION

The initial pyrolysis studies were conducted at the minimum temperature causing complete thermal degradation of the material as determined by thermogravimetric analysis (TGA). The three vinylidene fluoride and hexafluoropropene containing polymers were found to be similar in their thermal degradation characteristics. The slopes of the TGA curves for these materials were similar with little weight loss occurring before 450°C and complete thermal degradation taking place at 525-550°C. The TGA established 625°C as the temperature required for complete thermal decomposition of PTFE.

The results of animal exposure to the degradation products of the fluoropolymers are presented in Table I. A comparison of the toxicity of the 550°C pyrolysates from the vinylidene fluoride and hexafluoropropene containing polymers indicates that the VF /HFP copolymer is the least toxic when thermally degraded under these conditions. The addition of tetrafluoroethylene to the polymer increased the toxicity of the pyrolysate as did addition of the unidentified additives. However, all of the 500°C pyrolysates from the vinylidene fluoride and hexafluoropropene containing fluoropolymers were less toxic than the 625°C pyrolysate from the PTFE. No actual 30-minute $LD_{50}$ was established for PTFE due to the impracticality of attempting to

determine lethalities between 0.45 g which produced no deaths and 0.50 g which produced 100% lethality. An increase in the pyrolysis temperature to 800°C was accompanied by an increase in the toxicity of all the materials tested. Although the $VF_2$/HFP copolymer remained the least toxic, very little difference between the vinylidene fluoride and hexafluoropropene containing polymers was apparent. When PTFE was exposed to 800°C, a reduction in the exposure time to five minutes was required in order to establish an $LD_{50}$. As previously observed at lower temperatures, PTFE obviously produces the most toxic pyrolysate under the test conditions. At no time was visible smoke observed from the pyrolysis of any of these materials.

Gross and histological examination of tissues from the rats exposed to lethal levels of the pyrolysates exhibited lesions characteristic of the response following exposure to severe pulmonary irritants. This response was characterized by initial capillary damage leading to pulmonary edema with accompanying alveolar cell hypertrophy and desquamation. If the animal survived the first 24-48 hours, the edema was resolved and a proliferative phase began. The alveolar lining hypertrophied and histiocytes infiltrated the septa leading to partial alveolar collapse. By one week postexposure, resolution was complete and the lungs had returned to normal. The typical pulmonary response is illustrated graphically in Fig. 2. No other organ system was found to be involved. This picture is consistent with the early observations that survival for 48 hours assured recovery unless sequalae, such as secondary pneumonia, became complicating factors. Under the test conditions used to establish the $LD_{50}$'s, none of the animals died during the exposure, with a majority of the deaths occurring from 4-24 hours post-exposure.

Results from exposures to sublethal doses of the pyrolysates were similar to those following higher doses, differing principally in the degree of damage. The pulmonary changes following these exposures were defined as a more lobular pattern with the significant pathology often restricted to the respiratory bronchioles and adjacent alveolar walls. Resolution was complete within eight days with no subsequent effects noted at 16 or 32 days post-exposure.

Analysis of the pyrolysate from the chamber atmosphere immediately prior to introduction of the animals disclosed CO and $CO_2$ levels well below the lethal levels for 30-minutes exposure to rats. Carboxyhemoglobin determinations from rats exposed to both the $LD_{50}$ and $LD_{90}$ levels of the pyrolysates ranged from 18.3% to 52.5% which are well below lethal levels. Although carbon dioxide levels rose during the animal exposure, at no time did they approach a level which would cause incapacitation of the subjects.

Examination of the pyrolysate from PTFE by mass spectrometry revealed the presence of perfluorinated alkanes, alkenes, and alkadines of up to eight carbons in length as well as perfluorinated cyclic alkanes and alkenes. The pyrolysate from the vinylidene fluoride and hexafluoropropene containing elastomers consisted of similar compounds differing only by the presence of hydrogen in the molecule. Only in the pyrolysate from PTFE did the

concentration of any fluorocarbon species exceed 100 ppm. Publication of complete qualitative and quantitative data from these pyrolysates must await completion of this effort.

In the design of the pyrolysis system used in this study, special emphasis was given to rapid initiation of the thermal degradation and to minimum temperature variation during the pyrolysis. It was felt that these conditions had to be met in order to minimize the variation characteristic of studies such as these so that reproducible data could be obtained. Generally, the system accomplished the objective in that little difficulty was experienced in establishing reproducible lethality curves from the vinylidene fluoride and hexafluoropropene containing elastomers.

There are, however, some disadvantages with the use of this system that should be considered. The most obvious is the problem of condensation of the pyrolysate inside the pyrolysis and delivery tube. This results in the incomplete delivery of the pyrolysate into the exposure chamber. Pyrolysis of the material inside the exposure chamber could possibly eliminate this problem. Secondly, even though the thermocouple is in direct contact with the sample, insufficient information is available to accurately determine if the temperature registered at that point is the actual pyrolysis temperature of the sample. Finally, recent data indicates that a direct relationship between sample size and the quantity of the heavier molecular weight fluorocarbons in the pyrolysate may exist. This is especially true for PTFE, and along with the exposure time, may account for the difficulty in establishing a 30-minute $LD_{50}$ for this elastomer. Curie point pyrolysis could solve the problem of exact temperature definition as well as the sample size problem. However, the small sample required for this type of thermal degradation precludes its use in any animal exposure chamber of workable size.

Results of efforts to define the compounds responsible for the pulmonary irritation have been inconclusive. Carbonyl fluoride has been implicated as the primary cause of the pulmonary edema resulting from exposure to the 550°C pyrolysate produced from PTFE. Carbonyl fluoride was not quantitated directly but was derived through hydrolyzable fluoride determinations from the pyrolysate.[2,3] Our attempt to relate the hydrolyzable fluoride produced by these fluoropolymers with the relative lethalities of their pyrolysates indicated that no such correlation exists under these test conditions (Table I). On the other hand, the presence of fluoroalkenes in the pyrolysates may be of primary toxicological significance although, individually, they are present in relatively small concentrations. The potency of certain compounds of this group as pulmonary irritants, e.g., hexafluoropropene, perfluoroisobutylene, and 2H-heptafluorobutene-2, is well documented.[4] The degree to which these fluoroalkenes contribute to the total toxicologic response must await complete qualitative and quantitative analyses of the pyrolysates.

In conclusion, the relative toxicity of the pyrolysis products from three polymeric formulations containing vinylidene fluoride and hexafluoropropene has been determined. All three of these hydrogen containing

elastomers produced a less toxic pyrolysate than polytetrafluoroethylene. Identification of the compounds most probably responsible for the pulmonary damage induced by these pyrolysates is presently being carried out.

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TABLE I

| Polymer[a] | $LD_{50}$[b] | CO%[c] | $CO_2$%[c] | Fluoride[d] |
|---|---|---|---|---|
| | 550°C | | | |
| $VF_2$/HFP | 2.36 (2.27-2.45) | 0.05-0.07 | 0.20-0.28 | 10.57±0.66 |
| $VF_2$/HFP/TFE | 1.52 (1.46-1.60) | 0.04-0.10 | 0.14-0.48 | 5.23±0.60 |
| $VF_2$/HFP-A | 1.06 (1.00-1.12) | 0.02-0.05 | 0.13-0.26 | 0.19±0.50 |
| PTFE | 0.5[e] | 0.01 | 0.01 | 0.14±0.09 |
| | 800°C | | | |
| $VF_2$/HFP | 0.59 (0.56-0.62) | 0.01-0.02 | 0.06-0.10 | 2.55±0.38 |
| $VF_2$/HFP/TFE | 0.42 (0.34-0.54) | 0.02-0.04 | 0.05-0.08 | 1.34±0.46 |
| $VF_2$/HFP-A | 0.46 (0.43-0.50) | 0.01-0.02 | 0.05-0.07 | 2.49±0.20 |
| PTFE | 0.38 (0.34-0.43)[f] | 0.01 | 0.07-0.09 | 1.50±0.12 |

[a] $VF_2$/HFP - copolymer of vinylidene fluoride and hexafluoropropene;
$VF_2$/HFP/TFE - terpolymer of vinylidene fluoride, hexafluoropropene, and tetrafluoroethylene;
$VF_2$/HFP-A - copolymer of vinylidene fluoride and hexafluoropropene with additives;
PTFE - polytetrafluoroethylene.

[b] Grams of fluoropolymer and (95% confidence limits).

[c] Range of chamber concentrations at beginning of all exposures of $LD_{50}$ dose level.

[d] Mean ± SD mM of $F^-$ obtained from the pyrolysate of five burns at the $LD_{50}$ dose level.

[e] Pyrolysis temperature, 625°C.

[f] $LD_{50}$ for five-minute exposure.

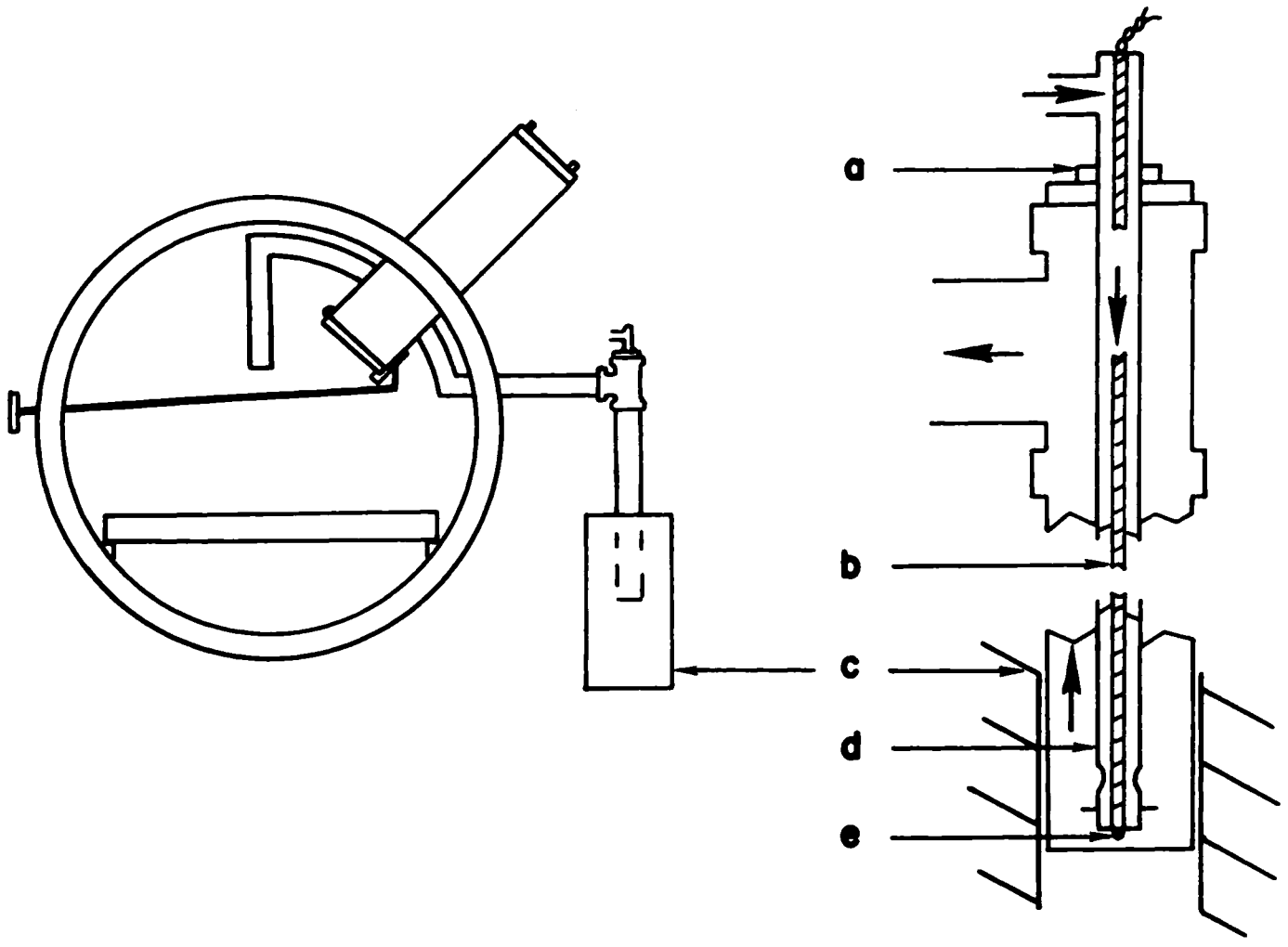


FIGURE 1. Diagram of chamber, pyrolysis tube, and furnace on the left and cross section of pyrolysis tube on the right. Arrows indicate direction of air flow. A - Swage-lock nut; B - Thermocouple wire; C - Furnace; D - 1/4 inch sample holder tube; E - Thermocouple.

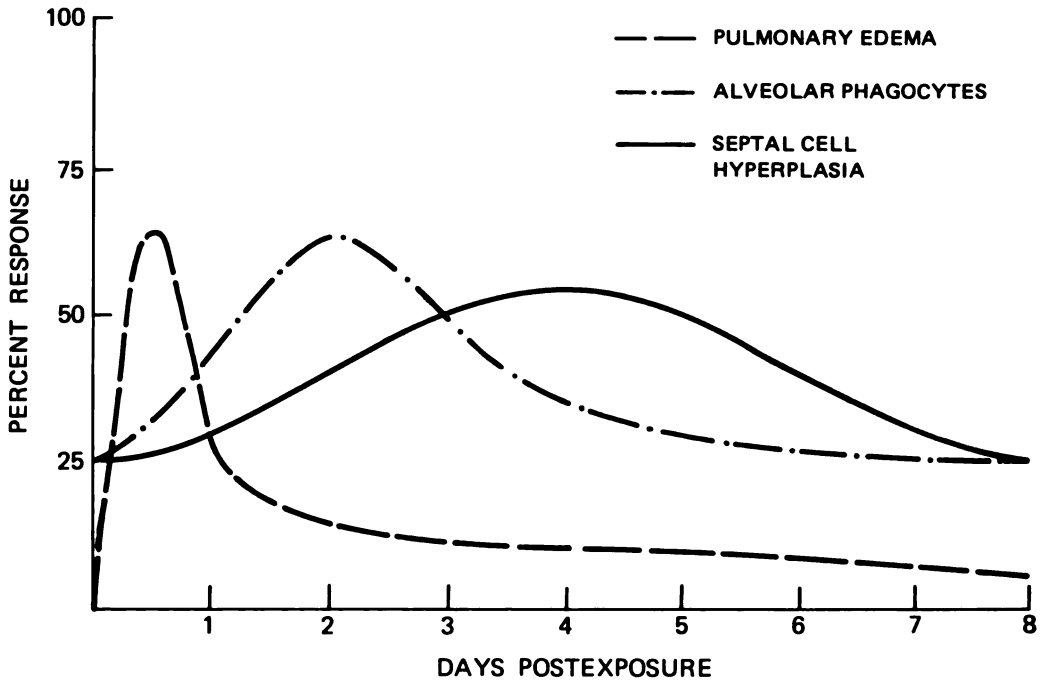


FIGURE 2. The acute rat lung response to sublethal fluorocarbon pyrolysis exposure.

# *SURVIVAL RESPONSE DURING FIRE EXPOSURE*

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## INTRODUCTION

When discussing survival from the effects of fire, it is customary to refer to mortality data. While these data give some indication of the severity of the problem, the factors responsible for mortality and morbidity remain undisclosed. A redefinition of the term "survival" is required, in order to take into account numerous factors which may prevent or impair escape from fire, or be responsible for long-term effects which do serious damage to the quality of life.

Since carbon monoxide is present in varying concentrations, along with other noxious gases, during the combustion of polymers and other materials, investigations conducted at the Flammability Research Center at the University of Utah have been concerned with the evaluation of "survival" in the presence of carbon monoxide. We have found that more exact knowledge of the action of carbon monoxide upon the nervous system has been very useful in the detection of effects produced by other intoxicants present in smoke.

In the following discussion I will first describe the levels of intoxication on exposure to carbon monoxide with respect to its effect on survival in fire and then consider the role of factors which may modify the response.

## METHODS

### The Animal Model

Our investigations have required the development of an animal model. The model permits monitoring of a variety of physiological functions and allows behavior to be monitored as well. Initial experiments were carried out on albino rats, allowed to move freely in a modified Skinner box, which served also as an exposure chamber (Fig. 1). An avoidance-conditioning paradigm was employed. The floor of the chamber was an electrified grid which delivered a shock to the animal's feet. A warning light was introduced into the experimental design. Unfortunately, less than half of the rats studied were capable of learning the avoidance response. Furthermore, it was quite difficult to assess the physiological status of the animals under these

conditions. However, as a consequence of observing behavior in these animals, five levels of intoxication were clearly defined, which have served as the basis for the development of a more manageable model. The five levels, in ascending order of intoxication, are as follows:

1. Production of distal limb and trunkal ataxia; reduction in exploratory behavior; decreased awareness.

2. Loss of an operant survival response.

3. Motor collapse; loss of postural tonus; failure to respond to electric shock.

4. Anoxic shock.

5. Death.

Assessment of the physiological status of the animal and analysis of levels of intoxication were greatly facilitated by placement of the animal in a sling, which permits free movement of the legs and head (Fig. 2). With this restraint and proper instrumentation it is possible to record the electroencephalogram, electrocardiogram, cortical evoked response, peripheral nerve conduction velocity, body temperature, respiratory rate and blood pressure. In order to determine the latter it was necessary to develop an arterial cannulation system which would not interfere with cerebral circulation, would permit continuous measurement of blood pressure, removal of small amounts of blood for determination of carboxyhemoglobin, oxyhemoglobin, etc., and remain patent for days or weeks (Fig. 3).[1] Finally, it was necessary to develop a measure of behavior which would require a level of nervous system integration essential for escape from a noxious stimulus. A simple avoidance behavior, withdrawal of the hind foot from an electrified plate, was selected. This behavior is learned uniformly in 10 to 15 minutes by virtually all rats. Its performance during exposure does not interfere with physiological monitoring. A number of psychological phenomena, including learning, recent and immediate recall and other operant-conditioned behavior can be investigated.

## THE EXPOSURE CHAMBER

It was also necessary to develop an exposure chamber which would provide uniform exposure to various concentrations of noxious gases. The chamber consists of an acrylic box with inside dimensions of 9" x 9" x 12", open at both ends. Funnel projections from the ends facilitate streamlined airflow through the chamber. One funnel is open into room air and the other is attached to an exhaust fan. Carbon monoxide is led into the open-ended funnel as room air is drawn through by the exhaust fan. A micro-flowmeter permits exact adjustment of carbon monoxide flow into the intake system. A pressure drop and baffle system interfaces the open-ended funnel with the central portion of the chamber. This promotes more complete mixing of carbon monoxide with incoming room air and an even distribution of carbon monoxide throughout the chamber. The entire system is air-tight with the exception of

the open-ended funnel through which room air is drawn. Numerous ports provide entry for electrical connections needed for physiological monitoring. The entire chamber is housed within an outer metal chamber, which is lined with sound- and vibration-attenuating foam rubber. For most experiments carbon monoxide concentration inside the chamber is 2500 to 3000 ppm, as determined by gas chromatography and a gas cell infrared carbon monoxide analyzer (Fig. 4).

## RESULTS AND DISCUSSION

### Levels of Intoxication

#### Level 1

This level is characterized by ataxia, reduced attentiveness and reduced exploratory behavior in rats. Most investigations in man have concentrated upon subtle changes in such measures as manual dexterity, visual evoked response,[2] etc., which are produced at levels of carboxyhemoglobin below 20%. Symptoms of headache, nausea, fatigue, somnolence and blurred vision may occur. Survival at this level is dependent upon the limitation of performance, which may threaten life. The driver of an automobile or operator of machinery may lose control at this level. Complex warning signs of fire may be ignored. Individual susceptibility to anoxia may be such that cardiac arrhythmia or convulsions may be produced even at this level. Taking into account all of the performance measures which are affected at this level, carboxyhemoglobin from 20% to 40% is estimated to exist at Level 1. Despite an impairment of psychomotor control, avoidance behavior is still possible at this level, i.e., it is still possible to escape.

#### Level 2

At 40% to 50% carboxyhemoglobin (mean 45.9%) avoidance behavior is lost. In Fig. 5 the mean and range of carboxyhemoglobin at the moment avoidance is lost is shown from experiments carried out on fifteen rats. Loss of avoidance is abrupt, with the animal readily responding to each shock with leg withdrawal. The level at which avoidance is lost may be shifted upward slightly if the animal is presented the stimulus continuously during exposure, and downward if no stimulus is applied during exposure, until carboxyhemoglobin values reach 30% to 40%. These results imply a greater effect of anoxia upon recent memory, as compared to immediate memory.[3]

From a functional standpoint the animal behaves as if decorticate. It is unable to integrate sensory input and provide an appropriate response which is based on previous experience. Despite the presence of warning signals and a clear exit, a man under similar circumstances would not initiate survival behavior.

The role of previous training must also be considered. A simple avoidance task such as the one used here consists of a simple flexion withdrawal of the leg, which requires integration only at the level of the

spinal cord. In other words, this protective response is inherent in the neuronal circuitry controlling the flexor muscles of the hind limb. The learned behavior consists of maintaining the state of flexion in anticipation of receiving a shock. This learned behavior may be more resistant to hypoxic insult when it is stored in remote rather than recent memory. For example, rats trained daily to perform this response over a period of several weeks may be more resistant to the effects of hypoxia than those trained for the 10 to 15 minutes prior to exposure. As indicated above, immediate memory of the avoidance response has a somewhat higher resistance to hypoxia than does memory.

The response to electrical stimulation, maintenance of postural tone and ambulation, normal peripheral nerve conduction velocity and electroencephalogram indicate that the cause of the failure to respond is not based upon failure in the sensory or motor systems *per se*, but in the assimilation of new information and its integration with past experience. Recent memory loss would seem to be the essential defect responsible for loss of avoidance behavior.

Level 3

As intoxication progresses there is gradual loss and finally disappearance of antigravity postural tone. This is accompanied by failure to withdraw the limb from an electric shock applied to the foot, and, of course, all movement to avoid a noxious stimulus is lost. Evaluation of conduction in the ventral caudal nerve reveals an abrupt fall in maximal velocity in both motor and sensory components of the nerve. Motor conduction is determined in the usual manner, by stimulating at two points along the nerve while recording the evoked muscle action potential. The distance over which conduction velocity is calculated is 10 cm. The evoked nerve action potential, conducted also over 10 cm, is utilized to determine sensory nerve conduction time (Fig. 6). Maximal motor velocity is unaffected until carboxyhemoglobin reaches 65 ± s.d. 5% carboxyhemoglobin (Fig. 6). At this point the response to proximal stimulation is lost. The relatively long conduction distance (10 cm) and possible involvement of the conductance properties at the nodes of Ranvier results in failure of the nerve to propagate an impulse. The long ventral caudal nerve is probably unique in this respect. At 2500 ppm CO loss of proximal conduction requires an exposure time of 80 ± 24 minutes. The evoked interosseus muscle potential, produced on stimulation at the distal point on the nerve, remains, although it is reduced to amplitude and polyphasic.[4]

Repetitive stimulation of 3, 10 and 30 Hz reveals a significant decrement in peak-to-peak amplitude. A gradual decrease in amplitude occurs, which indicates impairment of neuromuscular transmission. During the loss of proximal conduction the peak-to-peak amplitude in response to single shocks fluctuates in a step-wise manner, suggesting progressive loss of the action potential contribution by individual motor units.

It must be kept in mind that the exposure to 2500 ppm CO results in an equilibrium carboxyhemoglobin ranging from 65% to 70%. As can be seen in Fig. 6, animals attain this level before losing proximal conduction. A period of time at this level is required for conduction to disappear altogether. Time and the intensity of exposure must be taken into account. Exposure conditions were selected to allow for separation of the various levels of intoxication. At higher concentrations of ambient CO anoxic shock might develop very rapidly prior to involvement of ventral caudal nerve.

In summary, at Level 3 motor collapse occurs. There is evidence, at least in ventral caudal nerve, that this is associated with impairment of axonal conduction and a concomitant impairment of neuromuscular transmission. In our experiments this occurred at 65 ± 5% carboxyhemoglobin, but we do not know what the actual time-intensity relationship is for this function. We also do not know whether or not conduction is lost in larger and more proximal nerves, such as the sciatic. Very likely such nerves are more resistant to hypoxia.

Level 4

Following immediately upon the appearance of motor collapse is the development of anoxic shock. Up to this point there has been a gradual lowering of blood pressure, cardiac output, respiratory rate and tidal volume.[5] Slowing of the basic rhythm of the electroencephalogram has also occurred. The cortical evoked response to light flash is still present. The appearance of an isoelectric electroencephalogram, a drop in blood pressure below 50 to 60 mm of mercury, gasping respirations, bradycardia and ventricular arrhythmia can occur at any moment when carboxyhemoglobin exceeds 70%. It is unknown under the conditions of this experiment whether cerebral ischemia occurs at the moment the electroencephalogram becomes isoelectric. The duration of cerebral ischemia is of vital importance to the re-establishment of cerebral function.[6] The loss of the cortical evoked response occurs several minutes after the electroencephalogram becomes isoelectric. The somatosensory response is slightly less resistant than the visual evoked response.[7] Again, we do not know whether the onset of cerebral ischemia is associated with loss of the cortical evoked response. The electroencephalogram and cortical evoked response have been recovered after periods of cerebral ischemia as long as one hour.[7] Immediate resuscitation of humans brought to this level of intoxication may result in apparent recovery of neurologic function.

Level 5

This is the lethal level of intoxication. The actual event of death may appear to be a convulsion, respiratory arrest or cardiac arrhythmia. Pathological findings are of little help in ascertaining the event. Pathological findings as determined by light microscopy in the central nervous system following acute anoxic death are so minimal that researchers studying this process must "torture" the system in order to induce readily visible changes. Such methods include tying off the carotid artery in an already hypoxic

animal and allowing this state to exist for a time sufficient to produce pathological change.[8] Significantly more refinement in the approach to the investigation of the effects of anoxia is required. Since carbon monoxide produces a kind of "selective" damage to the central nervous system, animals may be kept at a given stage of intoxication for a time sufficient to produce pathological change.

## LONG-TERM EFFECTS—MORBIDITY

Survival must also be defined in terms of morbidity. Brief exposure to carbon monoxide sufficient to produce motor collapse (Level 3) results in subsequent impairment of the ability of rats to learn an avoidance response.[3]

Two-thirds of rats brought to Level 3 do not recover nerve conduction on the day of exposure. In the affected rats return of conduction requires from two to eight days. Late demyelination of the central nervous system probably requires exposure sufficient to produce at least Level 3 and perhaps more consistently Level 4.[9] Both anoxic anoxia and carbon monoxide-induced anoxia are sufficient to produce such effects. Critical limits for single acute, acute intermittent and chronic long-term exposure must be established. A variety of central nervous system disorders has been described following acute and chronic carbon monoxide intoxication.[10,11,12,13,14,15]

## CLINICAL APPLICATIONS

Carbon monoxide intoxication is often not diagnosed, despite the presence of suspicious circumstances, including fire. The classical findings of a rosy-red complexion depend upon a normal to high hemoglobin concentration, moderately high carboxyhemoglobin and a plethoric skin.[16] Carboxyhemoglobin determinations should be routine in the evaluation of undiagnosed coma and acute brain syndromes. The determination should be made, even when other causes seem obvious.

Tissue binding of carbon monoxide may alter the response to tissue injury. Carbon monoxide and other noxious gases may affect burn healing and be responsible for some of the phenomena, such as convulsions, which are now believed to result indirectly from the burn injury itself.[17]

## FACTORS MODIFYING RESPONSE TO CARBON MONOXIDE

Knowledge of the levels of carbon monoxide intoxication, with special attention to its behavioral effects, has permitted the detection of other intoxicants present in smoke. For example, motor collapse, coma and convulsions have been found in rats exposed to smoke produced during combustion of polyurethane foams to which brominated or phosphenated fire retardants have been added. Carboxyhemoglobin in all animals studied has been below 15%. For nonfire-retarded foams COHb levels were around 33% and no deaths or convulsions occurred. One can justifiably conclude that an additional intoxicant is active. The concept is emerging that, with respect to the toxicology of smoke, two categories of intoxicants are present: (1) those which produce a

clearly graded degree of intoxication and to which a human subject may become acclimated provided concentrations are low, such as hypoxia, carbon monoxide and $CO_2$; and (2) specific intoxicants, which, in very small concentrations, have special, sometimes lethal, effects and to which adaptation or acclimation is unlikely to occur. Based on knowledge of how the first category of intoxicants acts, it is possible to detect the presence of agents in the second group. Systematic analysis of their effects and a logical study of their chemical nature can then follow.

## COMMENT

Establishment of reference data for carbon monoxide, describing the time and intensity relations with respect to a given function, is essential in the investigation of the toxicology of smoke. We do not as yet know the critical time-intensity relationships for loss of avoidance behavior, motor collapse or anoxic shock. Exposure to very high concentrations of carbon monoxide may completely re-order the systemic hierarchy of sensitivity. These limits must be defined.

An ever-present question is the relevance of animal data to human survival. It is our contention, based on comparison of responses at lower ambient CO concentrations and a carboxyhemoglobin of less than 30%, that the essential difference between animal and man is the rate of blood and tissue equilibration with ambient CO. The rate of equilibration is primarily a function of metabolic rate. Where behavior can be roughly analogized, susceptibility is approximately the same. Animal studies will continue to be useful as a valuable bioassay of specific intoxicants in smoke.

## ACKNOWLEDGMENTS

This research, conducted at the Flammability Research Center of the University of Utah, has been supported by the National Science Foundation, Grant Number GI-33650, under its RANN (Research Applied to National Needs) program. The author is indebted to Dr. R. H. Long, Jr., the program monitor.

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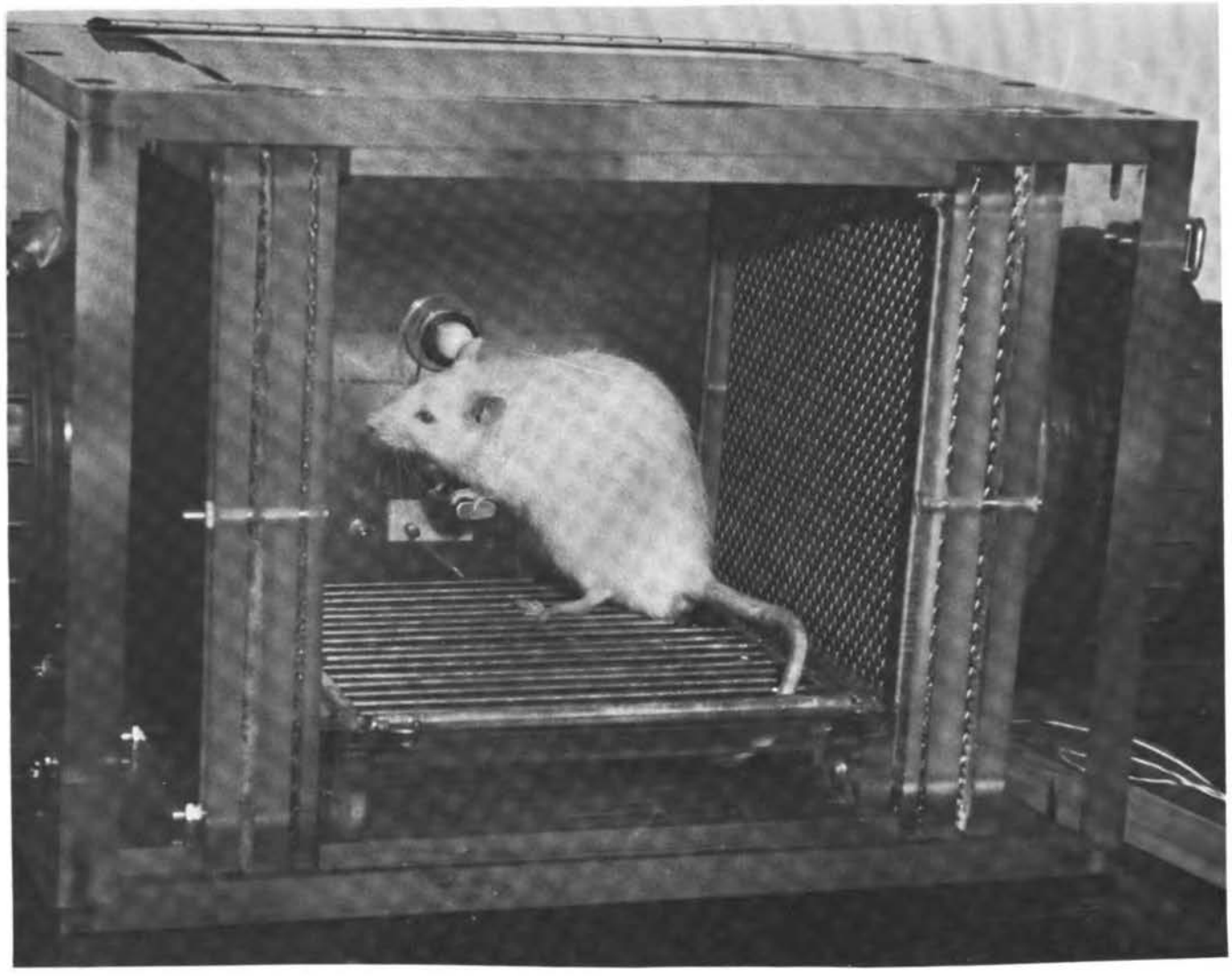

FIGURE 1. Modified Skinner box which serves as exposure chamber is shown. Failure of rat to depress manipulandum results in shock delivered through floor grid.

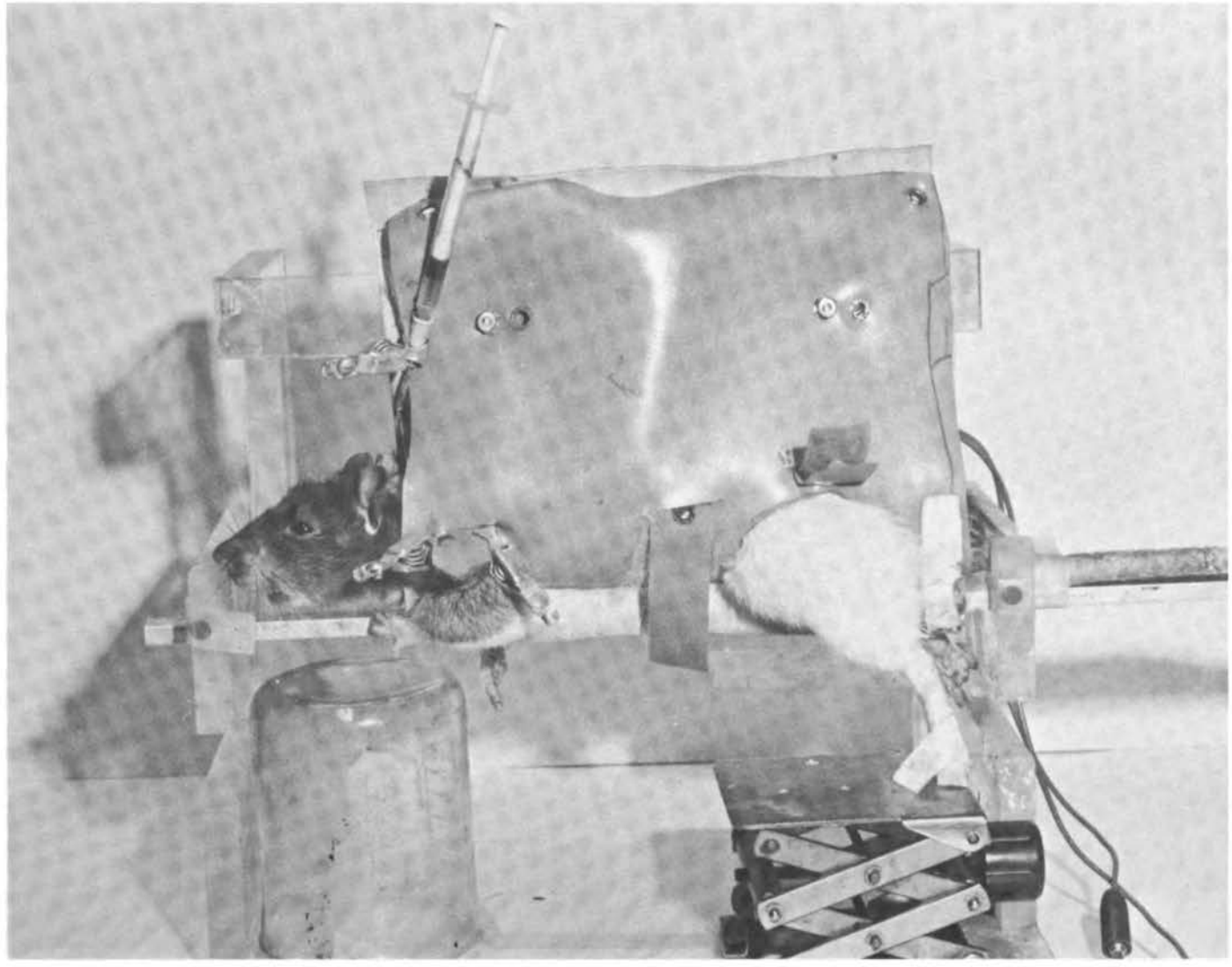

FIGURE 2. Rat mounted in sling and instrumented for exposure in chamber is shown.

FIGURE 3. Cannula is shown in place for sampling of arterial blood and measurement of blood pressure.

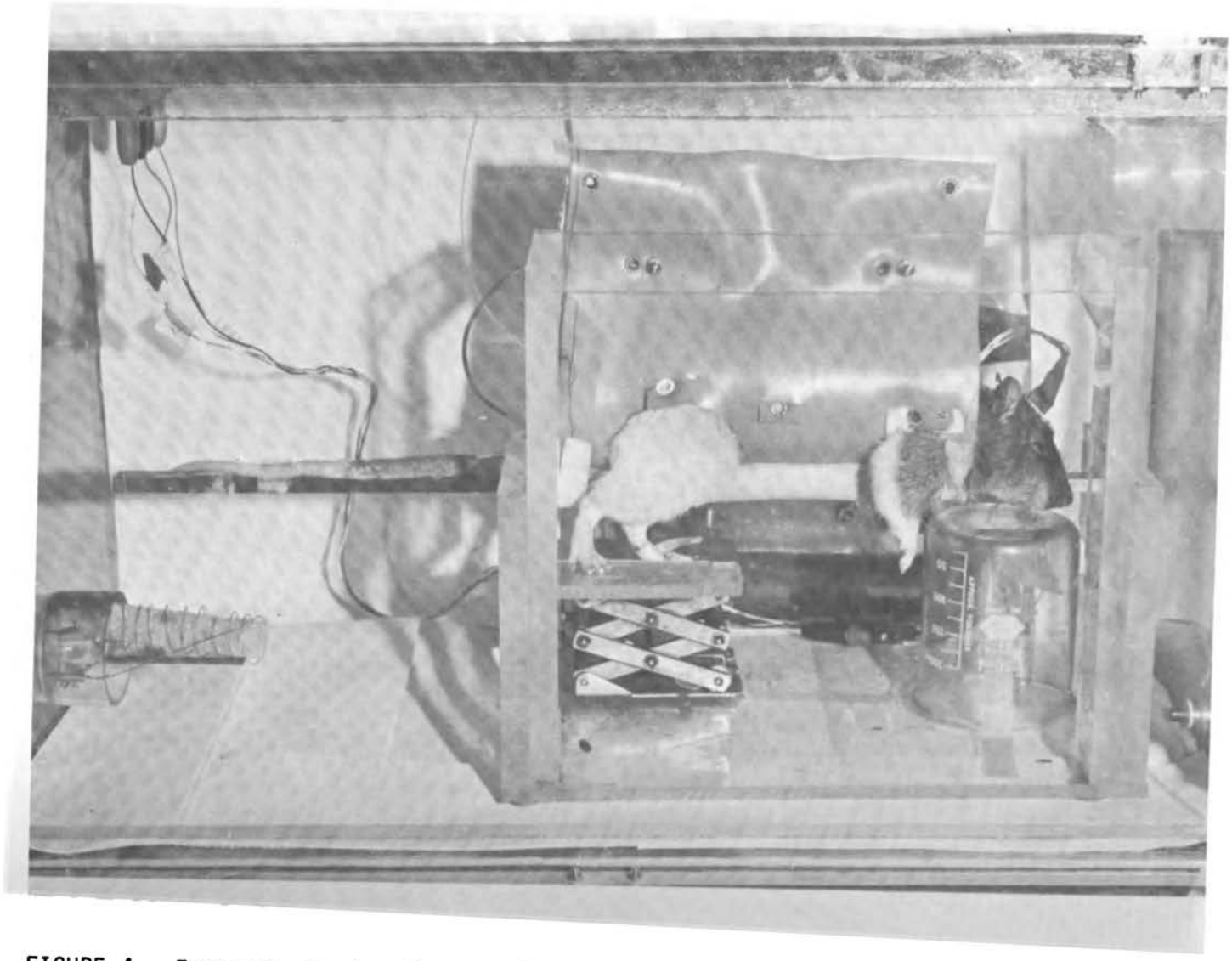

FIGURE 4. Exposure chamber for precise control of ambient atmosphere is shown.

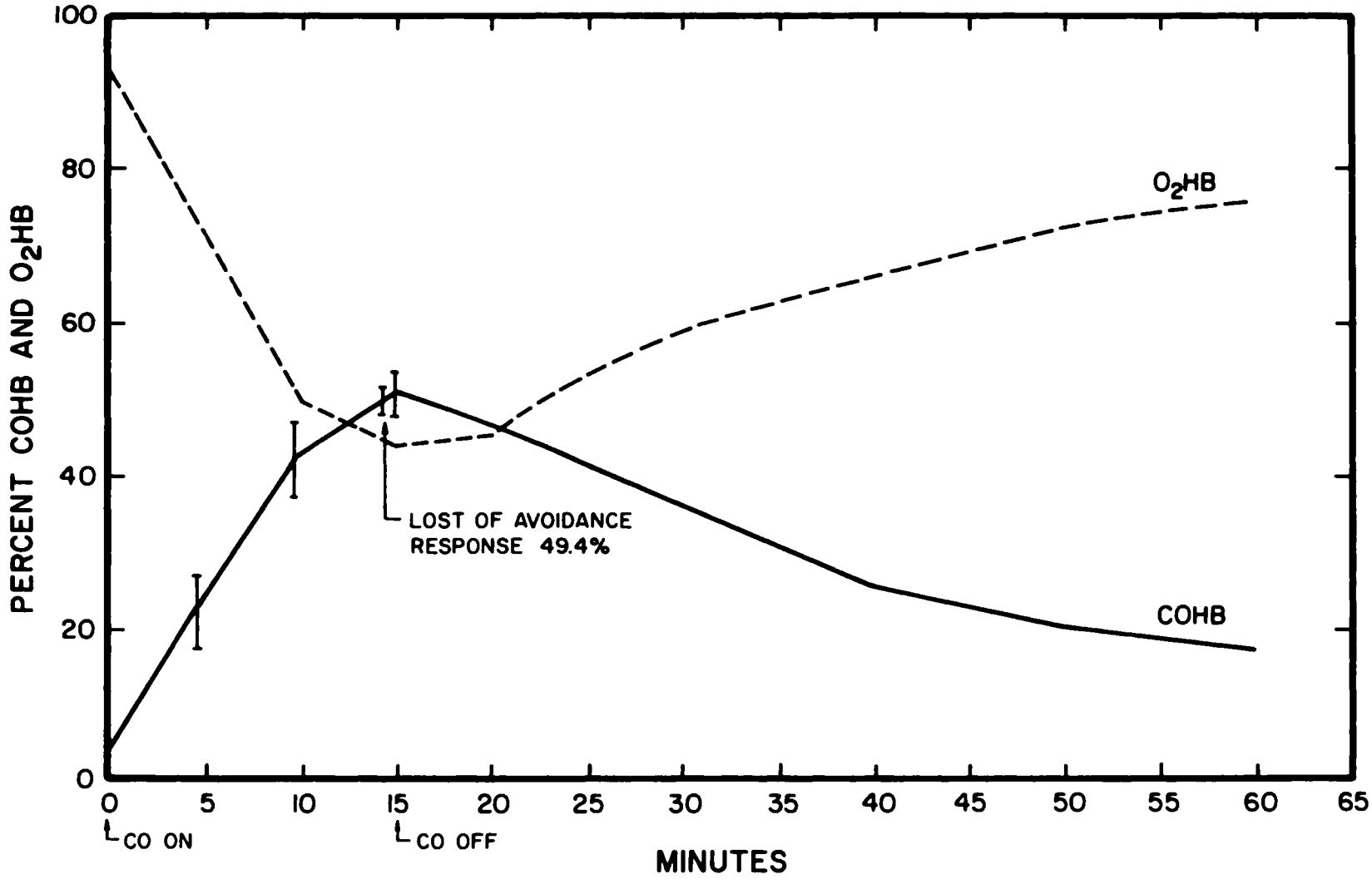


FIGURE 5. Mean percent carboxyhemoglobin and loss of avoidance response is shown versus time.

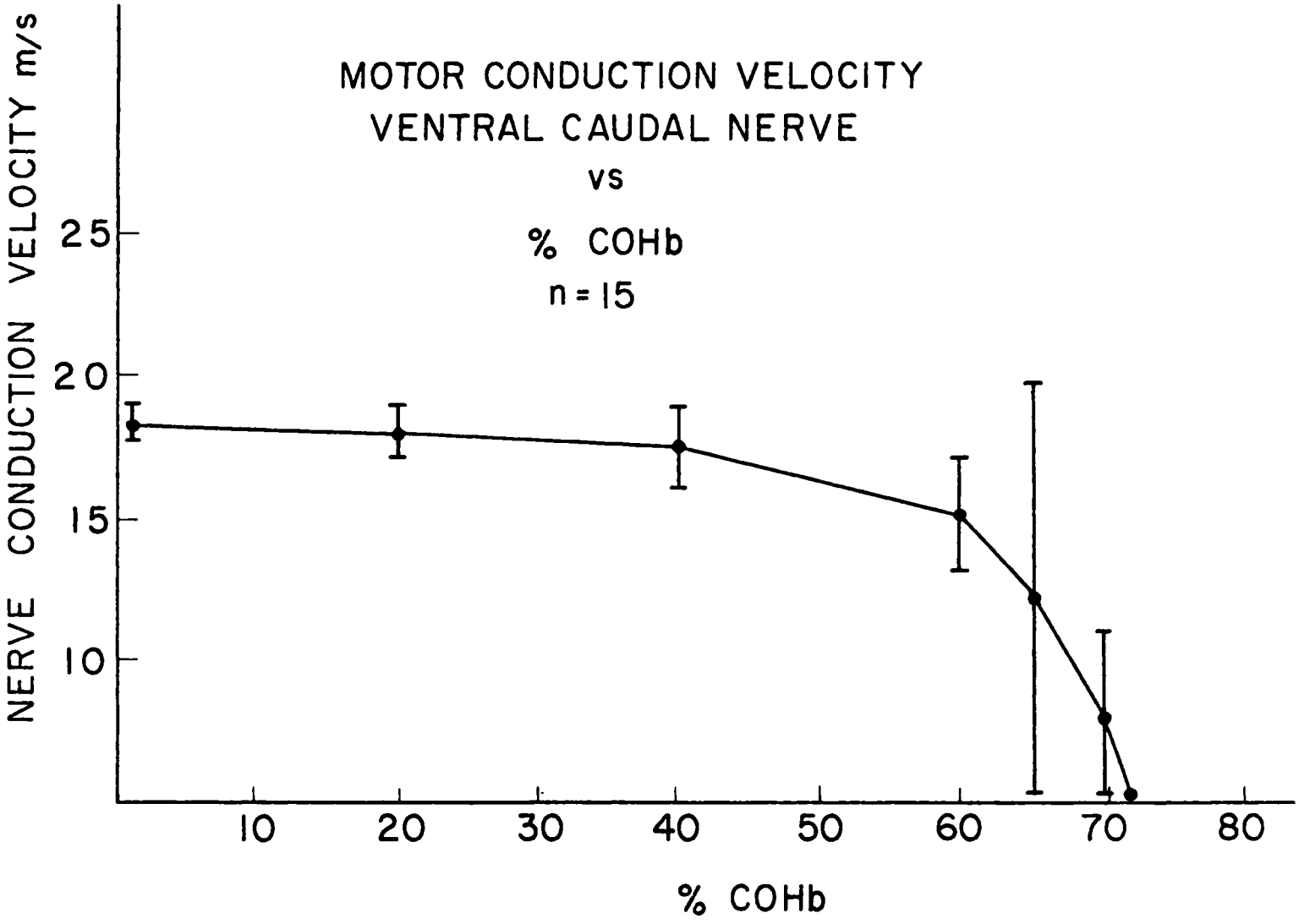


FIGURE 6. Mean and standard deviation of ventral caudal nerve conduction velocity is plotted versus percent carboxyhemoglobin.

# *LONG-TERM NERVOUS SYSTEM EFFECTS RESULTING FROM CARBON MONOXIDE EXPOSURE*

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When rats are exposed to the "anoxic shock" level of carbon monoxide intoxication they develop a carboxyhemoglobin level of from 60% to 80%. Clinically these animals show a decrease in blood pressure, decreased and irregular respiration, and cardiac arrhythmias. They also show a decrease in ventral caudal nerve conduction velocity, usually to zero. If removed from the CO environment at this time they survive. The ventral caudal nerve conduction velocity returns to normal in from two to eight days. Sensory conduction is most impaired. Although animals show no weakness during recovery, they are hypersensitive to stimuli.

Ventral caudal and peroneal nerves from rats exposed to the anoxic shock level of CO intoxication were examined 7, 10, 14, 21 and 28 days after exposure. Light microscopy of the nerves did not reveal any abnormalities to account for the decreased conduction velocity. However, with electron microscopy changes were seen at the node of Ranvier, both in large and small myelinated nerve fibers. The changes were more severe in large myelinated fibers than they were in small fibers.

The speed of progression of damage and repair depended on the size of the fiber and somewhat on the individual animal studied, but the steps in the progression appeared the same for small and large fibers. Most, though not all, nodes showed these changes.

Normally a thin sheet of cytoplasm from the Schwann cells on either side of the node or finger-like processes from the Schwann cells make contact at the center of the node. Each Schwann cell also sends finger-like processes vertically toward the axolemma (Fig. 1). After exposure to CO the major portions of cytoplasm from the Schwann cells from both sides of the node appear to retract and disengage. The many finger-like processes also appear to retract into the main mass of Schwann cell cytoplasm. The myelin terminals may remain attached to the axolemma in small fibers. Then myelin lamellae are stripped away from one another. The myelin terminals and adjacent myelin were completely or partially destroyed in many large fibers, with myelin figures appearing in the Schwann cell cytoplasm and axoplasm. The first-formed myelin terminals were sometimes preserved (Fig. 2). At the time of greatest

destruction the axon was frequently covered by only a thin sheath of Schwann cell cytoplasm and basement membrane or basement membrane alone. The axoplasm at this point was swollen. In larger diameter fibers microtubules and neurofilaments were disrupted in this area as well (Fig. 3). Large sized fibers were most severely damaged.

From seven to ten days after exposure the process of destruction seemed almost complete in large myelinated fibers but was still going on in smaller fibers. The beginning of reparative changes was late in larger diameter fibers, still being incomplete at 28 days. In smaller nerve fibers repair was often beginning by 14 days, though it too was not complete at 28 days. The first step in repair appeared to be swelling of the Schwann cell cytoplasm or production of processes to cover the bare node (Fig. 4). From 14 to 28 days the finger-like projections of Schwann cell cytoplasm began to reappear at the node (Fig. 5). Complete joining of thin layers of Schwann cell cytoplasm or finger-like processes from either side of the node overlying vertically-oriented finger-like processes was not often seen (Fig. 6). When joining was seen there was often reduplication of Schwann cell basement membrane over the node. The total process of reconstitution of destroyed myelin terminals and Schwann cell structures at the node was not observed. The node was rarely completely repaired by 28 days post exposure. In smaller fibers which showed less nodal damage myelin terminals tended to appear normal, with some myelin debris in the Schwann cell cytoplasm, but nodes still appeared elongated. Repair of myelin at node occurred after Schwann cell repair. Nerves from exposed rats before 7 and after 28 days must be examined before the entire process is clear. Only a few macrophages or inflammatory cells were seen in any nerves.

At seven days after exposure to CO Schwann cells showed some dilatation of smooth and rough endoplasmic reticulum with increased density and occasional vacuolization of mitochondria. These changes are more striking at 14 days postexposure, at which time evidence of protein synthesis within the dilated cysternae of the rough endoplasmic reticulum was seen (Fig. 7). These changes could still be seen 28 days after exposure.

Following exposure to CO a few unmyelinated axons exhibited swelling and loss of internal structure, but most appeared normal.

The changes described for carbon monoxide neuropathy are much like the early changes produced by intraneural injection of diphtheria toxin in rats, as described by Allt.[1] It, thus, appears as an early segmental demyelination.

It may be asked what these lesions in the peripheral nerves of the rat have to do with the nervous system changes produced by CO in the human. Peripheral neuropathies occur in humans following CO exposure but are thought to be rare.[2,3] However, they may be more common than is thought, since the neuropathy may be mild and fleeting, with central nervous system manifestation so severe that the neuropathy is overlooked. Also, rats rarely showed severe clinical evidence of neuropathy, so affected humans might not notice any neurological deficit.

The most common human consequences of CO poisoning are seen in the central nervous system if the patient survives long enough. This consists of loss of neurons, astrocytosis and sometimes necrosis of the globus pallidus, cerebral cortex, hippocampus and Purkinje cells of the cerebellum. But there is variation in pathology not completely related to the degree or length of exposure to CO. About 10% of patients who survive show "pseudo-recovery."[4] That is, they appear to awaken from coma and normalize, only to deteriorate mentally and neurologically weeks or months later. This is usually due to delayed demyelination and necrosis of the central nervous system white matter.

Recently we encountered a 21-year-old patient who was exposed to CO for an unknown period of time to an unknown COHb level. He was admitted to the hospital in a comatose condition and remained in coma for almost a month. Following this he began to regain function. Approximately a year after his admission he was able to return to college, although he had residual spasticity of the lower limbs, some inappropriateness and emotional lability. He drowned while scuba diving 18 months after admission. At autopsy the brain showed evidence of possible loss of neurons only in the globus pallidus, with other grey structures appearing intact. There was loss of myelinated fibers from the long tracts of the spinal cord and active demyelination of the cerebral white matter. The cause of the demyelination in CO poisoning is unknown, though it is well known that cyanide and hypoxia can also produce necrosis of white matter in primates and humans.[5,6,7,8,9]

In our experiments we have studied the brains and spinal cords of rats exposed to the anoxic shock level of CO intoxication at various times after exposure by light microscopy only. Six animals were studied seven days after exposure, two animals 14 days after exposure, two animals 21 days after exposure, nine animals 28 days after exposure and four animals two months after exposure. There was no apparent loss of neurons from the central nervous system grey matter or demyelination or necrosis of white matter in any animal. However, exposed rats did show difficulty in learning a conditioned response 30 days after exposure when compared with control rats. This is evidence that CO probably produced some central nervous system damage.

Recently Miyagishi has shown electron microscopic lesions in the cells of the white and grey matter of rats exposed acutely and chronically to CO.[10] These consisted of marked dilatation of the endoplasmic reticulum of the oligodendroglia, the central nervous system equivalent of the Schwann cell. Abnormalities of some myelin sheaths were also noted. Neurons showed some fragmentation or dilatation of rough endoplasmic reticulum and golgi but no mitochondrial changes. We are, therefore, about to begin an ultrastructural study of changes in central nervous system neurons and white matter in rats at various intervals after exposure to the anoxic shock level of CO intoxication.

ACKNOWLEDGMENTS

This research, conducted at the Flammability Research Center of the University of Utah and the University of Utah Medical Center, has been supported by the National Science Foundation, Grant Number GI-33650, under its

RANN (Research Applied to National Needs) program. The author is indebted to Dr. R. H. Long, Jr., the program monitor.

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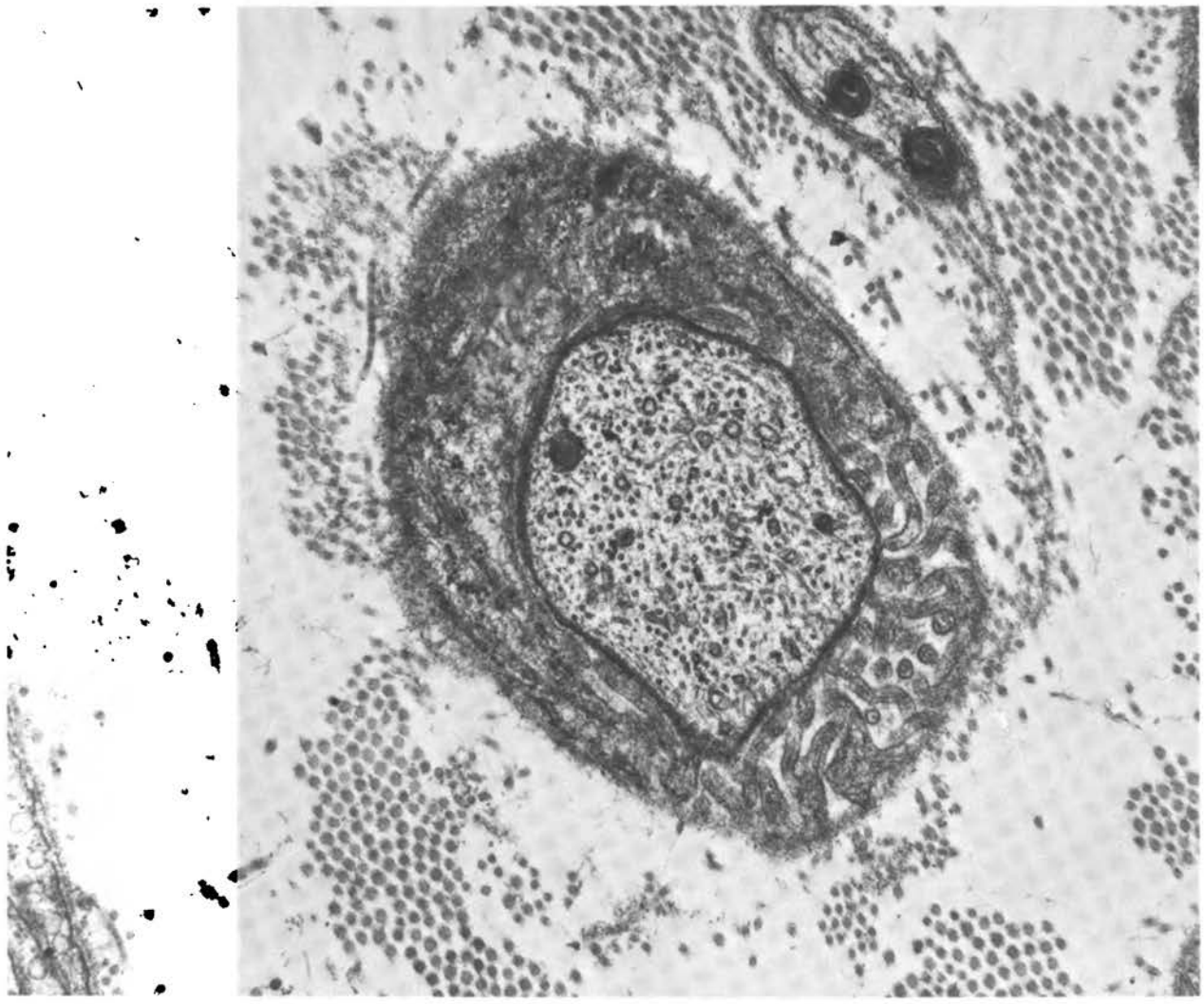

FIGURE 1. Normal node of Ranvier showing finger-like processes of Schwann cells extending toward the axoplasm (cross section X 16,000).

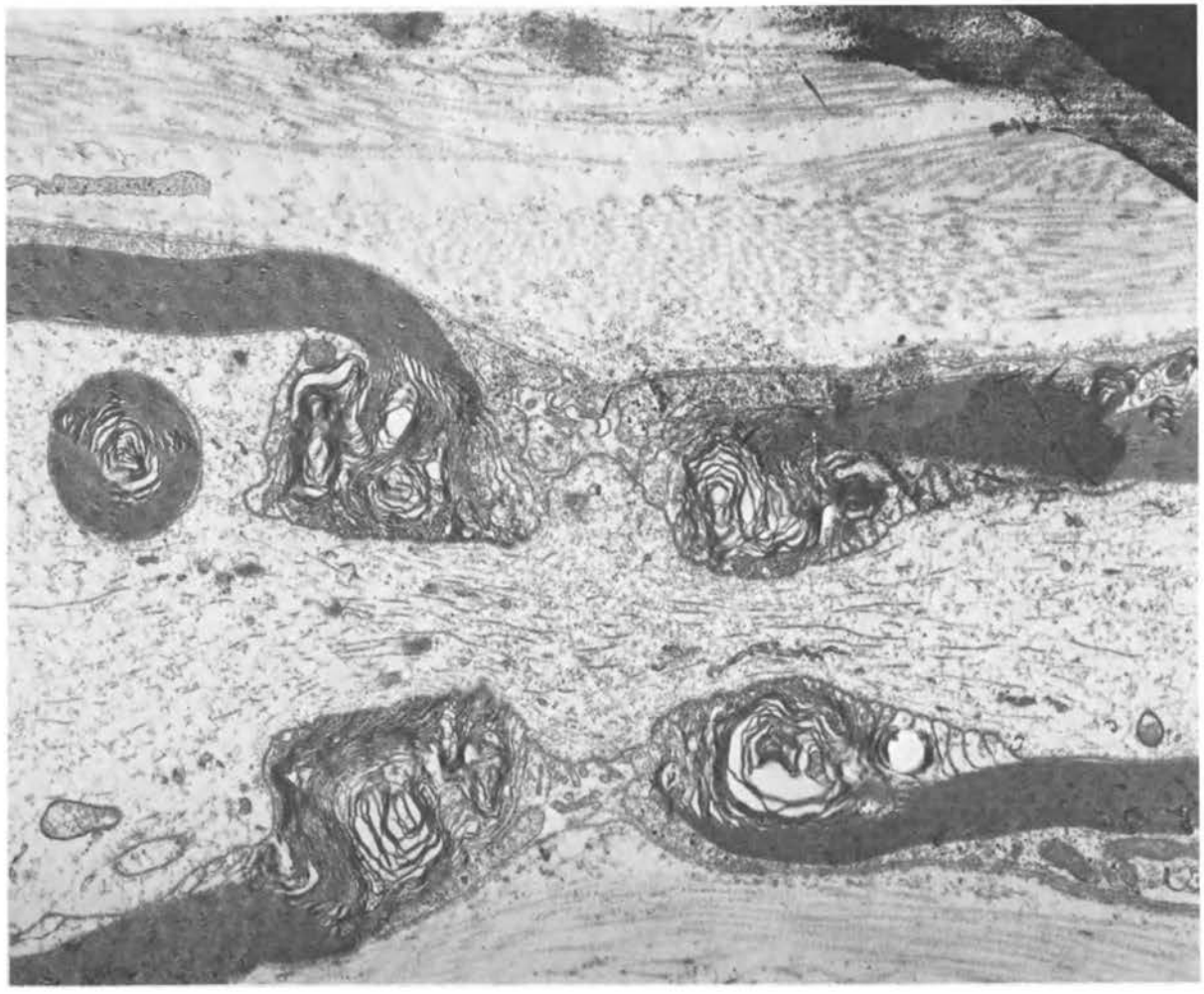

FIGURE 2. Node of Ranvier seven days after exposure to anoxic shock level of CO poisoning showing myelin degeneration and retraction of myelin terminals and Schwann cell cytoplasm and processes (longitudinal section X 6,000).

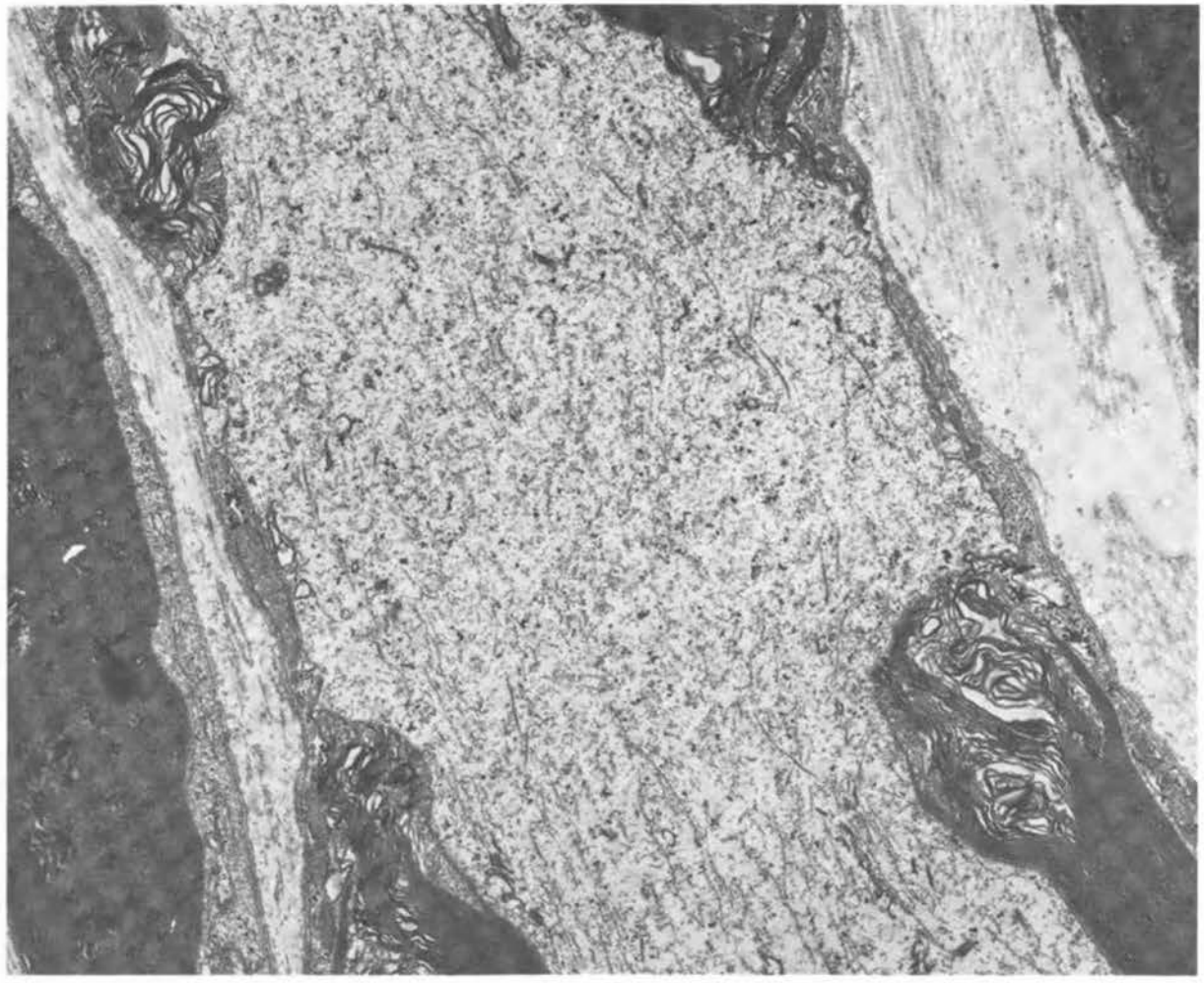

FIGURE 3. Node of Ranvier seven days after exposure to anoxic shock level of CO poisoning, with more severe changes showing loss of Schwann cell processes, swelling of axon and loss of covering of axoplasm (longitudinal section X 6,000).

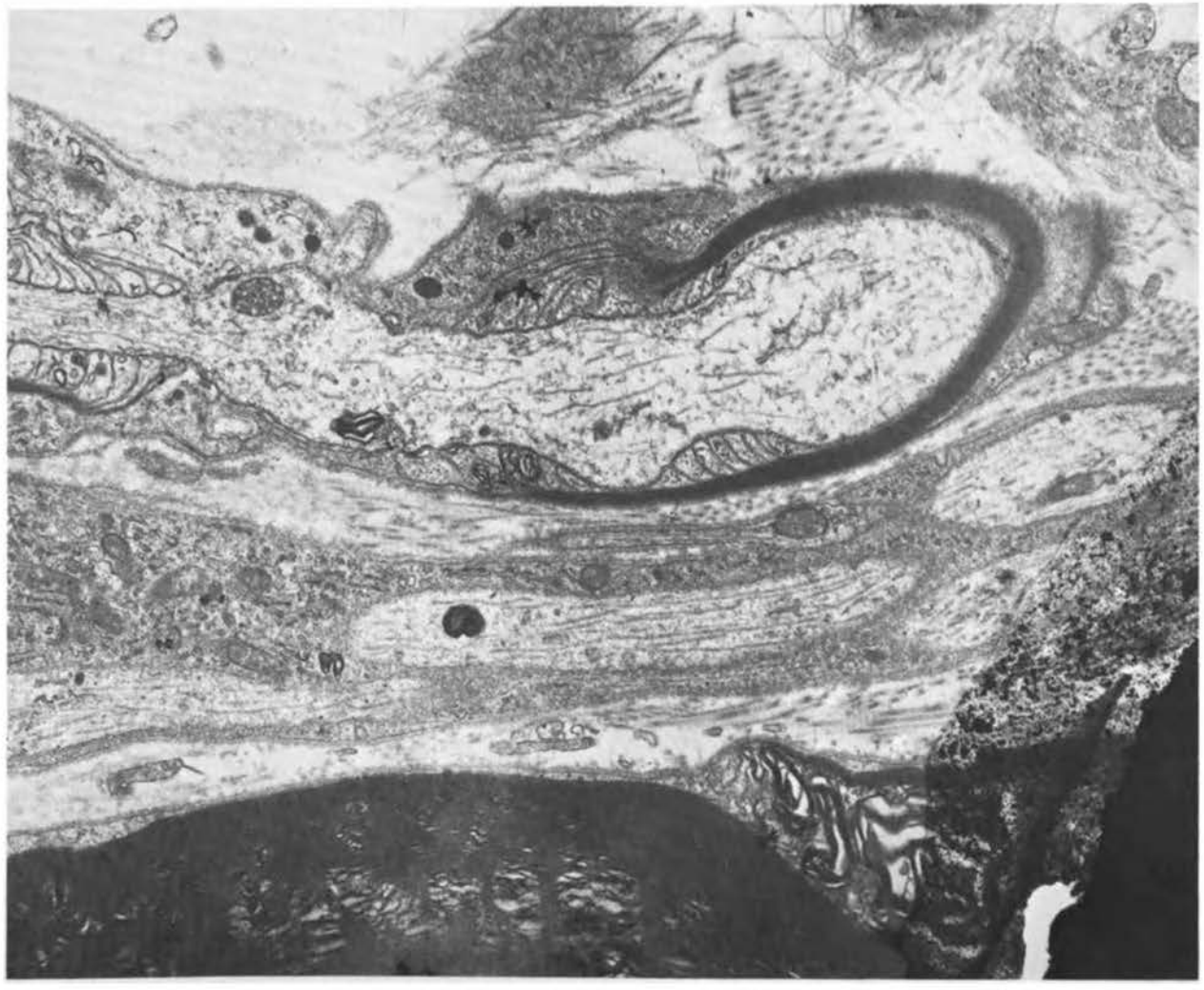

FIGURE 4. Node of Ranview 14 days after exposure to the anoxic shock level of CO poisoning, showing elongation of node and covering of the axon by swollen Schwann cell processes (longitudinal X 6,000).

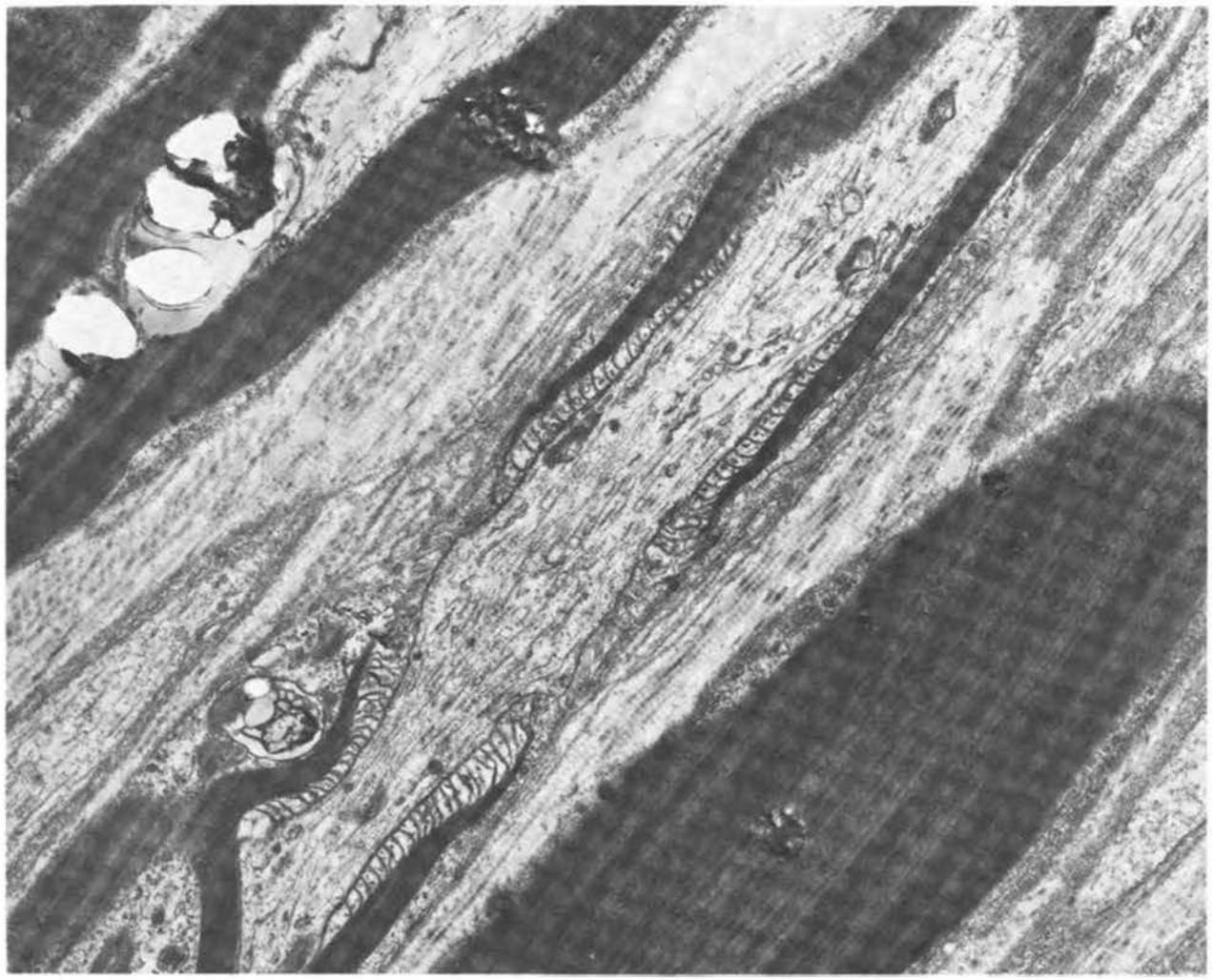

FIGURE 5. Node of Ranvier 21 days after exposure to the anoxic shock level of CO poisoning, showing return of finger-like processes, but an elongated node and myelin debris in the Schwann cell (longitudinal X 6,000).

FIGURE 6. Node of Ranvier 28 days after exposure to the anoxic shock level of CO poisoning, showing nearly normal Schwann cell processes, though myelin debris still remains (longitudinal X 6,000).

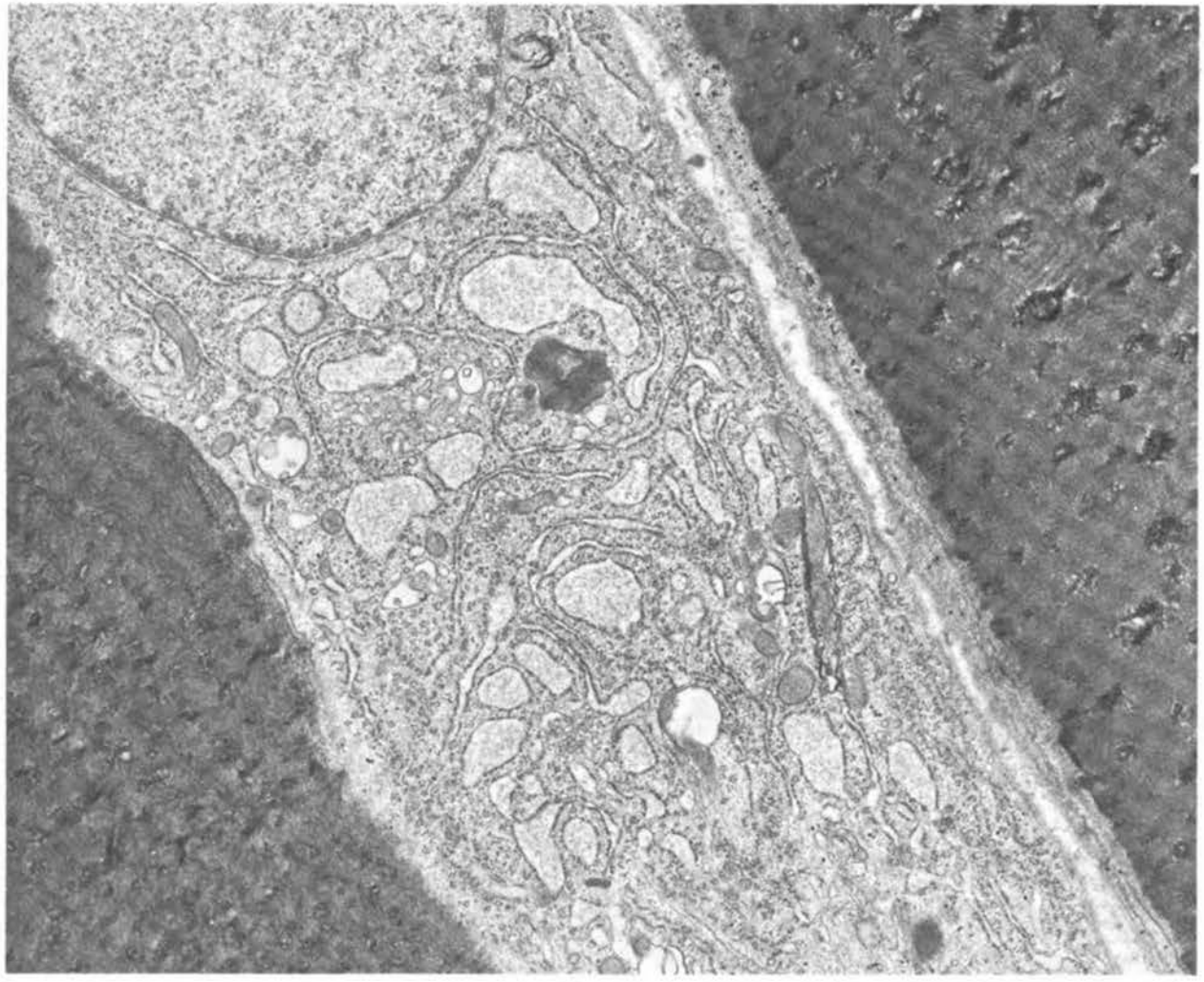

FIGURE 7. Schwann cell cytoplasm 14 days after exposure to anoxic shock level of CO poisoning, showing marked dilatation of cisternae of rough endoplasmic reticulum with evidence of protein synthesis (longitudinal X 6,000).

# *METHODOLOGY FOR TOXICOLOGICAL ANALYSES OF COMBUSTION PRODUCTS*

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Hazard due to fire is a complex phenomenon and the combined effect of many factors. The major factors causing death or serious incapacity of persons are compiled in Table I.

In actual fires, combustion or pyrolysis of single materials is seldom encountered. Thus, the toxicity potential of the total hazard is determined by the complex resultant of heat, anoxia, smoke, and gases or vapors. From these toxic factors combination effects can arise which can be additive, synergistic or antagonistic.

It is understandable that it is nearly impossible in a real fire situation to ascertain which agents may be responsible for hazards or deaths.

This paper is limited to the toxicity of gases and vapors derived by the pyrolysis of synthetic polymeric and some conventional materials. It could be shown that the toxicological characterization and the way of action of many important pyrolysis products are insufficient. Therefore it is difficult to calculate the toxicity based on two or more factors. These are problems in which all people concerned with the environment and determining allowable concentrations in air are involved. It would be unscientific to estimate the effects of two or more biological factors. Only animal tests can shed light on these problems by addition or calculation.

In his excellent paper "Toxicological Aspects of Flammability and Combustion of Polymeric Materials," John Autian[1] stated in 1970 that published information on toxicity tests conducted on thermal degradation products from burning materials is minimal, and this is still true in 1974. He also stressed that several different methods have been used to ascertain the toxic potential of thermodegradation products of a material obtained by fire or heat influence.

Being in agreement with Autian in this matter, I shall not report in detail about the laboratory experiments. For many reasons (for example, shape and weight of the materials tested, temperature of degradation, combustion equipment, air supply, exposure chamber and time, species of animals,

observation period, etc.) the test results found by different examiners cannot be compared with one another. But from a practical point of view, it is necessary to show that one material is more hazardous than another. If this would be possible, the products could then be classified in relation to their hazards.

Summaries of the results of animal experiments in most of the available papers were given in Autian's paper. In Table II I show the materials which have been tested up until now.

As mentioned before there are only a few data available which show the toxicity of the degradation products of materials by heating. Since we still know too little about the combined effect of several toxic substances occurring at the same time, data such as gas analyses alone are generally insufficient as a basis for drawing final conclusions as to the acute health hazards of fumes. It is also to be considered that the compounds produced by pyrolysis are dependent on the temperature. This is shown in plastic materials like polyvinyl chloride, polyurethanes and polyisocyanurate foam. For example, polyvinyl chloride yields more than 50 different gaseous compounds. However, as the temperature is increased, the number of gaseous species is rapidly diminished due to combustion. Thus, any reliable evaluation will only be possible via animal tests.

An appropriate way of assessing the hazards presented by the pyrolysis products of a synthetic material is to compare their toxic effects with that of the combustion products formed by a conventional material. In such investigations, reference can be made to equal amounts by weight, or equal surface areas of the substances to be burned. For an evaluation of combustion processes the second approach seems to be more in line with actual application conditions.

To be able to compare the hazards of synthetic products with conventional materials it is necessary to have a method where the products can be tested under exactly the same conditions. This question was raised in the Federal Republic of Germany some years ago. It was agreed that only smoldering conditions are of interest. That means pyrolysis of materials in a lack of oxygen. The time of exposure should be 30 to 60 minutes. In agreement with the German Commission of Standards, an apparatus of this kind was developed (DIN-Draft 53 436 of August 1966). It is used for animal tests at Münster (University, Institute of Hygiene), at Aix-la-Chapelle (Technical University, Institute of Hygiene), at Hamburg (University, Institute of Hygiene), and in the Institute of Toxicology of the chemical firms BASF, Ludwigshafen, BAYER AG, Wuppertal, and HOECHST AG, Frankfurt. Although the apparatus is likely to require further modification until it finally conforms with the requirements for a specification, it could readily be used for the investigations (Fig. 1). The apparatus essentially consists of a fused silica tube with a length of at least 1,000 mm, an outside diameter of 40 mm, and a wall thickness of 2 mm. An annular electric oven tightly enclosing the tube is moved along the tube. Its inner diameter must not exceed that of the tube by more than 1 mm. The air maintaining the pyrolysis process is introduced into the tube via a

flowmeter. The speed of the annular oven and, hence, the burning rate of the material is specified at 10 mm per minute. The stove moves against the air stream. The temperature of the test, the constancy of the temperature, and the distribution of the temperature are determined by a thermoelement (NiCr-Ni). In this way the average effective temperature in the tube can be controlled.

So far, this method of generation of the pyrolysis products of the materials is unique. In Germany the toxicologists have now come to an agreement on the following criteria:

1. Shape and dimensions of the test sample,
2. Length of the test sample, which gives the time of exposure,
3. Air supply,
4. Number of different effective temperatures to be tested,
5. Type of exposure chamber,
6. Number of exposed rats,
7. Analytical determination of the pyrolysis products.

The dimensions of the test samples in the above-mentioned institutes were: length 300 and 1,000 mm, width 10, 15 and 25 mm, and the thickness 2 and 5 mm. Velocity of the movement of the stove was 10 or 20 mm per minute. Times of exposure of 30 and 60 minutes were used. The amount of air supply was 100 or 300 liters per hour. The amount of air mixed with the pyrolysis products for diluting and cooling was also not constant. Dynamic flow systems were used, but the design of the exposure chambers was quite different. In one case, total body exposures of the animals, with the possibility of crowding, were used. In another, exposure of animals by inhalation only. The number of rats exposed varied between 5 and 20.

From these examples and there are more, of course, you can see that there are many technical problems to be solved. Nevertheless, the test results of the German investigators appear to be more reliable than those obtained elsewhere.

The above-mentioned institutes working with this apparatus have published some of their results (Reploh and co-workers,[2] Hofmann and Oettel,[3] Effenberger[4]).

In our Institute of Toxicology we have carried out a number of animal tests to study the toxicity of pyrolysis products of various conventional and synthetic materials. The conditions of testing which we normally used are shown in Table III.

The exposure chamber was developed ten years ago and has undergone improvements from time to time.[5] It can be used to study the acute and chronic toxicity of gases, vapors, aerosols and dusts in small animals (mice, rats, hamsters, and guinea pigs). Twenty animals are exposed in each test. They are exposed only to inhalation. We have experience on chronic inhalation studies on rats exposed daily for two years. The mortality rate after two years' exposure was 5% to 8%.

Besides analyzing the concentrations of the most important gases, observing the response of the test animals, and determining the COHb concentration in the blood of the animals, we also evaluate the death rate of the animals. For comparing the toxicity of the pyrolysis products of synthetic and conventional materials we use the range of pyrolysis temperatures which lies between those where no deaths occurred and those where deaths did occur. This is called "the critical temperature range" in which mortality in animals can be expected.

The results of tests with synthetic polymers and some natural materials are given in the following Tables IV - XI. They contain only the test results obtained with temperatures of pyrolysis within the critical temperature range. In most cases three or four different temperatures are used.

In most of the samples of rigid polyurethane foams tested, no mortalities of the exposed rats were found at a pyrolysis temperature of 600°C when standardized samples in the form of strips were used (volume basis). By testing the samples on a weight basis mortalities occurred at 400 and 450°C. The toxicity of the pyrolysis products of rigid polyurethane foams can be due mainly to the concentration of carbon monoxide or hydrogen cyanide, or both (see Table IV).

In testing rigid polyisocyanurate foams on a volume basis the critical temperature range was between 500 and 600°C or higher than 600°C (Table V). The corresponding range on a weight basis was between 350 and 450°C (Table VI).

Table VII shows the results of samples of semirigid polyurethane foams. The critical temperature range was between 400 and higher than 600°C (by volume) and between 350 and 400°C (by weight). The toxicity of the pyrolysis products of these samples was mainly due to carbon monoxide concentrations in the air.

The toxicity of the pyrolysis products of rigid foams based on an unsaturated polyester resin combined with expanded glass pellets in rats is given in Table VIII. The deaths of the rats cannot be related to carbon monoxide or hydrogen cyanide.

The results with thermoplastic materials are given in Table IX. The toxicity of the pyrolysis products of ABS foam cannot be explained by the concentrations of carbon monoxide and hydrogen cyanide in the air. The damage of the lungs and the delayed deaths confirm this argument, also. The

deaths of the rats from pyrolyzed polycarbonate are clearly due to carbon monoxide.

In Table X the mortality caused by the pyrolysis products of wood was due to carbon monoxide, but in the case of cork other gases were responsible for the deaths.

As shown before, an appropriate way of assessing the hazard presented by the pyrolysis products of plastics is in comparing their toxic effects with that of the toxic products formed by a conventional material. The comparison should be carried out using equal surface areas which correspond to practical conditions. Table XI indicates the relative toxicities of the pyrolysis products of several polymers compared with conventional products. This comparison can be made because all the tests were carried out under the same conditions. They are reproducible. The results show without doubt, that the pyrolysis products of most of the synthetic materials tested have a lower relative toxicity than those of wood and cork. Some are in the same range.

Tables XII and XIII show the toxicity of flexible polyurethane foams and conventional materials. In the furniture application both the equal volume and the equal weight basis can be realistic depending on the way of application. The mortalities caused by the pyrolysis products of flexible polyurethane foams were mainly due to carbon monoxide and hydrogen cyanide. Carbon monoxide from heated coconut fibre and cotton killed rats, and carbon monoxide and hydrogen cyanide were responsible for the deaths from combustion and pyrolysis products of flocks. The most important gas liberated from wool is hydrogen cyanide. In view of the absence of hydrogen cyanide and the low or medium content of carboxyhemoglobin in the blood, the deaths of rats exposed to the pyrolysis products of rubberized hair, lint wool and latex foam must be related to other toxic gases.

Table XIV indicates the relative toxicities (by weight) of the pyrolysis products of the tested materials used by the furniture industry. The toxicity of the pyrolysis products of flexible polyurethane foams generally seems to be lower than of conventionally used materials.

To conclude, the usefulness of our method is demonstrated in Table XV. It shows the toxic effects of the pyrolysis products of wood chipboard produced with different binders.

The method described is being recommended in Germany for the classification of noncombustible building materials (Class A). But now an international agreement on testing the toxicity of pyrolysis products is required. If the tests should contain other toxicological criteria (like performance tests, electrocardiograms, physical activities of the animals, swimming test, Time of Useful Function (TOF), pathological examinations, etc.) it should be discussed.

Recently we have been using a swimming test. In our preliminary results we were able to find a relationship between various concentrations of

carbon monoxide in the air and the swimming times of exposed rats before drowning (Table XVI).

In summary, the German method may not be the optimal one, but at the moment it seems to be very practical.

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TABLE I

MAJOR FACTORS CAUSING DEATH IN A FIRE

1. Direct consumption by the fire (flame contact)
2. Very high temperatures
3. Oxygen deficiency
4. Presence of carbon monoxide
5. Presence of other toxic gases
6. Presence of smoke
7. Development of fear, shock, and panic
8. Secondary fire effects due to mechanical reasons (trauma, bone fractures, etc.)

TABLE II

POLYMERIC MATERIALS TESTED ON ANIMALS DURING PYROLYSIS AND COMBUSTION

| Fields of Application | Polymeric Materials | |
|---|---|---|
| Building materials<br>Spacecraft materials<br>Aircraft materials | Polyvinyl chloride<br>Polystyrene<br>Polyurethane foam<br>Polyisocyanurate foam<br>Epoxy resin<br>Polychloropren | Polytetrafluoroethylene<br>Polychlorotrifluoro-ethylene<br>Fluorinated copolymers<br>Phenolformaldehyde resin<br>Rubber latex |

TABLE III

CONDITIONS OF TESTING THE TOXICITY OF PYROLYSIS PRODUCTS OF MATERIALS ON RATS
(Institute of Toxicology, BAYER AG, Wuppertal)

| | |
|---|---|
| Apparatus of pyrolysis ....: | Din-Draft 53 436 |
| Test material (comparison by volume) .....: | Length 300 mm,* width 10 mm, thickness 5 mm |
| Test material (comparison by weight) .....: | Same shape as above, but the same weight (gram/cm) as the material of comparison (wood, wool, etc.) |
| Temperature of pyrolysis ..: | 200 to 600°C (differences of 50°C) |
| Air supply .....: | 100 liters per hour |
| Air mixed with the pyrolysis products .....: | 100 liters per hour |
| Exposure chamber .....: | 20 liters capacity |
| Time of exposure .....: | 30 minutes |
| Observation period .....: | 7 days, if necessary extending to 14 days |
| Animals per test.....: | 20 male Wistar rats |

*The total length is about 40 to 50 mm longer because the exposure of the rats starts when development of smoke is fully in progress.

TABLE IV

TOXICITY OF THE PYROLYSIS PRODUCTS OF RIGID POLYURETHANE FOAMS ON RATS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM) AND EQUAL WEIGHT (1.2 GRAMS PER 10 MM)

| Sample | Fire Retardant | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|---|
| | | | CO ppm | HCN ppm | | |
| per Volume | | | | | | |
| 1 | no | 600 | 1,100 | 60 | 34.2 | 0 |
| 2 | yes | 600 | 1,000 | 75 | 43.6 | 0 |
| 3 | yes | 550 | 1,500 | 100 | 45.2 | 0 |
| | | 600 | 1,800 | 150 | 55.0 | 2 |
| per Weight | | | | | | |
| 1 | no | 300 | 450 | 25 | 22.9 | 0 |
| | | 400 | 1,900 | 75 | 48.0 | 4 |
| 2 | yes | 350 | 1,000 | 50 | 36.1 | 0 |
| | | 400 | 2,200 | 100 | 59.8 | 11 |
| 3 | yes | 400 | 2,000 | 60 | 55.2 | 0 |
| | | 450 | 4,400 | 100 | 62.4 | 20 |

TABLE V

TOXICITY OF THE PYROLYSIS PRODUCTS OF RIGID POLYISOCYANURATE FOAMS ON RATS. TEST WITH EQUAL VOLUME (300 BY 10 BY 5 MM)

| Sample | Fire Retardant | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|---|
| | | | CO ppm | HCN ppm | | |
| 1 | no | 500 | 1,100 | 150 | 43.5 | 0 |
| | | 550 | 3,000 | 150 | 58.9 | 13 |
| 2 | yes | 550 | 1,600 | 130 | 43.3 | 0 |
| | | 600 | 2,000 | 150 | 52.3 | 9 |
| 3 | yes | 600 | 1,200 | 75 | 32.8 | 0 |
| 4 | yes | 500 | 470 | 25 | 17.6 | 0 |
| | | 600 | 1,230 | 100 | 44.7 | 3 |
| 5 | yes | 500 | 500 | 45 | 29.6 | 0 |
| | | 550 | 1,300 | 120 | 47.6 | 11 |
| 6* | yes | 500 | 930 | 55 | 27.9 | 0 |
| | | 600 | 1,670 | 125 | 48.5 | 1 |

*Sample of rigid polyisocyanurate foam reinforced with foamed glass pellets.

TABLE VI

TOXICITY OF THE PYROLYSIS PRODUCTS OF RIGID POLYISOCYANURATE FOAMS ON RATS. TESTS WITH EQUAL WEIGHT (1.2 G PER 10 MM)

| Sample | Fire Retardant | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|---|
| | | | CO ppm | HCN ppm | | |
| 1 | no | 400 | 1,000 | 50 | 33.6 | 0 |
| | | 450 | 2,800 | 150 | 58.2 | 19 |
| 2 | yes | 350 | 1,100 | 50 | 23.7 | 0 |
| | | 400 | 3,500 | 150 | 39.2 | 15 |
| 3 | yes | 350 | 800 | 50 | 19.4 | 0 |
| | | 400 | 2,500 | 150 | 39.2 | 14 |
| 4 | yes | 300 | 450 | 10 | 17.0 | 0 |
| | | 350 | 1,470 | 45 | 42.6 | 4 |
| 5 | yes | 300 | 300 | 10 | 12.5 | 0 |
| | | 400 | 2,150 | 150 | 52.6 | 12 |

TABLE VII

TOXICITY OF THE PYROLYSIS PRODUCTS OF SEMIRIGID POLYURETHANE FOAMS ON RATS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM) AND EQUAL WEIGHT (1.2 GRAMS PER 10 MM)

| Sample | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|
| | | CO ppm | HCN ppm | | |
| per Volume | | | | | |
| 1 | 400 | 1,000 | 65 | 41.7 | 0 |
| | 500 | 1,400 | 150 | 57.6 | 4 |
| 2 | 600 | 3,000 | 80 | 62.0 | 0 |
| 3 | 500 | 1,600 | 90 | 49.7 | 0 |
| | 600 | 2,500 | 100 | 56.0 | 1 |
| per Weight | | | | | |
| 1 | 350 | 3,000 | 70 | 63.2 | 0 |
| | 400 | 7,000 | 150 | 71.2 | 20 |
| 2 | 350 | 1,300 | 10 | 34.0 | 0 |
| | 400 | 3,500 | 75 | 68.9 | 6 |
| 3 | 350 | 2,000 | 70 | 61.2 | 0 |
| | 400 | 6,600 | 100 | 71.5 | 9 |

TABLE VIII

TOXICITY ON RATS OF THE PYROLYSIS PRODUCTS OF RIGID FOAMS BASED ON AN UNSATURATED POLYESTER RESIN COMBINED WITH EXPANDED GLASS PELLETS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM) AND EQUAL WEIGHT (1.2 GRAMS PER 10 MM)

| Sample | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|
| | | CO ppm | HCN ppm | | |
| per Volume | | | | | |
| 1 | 600 | 1,830 | 100 | 39.4 | 0 |
| 2 | 600 | 3,000 | 50 | 57.9 | 0 |
| per Weight | | | | | |
| 1 | 300 | 280 | 0 | 8.2 | 0 |
| | 350 | 470 | 0 | 13.3 | 2 |
| 2 | 300 | 270 | 0 | 6.2 | 0 |
| | 400 | 900 | 0 | 23.8 | 5 |

TABLE IX

TOXICITY OF THE PYROLYSIS PRODUCTS OF THERMOPLASTIC MATERIALS ON RATS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM)

| Sample | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|
| | | CO ppm | HCN ppm | | |
| ABS | 350 | 150 | 100 | 5.8 | 0 |
| | 400 | 350 | 150 | 14.2 | 13 |
| ABS Foam | 350 | 100 | 20 | 6.2 | 0 |
| | 400 | 100 | 100 | 7.3 | 2 |
| Polycar-bonate | 500 | 3,500 | 0 | 44.0 | 0 |
| | 600 | 24,000 | 0 | 71.5 | 20 |

TABLE X

TOXICITY OF THE PYROLYSIS PRODUCTS OF SPRUCE WOOD AND CORK ON RATS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM)

| Sample | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|
| | | CO ppm | HCN ppm | | |
| Spruce wood | 300 | 1,000 | 0 | 27.6 | 0 |
| | 350 | 7,500 | 0 | 72.7 | 19 |
| Cork | 250 | 600 | 0 | 20.7 | 0 |
| | 300 | 1,000 | 10 | 21.5 | 2 |

TABLE XI

COMPARISON OF THE RELATIVE TOXICITIES OF THE PYROLYSIS PRODUCTS OF SYNTHETIC AND NATURAL BUILDING MATERIALS. TESTS WITH EQUAL VOLUME

| Materials | Lowest Temperature (°C) Indicating Mortality |
|---|---|
| Rigid Polyurethane Foam | 600 and >600 |
| Rigid Isocyanurate Foams | 550, 600, and >600 |
| Semirigid Polyurethane Foams | 500, 600, and >600 |
| Foams based on UP resins combined with expanded class pellets | >600 |
| ABS | 400 |
| Polycarbonate | 600 |
| Spruce wood | 350 |
| Cork | 300 |

TABLE XII

TOXICITY OF THE PYROLYSIS PRODUCTS OF FLEXIBLE POLYURETHANE FOAMS ON RATS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM) AND EQUAL WEIGHT (1.2 GRAMS PER 10 MM)

| Sample | Fire Retar-dant | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|---|
| | | | CO ppm | HCN ppm | | |
| **per Volume** | | | | | | |
| Polyether Foam | | | | | | |
| Sample 1 | no | 600 | 1,200 | 60 | 28.2 | 0 |
| Sample 2 | no | 300 | 1,500 | 28 | 40.3 | 0 |
| | | 400 | 3,930 | 38 | 53.1 | 9 |
| Sample 3 | yes | 300 | 1,500 | 15 | 41.3 | 0 |
| | | 400 | 2,200 | 38 | 48.5 | 9 |
| Polyester Foam | no | 600 | 300 | 15 | 13.7 | 0 |
| High Resilience Foam | no | 400 | 1,250 | 25 | 33.5 | 0 |
| | | 500 | 2,100 | 138 | 60.2 | 15 |
| **per Weight** | | | | | | |
| Polyether Foam | | | | | | |
| Sample 1 | no | 250 | 180 | 10 | 7.7 | 0 |
| | | 300 | 3,000 | 25 | 62.5 | 5 |
| Sample 2 | no | 300 | 1,500 | 28 | 40.3 | 0 |
| | | 400 | 3,930 | 38 | 53.1 | 9 |
| Sample 3 | yes | 300 | 1,500 | 15 | 41.3 | 0 |
| | | 400 | 2,200 | 38 | 48.5 | 9 |
| Polyester Foam* | no | 450 | 900 | 35 | 31.8 | 0 |
| High Resilience Foam | no | 400 | 1,250 | 25 | 33.5 | 0 |
| | | 500 | 2,100 | 138 | 60.2 | 15 |

*Only tested up to 450°C.

TABLE XIII

TOXICITY OF PYROLYSIS PRODUCTS OF CONVENTIONAL UPHOLSTERY MATERIALS ON RATS. TEST WITH EQUAL WEIGHT (1.2 GRAMS PER 10 MM)

| Sample | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|
| | | CO ppm | HCN ppm | | |
| Coconut Fibre | 200 | 20 | 0 | | 0 |
| | 250 | 6,500 | 0 | 48.7 | 8 |
| Rubberized Hair | 250 | 200 | 0 | 10.5 | 0 |
| | 300 | 2,000 | 10 | 41.5 | 5 |
| Latex Foam | 200 | 260 | 0 | 13.0 | 14 |
| Lint Wool | 250 | 2,930 | 0 | 56.8 | 0 |
| | 300 | 3,350 | 0 | 56.9 | 7 |
| Flocks | 250 | 1,460 | 85 | 36.0 | 0 |
| | 300 | 3,300 | 150 | 50.1 | 5 |
| Wool | 250 | 450 | 50 | 4.4 | 0 |
| | 300 | 600 | 150 | 6.6 | 3 |
| Cotton | 200 | 50 | 0 | 4.7 | 0 |
| | 250 | 8,000 | 10 | 69.0 | 11 |

TABLE XIV

COMPARISON OF THE RELATIVE TOXICITIES OF THE PYROLYSIS PRODUCTS OF SYNTHETIC AND NATURAL UPHOLSTERY MATERIALS. TESTS WITH EQUAL WEIGHT

| Materials | Lowest Temperature (°C) Indicating Mortality |
|---|---|
| Flexible Polyurethane Foam (Polyether) | 300 and 400 |
| Flexible Polyurethane Foam (Polyester) | >450 |
| High Resilience Foam | 500 |
| Coconut Fibre | 250 |
| Rubberized Hair | 300 |
| Latex Foam | <200 |
| Flocks | 300 |
| Lint Wool | 300 |
| Wool | 300 |
| Cotton | 250 |

TABLE XV

TOXICITY OF THE PYROLYSIS PRODUCTS OF WOOD CHIPBOARD PRODUCED WITH DIFFERENT BINDERS ON RATS. TESTS WITH EQUAL VOLUME (300 BY 10 BY 5 MM)

| Sample | Binder | Temp. °C | Concentration in Air | | COHb % | Number of Deaths out of 20 |
|---|---|---|---|---|---|---|
| | | | CO ppm | HCN ppm | | |
| 1 | Urea Formaldehyde | 200 | 1,300 | 13 | 26.5 | 0 |
| | | 250 | 4,200 | 42 | 55.3 | 6 |
| 2 | Phenol Formaldehyde | 200 | 4,200 | 1 | 44.8 | 0 |
| | | 250 | 6,300 | 3 | 70.4 | 19 |
| 3 | Phenol Formaldehyde Isocyanate | 200 | 900 | 1 | 3.6 | 0 |
| | | 250 | 4,200 | 5 | 54.8 | 4 |
| 4 | Isocyanate | 250 | 3,700 | 8 | 49.8 | 0 |
| | | 300 | 10,000 | 28 | 70.7 | 20 |

TABLE XVI

EVALUATION OF THE SWIMMING TIME OF RATS EXPOSED FOR 30 MINUTES TO VARIOUS CARBON MONOXIDE CONCENTRATIONS IN AIR

| Carbon Monoxide Concentration in Air ppm | Swimming Times min. |
|---|---|
| 0 | 50.0 |
| 1,000 | 28.2 |
| 2,000 | 21.6 |
| 3,000 | 10.5 |

Explanations of products being described in:

Table IV

The rigid polyurethane foams tested are commercial products.

Sample 1: foam based on polyfunctional polyols and polymeric MDI, not flame-retarded.

Sample 2: foam based on polyfunctional polyols with reactive flame-retarding agent, and on polymeric MDI.

Sample 3: rigid foam based in trifunctional polyols with nonreactive flame-retarding agent, and on polymeric MDI.

Blowing agent: fluorotrichloromethane.

Tables V and VI

The polyisocyanurate foams are development products which were formulated in one case without, and in the others with, an addition of solid or liquid, nonreactive flame-retarding agents.

Blowing agent: fluorotrichloromethane.

Table VII

The semirigid foam systems tested are products based on long-chain bi- and trifunctional polyols with an addition of short-chain crosslinking agents, and on polymeric MDI.

Blowing agent: Samples 1 and 2: carbon dioxide.
Sample 3: fluorotrichloromethane.

Table XII

Samples 1 and 2: foams based on a long-chain polyether and TDI.

Sample 3: same as samples 1 and 2, but with an addition of a nonreactive flame-retarding agent.

Sample 4: (polyester foam) foam based on a long-chain polyester and TDI.

Sample 5: (high-resilience foam) foam based on a trifunctional long-chain polyether and modified TDI.

Blowing agent: used in all cases, carbon dioxide.

Table XV

Sample 3: isocyanate (polymeric MDI) as binder in the central layer of the particle board and phenol formaldehyde resin in the skin zones.

Sample 4: polymeric MDI.

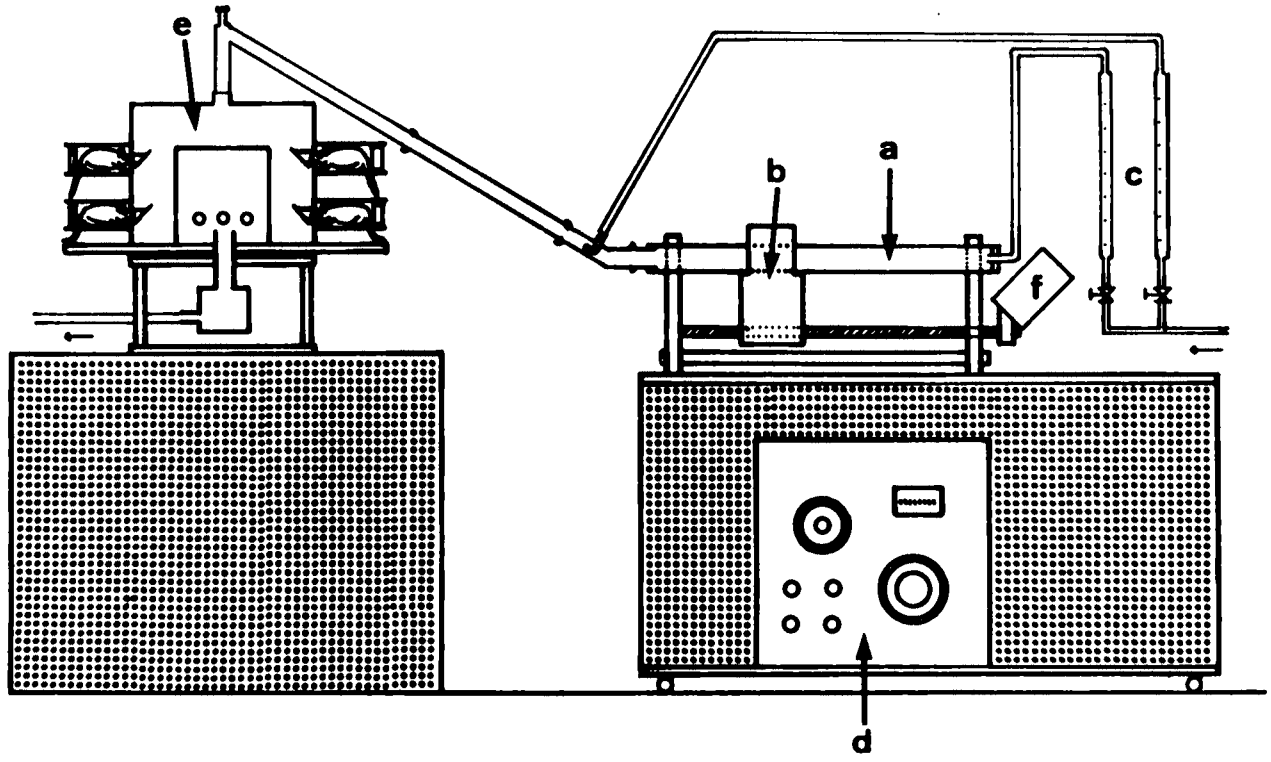


FIGURE 1. Schematic Diagram of the Apparatus for Pyrolysis and Inhalation Chamber.

a - Silica tube
b - Electric oven
c - Rotameter
d - Electric device
e - Exposure chamber
f - Motor

# *USE OF ANIMALS IN EXPERIMENTS TO PREDICT HUMAN RESPONSE*

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From the standpoint of toxicology, the greatest immediate threat to victims exposed to fire is represented by the smoke and combustion products formed during the burning of a variety of materials. In some circumstances, however, there can exist a threat from inhalation of substances other than combustion products, e.g., inhalation of substances used to combat the fire. A number of halogen-containing compounds are being developed as components of fire extinguishers and fire-suppressing apparatus and release of these substances contributes to the total toxicologic hazard faced by either victims or persons involved in combating the fire itself. In fact, as more efficient fire-fighting systems evolve, it is possible that exposure to fire extinguishers and suppressants and their combustion products will constitute more of a hazard than that posed by exposure to combustion products indigenous to the fire itself. In a study of eight fluorinated hydrocarbon compounds that were possible fire extinguishers, Chambers *et al.* (1950) reported that the pyrolysis products of six compounds were more lethal to rats exposed for 15 minutes to atmospheres containing them than the original compounds, in one case by nearly 79 times.

Depending on the nature of the facility to be protected and the type of fire-combating material employed, there are at least two situations representing rather different toxicologic problems. An acute danger could ensue from the deployment, particularly in a closed space, of a halogenated substance emitted by a fire extinguisher. This could occur in aircraft cockpits or cabins, compartments of ships, warehouses, etc. On the other hand, chronic exposure could follow the use of a halogenated fire suppressant, incorporated into the atmosphere of a vessel, such as a spacecraft, to prevent even the initiation of fire. In this situation, exposure could be continuous for several weeks or even several months.

Both of these problems in toxicology are under investigation in our laboratories and some of the halocarbons we have investigated are shown in Table I. With the exception of Freon C-318 ($C_4F_8$), all are fluorinated methanes or ethanes. Despite the obvious structural similarities, they produce a variety of pharmacologic effects in laboratory animals ranging from violent convulsions and death following exposure to Halon 1202 ($CBr_2F_2$) to anesthesia induced by Halothane ($CHBrCl\text{-}CF_3$) which is, in fact, a widely used

anesthetic mentioned here for comparison purposes only. Others induce myocardial sensitization to catecholamines, e.g., Freon 12 ($CCl_2F_2$), while others such as Freon 116 ($C_2F_6$), affect biological systems only subtly if at all. In general, the observations we have made have been in accordance with the generally accepted principle relating the chemical constitution with the toxicity of many of the fluoroalkanes, namely a lower toxicity is associated with an increasing number of fluorine atoms in the molecule (Clayton, 1966). The low order of toxicity in highly fluorinated alkanes is a reflection of low-chemical reactivity and low-biological activity.

Experiments with laboratory animals have shown also that physical chemical characteristics of halocarbons can influence biological activity. For example, blood levels of gaseous compounds such as Halon 1301 ($CBrF_3$) and Freon 116 ($C_2F_6$) decrease rapidly during the first few minutes after exposure is terminated (Fig. 1). Exposure to a less volatile material, such as Halon 2402 ($CBr_2F_4$), which is liquid at room temperature, leaves substantial residues in the blood (and other tissues) even several hours after the exposure is terminated (Table II).

One of our objectives has been to determine the extent to which toxicologic studies in laboratory animals can aid in the selection of halocarbons suitable for long-term continuous exposure in humans inhabiting closed atmospheres containing substantial quantities of a halocarbon. To aid in the selection, a variety of halocarbons were first studied in short-term exposures to disclose which were the least acutely toxic. The results of such studies have been reported elsewhere (Griffin *et al.*,1972). One compound, Freon 116, showed a remarkable lack of biological activity in these acute investigations and long-term, continuous exposures were initiated. Freon 116 has also been shown to be potentially useful as a fire suppressant (Huggett, 1973). In our acute studies, Freon 116 was well tolerated by rats and rabbits. No immediate toxic effects were observed even when the concentration of Freon 116 was raised to 20% (v/v) for periods up to one hour. With this background and with the goal of obtaining definitive data during more prolonged exposure, an experiment was devised in which rats would be exposed to the fluoralkane almost continuously for an extended period. We planned an atmospheric concentration of 20% (v/v), an effective concentration of Freon 116 for use as an inerting gas. A concentrated multidisciplinary approach was taken because only a limited number of animals would be available.

Hematological and biochemical serum examinations were selected with a two-fold purpose in mind. In the first place, we wanted data on the general health and clinical status of the animals and this was obtained through a battery of tests performed at the termination of the exposure. Secondly, some selected tests were incorporated with the hope of showing specific sensitivity to exposure to the halocarbon. Osmotic fragility studies were employed to demonstrate whether the fluorocarbon had any effect on the integrity of the erythrocyte membrane. It was hoped that studies of enzyme activities within serum would disclose liver or other tissue damage.

Preliminary studies employing acute exposures had not revealed lability of the carbon-fluorine bond as determined by measurements of concentrations of fluoride in samples of urine obtained after the exposures. This approach to studying the metabolism of Freon 116 was employed in the present experiment also. We felt also that, since excess fluoride accumulates in bone, a study of that tissue was necessary and would provide a more sensitive indicator of metabolism. Fluoride levels in bone were studied at the termination of the experiment.

Previous biochemical studies had shown that acute exposure to Freon 116 had no effect on mitochondria (Griffin *et al.*, 1972). Since then, the biochemical emphasis has been to determine whether perfluorinated hydrocarbons have any effect on the endoplasmic reticulum of the liver. Mixed function oxidase and cytochrome P-450 were measured to determine whether the Freon 116 would induce production of processing enzymes in the microsomes.

For exposure of the animals, a portable animal exposure chamber as described by Hinners *et al.* (1968) was modified to operate in a "closed dynamic" mode for control of the gaseous content. This manner of operation was utilized because of long-term exposure studies involving concentrations of Freon 116 of the order of 20% would necessitate the use of large quantities of the fluorocarbon if the open dynamic system of exposure were used. Such a system would not be economically feasible because of the high cost of the fluorocarbon; therefore, a recirculating system, based on that described by Clayton *et al.* (1960), permitting the recovery and reuse of Freon 116 was devised. In our variation of the closed dynamic system chamber gases were circulated through a bed of lithium hydroxide to remove carbon dioxide. For each mole of carbon dioxide removed, it was necessary to add back an equivalent amount of oxygen. This was accomplished by metering oxygen to the changer at a rate required to maintain the concentration at about 20%. Freon 116 was also metered to the chamber at the rate required to maintain a 20% by volume concentration.

The chamber was operated for 23 hours a day, seven days a week. The daily one hour down time was used for animal care and for cleaning. In order to prevent the buildup of ammonia and other odorous materials, strict standards of cleanliness were observed. Animal cages and the trays for collecting urine and feces were changed daily and the inside of the chamber was washed each day. Urine and any spillage from water bottles was absorbed on Pel-E-Cel (Paxton Products). In addition, boric acid crystals were sprinkled on the trays holding the absorbent to help prevent the formation of ammonia. Finally, the chamber was purged at a slow rate throughout the exposure period to help prevent the buildup of foreign materials in the atmosphere.

With the chamber operating in this manner, we determined that its size limited the exposure capability to about 12 rats; therefore, the experiment was started with that number of male Sprague-Dawley rats in the chamber. Twelve male animals housed outside the exposure chamber served as controls for the study. The exposure lasted for 37 weeks, or nearly nine months.

The concentrations of Freon 116, oxygen, and carbon dioxide measured during the study are shown in Fig. 2. The grand average of the weekly Freon 116 concentrations was 20.7% and the averages of those of oxygen and carbon dioxide were 20.1% and 0.39%, respectively. The system of exposure proved to be a suitable one for maintaining the gaseous concentrations at the desired levels, although there were some variations. Carbon dioxide concentrations were maintained well below the upper limit of 1% which would have been tolerated. Lesser variations in the concentration of $CO_2$ after the 15th week of exposure followed an improvement in the design of the lithium hydroxide scrubber. The temperature within the chamber reflected the temperature outside the chamber except that conditions were 2 to 3°F higher inside.

The general response of the animals to the exposure conditions is indicated by the body weights shown in Fig. 3. Although the weights were equivalent at the beginning of the experiment, growth of the exposed animals was retarded somewhat at the beginning of the exposure and again at about the 18th week of exposure.

Of the original 12 rats admitted to the chamber at the beginning of the exposure, eight survived until the experiment was terminated after 37 weeks of exposure. Two of the animals died during anesthesia for drawing blood samples from the orbital sinus. Two others died from causes which could not be determined from gross examination at autopsy. Autolysis prevented a definitive histopathological examination for determination of the cause of death. Of the twelve animals which entered the study as controls, only six survived, the others dying from respiratory infection.

The general health and well-being of the animals is reflected in the hematology data shown in Fig. 4. All the parameters examined were well within the normal range as determined in our laboratory, although there was some evidence of hemo-concentration in the exposed rats during the early stages of the experiment. This was indicated by slightly elevated hematocrits and erythrocyte counts in the exposed animals during the 18-week exposure period. However, after 37 weeks of exposure there were no differences between the exposed and the control groups. Total erythrocyte counts were well within the normal ranges at all times and there was no evidence that exposure to Freon 116 elicited a change in the osmotic fragility of erythrocytes. At the termination of the experiment, no differences were observed in the concentrations within serum of glucose, urea nitrogen, sodium or potassium (Table III).

With the exception of alkaline phosphatase activity, there were essentially no differences between the exposed and the control groups in the activities of those serum enzymes shown in Table IV. In the exposed group, the average alkaline phosphatase activity was slightly higher than that of the controls. This difference was statistically significant ($p<0.05$). LDH activities in the sera of the rats are shown in Fig. 5. Significant increases were observed in the exposed rats after 18 weeks of exposure, but this change was no longer present after 37 weeks of exposure to Freon 116.

The biochemical studies of enzymes and levels of cytochrome P-450 in liver (Fig. 6) did not show any evidence of the induction common following exposure to many chlorinated compounds.

Concentrations and total daily excretions of fluoride ion in the urines of the rats are shown in Fig. 7. Regardless of the manner of expression, the absolute values are slightly lower in the exposed group although there were no significant differences between exposed and control groups of animals. Fluoride ion in the bone of the animals, expressed as per cent of the bone ash, is also shown in Fig. 7. There was no significant difference between exposed and control groups.

## DISCUSSION

It has been pointed out that the low order of toxicity of highly fluorinated alkanes is a reflection of a low order of chemical reactivity and a low order of biological activity. The experiment described herein confirmed a low order of toxicity of a perfluoroalkane and demonstrated that in spite of the relatively high exposures over a long period of time and under almost continuous conditions of exposure, there was remarkably little response of any kind by the rats that could be attributed to the fluorocarbon. With the chamber containing 20% by volume of Freon 116, the animals were being exposed by the respiratory route to rather enormous quantities of the chemical. Taking the vapor density of Freon 116 to be 5.7 g/liter and the respiratory minute volume of the rats to be 0.073 liters/min (Altman and Dittmer, 1964), a conservative estimate for rats weighing 200g or more, it can be calculated that the animals were exposed to about 115g of Freon 116 per day. Certainly, not all was absorbed; in fact, we have shown (Griffin *et al.*, 1972) that rabbits undergoing exposure to 20% Freon 116 had a mean concentration of the Freon in their bloods of less than 1.0µg/g.

The small fluctuations in the activities of serum enzymes, and particularly the elevation of LDH activity during the initial phase of exposure, may have been related to factors other than exposure to the fluorocarbon. In our preliminary studies, in which groups of rats were exposed to fluoroalkanes continuously in the closed dynamic chamber for periods up to 30 days, and in control experiments, in which rats were confined to the chamber for 30 days but were not exposed to a fluoroalkane, elevated LDH activities in serum were often observed. In these experiments, we also noted that slight retardation of growth accompanied confinement of the rats to the chamber and thus did not necessarily result from exposure to a fluoroalkane. It is difficult to conclude what might be the cause of these apparent effects from confinement in the chamber. Their presence within the chamber did expose the rats to an atmosphere different from that of the control animals in more respects than just the presence of Freon 116. As noted above, the temperature was slightly higher and we also observed that the chamber air was often more humid than the air outside. Many closed, environmentally-controlled chambers incorporate activated charcoal scrubbers and catalytic burners to remove trace amounts of methane, ammonia, odors. etc., from chamber atmospheres, but we could not use such systems because of possible interactions with Freon 116. Hence, the slow

purge of chamber gases was adopted to maintain low levels of such materials, but, even so, minute quantities could still have been present. All of these factors taken together may account for some of the slight differences between control and exposed groups of rats.

Since Freon 116 is a nonpolar organic compound and soluble in many organic substances, it is conceivable that the material might be transported to membrane systems and there interact with the lipid components of such systems. It could be anticipated that incorporation of Freon 116 into membranes of cells or components of mixed function oxidase systems might change the integrities of cell membranes or the activities of cellular enzyme systems. If such an interaction occurred in the erythrocyte cell membrane, it was not expressed by a change in the osmotic fragility of the cells.

Decomposition of Freon 116 within the bodies of the rats was not detected by measuring the excretion of the fluoride ion in the urine. Excess fluoride ion accumulates in bone; our failure to find increased concentrations of fluoride in the skeletons of the exposed rats is even stronger evidence that breakage of the carbon-fluoride bond did not occur in these animals. Urinary concentrations of fluoride were low in both the exposed and the control groups and were lower in the exposed group, although by only a small margin; the observed differences were not statistically significant. However, it is interesting that the trend was lower in the exposed group; this may have been related to differences in dietary intakes of fluoride ion. The growth was slightly retarded in the exposed animals, which may have been due to a decreased consumption of food and a consequent decreased intake of fluoride ion.

Many gaseous hydrocarbons are known to sensitize the myocardium to the effect of catecholamines; although this phenomenon was not studied specifically in our experiment, it is worthy of mention. Studies of this effect of haloalkanes are under study in our laboratories, a preliminary investigation having showed only a low order of catecholamine-induced cardiac arrhythmia caused by Freon 116 (Wills *et al.*, 1972). A comprehensive study of cardiac arrhythmias induced by a variety of halogenated as well as nonhalogenated hydrocarbons was reported by Reinhardt *et al.* (1971), but Freon 116 was not included in this study. In a more recent communication from the same laboratory (Trochimowicz, 1972), Freon 116 at 20% v/v in air was reported not to induce cardiac arrhythmia in dogs. We plan exposures of rhesus monkeys in a study to be conducted in a manner similar to the one reported here for rats but to include also investigations of cardiac sensitivity.

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TABLE I

COMPOUNDS OF INTEREST

| | | |
|---|---|---|
| A. Substituted methanes | | |
| Halon 1301 | Bromotrifluoromethane | $CBrF_3$ |
| Halon 1202 | Dibromodifluoromethane | $CBr_2F_2$ |
| Freon 11 | Trichlorofluoromethane | $CCl_3F$ |
| Freon 12 | Dichlorodifluoromethane | $CCl_2F_2$ |
| B. Substituted ethanes | | |
| Freon 116 | Hexafluoroethane | $CF_3$-$CF_3$ |
| Freon 115 | Chloropentafluoroethane | $CClF_2$-$CF_3$ |
| Freon 114 | 1,2-Dichlorotetrafluoroethane | $CClF_2$-$CClF_2$ |
| Halon 2402 | 1,2-Dibromotetrafluoroethane | $CBrF_2$-$CBrF_2$ |
| Freon 113 | 1,1,2-Trichloro-1,2,2-trifluoroethane | $CCl_2F$-$CClF_2$ |
| Halothane | Bromochlorotrifluoroethane | $CHBrCl$-$CF_3$ |
| C. Other | | |
| Freon C-318 | Octafluorocyclobutane | $C_4F_8$ |

TABLE II

LEVELS OF HALON 2402 IN RAT TISSUE FOLLOWING EXPOSURE BY INHALATION

| Tissue | Post-inhalation Interval (hrs) | | | |
|---|---|---|---|---|
| | 0 | 1-1/2 | 3 | 24 |
| Liver | 258* | 5 | 2 | 0.28 |
| Lung | 44 | 18 | 2 | 0.18 |
| Brain | 0.70 | 2.1 | 0.78 | 0.36 |
| Kidney | 82 | 27 | 23 | 0.33 |
| Heart | 24 | 2.1 | 2 | 1.1 |
| Muscle | 73 | 19 | 2.8 | 1.0 |
| Fat | 365 | 469 | 410 | 11 |
| Blood | 87 | 7 | 0.23 | 0.22 |

*All values shown are in μg Halon 2402/g tissue.

TABLE III

SERUM CHEMISTRY

| | Control | Exposed |
|---|---|---|
| Glucose, mg/100ml | 131 ± 16 | 140 ± 18 |
| Urea Nitrogen, mg/100ml | 18 ± 2 | 16 ± 3 |
| Sodium, meq/l | 145 ± 2 | 144 ± 1 |
| Potassium, meq/l | 5.1 ± 0.2 | 5.3 ± 0.4 |

TABLE IV

SERUM ENZYMES

| | Control | Exposed |
|---|---|---|
| Alk. Phos, K-A units | 14.4 ± 5.9 | 24.0 ± 7.2 |
| Acid Phos, K-A units | 11.0 ± 2.0 | 11.1 ± 2.7 |
| GOT, S-F units | 65.0 ± 12.0 | 67.0 ± 21.0 |
| GPT, S-F units | 28.0 ± 12.0 | 23.0 ± 6.0 |
| CPK, units | 38.0 ± 24.0 | 32.0 ± 15.0 |

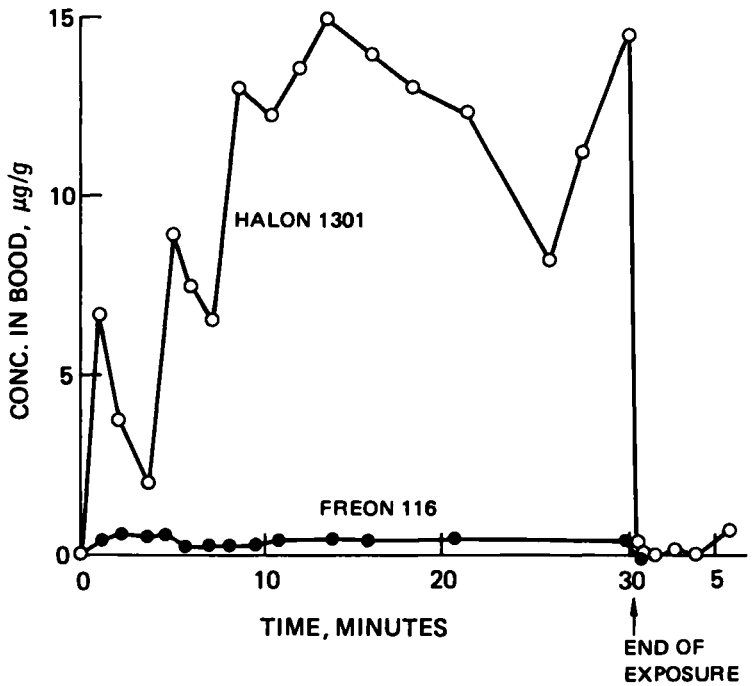


FIGURE 1. Halocarbons in Blood of Rabbits During 5% Atmospheric Exposures. Blood samples were withdrawn from the animals before, during and after exposures to either Halon 1301 (open circles) or Freon 116 (solid circles). Concentrations of the halocarbons in blood were determined by gas-liquid chromatography.

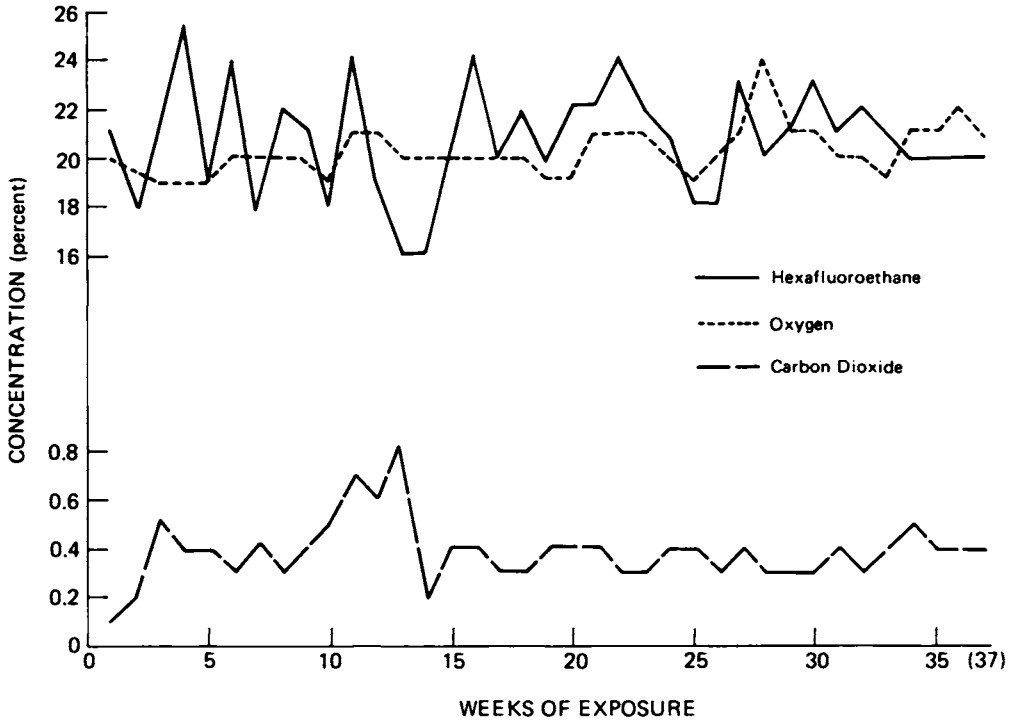


FIGURE 2. Conditions of Exposure.

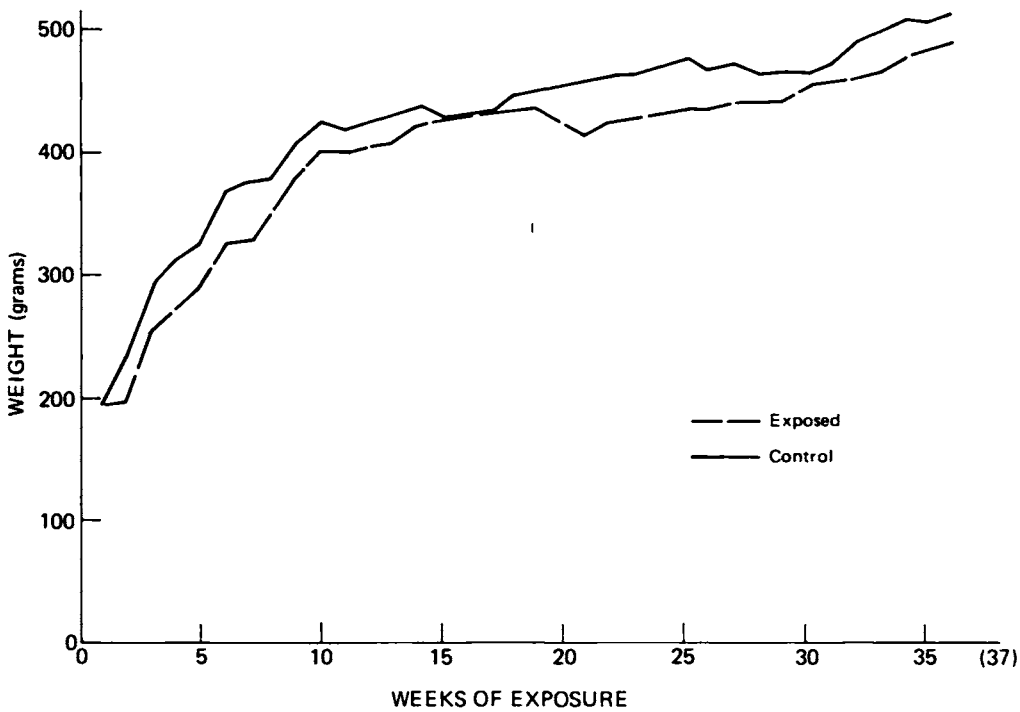


FIGURE 3. Growth.

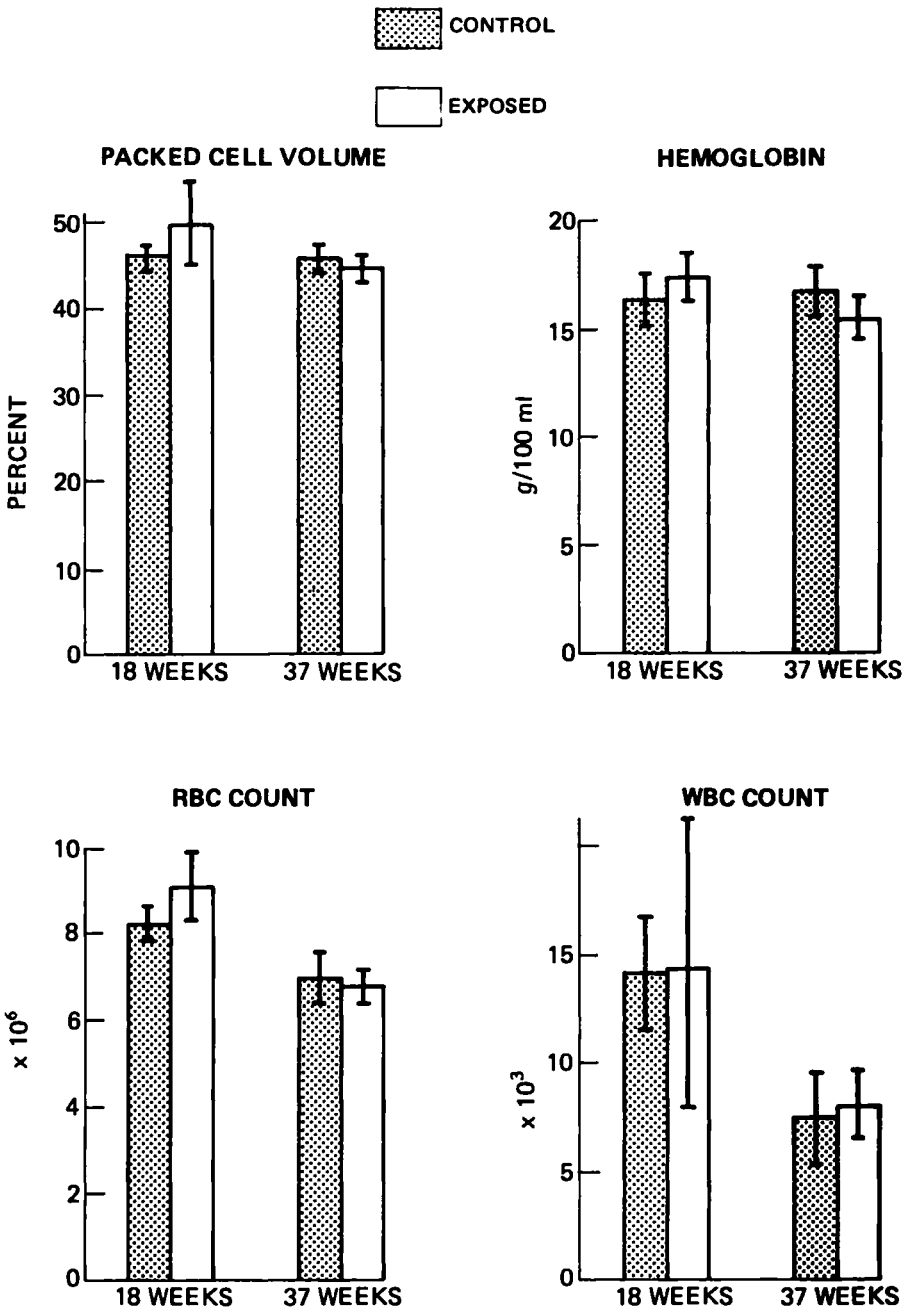


FIGURE 4. Hematology Data.

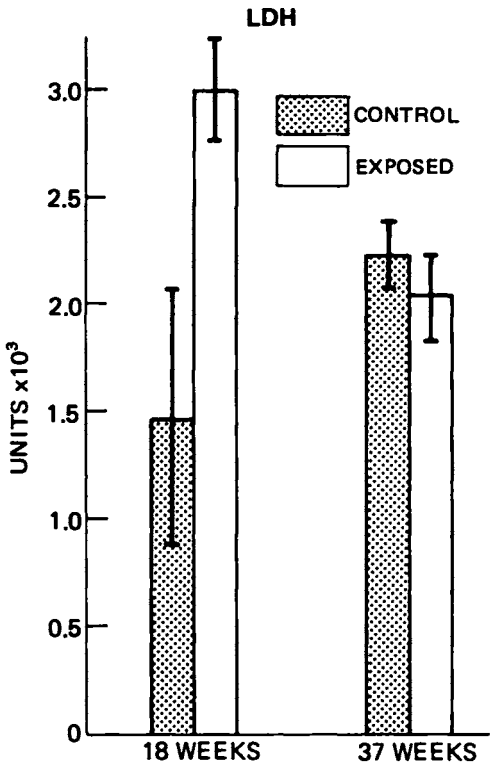


FIGURE 5. LDH Activities in the Sera of Control and Exposed Rats.

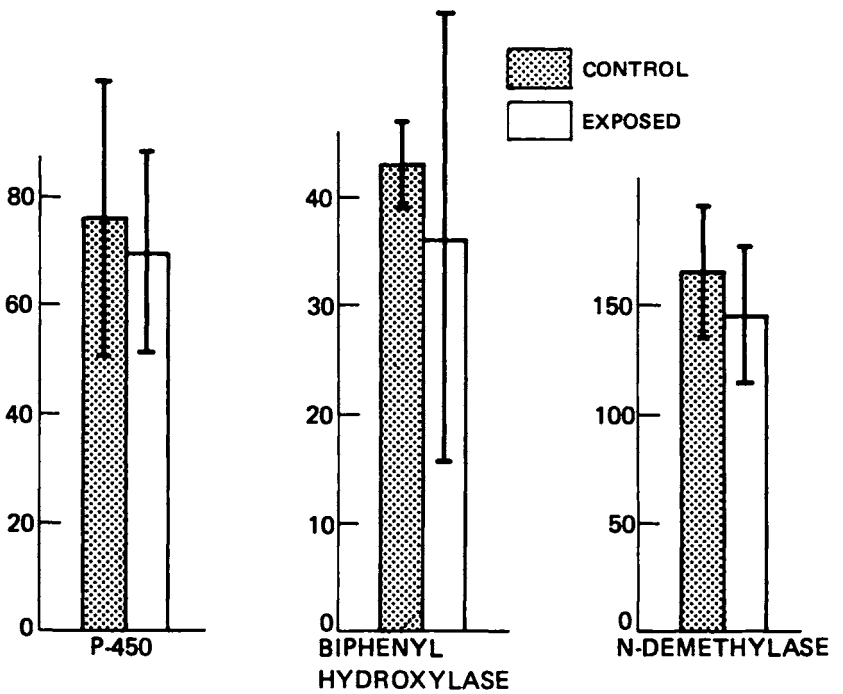


FIGURE 6. Liver Enzymes.

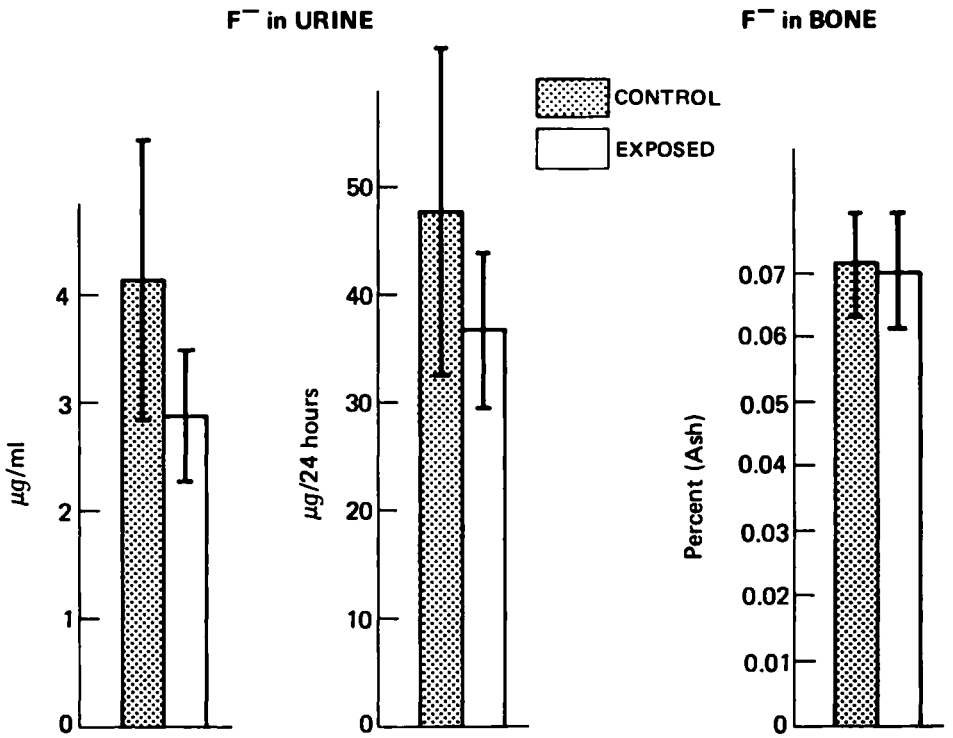


FIGURE 7. Concentration of Fluoride Ions in Urine and Bone.

*SESSION IV*

# SMOKE: ITS DEVELOPMENT AND CHARACTERIZATION

Moderator:

Robert M. Fristrom
Applied Physics Laboratory

# *FACTORS AFFECTING SMOKE DEVELOPMENT AND MEASUREMENT*

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## INTRODUCTION

Smoke may be defined as the airborne products from smoldering or burning materials. Thus, besides gas, smoke may contain various types of solid and liquid particulate matter, such as carbonaceous particles, ash, spalled fragments, tarry droplets, and other condensed matter. Indeed, the layman would not normally refer to a colorless gas containing no light-scattering particulates as smoke.

Perhaps the most important factor affecting life and property in a building fire is smoke.[1] It can damage property, prevent easy exit of occupants, and cause injury or even death by inhalation. Thus, it is imperative that the factors affecting smoke development be well understood.

Research in the area of smoke has been directed mainly towards the determination of the physical characteristics of smoke. A number of relatively small-scale tests have been developed and utilized to measure the obscuration of light due to smoke particulates. These include the ASTM E-84-70 Tunnel Test,[2] the ASTM E-286-69 8-ft Tunnel Test,[3] the ASTM D-2843-70 Smoke Test[4] based on the Rohm and Haas Co. XP-2 smoke-density chamber,[5] the ASTM E-162-67 Radiant Panel Test,[6] the NBS Smoke-density Chamber,[7] and a modified NBS chamber.[8] With the NBS Smoke-density Chamber, the problem of smoke stratification is minimized and reasonably reproducible results can be achieved as reported by Lee.[9]

In the work reported here, an NBS-Aminco Smoke-density Chamber was modified to permit the simultaneous measurement of sample weight loss and light obscuration. In addition, a new radiant heater was developed to permit operation of the chamber over a nominal energy flux range of 1 to 7.5 watts/cm$^2$. Experimental data from the modified chamber are presented for several natural, synthetic, and fire-retarded polymers in terms of specific optical density, and a mass optical density which accounts for the sample mass loss.

## IMPORTANT FACTORS AFFECTING SMOKE DEVELOPMENT

Smoke may be characterized by both chemical and physical factors, but only the latter are considered here. Some of the important physical characteristics related to smoke development from smoldering or burning materials are the magnitude and rate of airborne mass loss, the fraction of that mass loss that is particulates, and the nature of the particulates (shape, phase state, size distribution, and coalescing and sticking tendencies). These characteristics may be considered as dependent variables that are related in some analytical fashion to a single characteristic parameter termed the optical density. Several different formulations of the optical density have been proposed.[10] Both the maximum magnitude and the time dependence of the optical density are of interest.

The physical characteristics of smoke and the optical density can be related to a number of independent variables, which may be classified into two groups, material variables and environmental variables. Most often, the relationship must be determined experimentally. The material variables include polymer type, exposed area, thickness, weight, density, orientation, and presence of fire retardant(s) and/or surface coating(s). The environmental variables include the enclosure volume, degree of ventilation (if any), oxygen partial pressure, space temperature within the enclosure, and energy flux incident on the exposed surface of the material. Recent experimental studies of the manner in which these independent variables affect the dependent variables and the optical density are summarized by Seader and Chien.[11]

## OPTICAL DENSITY

In the standard procedure for conducting a smoke-density test with the NBS-Aminco Chamber,[7] the percent light transmission, T, is determined as a function of time until the minimum value is attained. As discussed above, T depends on the independent variables, some of which are fixed by the standard test procedure. The data are then converted to a specific optical density, $D_s$, where

$$D_s = \frac{V}{AL} [\log_{10}(100/T)]. \qquad (1)$$

The chamber volume, V, is 18 $ft^3$, the light path length, L, is 3 ft, and the exposed material surface area, A, is 0.0456 $ft^2$. The optical system of the NBS-Aminco Chamber is capable of detecting values of $D_s$ as high as 660 (0.001% light transmittance) with reasonable accuracy. Of particular importance is the maximum value of $D_s$ after correction for any deposits on the photometer windows. This value is termed $D_m$(corr.).

Seader and Chien[12] have shown that if certain simplifying assumptions are made, radiative transport theory can be applied to relate the percent

light transmission, T, to the dependent variables and certain particulate physical properties. The resulting expression is

$$\log_{10}\left(\frac{100}{T}\right) = \frac{Lm\Gamma_1}{V} f\{\bar{r},\bar{\lambda},n,\rho_p\} \tag{2}$$

where m is the airborne mass loss of the sample, $\Gamma_1$ is the fraction of sample mass loss that is particulates, $\bar{r}$ is the average or effective particulates radius, $\bar{\lambda}$ is the average or effective wave length of the light beam, n is the complex refractive index of the particulates and $\rho_p$ is the density of the particulates.

If Equations (1) and (2) are combined, the following alternate optical density termed the mass optical density (MOD), may be defined:

$$MOD = D_s A/m = \frac{V}{mL} \log_{10}\left(\frac{100}{T}\right) = \Gamma_1 f\{\bar{r},\bar{\lambda},n,\rho_p\} \tag{3}$$

It should be noted that the right side of Equation (3) contains no geometrical parameters of the enclosure or of the sample material being exposed, but rather only properties of the light beam and the particulate matter, as well as the fraction of polymer mass loss that is particulate matter. By comparison, the corresponding expression for the specific optical density is

$$D_s = \rho_s t \Gamma_1 \Gamma_2 f\{\bar{r},\bar{\lambda},n,\rho_p\}, \tag{4}$$

where $\rho$ is the density of the sample, t is the thickness of the sample, and $\Gamma_2$ is the mass fraction of the sample that becomes airborne. Thus, as seen, $D_s$ depends directly on the sample thickness and sample density as well. Seader and Chien[12] show that this direct dependence is verified by experimental data reasonably well provided that the light transmission is not less than about 1%. Thus, it appears that MOD represents a more fundamental smoke index than $D_s$. However, MOD can be determined only if the airborne mass loss is measured.

## MEASUREMENT OF AIRBORNE MASS LOSS

The suggested report form for the NBS Smoke-density Chamber[9] calls for weighing the sample before and after a test in order that the mass loss can be determined. If no dripping of the sample occurs, this decrease in weight corresponds to the airborne mass loss. Unfortunately, values for this quantity have seldom been reported. Recently Mickelson and Traicoff[13]

developed a chamber which permitted continuous measurement of sample weight during heat flux exposure by using a force transducer. They discussed the advantages of using such a technique. We have added such a force transducer to our NBS-Aminco Chamber to permit the simultaneous measurement of light transmittance and sample weight. Details of the development of the technique employed are given by Chien, Seader, and Birky.[14] Data have been obtained for a number of different materials, mainly in the nonflaming region with some results in the flaming region. Values of $D_m$(corr.) and corresponding values of mass loss and % mass loss for nonflaming conditions are given in Table I. A time history of $D_s$ and mass loss, m, is shown for a TDI-based flexible polyurethane foam in Fig. 1. Nonflaming conditions at a heat flux of 2.5 watts/cm$^2$ were utilized. In Fig. 1, the dependent variable scales have been chosen to force both ordinates to cover the same height so as to best compare the time effect on specific optical density and sample weight loss. The sample is seen to lose weight almost immediately upon initiation of the test. The response of light transmission initially lags the change in sample weight. This is due, in part at least, to the time required for the smoke particulates to spread throughout the chamber. The lag gradually increases during the early period of rapid sample weight loss until a maximum lag is attained at about 160 seconds. Thereafter, the lag rapidly diminishes.

The maximum value of the mass optical density is included in Table I with the ten materials ranked in the order of decreasing values of this index, $D_m$(corr.)/(m/A). Figure 2 is a plot of the mass-optical density as a function of time for several materials. All these tests were conducted at standard nonflaming conditions. Significant differences in the positions of the curves are noted with the woods occupying a central position.

## HIGH ENERGY FLUX EFFECTS

The NBS-Aminco Smoke-density Chamber is equipped with a nichrome-wire radiant heater, capable of operation over a narrow energy flux range of 1.7 to 2.9 watts/cm$^2$. The standard nonflaming test utilizes an energy flux of 2.5 watts/cm$^2$, which is sufficient to prevent ignition of $\alpha$-cellulose, as well as most other materials. We have recently made studies of the effect of radiant energy flux on the specific optical density by replacing the standard heater assembly with newly designed heaters by Deltech, Inc. for higher temperature service. Results for 1/4-inch thick Douglas fir are shown in Fig. 3 where data are plotted for an energy flux range of approximately 1.2 to 7.5 watts/cm$^2$. An asymptotic calorimeter (Hy-Cal Model C-1301-A-120) was used to determine the energy flux, which was controlled automatically by a temperature controller (Alnor Model 611-040-XXX) during each test by a thermocouple located near the electrically heated wire. As seen in Fig. 3, the maximum specific optical density appears to go through a maximum just prior to ignition. In the flaming region, $D_m$ values are generally significantly reduced below those for most of the nonflaming region. Thus, the energy flux required to cause ignition of the sample is a significant factor with respect to smoke density.

## EFFECT OF FIRE RETARDANTS

A fire-retarded rigid-urethane foam (designated as TMP-312/PAPI) was formulated and utilized in a smoke study. The major components of the foam are polymethylene polyphenyl isocyanate (PAPI) and propoxylated adduct—trimethylolpropane (TMP-312). Three different fire-retardant compounds were blended with the polymer to a maximum of 18% by weight. One reactive type was used, o,o-diethyl N,N'-bis (2 hydroxyethyl) aminomethyl phosphonate, which contains phosphorus. Two nonreactive types were used, tris(2,3-dichloropropyl) phosphate and tris(2,3-dibromopropyl) phosphate, both of which contain both halogen and phosphorus.

Figure 4 shows the relationship between the final mass optical density and the fire-retardant concentration at 2.5 watts/cm$^2$ for data obtained with the NBS-Aminco Smoke-density Chamber. A general trend is observed. The maximum value of the final mass optical density occurs somewhere in the middle region rather than at the extremes of the fire-retardant concentration level. However, the addition of fire retardants always yielded more smoke.

Figure 5 shows the relationship between the final mass optical density and the percent fire retardant at an energy flux of 7.5 watts/cm$^2$. Even at this relatively high energy flux level, ignition did not occur as it did with Douglas fir as shown in Fig. 3. Also included in Fig. 5 is the percent char residual. The brominated fire retardant system gives more smoke. The reactive phosphorous system results in less smoke, but the char residual is greatest.

Figure 6 shows the final mass optical density for 7.5 watts/cm$^2$ as a function of the limiting oxygen index (LOI). When these data are compared to Fig. 5, it is seen that the less flammable polymers (with high LOI) give more smoke. Figure 7 shows the relationship between the final mass optical density and the fire-retardant concentration for the brominated system at different energy-flux levels (insufficient to achieve ignition). The trend observed is an increasing smoke level as the energy flux is increased.

## CONCLUSIONS

A large number of factors can influence the development of smoke. In order to reduce to a reasonable number the factors that need to be studied experimentally, it is important to apply theoretical considerations where possible. The use of the mass optical density as a correlating parameter appears to eliminate, or at least reduce, the need to study effects of sample thickness and sample density. This requires that the sample mass loss be monitored simultaneously or at least determined at the conclusion of a smoke test. A means for accomplishing this has been developed.

An important factor which heretofore has been largely ignored is the energy flux incident on the sample. Heaters have been developed which can deliver up to 7.5 watts/cm$^2$. This is sufficient to cause spontaneous ignition of cellulosic materials, but not polyurethanes.

The addition of fire retardants to the TMP 312/PAPI rigid urethane system was studied at different energy-flux levels. The maximum final mass optical density occurs at intermediate levels of fire-retardant concentrations. The correlations between final mass optical density and limiting oxygen index as well as percent residual char at a moderately high energy flux level of 7.5 watts/cm$^2$ are of interest.

ACKNOWLEDGMENTS

The work supported here was supported by the National Science Foundation under Grant GI-33650. Gratitude is expressed to Professor I. N. Einhorn of the University of Utah, Dr. Merritt M. Birky of the NBS, and to James R. Gaskill of the University of California for their helpful suggestions. The smoke density chamber was made available for our use by the American Instrument Company through Samuel Greenberg. The high-energy flux heaters were supplied by Deltech, Inc. through Calvin L. Stevenson and Richard G. Davis. The fire retardant samples and oxygen index measurements were made by Dr. M. D. Kanakia, D. M. Hou, and Cheu-Wu Yo.

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TABLE I

MAXIMUM OPTICAL DENSITIES AND MASS LOSS FOR SEVERAL MATERIALS

| Material | $D_m$(corr.) | m,gm | % Mass Loss | $\frac{D_m(\text{corr.})}{m/A}$, $cm^2/gm$ |
|---|---|---|---|---|
| Polyvinyl chloride, plasticized | 350 | 2.30 | 63.7 | 6430 |
| Neoprene | 877 | 6.75 | 14.1 | 5490 |
| TDI-based flexible polyurethane | 198 | 1.78 | 97.2 | 4690 |
| TMP-312/PAPI rigid-urethane foam (no fire retardant) | 100 | 1.17 | 53.0 | 3620 |
| α-Cellulose | 173 | 2.19 | 87.6 | 3340 |
| Douglas fir marine plywood | 533 | 7.73 | 47.0 | 2910 |
| Douglas fir | 620 | 9.39 | 53.7 | 2790 |
| Redwood | 436 | 8.38 | 53.9 | 2230 |
| Polymethylmethacrylate | 724 | 20.0 | 40.4 | 1530 |
| Polyvinyl chloride | 177 | 6.40 | 53.1 | 1170 |

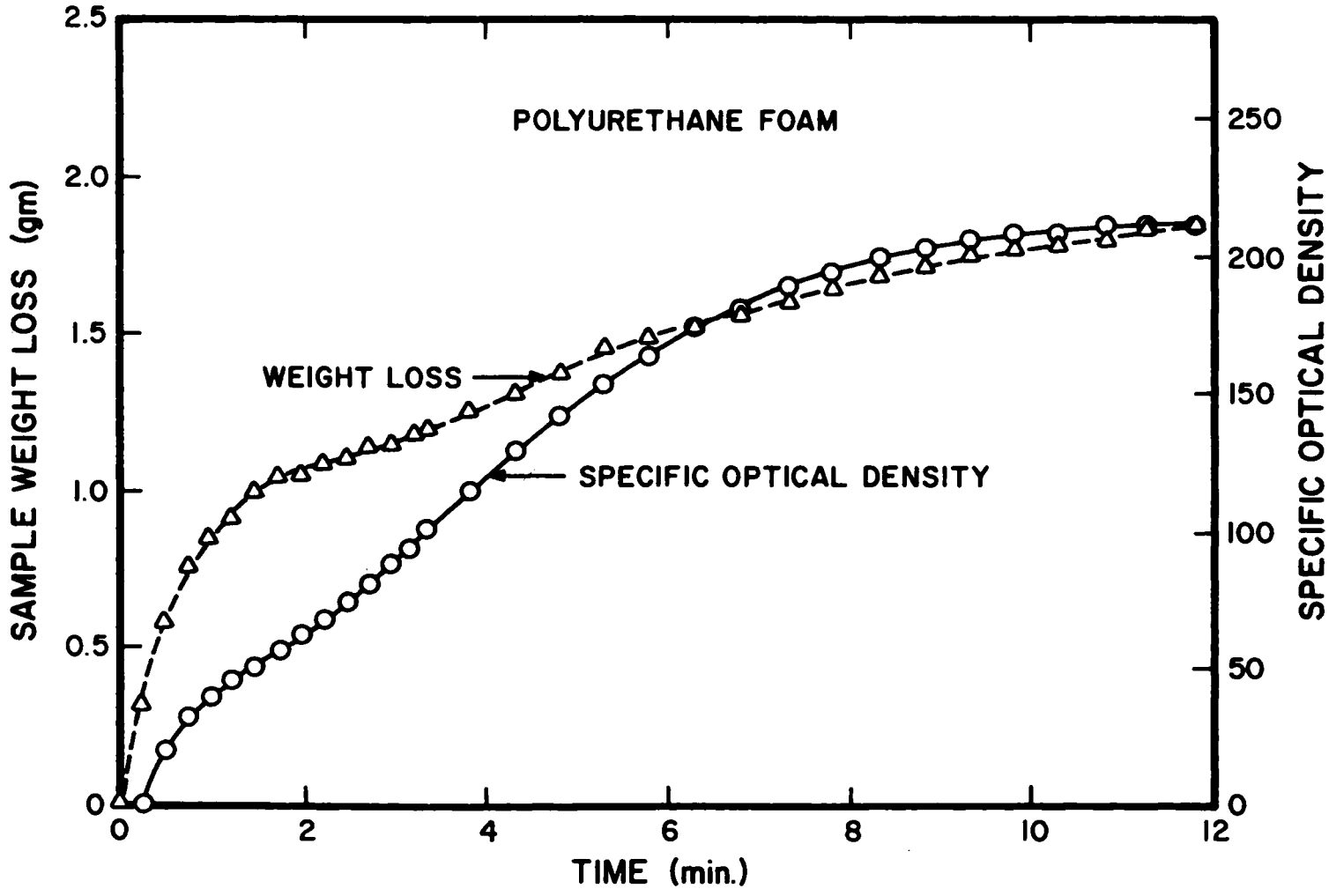


FIGURE 1. Specific Optical Density (Uncorrected) and Sample Weight Loss Curves for Polyurethane Foam.

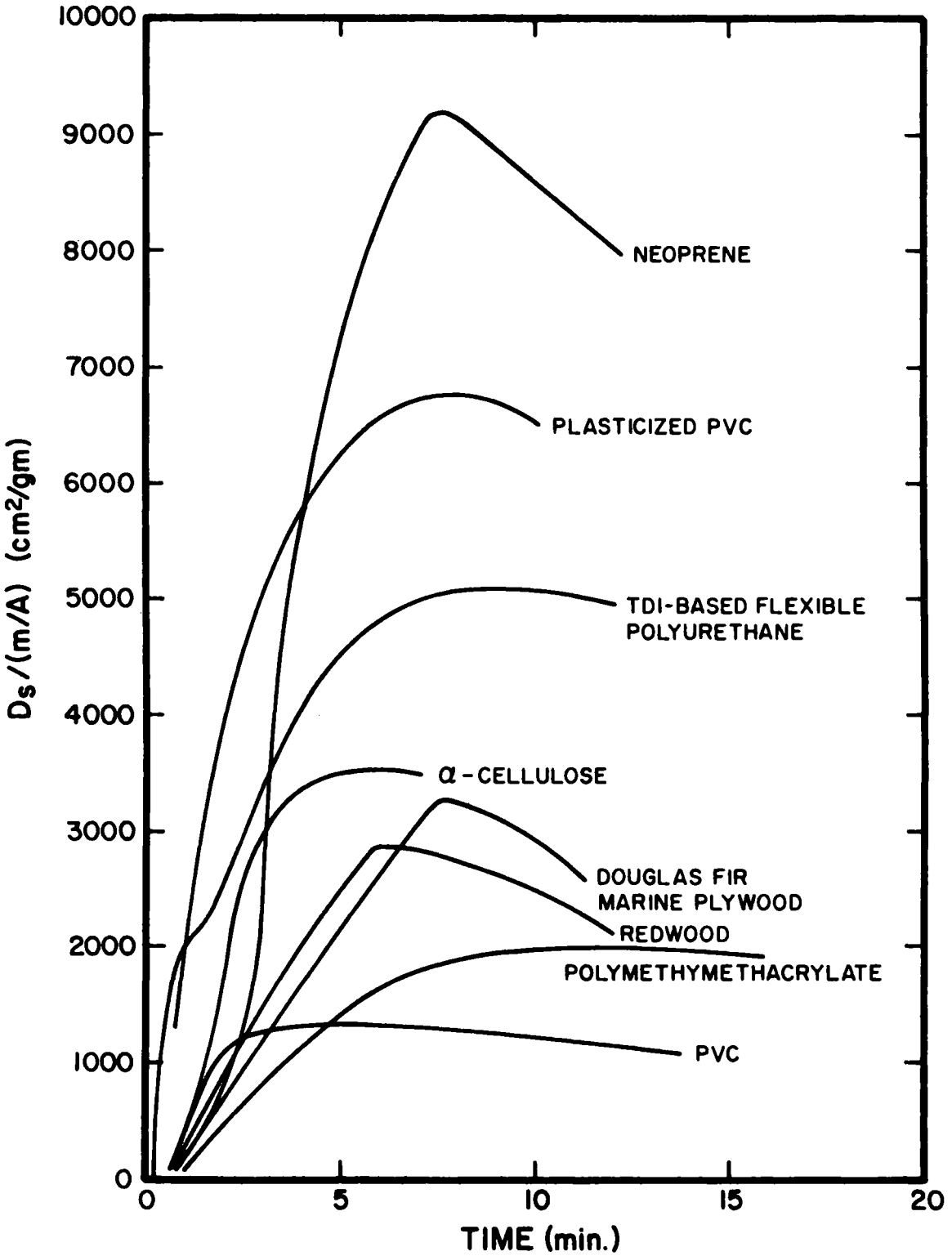


FIGURE 2. Mass Optical Density of Several Materials.

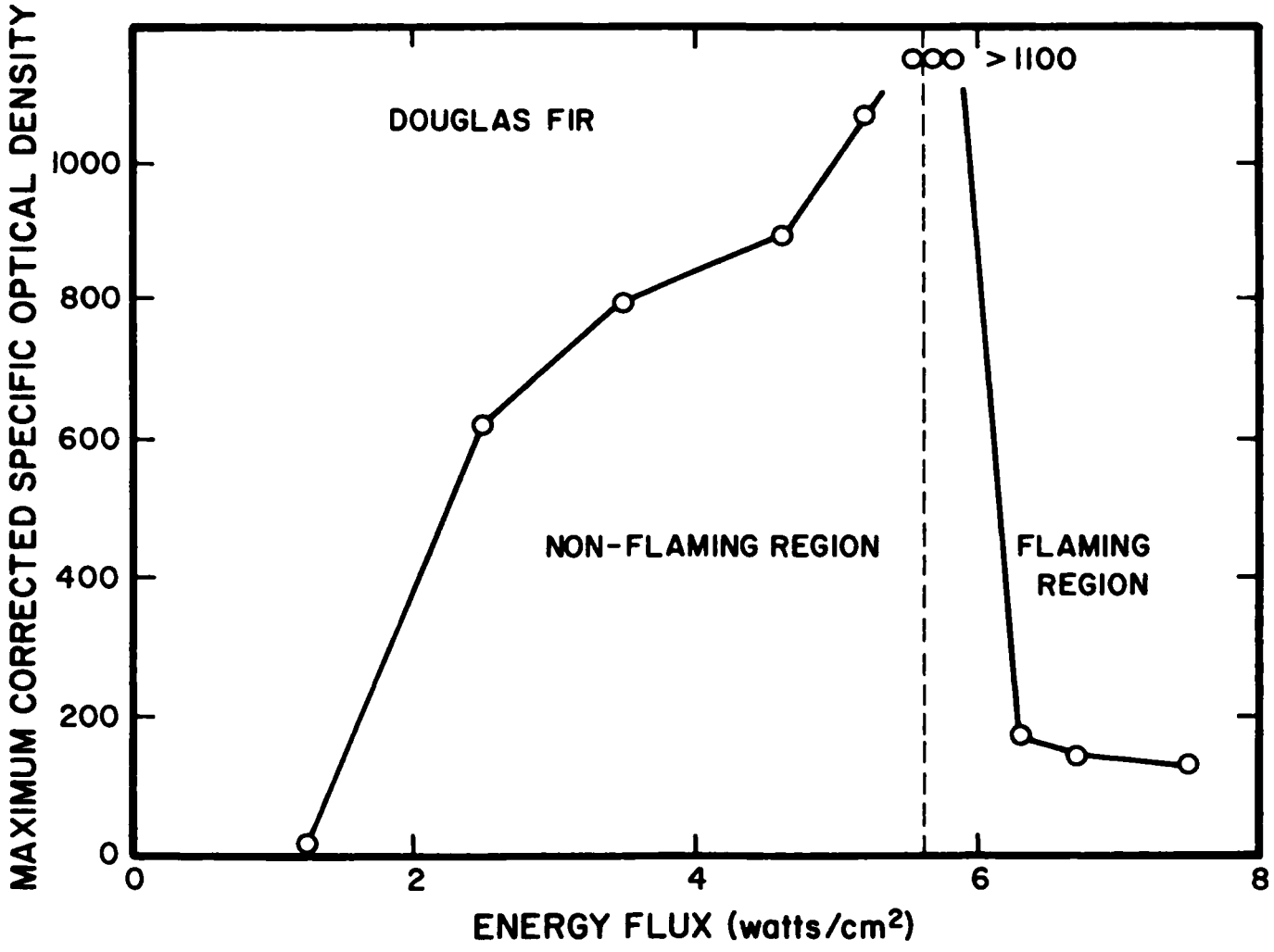


FIGURE 3. Effect of Energy Flux on Maximum Corrected Specific Optical Density of Douglas Fir.

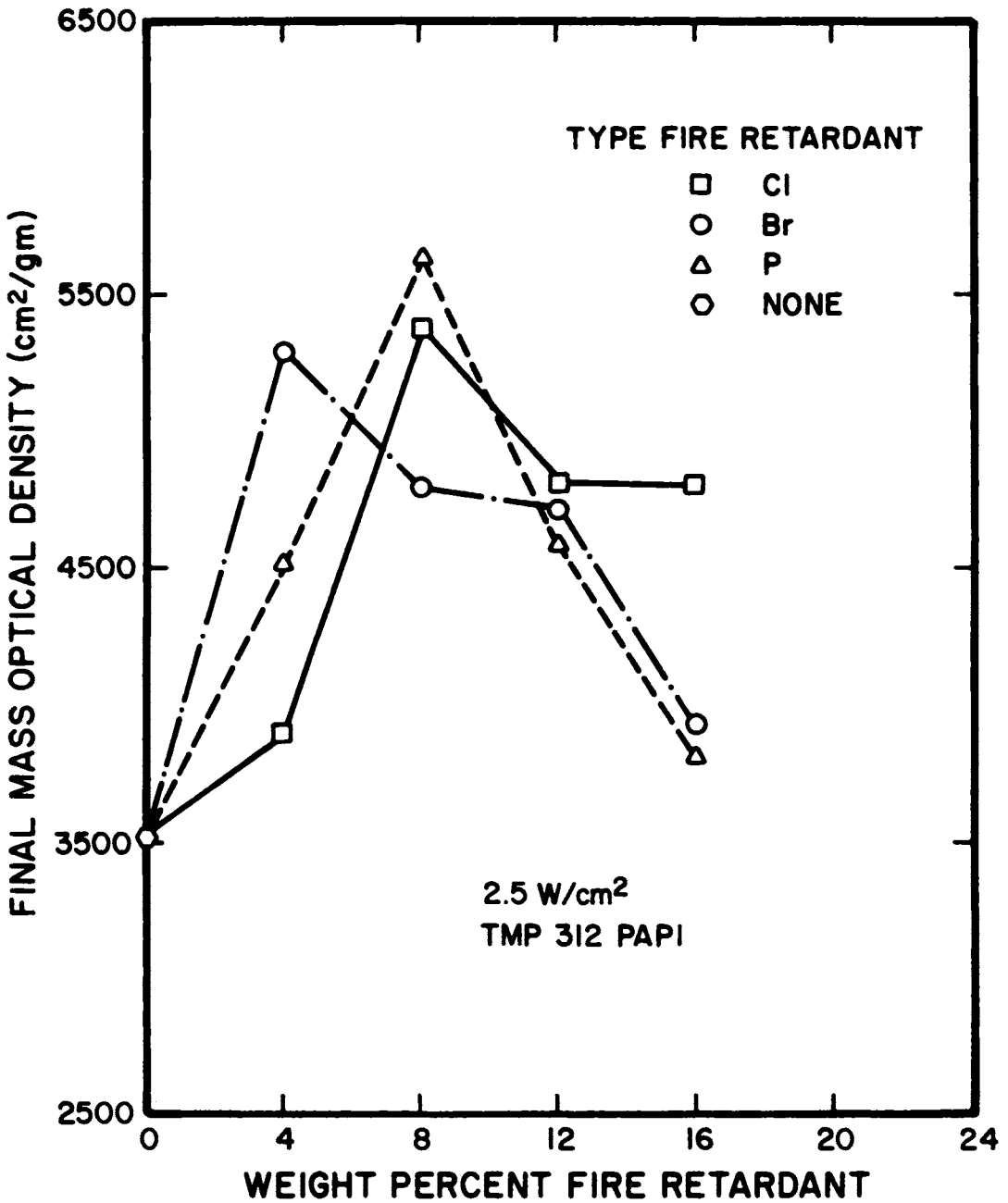


FIGURE 4. Effect of Fire Retardant Concentration on Final Mass Optical Density at 2.5 watts/cm$^2$.

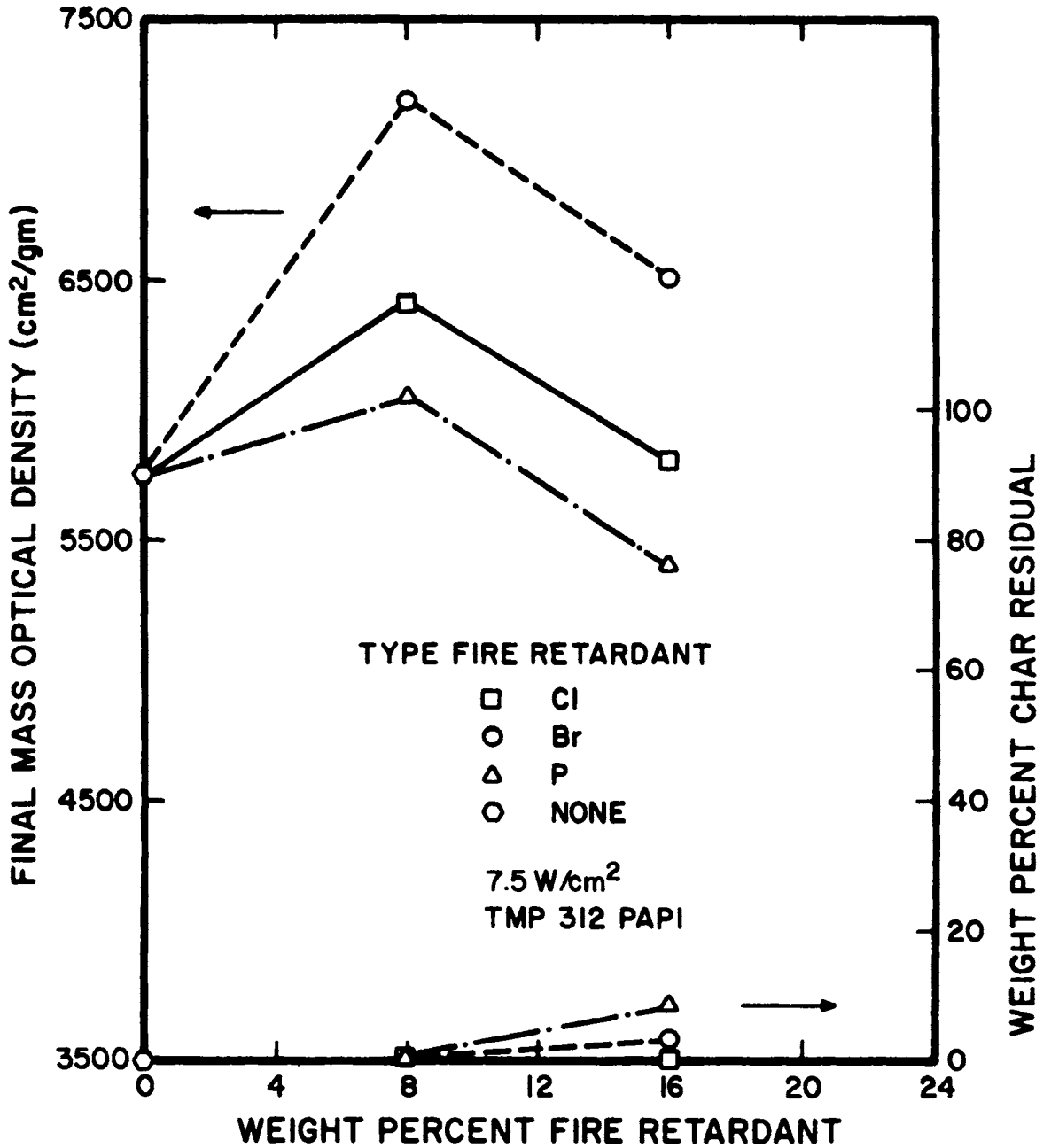


FIGURE 5. Effect of Fire Retardant Concentration on Final Mass Optical Density at 7.5 watts/cm$^2$.

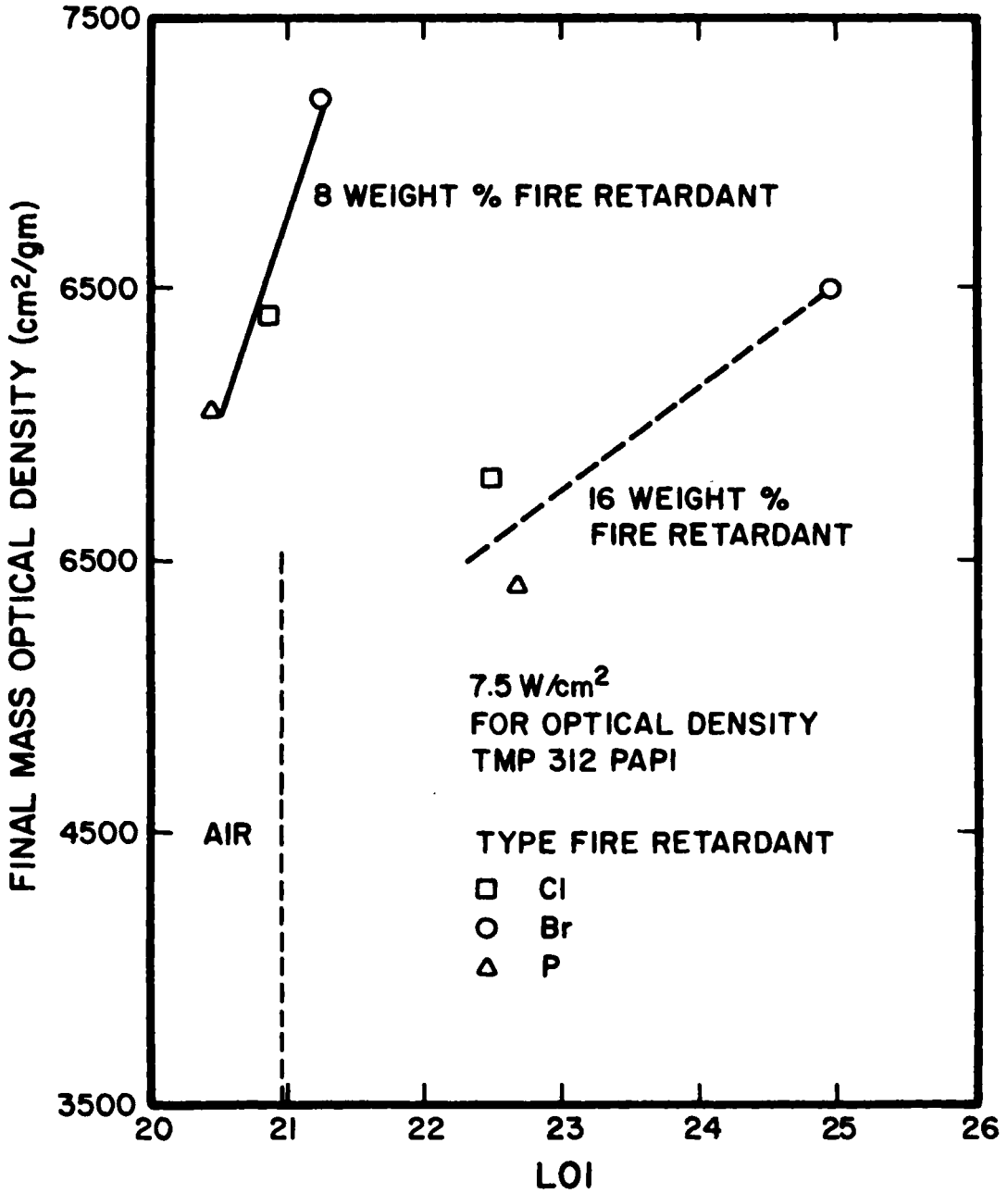


FIGURE 6. Correlation of Final Mass Optical Density with LOI.

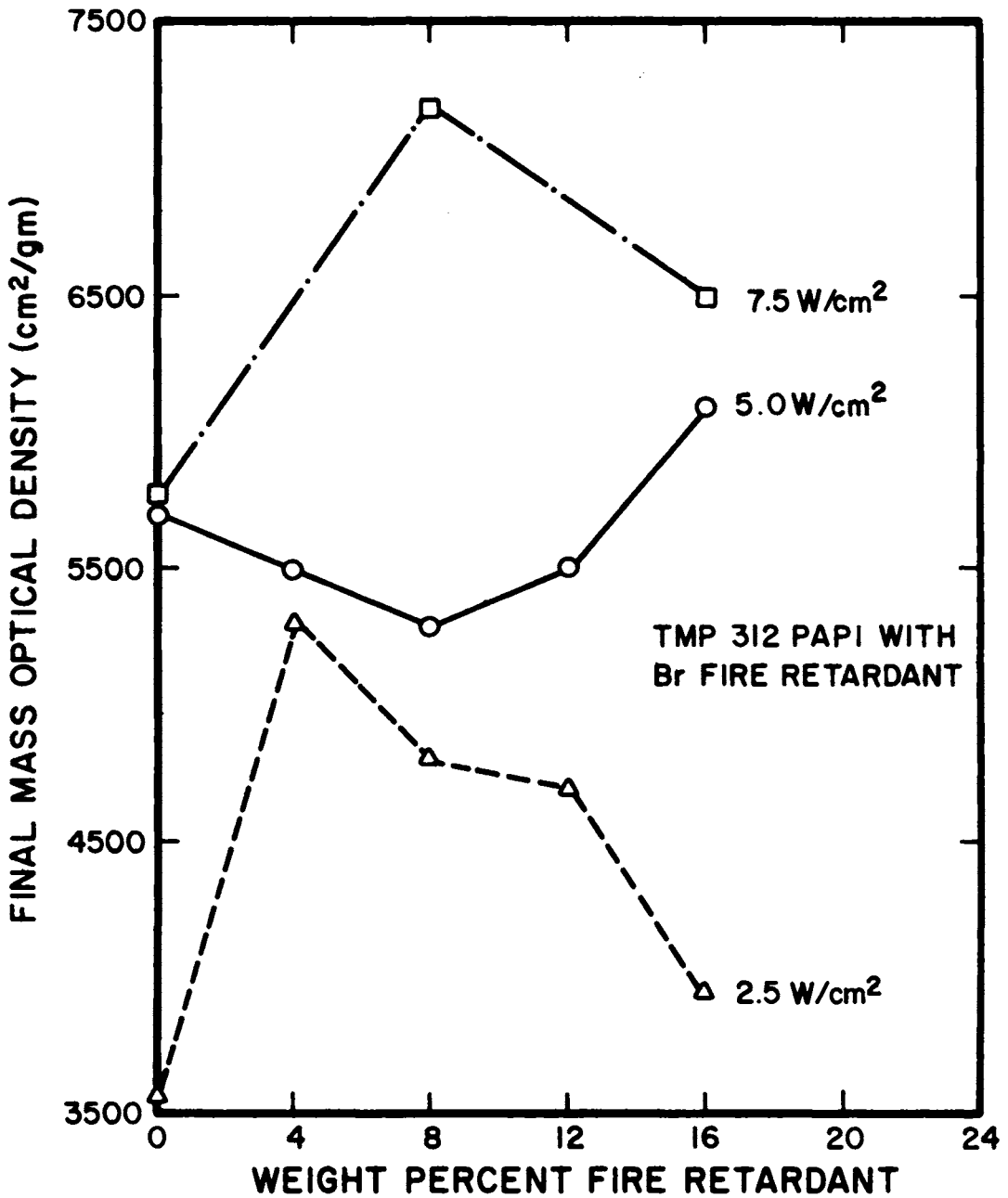


FIGURE 7. Effect of Energy Flux on Final Mass Optical Density for Different Fire Retardant Concentrations.

# *COMPUTERIZED ANALYTICAL SYSTEM FOR THE ANALYSIS OF THERMAL DECOMPOSITION PRODUCTS**

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## INTRODUCTION

Flexible- and rigid-urethane foams, representative of typical formulations used in commerce, were selected as materials for study because of the involvement of this class of polymers in numerous fires. When so involved, there is a concern that urethane foam may release toxic and noxious products, such as cyanides, carbon monoxide, aldehydes, isocyanates, alcohols, and low-molecular-weight alkanes, alkenes, and aromatic compounds, as a consequence of pyrolysis, oxidative degradation, and flaming combustion. The methodology developed for the identification and quantification of the principal products of thermal degradation will be described within the scope of this paper.

## MATERIAL SELECTION

### Polymer System

A flexible-urethane foam, produced on a commercial production facility, was obtained and characterized. Table I presents a summary of the foam's formulation and chemical composition. Table II summarizes the physical properties and flammability characteristics of this flexible-urethane foam.

A rigid-urethane foam, based on polymethylene polyphenyl isocyanate and the 312-molecular-weight propoxylated adduct trimethylolpropane, was prepared in the laboratory and characterized. Table III presents a summary of the rigid foam's formulation and chemical composition. Table IV summarizes the physical properties and flammability characteristics of this rigid-urethane foam.

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*See also "Pyrolysis of a Flexible-Urethane Foam" in the Appendix.

## COMPUTERIZED ANALYTICAL SYSTEM

The computerized analytical system employed in this study is shown schematically in Fig. 1. Weighed samples of the urethane foam (2 mg) were placed in the pyrolysis probe and pyrolyzed directly into the inlet of the gas chromatograph. A delayed mode was used during which time, the same was permitted to reach equilibrium with its environment. Pyrolysis temperatures ranging from 300°C to 1000°C were obtained by varying the amperage supplied to the filament probe. A 6-inch stainless-steel insert, filled with glass wool, was connected as a precolumn filter to prevent large particles from reducing the flow rate of the effluents. A research gas chromatograph, capable of subambient operation, was employed for the separation of the decomposition products. The system has available three detectors, a thermal conductivity detector (TC), a flame ionization detector (FID), and an electron capture detector (EC). Any two of these detectors can be used simultaneously by using a splitter. For the results reported here, the thermal conductivity detector and flame ionization detector were used. A splitting ratio (TC to FID) of 10 to 1 was employed.

A list of columns commonly employed to separate the products resulting from thermal decomposition of urethane polymers is presented in Table V. Within the scope of this paper, the products of thermal decomposition, principally low-molecular-weight gases, were separated using a 1/8-inch O.D. by a 16-foot stainless-steel Chromosorb 101 column. The higher-molecular-weight degradation products were separated using a 1/8-inch O.D. 8-foot stainless-steel Dexsil column.

An automatic electronic integrator with printout facilities was used to record chromatographic peak areas and retention time for quantitative analysis. Internal standards were injected into the gas chromatograph in varying amounts to produce a plot of standard weight vs. peak area. A table of response factors for gas chromatographic analysis[1] was used to calculate the quantitative amounts of products obtained from the thermal degradation of the urethane foams being studied.

The identification of materials separated by the gas chromatograph was obtained using a modified quadropole (known as a dodecapole) mass spectrometer. This system completely scans a mass range of 1 to 600 amu in two seconds. This is fast enough to permit spectral identification of separate GC peaks in real time. In the periodic scan computer-controlled mode, the mass spectrometer repetitively scans a complete GC run and stores all of the data on a disk.

Many studies have been reported in the literature on the use of computers for the interpretation of chemical spectra.[2,3,4,5,6,7,8] Several computer search techniques have been employed in the process of identification of compounds from these mass spectra, as follows:

1. The identification of compounds by a comparison of the five most intense peaks in the unknown spectrum with the five most intense peaks of the reference spectra.[9]

2. A similar searching technique, as reported by Crawford and Morrison,[10] makes use of the six most intense peaks in the spectra.

3. A modified technique, as evaluated by Einhorn, Ramakrishnan, and Hileman,[11] utilizes the ten major peaks and their relative abundances in the search program.

4. A similarity index used by Einhorn, *et al.*,[12] is based on the n strongest peaks in each interval of m amu. (This comparison depends on the position of each selected peak in the spectrum from the reference file, positions of i and j being determined by decreasing ion intensity.

For practical reasons, any collection of reasonable size will consist of mass spectra taken in different laboratories on different types of instruments and under different experimental conditions. Thus, a reliable search technique which will provide a good match in the identification process should use the gross feature of the entire spectrum, rather than depending on an excellent match of a few intense peaks which may come from a limited window of the total spectrum.

In order to shorten the storage of data and reduce the time required for each search, while deleting as little information of structural significance as possible, the library search routine is based on a series of spectra that are abbreviated by selecting the two largest peaks in each 14-mass unit interval throughout the spectrum, 14 being the mass of the methylene group. The Bieman System[4] is used, wherein the first interval is 6-19 followed by 20-33, 34-47, etc. This grouping was selected to prevent the difficulties that were encountered in earlier work when the boundaries split common peak clusters such as 42, 43, etc.

Selecting the two most intense peaks in consecutive regions of 14-mass units assures that the significant peaks belonging to a homologous series of ions are retained. This abbreviation technique assures that the molecular ion (if it is present in the complete spectrum) is not deleted, because the heaviest fragment ion cannot possibly be in the same group as the molecular ion (exceptions due to the loss of hydrogen). To ensure greater comparability, the two most intense ions in each region are retained in the abbreviated spectrum. Inasmuch as the low mass region contains many intense peaks, this search technique clearly discriminates against ions in this region and thus, permits a more accurate overall match.

The actual search proceeds through a comparison of the unknown spectra with the complete set of library spectra on some selected subset of these spectra. The selection of the subset of the library usually involves some

known characteristics of the unknown compound, such as molecular weight, or compounds containing oxygen, sulfur, etc.

The principal aim of the search routine is to relate known spectra which "best fit" the unknown, according to a preselected criterion. Further verification techniques include the use of Kovats' constants or peak verification through the use of known standards.

## THERMOANALYTICAL SYSTEM

The analysis of the gaseous decomposition products is required for the study of the kinetics and reaction mechanism in thermal degradation. Simultaneous thermogravimetric analysis (TGA), derivative thermogravimetry (DTG), differential thermal analysis (DTA), and effluent gas analysis (EGA), permit a more thorough investigation of the degradation process than individual pyrolysis techniques. It is possible to obtain a mass balance by determining the elemental analysis of the material being studied prior to degradation followed by quantitation of the effluent gases and analysis of any remaining residue. Figure 2 shows the schematic drawing of the Mettler Thermoanalyzer 1 used in this study. This system described by Wiedemann[13] makes use of a furnace tube of only 35 $cm^3$. The small volume is essential, in order to keep the time delay low during gas transport. As the degradation process proceeds the products eluted from the thermoanalyzer are concentrated in a cold trap then flushed into the GC/MS system for individual product identification. Figure 3 presents a schematic layout of the interfaced thermoanalyzer and the mass spectrometer.

The mass spectrometer may be coupled as shown in Fig. 3 to the thermoanalyzer or may be interfaced with the gas chromatograph which is coupled directly to the thermoanalyzer. The latter arrangement facilitates separation of complex mixtures of effluents resulting from the degradation process. The thermobalance is shown in the center of Fig. 3, enclosed in a vacuum-tight tank, with a thermostatically-controlled water jacket. The reaction chamber (R) is surrounded by the furnace and is clearly separated from the balance housing by a diffusion baffle. The diffusion pumps (K) evacuate the balance housing and the reaction chamber when the thermoanalyzer is used in the vacuum mode. If the thermal studies are conducted in an environment other than vacuum, the carrier gas is eliminated by means of a silicone separator prior to introduction into the ion source of the mass spectrometer. The decomposition gases pass the mass analyzer (F) which, in turn, is connected to a recorder (J) through the mass spectrograph control panel. The total pressure required for detailed interpretation of the mass spectra is determined with an ionization gauge (S). The gas inlet system (A, B, C) is used for calibration purposes. The relation between measured total partial pressure and the ion current of an injected specific gas permits calibration of the mass spectrometer in absolute partial pressure units or amps/ton.

## EXPERIMENTAL

### Pyrolysis Studies

Pyrolysis was selected as an example in this study because this technique affords an easy and reproducible way to fractionate a polymer into a complex series of characteristic degradation products. Two milligram samples of the rigid- and flexible-urethane foams were pyrolyzed at 300°C, 500°C, 750°C, and 1000°C. Figure 4 shows a typical separation of degradation products on a 16' Chromosorb-101 column programmed from 0°C to 200°C at 10°C per minute using both thermal conductivity (Fig. 4a) and flame ionization (Fig. 4b) detection with peak identification given in Table VI. Figures 5 and 6 show a composite of separations of degradation products for the flexible foams pyrolyzed at several temperatures utilizing the Chromosorb-101 and Dexsil columns respectively.

Figure 7 is a chromatogram obtained by pyrolyzing the rigid-urethane foam at 1000°C in helium utilizing the Chromosorb-101 column programmed from 0°C to 200°C at a rate of 10°C per minute. Table VII lists the products shown in Fig. 7.

Since the mass spectrometer was continually scanning the GC peaks as they eluted, it was possible to reconstruct the chromatogram from the mass spectral data as shown in Fig. 8. This reconstructed chromatogram can then be used to qualitatively identify the eluted peaks. As an example, peak 10 of Fig. 4 was related by retention time to the reconstructed chromatogram with a scan number 205. The mass spectrum of scan 205 is shown in Table VIII. The search routine can then be used to identify the compound in scan 205 as shown in Table IX. It is apparent from Table IX that acetaldehyde has the best match, however, ethylene oxide is also a reasonable candidate on the basis of GC retention time and mass spectra. A positive identification was obtained when a mixture of acetaldehyde and ethylene oxide was injected into the column. The retention time and spectra clearly indicate the unknown to be acetaldehyde.

Quantitative analysis of the major components of degradation was obtained using the ethanol peak as an internal standard. Various amounts of ethanol were injected into the gas chromatograph and a plot of weight versus peak area was prepared. The response factors given by Dietz[1] were then used to quantify all major peaks. Table X summarizes the quantities of degradation products obtained from the flexible foam at the various pyrolysis temperatures.

## THERMOANALYTICAL STUDIES

### Sample Preparation

Selected samples of flexible- and rigid-urethane foams, previously described, were embrittled using liquid nitrogen, reduced to a fine powder, and ground to desired size using a mortar and pestle. Particle size was

controlled by utilizing the portion of the samples which passed a 40-mesh copper screen. All samples were dried under vacuum at 60°C for 45 minutes and stored in a dessicator in sealed vials prior to analysis. To minimize experimental variables all samples were weighed to 10 ± .05 mg. A single platinum crucible was used on the sample holder throughout this study. An $Al_2O_3$ Standard Mettler Sample 10 ± .05 mg was employed as the reference material.

## Thermal Analysis

Figure 9 illustrates the simultaneous DTA, TGA, DIG, and temperature traces obtained during a typical single analysis utilizing the rigid-urethane foam sample.

As the test specimens are heated in the crucible in the dynamic mode, a buoyancy effect is encountered with alters the observed sample weight. A computer program has been developed which corrects for this phenomena. Figure 10 illustrates the corrected and original noncorrected TGA thermograms obtained in helium and oxygen environments.

## Effect of Heating Rates

Considerable controversy exists as to possible correlations which might be developed utilizing pyrolysis and oxidative degradation experiments carried out at slow heating rates to predict the response of materials during flaming combustion under rapid heating conditions. The general nature of thermograms may be illustrated by briefly considering several examples. If the degradation process is a simple, irreversible reaction, then the rate can often be described by a power law function:

$$-\frac{1}{w_o}\frac{dw}{dt} = k\left(\frac{w-w_R}{w_o}\right)^n \tag{1}$$

or

$$-\frac{dW}{dt} = k\,W^n, \tag{2}$$

where

$w$ = instantaneous weight of sample during the degradation process,
$w_o$ = initial weight of sample,
$w_R$ = final weight of the residue upon completion of reaction,
$W = (w-w_R)/w_o$
$t$ = time,
$n$ = kinetic order of reaction, and
$k$ = specific rate constant.

The reaction rate constant, k, may depend on the absolute temperature according to the Arrhenius law:

$$k = Ae^{-E/RT}, \tag{3}$$

where

A = pre-exponential factor,
E = activation energy,
R = universal gas constant, and
T = absolute temperature.

As the heating rate is increased, the degradation process occurs predominantely at higher temperatures and the thermograms become less steep. For power-law degradations with lower activation energies and/or higher kinetic orders, thermogram slope is also decreased. Figure 11 illustrates this phenomena as obtained when the flexible-urethane polymer was subjected to oxidative degradation in air at heating rates of 10°C, 25°C, and 100°C/minute.

Consideration must be given to potential thermal gradients which may develop during rapid heating, i.e., greater than 10°C/minute. Figure 12 is a composite of differential thermograms obtained at rates varying from 6°C to 100°C/minute, dramatically illustrating this effect.

Effect of Atmosphere

The effect of environment can greatly influence the mode of thermal decomposition. Samples of flexible- and rigid-urethane foams were evaluated in dynamic environments of helium, air, and oxygen. Figure 13 shows a rapid degradation occurred in an oxidizing environment. A substantially slower degradation occurred in the inert helium environment. Figure 14 shows a typical bimodal degradation process normally encountered in the thermal degradation of polymers which exhibit char formation.

## PRELIMINARY APPLICATIONS OF THE COMPUTERIZED ANALYTICAL SYSTEM TO THE FIRE PROBLEM

Chemical analysis of degradation products resulting from the fire exposure of materials is extremely complex inasmuch as numerous degradation processes may be encountered simultaneously. In addition, certain effluents may serve to autocatalyze or retard the degradation of materials undergoing decomposition. In the dynamic fire environment there is often a rapid heat buildup which is greatly influenced by the presence of an excess supply of oxygen. Under some conditions the degradation products may also provide a fuel-rich environment and this affects the combustion process. In an attempt to develop a model which will permit simulation of the fire environment, laboratory studies encompassing pyrolysis, oxidative degradation, and flaming combustion have been carried out. Figure 15 presents the chromatograms

obtained during the laboratory-scale pyrolysis and combustion experiments involving the flexible-urethane foams. Initial examination of the chromatograms presented in this figure showed definite changes in the nature of decomposition products. Of particular significance is the peak identified as hydrogen cyanide in the combustion chromatograph that does not appear in the pyrolysis chromatogram.

## CONCLUSIONS

The methodology has been developed for the identification and quantification of the products of combustion of polymeric materials using a computerized analytical system. This analytical system will permit simultaneous differential thermal analysis, thermogravimetric analysis, and derivative thermogravimetric analysis, thus facilitating the study of mechanism and kinetics of polymer degradation. Additional studies are also under way using this analytical system to determine the effect of fire retardant type and concentration on the degradation process as well as the effect of heat-flux on the smoking tendency of polymers. The computerized analytical system will be further employed in the development of a realistic fire model which may be suitable for scaling purposes in the study of the physiological and toxicological aspects of human exposure under real fire conditions.

## ACKNOWLEDGMENTS

This research, conducted at the Flammability Research Center of the University of Utah, has been supported by the National Science Foundation, Grant Number GI-33650, under its RANN (Research Applied to National Needs) program. The authors are indebted to Dr. R. H. Long, Jr., the program monitor.

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TABLE I

FLEXIBLE-URETHANE FOAM — CHEMICAL CHARACTERISTICS

Formulation

| Ingredients | pbw |
|---|---|
| Propoxylated triol (3000 M.W.) | 100.00 |
| Water | 3.19 |
| Silicone surfactant (L-540)[a] | 0.90 |
| Bis-dimethylaminoethyl ether (A-1)[b] | 0.08 |
| Stannous octoate[c] | 0.45 |
| Tolylene diisocyanate (80:20 mixture of 2,4/2,6 isomers) | 46.19 |

Elemental Analysis

| Element | % by weight |
|---|---|
| Carbon | 61.66 |
| Oxygen | 23.12 |
| Nitrogen | 5.63 |
| Hydrogen | 8.74 |
| Silicon, tin | (0.005 - 0.05%) |
| Calcium, sodium, magnesium, aluminum, and potassium | (less than 0.01%) |
| Iron | (4.5 ppm) |
| Copper | (5.0 ppm) |

[a]Silicone Division, Union Carbide Corporation

[b]Union Carbide Corporation

[c]Metal and Thermit Corporation

TABLE II

FLEXIBLE-URETHANE FOAM — PHYSICAL PROPERTIES AND FLAMMABILITY CHARACTERISTICS

| | |
|---|---|
| **Physical Properties** | |
| Density, lbs/ft$^3$ | 2.0 |
| Tensile strength, psi | 19.0 |
| Elongation, % | >300 |
| Compression Set, % (75% deflection, 22 hrs at 158°F, 30 minutes recovery) | <10 |
| Load Bearing, 50 square inches | |
| (psi at 25% deflection) | 26 |
| (psi at 65% deflection) | 52 |
| **Flammability Characteristics** | |
| Smoke Development (ASTM D-2843T) | |
| Time - Seconds To: 10% LO | 4.1 |
| Time - Seconds To: 20% LO | 7.6 |
| Time - Seconds To: 30% LO | 14.5 |
| Ignition and Propagation (ASTM D-1692 -45°) | |
| Self-Extinguishing | ---- |
| Burning rate, inches/minute | 26.5[a] |
| Flame Penetration Test (Modified Bureau of Mines Test) | |
| Burn-Through Time, seconds | 5.0 |
| Limiting Oxygen Index Test | |
| $n_{O_2} / (n_{O_2} + n_{N_2})$ | 0.168 |

[a]Samples completely consumed during testing.

TABLE III

RIGID-URETHANE FOAM — CHEMICAL CHARACTERISTICS

Formulation

| Ingredients | pbw |
|---|---|
| Polymethylene polyphenyl isocyanate | 134.70 |
| Propoxylated adduct - trimethylolpropane (M.W. 312) | 100.00 |
| Silicone surfactant | 1.00 |
| Trichlorofluoromethane | 30.00 |
| Triethylene diamine | 3.53 |

Elemental Analysis (Calculated)

| Element | % by weight |
|---|---|
| Carbon | 69.0 |
| Oxygen | 17.0 |
| Nitrogen | 9.0 |
| Hydrogen | 5.0 |

TABLE IV

RIGID-URETHANE FOAM — PHYSICAL PROPERTIES
AND FLAMMABILITY CHARACTERISTICS

| | |
|---|---|
| **Physical Properties** | |
| Density, lbs/ft$^3$ | 2.1 |
| % Closed Cells, Corrected | 92.0 |
| Compression Strength, psi yield, (70°F, 0.02" minutes) | 32 |
| **Flammability Characteristics** | |
| **Smoke Development (ASTM D-2843T)** | |
| Time - Seconds To: 10% LO | 12.5 |
| Time - Seconds To: 20% LO | 18.5 |
| Time - Seconds To: 30% LO | 24.0 |
| Time - Seconds To: 40% LO | 30.0 |
| Time - Seconds To: 50% LO | 35.5 |
| Time - Seconds To: 60% LO | 42.0 |
| **Ignition and Propagation (ASTM D-1692 -45°)** | |
| Inches burned/minute | 11.0 |
| **Char Structure** | Weak |
| **Flame Penetration Test (Modified Bureau of Mines Test)** | |
| Burn-Through Time, seconds | 14.2 |
| % Weight Loss | 61.3 |
| **Limiting Oxygen Index Test** | |
| $n_{O_2} / (n_{O_2} + n_{N_2})$ | 0.187 |

TABLE V

GAS CHROMATOGRAPHIC COLUMNS SELECTED FOR ANALYTICAL STUDIES

| Column | Specification | Gas Species |
|---|---|---|
| Porapak Q series<br>Porapak R | 8' x 1/8" 80/100 ss<br>8' x 1/8" 80/100 ss | $H_2$,$N_2$,$O_2$,Ar,CO,NO,$N_2O$,$CH_4$,<br>$CO_2$,$H_2O$,$H_2S$,$SO_2$,$C_2H_6$,COS,HCN |
| Chromosorb 101 | 8' x 1/8" 80/100 ss | glycols, alcohols, free acids |
| Chromosorb 103 | 8' x 1/8" 80/100 ss | amines |
| 10% SP-1000 on washed Chromosorb W | 8' x 1/8" 100/120 ss | alcohols, free acids |
| 20% DEGS on Chromosorb W (AW) | 8' x 1/8" 80/100 ss | $C_8$-$C_{24}$ fatty acids |
| 10% Apiezon L wax on Chromosorb W (HP) | 8' x 1/8" 80/100 ss | hydrocarbons - aromatics |
| 10% Carbowax 20M on Chromosorb W (HP) | 8' x 1/8" 80/100 ss | alcohols, ketones, acetates, essential oils |
| Tenax GC | 8' x 1/8" 80/100 ss | high boiling polar compounds, alcohols, polyethylene glycol compounds, diols, phenols, mono- and diamines, ethanolamines, amides, aldehydes, and ketones |
| OV-1 | 8' x 1/8" 80/100 ss | hydrocarbons |
| 3% Dexsil 300 GC | 8' x 1/8" 100/120 ss | high temperature hydrocarbons |

TABLE VI

PEAK IDENTIFICATION — THERMAL DECOMPOSITION PRODUCTS RESULTING FROM FLEXIBLE-URETHANE FOAM

| Peak Number | Formula | Name |
|---|---|---|
| 1 | CO | Carbon monoxide |
| 2 | $CH_4$ | Methane |
| 2A | $N_2O$ | Nitrous oxide |
| 3 | $CO_2$ | Carbon dioxide |
| 4 | $C_2H_4$ | Ethylene |
| 5 | $C_2H_2$ | Acetylene |
| 6 | $C_2H_6$ | Ethane |
| 7 | $H_2O$ | Water |
| 8 | $C_3H_6$ | Propylene |
| 9 | $CH_4O$ | Methanol |
| 10 | $C_2H_4O$ | Acetaldehyde |
| 11 | N.I. | --- |
| 12 | $C_2H_6O$ | Dimethyl ether |
| 13 | $C_2H_6O$ | Ethanol |
| 14 | $C_3H_6O$<br>$C_3H_6O$ | Propionaldehyde and<br>Acetone |
| 15 | $C_3H_8O$ | Propanol |
| 16 | N.I. | --- |
| 17 | $C_4H_{10}O_2$ | 2-Ethoxyethanol |
| 18 | N.I. | --- |
| 19 | N.I. | --- |
| 20 | N.I. | --- |
| 21 | N.I. | --- |
| 22 | N.I. | --- |
| 23 | N.I. | --- |
| 24 | N.I. | --- |
| 25 | N.I. | --- |
| 26 | $C_6H_{14}O_2$ | 2-Isopropoxy-1-propanol |
| 27 | $C_6H_{14}O$ | Di-N-propyl ether |

TABLE VII

PEAK IDENTIFICATION — THERMAL DECOMPOSITION PRODUCTS RESULTING FROM RIGID-URETHANE FOAM

| Peak Number | Formula | Name |
|---|---|---|
| 1 | $CO$ | Carbon monoxide |
| 2 | $CH_4$ | Methane |
| 3 | $CO_2$ | Carbon dioxide |
| 4 | $C_2H_4$ | Ethylene |
| 5 | $C_2H_6$ | Ethane |
| 6 | $H_2O$ | Water |
| 7 | $C_3H_6$ | Propylene |
| 8 | $C_3H_8$ | Propane |
| 9 | $C_2H_6O$ | Ethanol |
| 10 | $CFCl_3$ | Trichlorofluoromethane |
| 11 | $C_3H_6O$ | Propionaldehyde |
| 12 | N.I. | --- |
| 13 | N.I. | --- |
| 14 | $C_5H_6O$ | 2-Methyl furan |
| 15 | N.I. | --- |
| 16 | $C_5H_8O$ | Dihydropyran |
| 17 | $C_6H_{10}O$ | 2-5 Dimethylfuran |
| 18 | N.I. | --- |
| 19 | $C_5H_{10}O$ | 2-Penten-1-ol |
| 20 | N.I. | --- |
| 21 | $C_7H_8$ | Toluene |
| 22 | $C_7H_{12}O_2$ | 2-Penten-1-acetate |
| 23 | N.I. | --- |
| 24 | N.I. | --- |
| 25 | Structure unknown | Compounds resulting from isocyanate degradation |
| 26 | Structure unknown | |
| 27 | Structure unknown | |
| 28 | Structure unknown | |
| 29 | Structure unknown | |

TABLE VIII

MASS SPECTRUM OF CHROMATOGRAPHIC PEAK 10

| MASS | ABUND |
|---|---|
| 12 | 1.3 |
| 13 | 4.5 |
| >14 | 15.8 |
| 16 | 2.6 |
| 25 | 2.2 |
| 26 | 4.9 |
| 27 | 2.9 |
| >28 | 12.7 |
| >29 | 90.4 |
| 30 | 1.1 |
| 40 | 1.2 |
| 41 | 5.9 |
| >42 | 14.9 |
| >43 | 56.2 |
| >>44 | 100.0 |
| 45 | 3.4 |

TABLE IX

COMPUTER IDENTIFICATION OF UNKNOWN SPECTRA

| Number of Possible Criteria | Number Criteria Found | Identification |
|---|---|---|
| 8 | 6 | ETHANAL (ACETALDEHYDE)<br>44 10164 3000 $C_2H_4O$<br>100,117, API 293 |
| 12 | 5 | N-METHYLOL ACRYLAMIDE<br>101 7296 3000 $C_4H_7NO_2$<br>101,113,126, AST 1341 |
| 12 | 5 | EPOXYETHANE<br>44 4869 3000 $C_2H_4O$<br>103,110, MSC 4869 |
| 8 | 3 | ETHANAL (ACETALDEHYDE)<br>44 10244 3000 $C_2H_4O$<br>100,117, API 373 |
| 8 | 2 | 1,1-DIETHOXYETHANE (DIETHYLACETAL: ACETAL)<br>118 10195 3000 $C_6H_{14}O_2$<br>100,115, API 324 |
| 8 | 2 | NOR-BUTANAL (NOR-BUTYRALDEHYDE)<br>72 10166 3000 $C_4H_8O$<br>100,117, API 295 |
| 8 | 2 | ETHENE (ETHYLENE)<br>28 9936 3000 $C_2H_4$<br>101, API 65 |
| 12 | 3 | GLYOXAL<br>58 7948 3000 $C_2H_2O_2$<br>117, DOW 46 |
| 12 | 3 | ACETALDEHYDE<br>44 7926 3000 $C_2H_4O$<br>100,117, DOW 24 |
| 8 | 2 | ETHYLENE<br>28 7907 3000 $C_2H_4$<br>101, DOW 5 |

TABLE X

PRODUCTS OBTAINED DURING THE PYROLYSIS OF FLEXIBLE-URETHANE FOAM

| Product | 300°C | | 500°C | | 750°C | | 1000°C | |
|---|---|---|---|---|---|---|---|---|
| | µg | % | µg | % | µg | % | µg | % |
| CO | -- | -- | -- | -- | 4.45 | 0.19 | 44.20 | 1.65 |
| $CH_4$ | -- | -- | 1.42 | .06 | 9.35 | 0.41 | 46.60 | 1.74 |
| $CO_2$ | 6.44 | 0.21 | 12.47 | .55 | 3.00 | 0.13 | 29.80 | 1.12 |
| $C_2H_4$ | -- | -- | 0.36 | .02 | 14.07 | 0.62 | 39.50 | 1.49 |
| $C_2H_2$ | -- | -- | -- | -- | -- | -- | 0.14 | 0.05 |
| $C_2H_6$ | -- | -- | 0.84 | .04 | 14.40 | 0.68 | 12.00 | 0.45 |
| $H_2O$ | 2.95 | 0.10 | -- | -- | 12.66 | 0.55 | 12.40 | 0.47 |
| $C_3H_6$ | 2.49 | 0.08 | 31.17 | 1.38 | 10.66 | 0.47 | 142.10 | 5.35 |
| $CH_3OH$ | -- | -- | -- | -- | 0.49 | 0.02 | 1.51 | 0.06 |
| $CH_3CHO$ | Trace | | 16.05 | 0.71 | 42.37 | 1.86 | 126.40 | 4.75 |
| $CH_3CH_2OH$ | -- | -- | 1.13 | 0.05 | 2.52 | 0.11 | 4.57 | 0.17 |
| $CH_3CH_2CHO$ | Trace | | 10.73 | 0.47 | 24.05 | 5.46 | 279.50 | 10.40 |
| $CH_3CH_2CH_2OH$ | -- | -- | 1.75 | 0.08 | 4.39 | 0.19 | 10.31 | 0.38 |
| TDI | 7.15 | 0.23 | 14.28 | 0.63 | 6.35 | 0.28 | 11.55 | 0.41 |

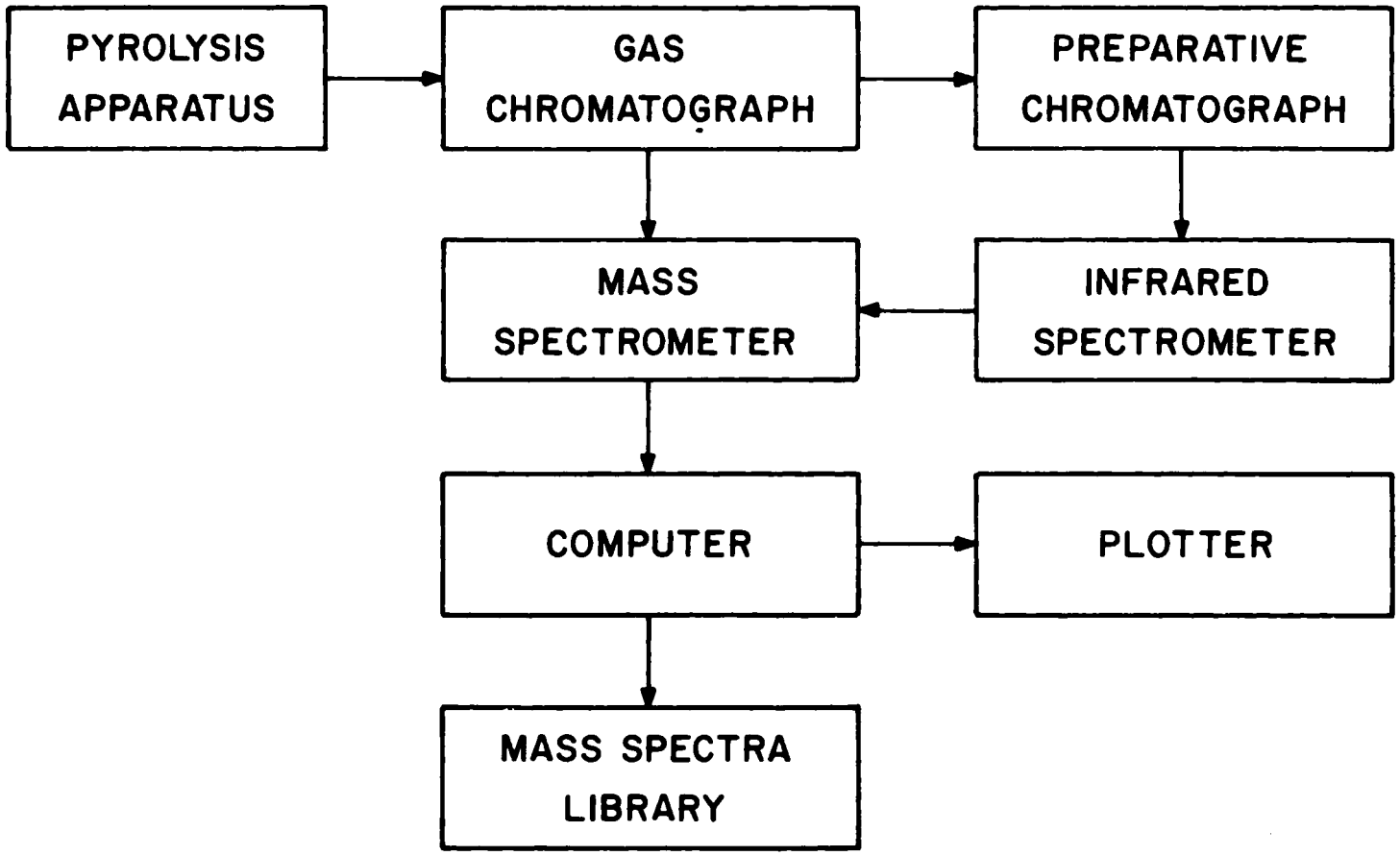


FIGURE 1. Schematic Representation of Computerized Analytical System.

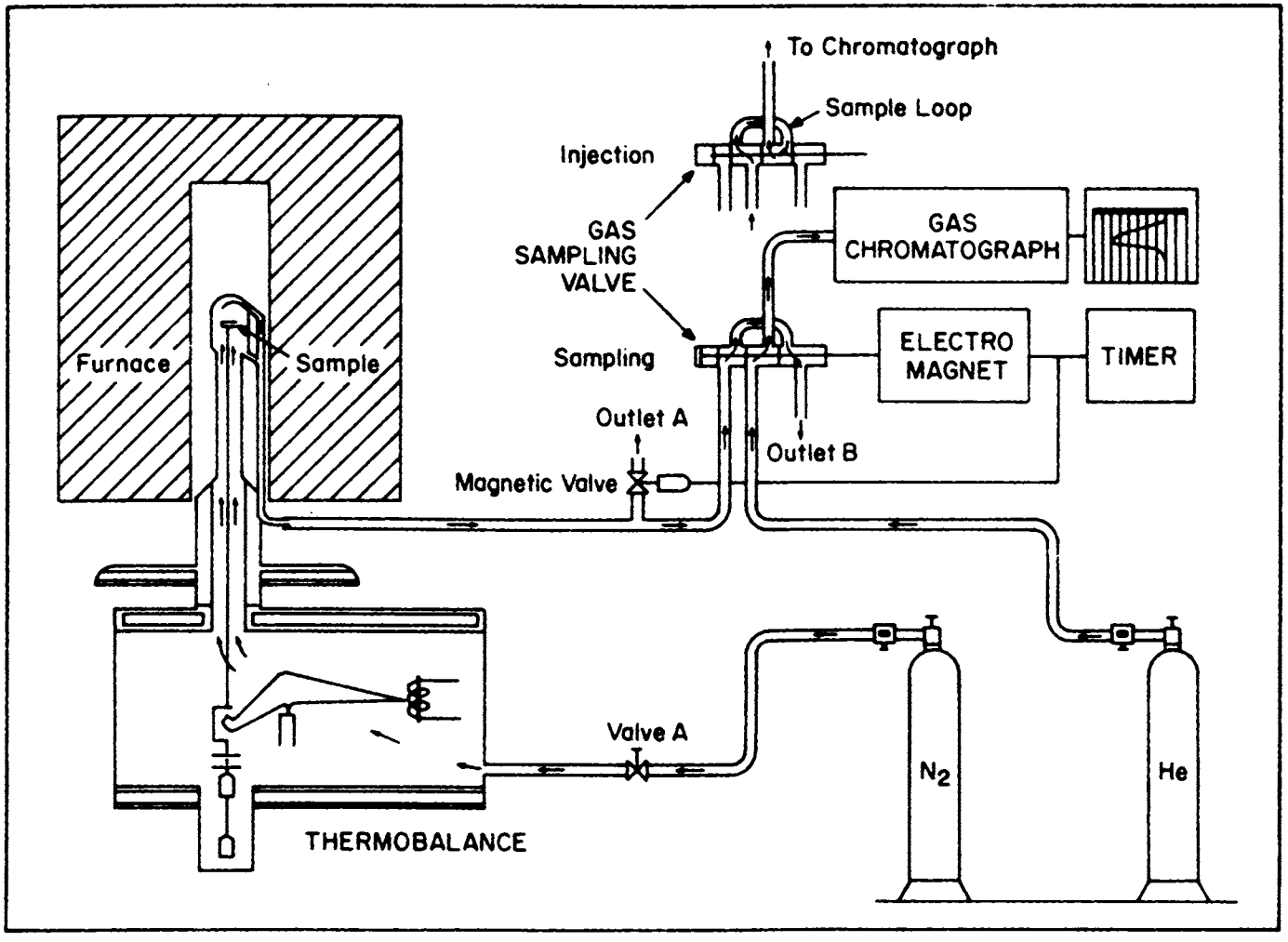


FIGURE 2. Schematic Layout for Simultaneous DTA, TGA, DTG, and EGA.

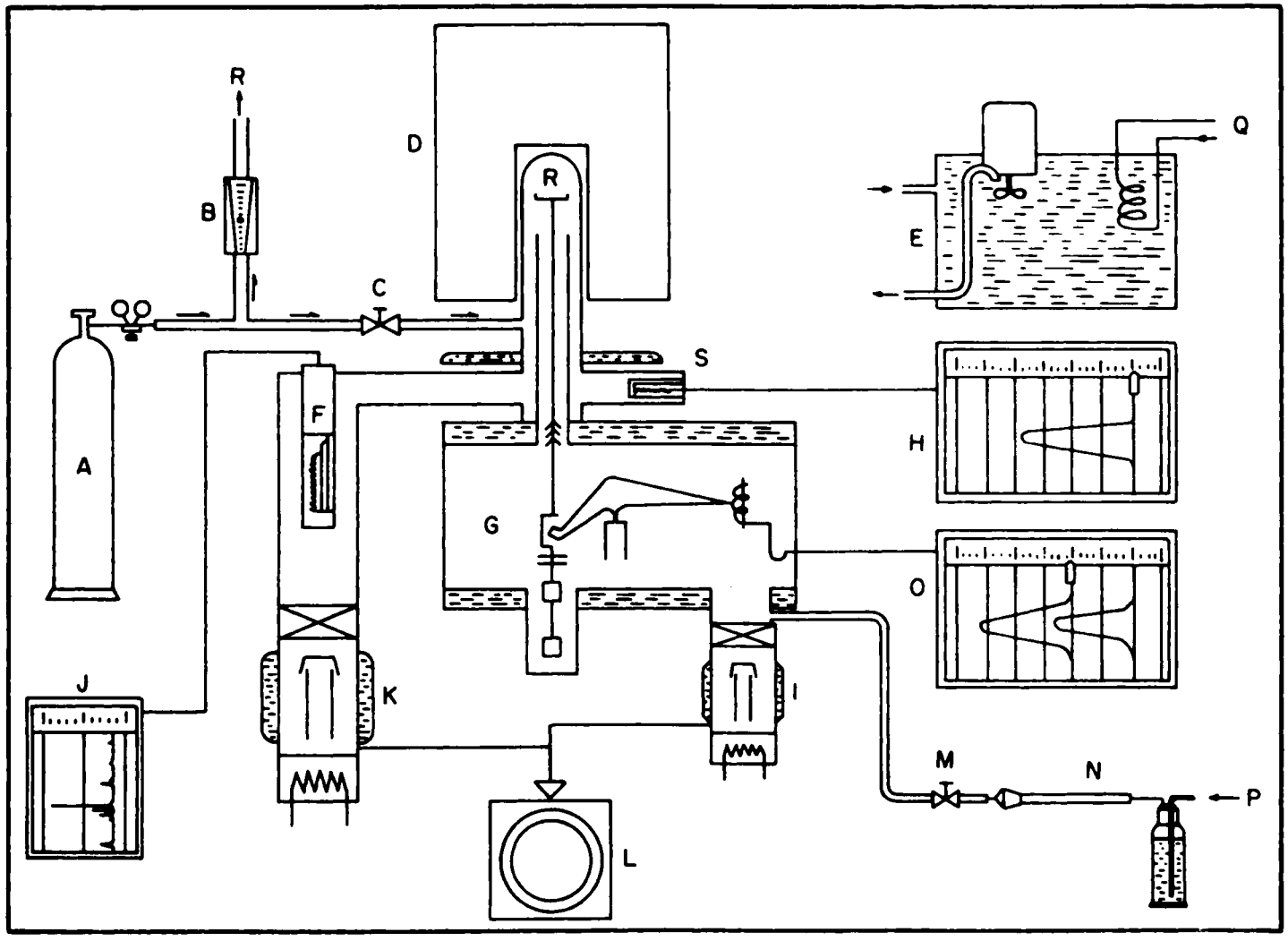


FIGURE 3. Simultaneous Thermal Analysis and Mass Spectrometry.

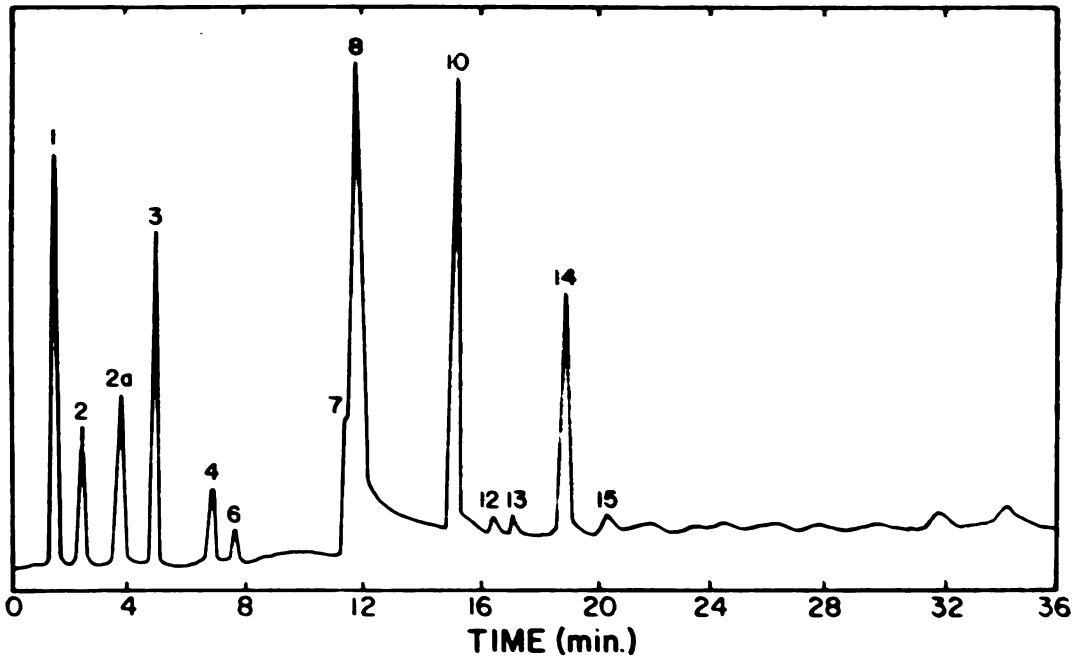


Figure 4a. Thermal Decomposition - Chromosorb-101 (TC).

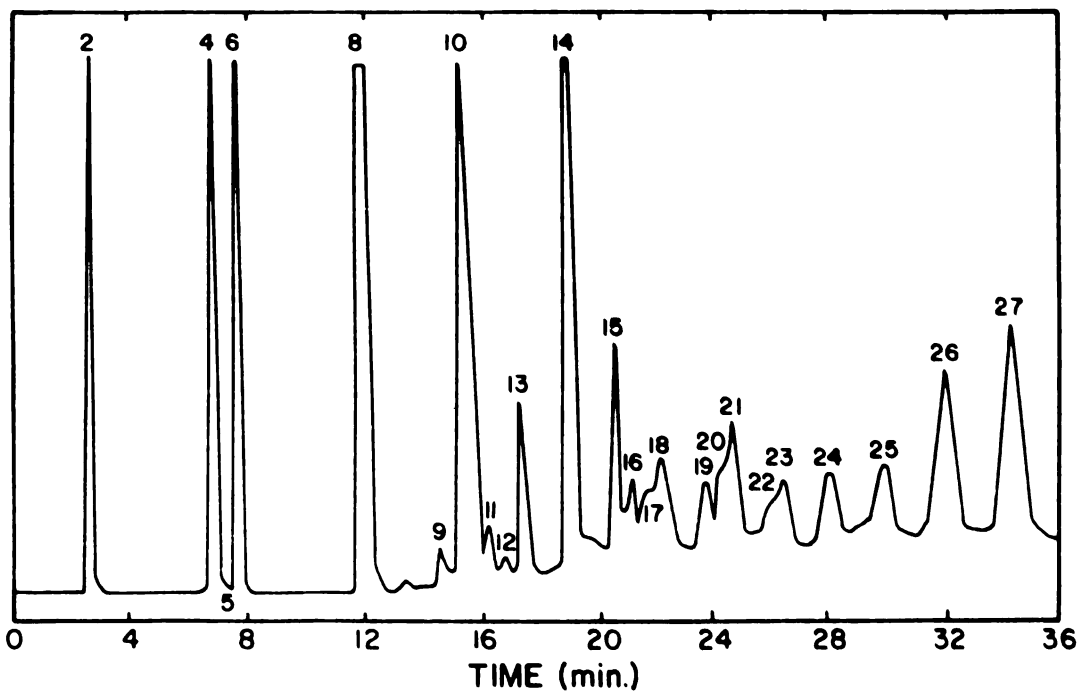


Figure 4b. Thermal Decomposition - Chromosorb-101 (FID).

FIGURE 4. Thermal Decomposition of Flexible-Urethane Foam.

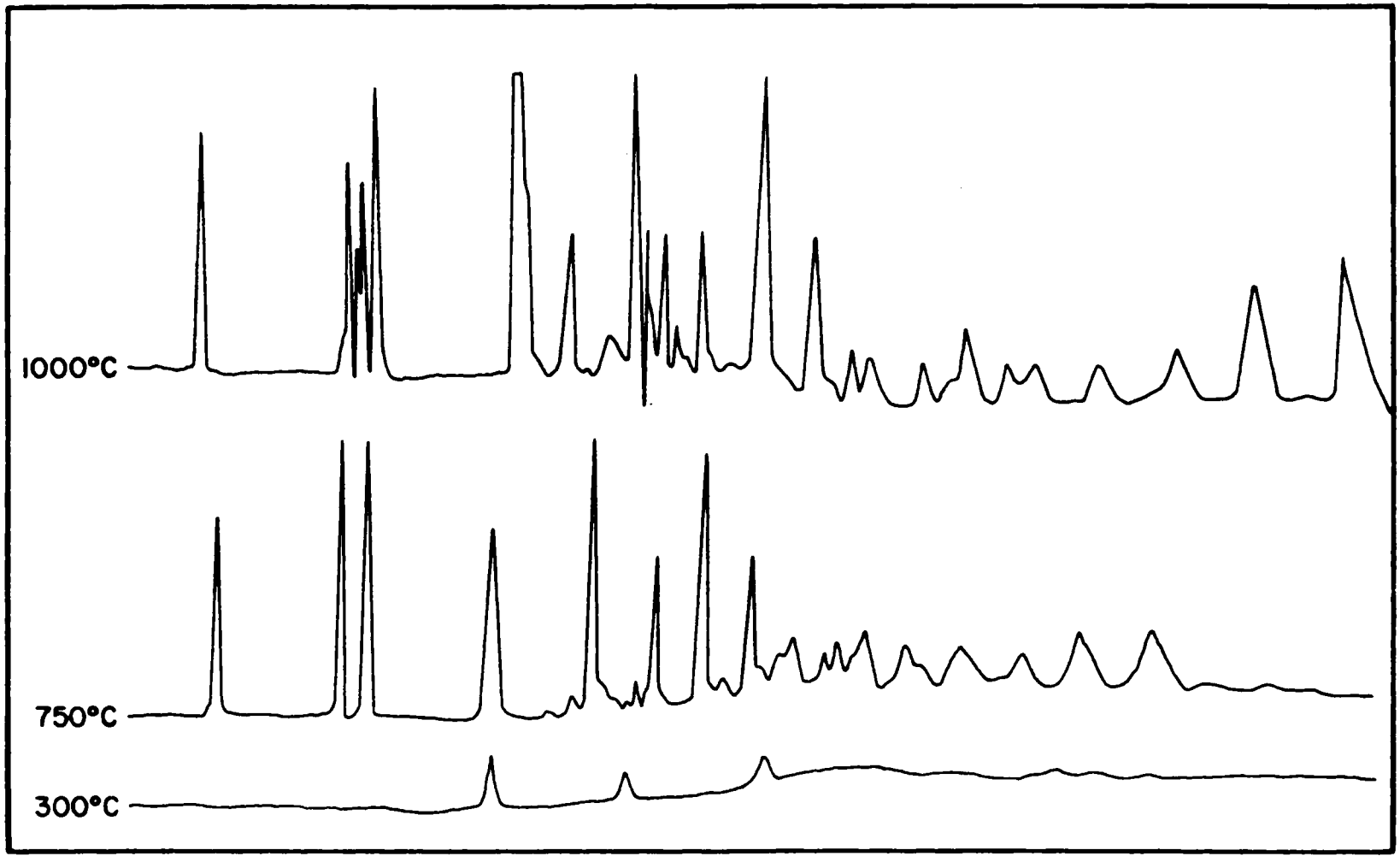


FIGURE 5. Effect of Temperature on Pyrolysis Products. Chromosorb-101 Chromatograms of Flexible-Urethane Decomposition Products (FID).

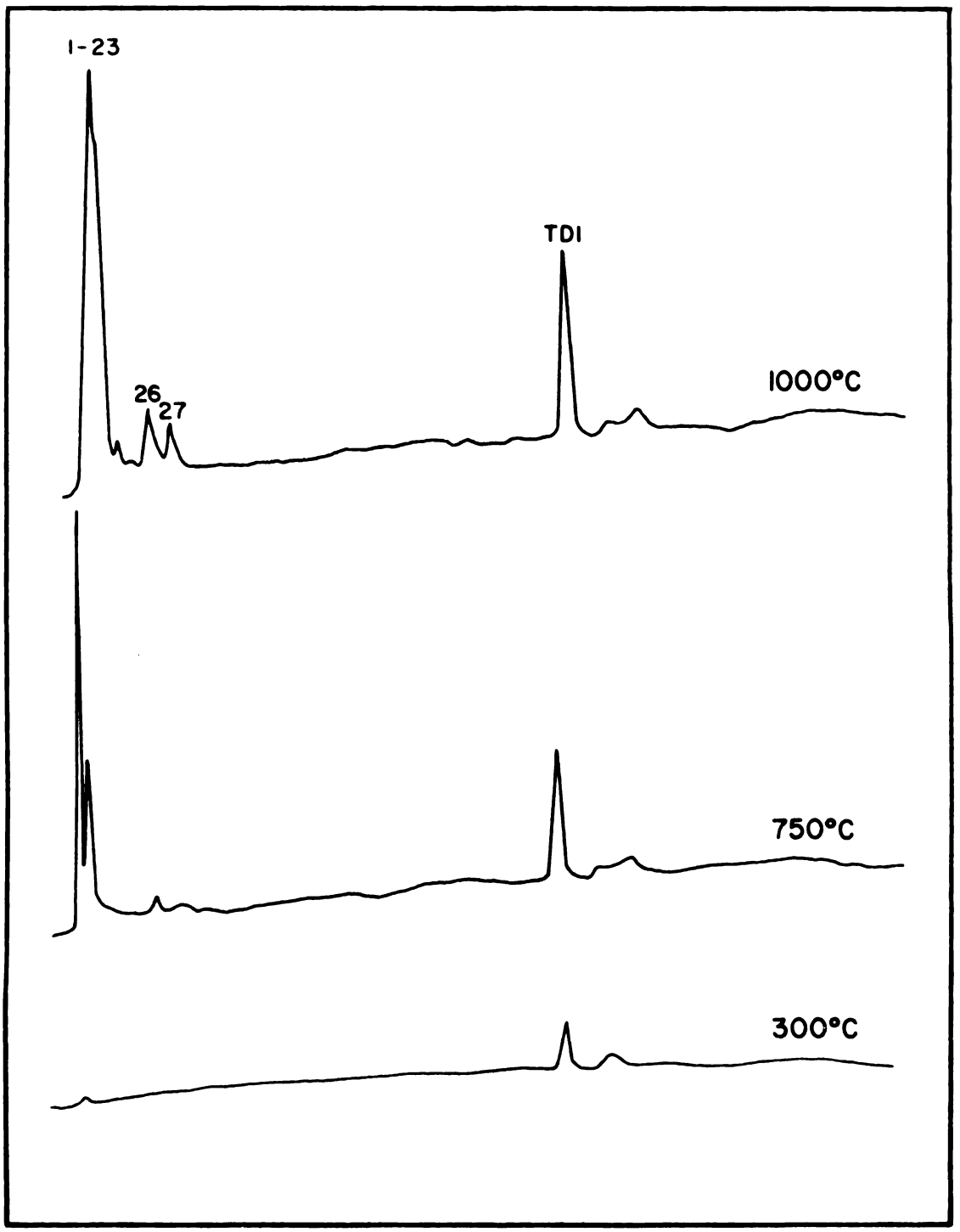


FIGURE 6. Effect of Temperature on Pyrolysis Products from Flexible-Urethane Foam - Dexsil Chromatograms (TC).

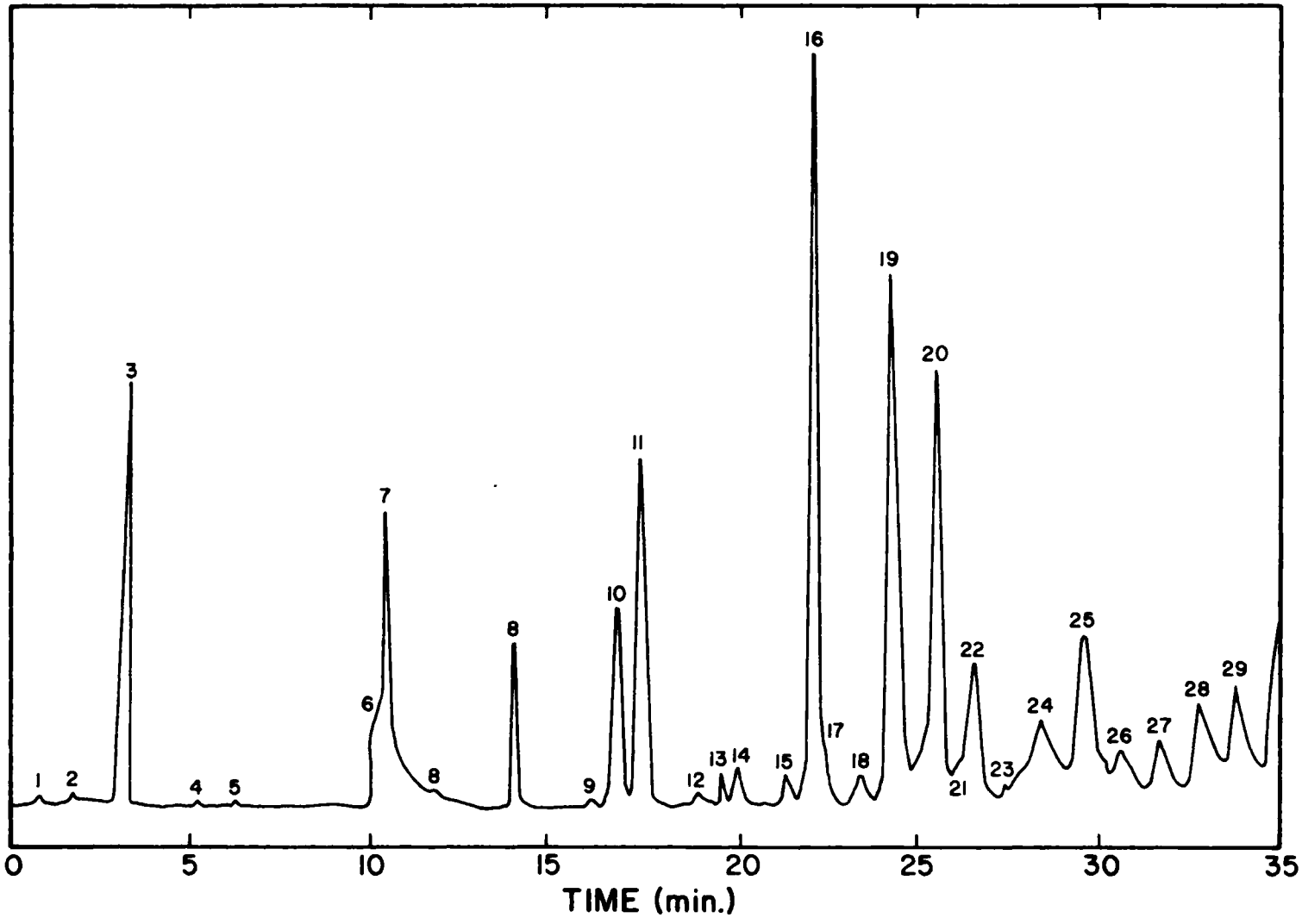


FIGURE 7. Thermal Decomposition of Rigid-Urethane Foam - Chromosorb-101 (TC).

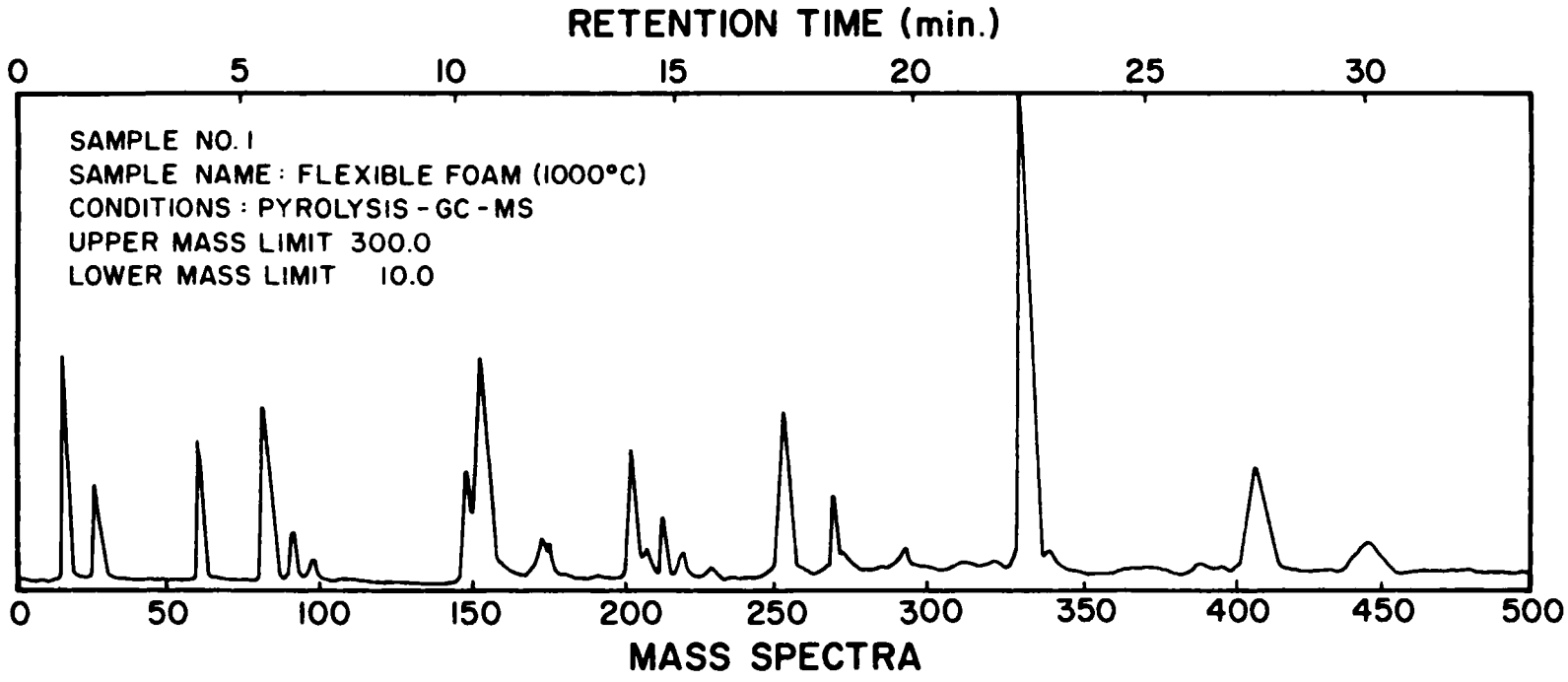


FIGURE 8. Computer Reconstructed Mass Chromatogram.

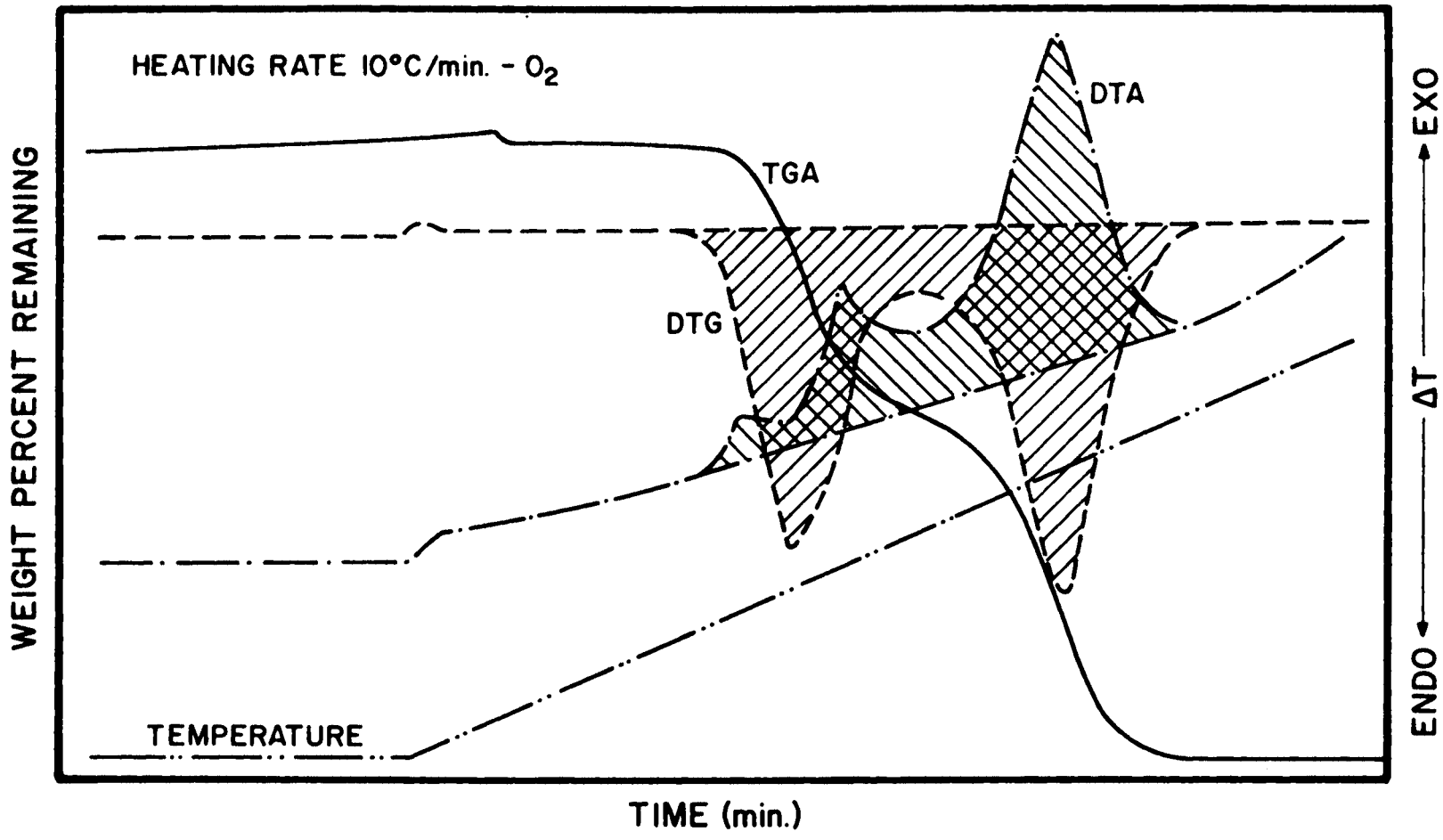


FIGURE 9. Thermal Decomposition of Rigid-Urethane Foam.

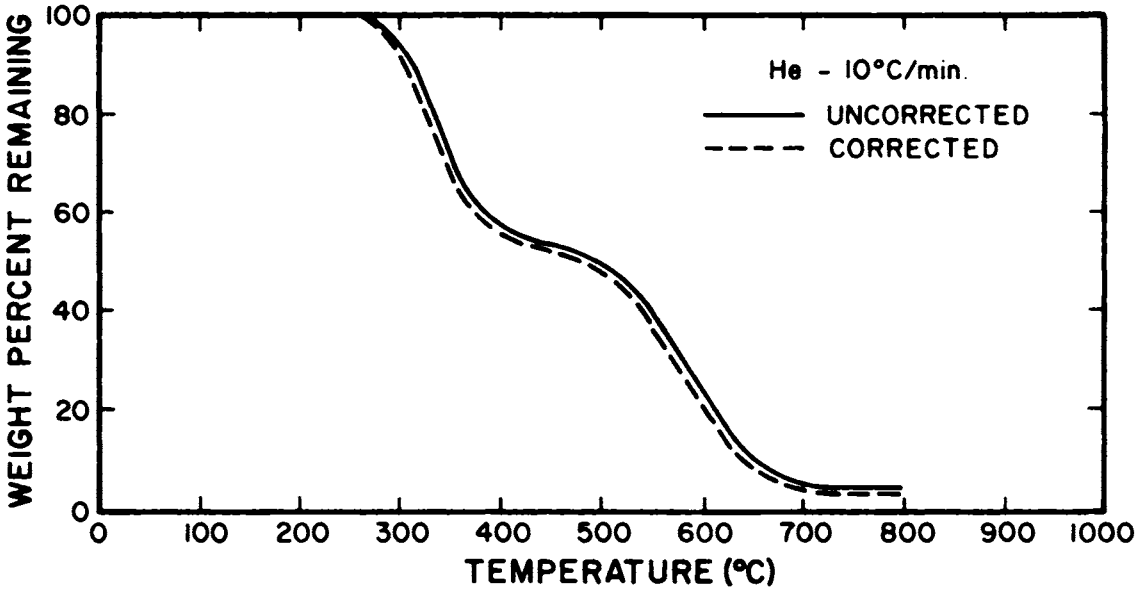


Figure 10a. TGA Rigid-Urethane Foam Thermal Decomposition Showing Buoyancy Correction.

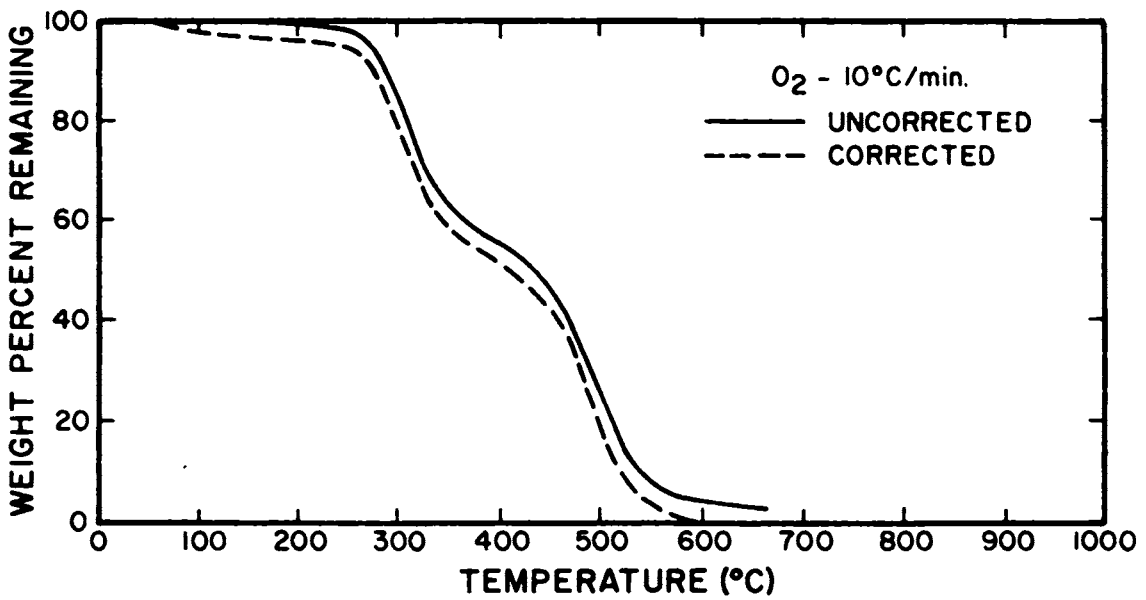


Figure 10b. TGA Rigid-Urethane Foam Thermal Decomposition Showing Buoyancy Correction.

FIGURE 10. Computer Corrections for Buoyancy Occurring During Thermal Decomposition.

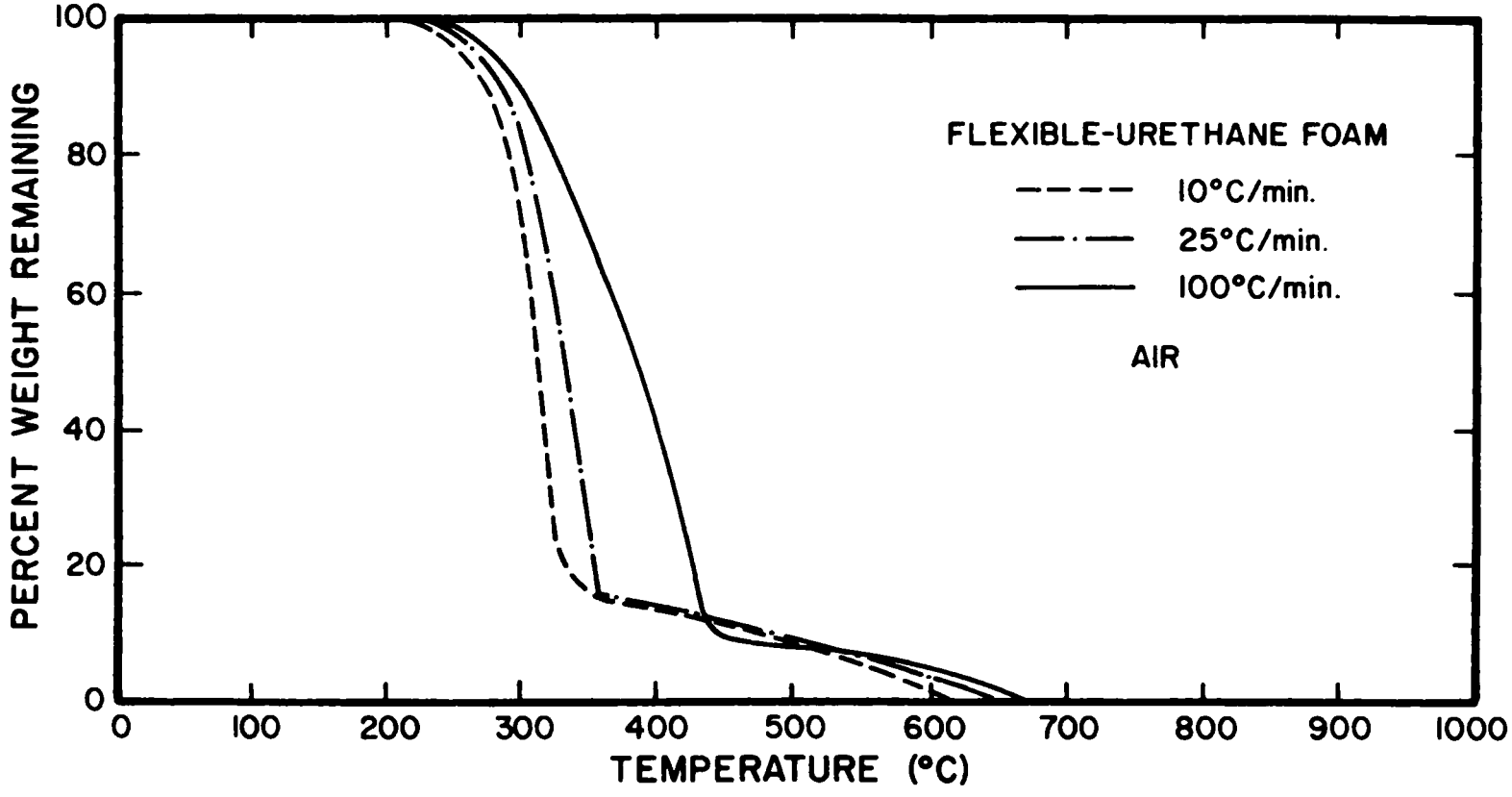


FIGURE 11. Effect of Heating Rate on Thermal Degradation.

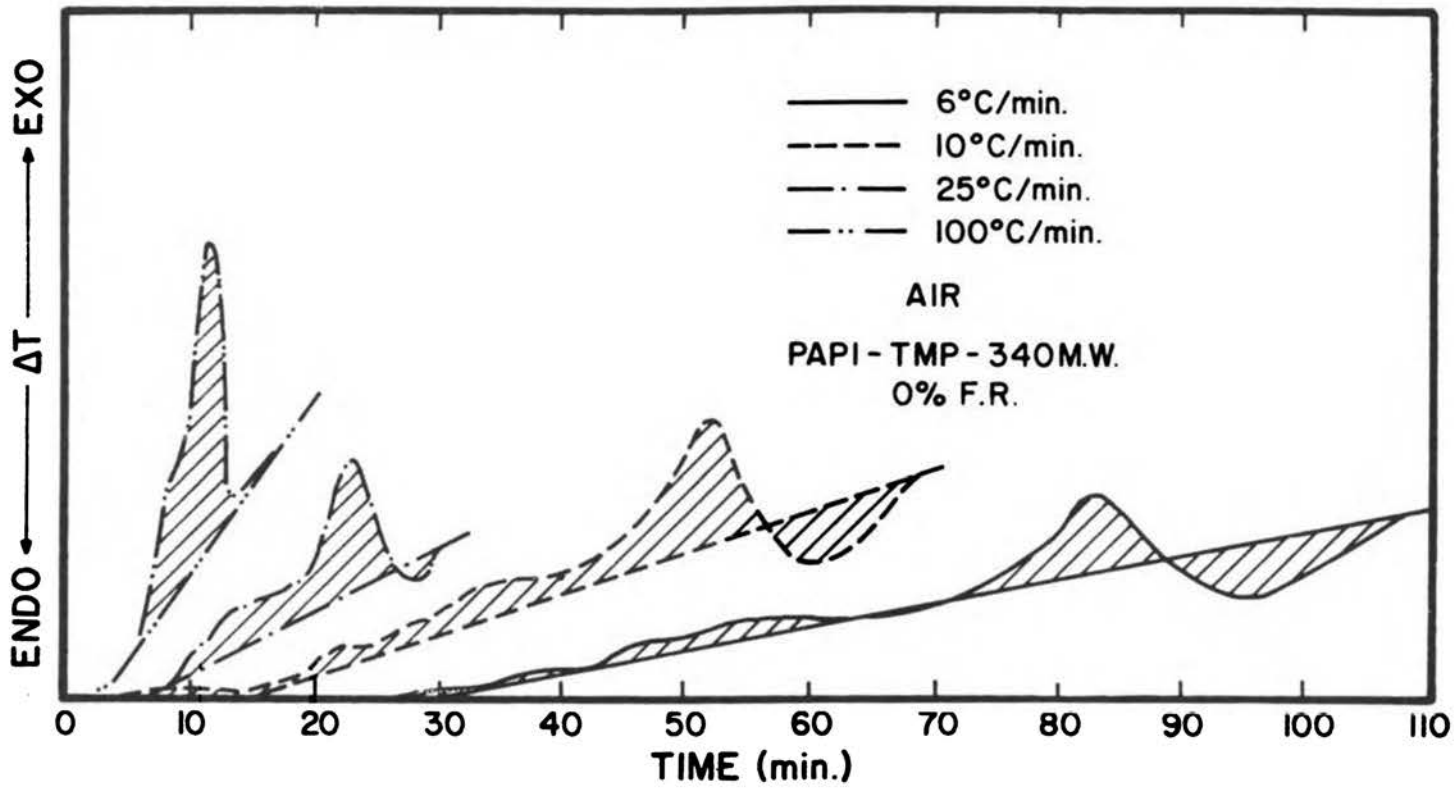


FIGURE 12. Effect of Heating Rate on DTA Curves.

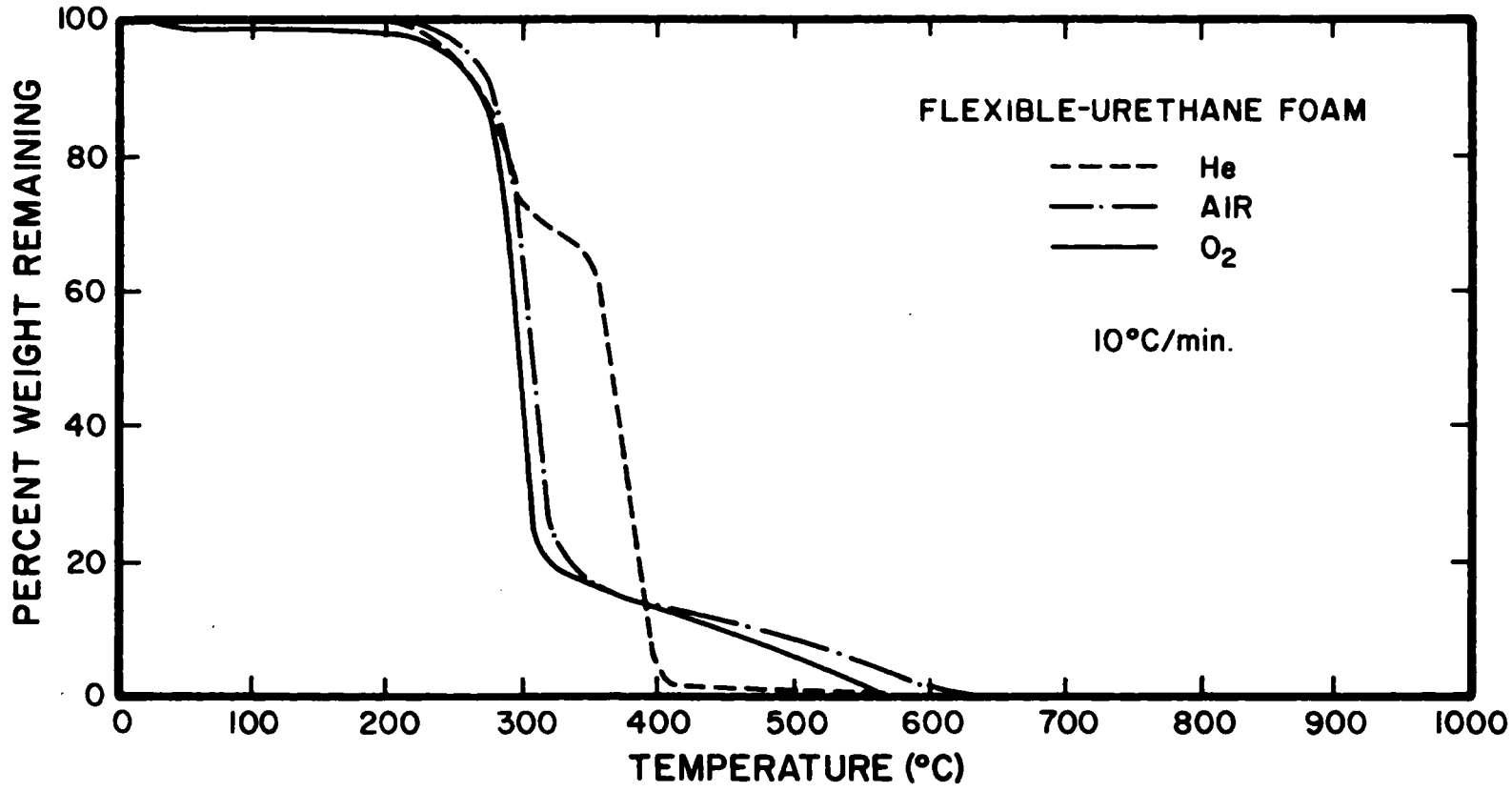


FIGURE 13. Effect of Environment on Thermal Degradation.

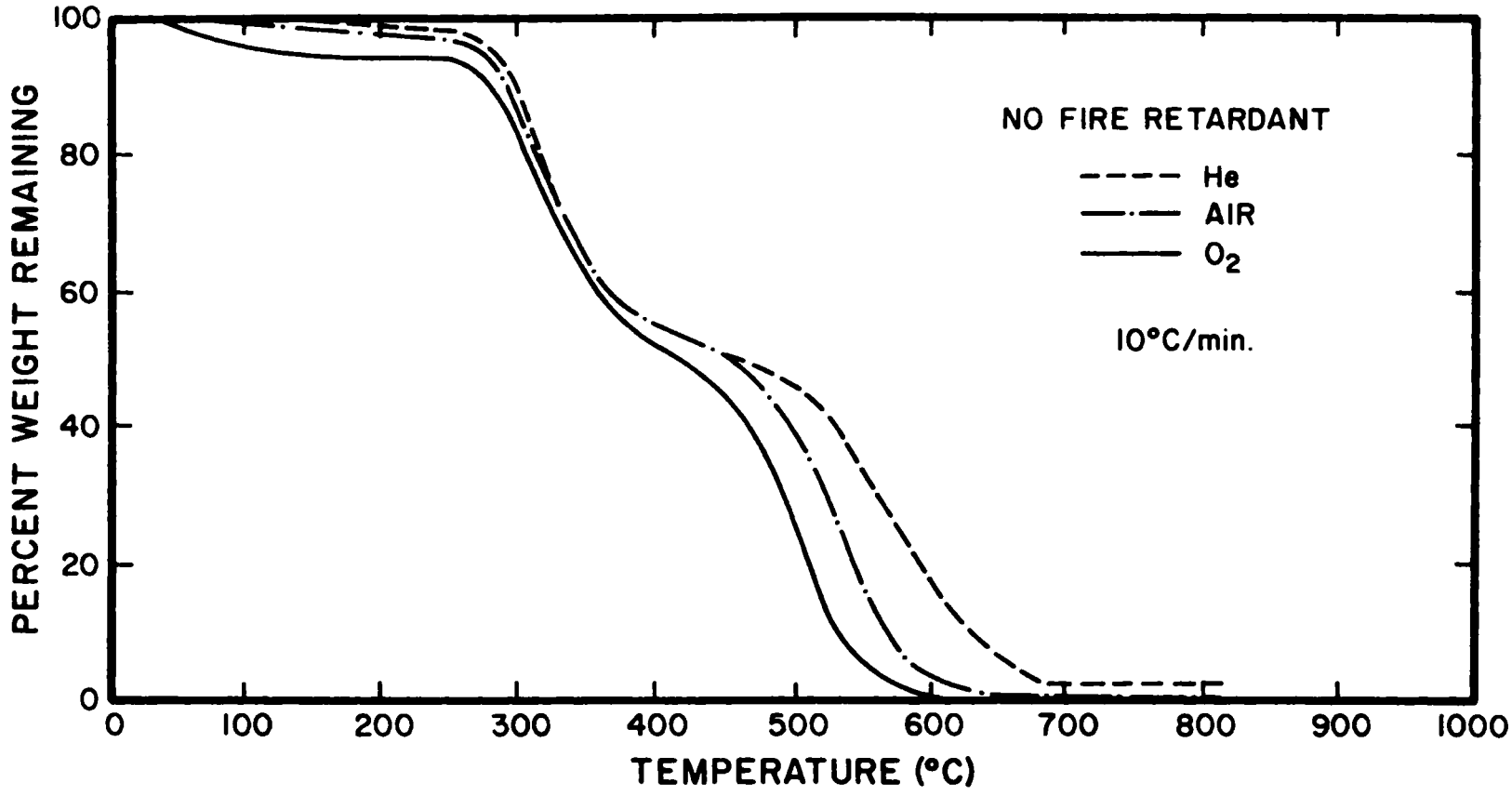


FIGURE 14. Effect of Environment on Thermal Degradation.

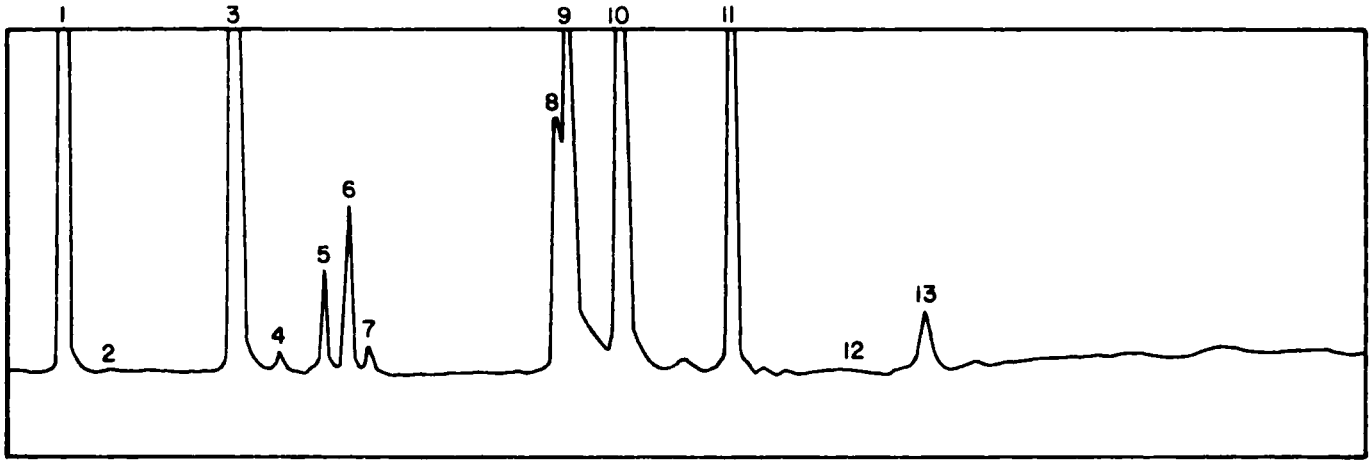


Figure 15a. Combustion of Flexible-Urethane Foam.

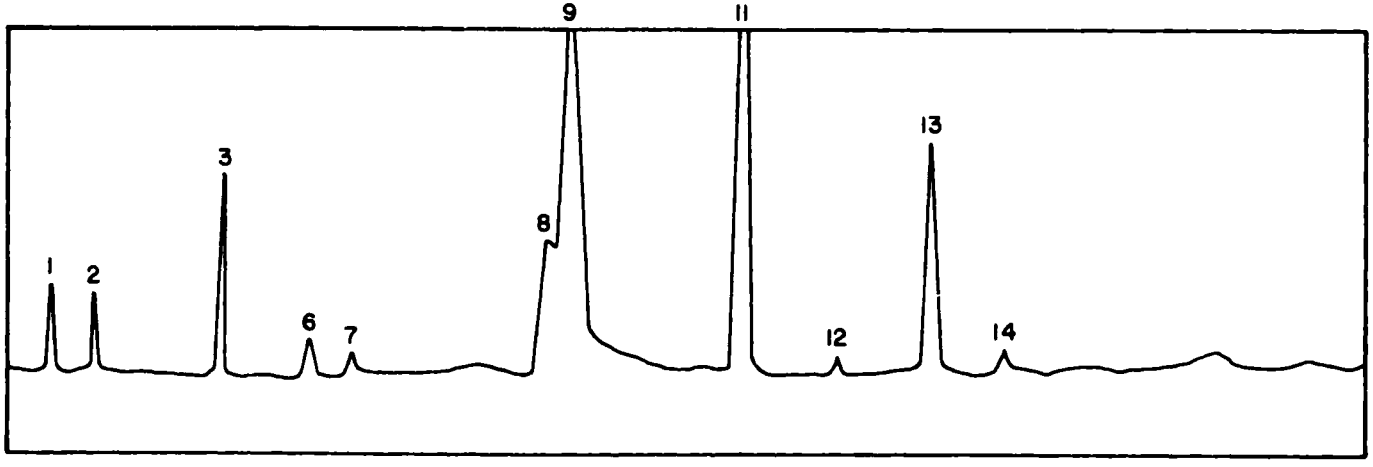


Figure 15b. Pyrolysis of Flexible-Urethane Foam.

1. CO
2. $CH_4$
3. $CO_2$
4. $N_2O$
5. $C_2H_4$
6. $C_2H_2$
7. $C_2H_6$
8. $H_2O$
9. $C_3H_6$
10. HCN
11. $C_2H_4O$
12. N.I.
13. $C_6H_6$

FIGURE 15. Comparison Degradation Products from Combustion and Pyrolysis of Flexible-Urethane Foam.

# APPENDIX

# *BIOMEDICAL RESEARCH OF THE DEFENSE CIVIL PREPAREDNESS AGENCY*
(A Literature Overview)

James W. Kerr
Emergency Operations Systems Division
Defense Civil Preparedness Agency
Washington, D. C.

The 1950 Act of Congress that set up a civil defense organization and the numerous executive orders that have served over the decades to implement the legislation, have consistently focused at the federal level on nuclear war. In contrast, states, counties and cities have had the job of preparing for peacetime disasters as well. This partial partnership was somewhat remedied in 1972 when DCPA moved into helping plan for peacetime events.

A key word here is PLAN. DCPA has no "troops"; there is no federal "Civil Defense Corps" as seen in some foreign countries. Even the local CD people are largely volunteers. Federal operating budgets have ranged from trivial to moderate, with the average on the small side. What we have been able to produce, in addition to grants and transfers of federal property, is advice and uniform guidance that interprets and transmits to local users the output of advanced technology and high-level decisions.

DCPA concern with the toxicology of combustion products is obvious. The Agency has national responsibility for the well-being of the population in times of great threat to life and property. A population that is moving into or taking refuge in shelter (from radioactive fallout, flood, explosion, conflagration, or what have you) is in a vastly different vulnerability posture from a population at home or at work. What effect has this shift in posture on the life safety of the people of the U.S.? What operational constraints are posed by the presence of active threats such as fallout, or CO, or HCN? How is the performance of public safety forces degraded (physically or psychologically) by the presence of these threats or by the measures that must be taken to counteract their presence or to coexist with it? To get a bit more specific, what sort of case load must medical planners contemplate, as they prepare to cope?

Over the years we have sponsored or actually carried out a number of tests and studies leading up to Federal Preparedness Guide inputs. Many of these have become classics in the field. Perhaps our greatest leadership has been in the nuclear radiation area, but the literature is full of citations of civil preparedness research on combustion products and their effects on people and organizations. It might not be obvious to a technically

oriented audience, but it is a simple fact that if we do not have some feel for what the disaster environment is, we cannot talk sense about a disaster organization. Thus, DCPA analyses often concentrate on organizational performance, much to the amazement of, say, a biophysicist we are paying to test CO on mice. Major technical programs relevant to this symposium have been funded by DCPA at IIT Research Institute, Southwest Research Institute, Stanford Research Institute, and in various government laboratories.

Distillation of the results is to be found in guidance material now moving into the field. We have structured the operating environment in such a fashion as to provide detailed checklists that allow local planners to prepare contingency plans for all the major threats to their locality. The first such checklist addressed the nuclear war problems of a city. Others cover natural disasters, and still others the problem of relocating part or all of the population prior to the disaster event. Full integration of the various checklists is a study topic now, and the relocation concept is now receiving operating and planning emphasis. Feedback from the field enables us to upgrade the product.

The rather extensive bibliography appended hereto was prepared as an adjunct to our in-house study project on medical support programs. We think the scope is impressive, and that it illustrates quite well both the nature of our federal effort and the depth of the supporting research program.

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# *PYROLYSIS OF A FLEXIBLE-URETHANE FOAM**

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## INTRODUCTION

Flexible-urethane foams, representative of typical formulations used in commerce, were selected as materials for study because of their involvement in numerous fires. The principle concern with this product is that when it is burned, it may release toxic and noxious products such as cyanides, carbon monoxide, aldehydes, isocyanates, alcohols, and low-molecular-weight alkanes, alkenes, and aromatic compounds. This paper discusses the results of the analysis of the inert atmosphere pyrolysis products of this foam in an attempt to establish the methodology for continued studies in the oxidative degradation and flaming combustion product analysis.

## EXPERIMENTAL

### Polymer System

A flexible-urethane foam produced on a commercial production facility was obtained and characterized. Table I presents a summary of the foam's formulation and chemical composition. Table II summarizes the physical properties and flammability characteristics of this flexible-urethane foam.

### Instrumentation

Polymer samples were pyrolyzed, using a Hewlett-Packard Model 80 pyrolyzer, directly in the injection port of a Hewlett-Packard Model 7620 gas chromatograph. All eluted peaks were split 10:1 between a thermal conductivity and flame ionization detector with the responses simultaneously recorded on a dual pen recorder. A Hewlett-Packard Model 5930A mass spectrometer was connected in series after the thermal conductivity detector and was used for qualitative identification of all eluted peaks. In order to facilitate data acquisition and refinement, the mass spectrometer was coupled

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*Portions of this paper were used by Professor Einhorn in the presentation "Computerized Analytical System for the Analysis of Thermal Decomposition Products."

to a Hewlett-Packard 5933A system equipped with the appropriate data handling software.

When applicable, a Varian MAT CH-7 mass spectrometer, modified for chemical ionization,[1] was used to identify materials for which conventional methods were insufficient.

Carbon, hydrogen, and nitrogen analyses were performed on an F&M Model 185 carbon-hydrogen-nitrogen analyzer. A Perkin-Elmer Model 237B grating infrared spectrometer was used for all IR analyses.

Procedure

Typically, 2 to 3 mg of the polymer was pyrolyzed at temperatures ranging from 300°C to 1000°C. The pyrolysis products were separated on a variety of columns including Chromosorb-101, Chromosorb-103, Porapak Q, QV-1, Dexsil 300 GC, and Carbowax 20M. Identification of the products was obtained from the tabulation of the corresponding mass spectrum of a particular GC peak. To aid in identification, the Aldermaston library of mass spectra data was available for computer searching. As an additional aid, retention indices based on the principle of Kovats[2,3,4] were determined for all compounds on the columns used. Thus, the combination of retention index and mass spectral data allowed identification of all major pyrolysis products. Quantitative analysis of the pyrolysis products was facilitated utilizing the peak area recorded by the interfaced electronic integrator. The peak areas were corrected for response factors[5] and referenced to ethanol as an internal standard for quantification.

Whenever possible, the residue remaining after pyrolysis was weighed in order to obtain a mass balance. In addition, CHN and IR analyses were made on the residue to check on elemental composition and the presence of various functional groups. The components of the residue were frequently separated, using thin layer chromatography with the components being identified using either electron impact or chemical ionization mass spectrometry.

RESULTS

In the initial analysis of the pyrolysis products, a 1/8" x 16' stainless-steel column packed with Chromosorb-101 was used for component separation. Figure 1 shows a typical chromatogram with the major peaks numbered and the identification of those peaks given in Table III. Table III also shows the method of identification for each peak. The chromatograms from several runs at a variety of pyrolysis temperatures is shown in Fig. 2, and a summary of the quantitative data are presented in Table IV.

From the results given above, it is apparent that the major constituents being eluted from the Chromosorb-101 column are oxygen-containing compounds or compounds that would result principally from the breakdown of the polyol. This is pointed out by the fact that no nitrogen-containing compounds were found. When a sample of the pure polyol was pyrolyzed (Fig. 3 and Table

III),[6] oxygen-containing compounds such as ethanal, propanal, and acetone were eluted. Pyrolysis of TDI, conducted at 1000°C utilizing a Chromosorb-101 column, led to separation of components 1-10 (Table III) plus small amounts of benzene and toluene.

In order to ensure that no nitrogen-containing compounds were being irreversibly absorbed onto the Chromosorb-101, a different porous polymer, Chromosorb-103, designed to separate nitrogen-containing compounds, was used. When the pyrolysis mixture was separated on a 1/8" x 8' stainless-steel column, packed with this support (Fig. 4 and Table V), several nitrogen-containing compounds, such as propanitrile and benzonitrile are eluted which had not shown significant peaks on the Chromosorb-101 column. However, it must be noted that all peaks observed on the Chromosorb-101 are also observed on the Chromosorb-103.

An interesting comparison of the pyrolysis of the TDI, polyol and urethane, respectively, is shown in Fig. 5. These pyrolyses products were all separated on the same Chromosorb-103 column. It is interesting to note that peaks such as methane, ethane, ethene, propene, propyne, and allene result from the decomposition of both the TDI and the polyol. In contrast, carbon dioxide appears to come principally from the decomposition of the TDI since little or no $CO_2$ is detected from the pyrolysis of the polyol on either the Chromosorb-101 or Chromosorb-103. The oxygen-containing compounds such as aldehydes and ketones result from the decomposition of the polyol. The nitrogen-containing compounds, such as benzonitrile, and other aromatics—benzene, toluene and styrene—result from the decomposition of the TDI. What is most evident from Fig. 5 is that the pyrolysis products of the urethane can be obtained by combining the pyrolysis products of the individual constituents. This strongly suggests that the decomposition process is relatively independent of urethane linkage.

Additional columns were used to ensure that no higher boiling components were being missed due to excessive retention times. The chromatograms in Fig. 6 show the results of the pyrolysis of the urethane at various temperatures with the components being separated on a 1/8" x 8' three percent Dexsil column. As a point of reference, peaks 26 and 27 of Fig. 6 correspond to those of the same number in Fig. 1. The major additional peak eluted on this column is TDI.

The remaining residue after completion of the pyrolysis was recovered and examined. This residue usually consisted of deep yellow crystals. Carbon, hydrogen, and nitrogen analyses were performed on these crystals and the following results were obtained:

| | C | H | N | O |
|---|---|---|---|---|
| Polymer | 61.66 | 8.74 | 5.63 | 23.12 |
| Yellow crystals | 47.95 | 5.47 | 8.52 | 38.06 |

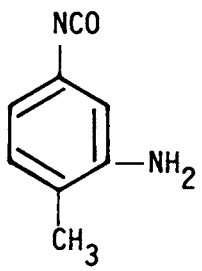
For comparative purposes, the CHN results were given for both the original starting polymer and for the residue. The high-nitrogen content of residue

points out that the nitrogen is being concentrated in the residue during pyrolysis. This, in turn, may account for the low percentage of nitrogen-containing compounds which are eluted via the GC.

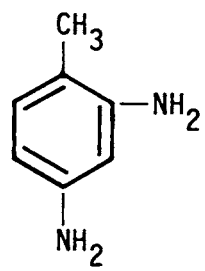
When the residue was subjected to infrared analysis the spectra shows the presence of the carbonyl band at 1725 $cm^{-1}$ and the ether stretch at 1100 $cm^{-1}$ which is characteristic of the original polymeric material. When these yellow crystals were, in turn, pyrolyzed (Fig. 7), the products were readily comparable to the pyrolysis chromatogram of the original urethane. This suggests that the yellow crystals were simply smaller molecular-weight pieces of the original foam.

The total weight percent of volatiles from Table IV comprises approximately 30% of the original weight of the sample. The amount of residue remaining, if repyrolyzed, would readily bring the mass balance to nearly 100%.

When the residue was analyzed by electron impact mass spectrometry, the resulting spectra obtained were so complex as to be of little use. This resulted from the residue being a mixture of components which would subsequently give overlaying spectra. In order to resolve this problem, the principle of chemical ionization mass spectrometry was used. Using methane as the ionizing reagent gas, the major peaks of the volatile materials occur at masses 175, 149, and 123, respectively. These correspond to the protonated compounds having molecular weights of 174, 148, and 122. The mass at 174 corresponds to the free TDI, part of which has been eluted and detected using the Dexsil column. The lower masses correspond to succeeding degradation of the TDI to form 3-amino-4-methylphenylisocyanate, 1, and 2,4-diaminotoluene, 2.




1




2

DISCUSSION

The presence or absence of certain compounds present additional insight to the thermal breakdown of this urethane. Three major pathways[7] are postulated as occurring in urethane decompositions: (1) disassociation to the original polyol and isocyanate, (2) cleavage by a concerted reaction to produce a carbamic acid and an olefin from the polyol, and (3) a cleavage followed by loss of $CO_2$ followed by a probable intermolecular recombination of the olefin and primary amine to give a secondary amine. Products identified

in this study supports at least two of these primary decomposition modes. The results from the chemical ionization spectroscopy study of pyrolysis residue have clearly shown that a substituted isocyanate, 1, and diaminotoluene, 2, were present. The formation of these compounds could have arisen from either pathway 1 or 2 as shown in Fig. 8.

Since the polyol used in this study contains a propoxylated terminal end, two types of products could be expected from the concerted reaction, pathway 2. The β-hydrogen can be removed from either C-1 or C-3 position; thus, in one case producing an unsaturated ether 3 and in the other process producing a substituted vinyl ether 4. The absence of identification of the vinyl ethers is not surprising since these compounds are easily pyrolyzed[8] to produce propanal and another vinyl ether or a propenyl ether. The mode of decomposition of propenyl ethers has been extensively studied by Molera and co-workers.[9] Based on their work, the propenyl ethers should pyrolyze to propene plus a keto ether. The keto ether should fracture to produce acetone plus another keto ether. Although experimental evidence is not available, it is felt that ethanal must also result from the propenyl ethers.

The strongest evidence in favor of pathway 1 results from the 300°C pyrolysis, Table IV, producing as the major volatile products, propene, $H_2O$, and TDI. A trace of ethanal and propanal were also observed. This suggests that at 300°C the major route of decomposition is disassociation to produce TDI, then dehydration of the alcohol followed by cleavage to produce propene. The question of water reacting with the isocyanate is a possibility, since substantial amounts of $CO_2$ were identified. The amine produced from isocyanate and $H_2O$ would not be volatile enough to pass through the GC column. The presence of the propanal seems to indicate at 300°C there is only a small contribution of pathway 2. As the temperature is raised to above 300°C, both of these pathways are operative plus other secondary decompositions. It also should be noted that each of these mechanisms lead into a common intermediate, 4.

Although special techniques have been employed for the identification of nitrogen compounds, no 2° or 3° amines were observed. This seems to indicate that the recombination mechanism is nonoperative or contributes only a very minor amount to the overall reaction scheme.

As can be attested by the complexity of the product array resulting from a 1000°C pyrolysis, there are many reactions occurring simultaneously. To attempt to explain the formation of each of these compounds at this point, would only be at best very speculative.

## CONCLUSIONS

The combustion of polymeric materials is complex and often involves simultaneous pyrolysis, oxidative degradation, and flaming combustion processes. This paper summarizes the results obtained when flexible-urethane foams were subjected to degradation under inert pyrolysis conditions. Continuing research programs are directed to the study of urethane polymer

degradation under oxidative degradation and flaming combustion conditions. When completed, the results will be used to provide a greater insight into the total combustion process.

ACKNOWLEDGMENTS

This research, conducted at the Flammability Research Center of the University of Utah, has been supported by the National Science Foundation Grant GI-33650, under its RANN (Research Applied to National Needs) program. The authors are indebted to Dr. R. H. Long, Jr., the program monitor.

The authors further wish to acknowledge the many contributions by Professors M. M. Birky, J. H. Futrell, and J. D. Seader. Further acknowledgment is made to the analytical studies provided by Messrs. L. H. Wojcik and P. W. Ryan.

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4. Nau, H. and Bieman, K., "Computer-Assisted Assignment of Retention Indices in Gas Chromatography-Mass Spectrometry and Its Application to Mixtures of Biological Origin," *Anal. Chem., 46:*426-434 (March 1974).

5. Dietz, W. A., "Response Factors for Gas Chromatographic Analysis," *J. of Gas Chromatography* (February 1967), pp. 68-71.

6. All components separated on Chromosorb-101 are referenced to Table III by numbers above the peaks. Likewise, all components separated on Chromosorb-103 are referenced to Table V.

7. Saunders, J. H., *Rubber Chem. and Tech., 32:*327 (1959).

8. For an excellent review, see Smith, G. G. and Kelly, F. K., *Progress in Physical Org. Chem.,* Vol. 8, A. Streitwieser and R. W. Tafts, eds., New York: Wiley Interscience, 1971, pp. 195-199.

9. Molera, M. J., Gamboa, J. M., Garcia Dominques, J. A. and Couto, A., *J. Gas Chrom., 6:*594 (1968).

TABLE I

FLEXIBLE-URETHANE FOAM — CHEMICAL CHARACTERISTICS

Formulation

| Ingredients | pbw |
|---|---|
| Propoxylated triol (3000 M.W.) | 100.00 |
| Water | 3.19 |
| Silicone surfactant (L-540)[a] | 0.90 |
| Bis-dimethylaminoethyl ether (A-1)[b] | 0.08 |
| Stannous octoate[c] | 0.45 |
| Tolylene diisocyanate (80:20 mixture of 2,4/2,6 isomers) | 46.19 |

Elemental Analysis

| Element | % by Weight |
|---|---|
| Carbon | 61.66 |
| Oxygen | 23.12 |
| Nitrogen | 5.63 |
| Hydrogen | 8.74 |
| Silicon, tin | (0.005 - 0.05%) |
| Calcium, sodium, magnesium, aluminum, and potassium | (less than 0.01%) |
| Iron | (4.5 ppm) |
| Copper | (5.0 ppm) |

[a]Silicone Division, Union Carbide Corporation

[b]Union Carbide Corporation

[c]Metal and Thermit Corporation

TABLE II

FLEXIBLE-URETHANE FOAM — PHYSICAL PROPERTIES AND FLAMMABILITY CHARACTERISTICS

| | |
|---|---|
| **Physical Properties** | |
| Density, lbs/ft$^3$ | 2.0 |
| Tensile strength, psi | 19.0 |
| Elongation, % | >300 |
| Compression set, % (75% deflection, 22 hrs. at 158°F, 30 minutes recovery) | <10 |
| Load Bearing, 50 square inches | |
| (psi at 25% deflection) | 26 |
| (psi at 65% deflection) | 52 |
| **Flammability Characteristics** | |
| Smoke Development (ASTM D-2843T) | |
| Time - Seconds to: 10% LO | 4.1 |
| Time - Seconds to: 20% LO | 7.6 |
| Time - Seconds to: 30% LO | 14.5 |
| Ignition and Propagation (ASTM D-1692 - 45°) | |
| Self-extinguishing | -- |
| Burning rate, inches/minutes | 26.5[a] |
| Flame Penetration Test (Modified Bureau of Mines Test | |
| Burn-through time, seconds | 5.0 |
| Limiting Oxygen Index Test | |
| $n_{O_2} / \left(n_{O_2} + n_{N_2}\right)$ | 0.168 |

[a]Samples completely consumed during testing.

TABLE III

PEAK IDENTIFICATION — THERMAL DECOMPOSITION PRODUCTS RESULTING FROM FLEXIBLE-URETHANE FOAM ON CHROMOSORB-101

| Peak No. | Name | Method[a] |
|---|---|---|
| 1 | Carbon monoxide | GC/MS |
| 2 | Methane | GC/MS |
| 2A | Nitrous oxide | GC/MS |
| 3 | Carbon dioxide | GC/MS |
| 4 | Ethylene | GC/MS |
| 5 | Acetylene | GC/MS |
| 6 | Ethane | GC/MS |
| 7 | Water | GC/MS |
| 8 | Propylene | GC/MS |
| 9 | Methanol | GC/MS |
| 10 | Acetaldehyde | GC/MS |
| 11 | --- | |
| 12 | Dimethyl ether | GC/MS |
| 13 | Ethanol | GC/MS |
| 14 | Propionaldehyde and acetone | GC/MS |
| 15 | Propanol | |
| 16 | --- | |
| 17 | 2-Ethoxyethanol | MS |
| 18 | --- | |
| 19 | --- | |
| 20 | --- | |
| 21 | --- | |
| 22 | --- | |
| 23 | --- | |
| 24 | --- | |
| 25 | --- | |
| 26 | 2-Isopropoxy-1-propanol | MS |
| 27 | Di-N-Propoxymethane | MS |

[a]GC - indicates confirmation using Kovats' Indices.
MS - indicates confirmation using mass spectral data.

TABLE IV

PRODUCTS OBTAINED DURING THE PYROLYSIS OF FLEXIBLE-URETHANE FOAM
(Chromosorb-101 and Dexsil Columns)

| Product | 300°C | | 500°C | | 750°C | | 1000°C | |
|---|---|---|---|---|---|---|---|---|
| | µg | % | µg | % | µg | % | µg | % |
| CO | -- | -- | -- | -- | 4.45 | 0.19 | 44.20 | 1.65 |
| $CH_4$ | -- | -- | 1.42 | .06 | 9.35 | 0.41 | 46.60 | 1.74 |
| $CO_2$ | 6.44 | 0.21 | 12.47 | .55 | 3.00 | 0.13 | 29.80 | 1.12 |
| $C_2H_4$ | -- | -- | 0.36 | .02 | 14.07 | 0.62 | 39.50 | 1.49 |
| $C_2H_2$ | -- | -- | -- | -- | -- | -- | 0.14 | 0.05 |
| $C_2H_6$ | -- | -- | 0.84 | .04 | 14.40 | 0.68 | 12.00 | 0.45 |
| $H_2O$ | 2.95 | 0.10 | -- | -- | 12.66 | 0.55 | 12.40 | 0.47 |
| $C_3H_6$ | 2.49 | 0.08 | 31.17 | 1.38 | 10.66 | 0.47 | 142.10 | 5.35 |
| $CH_3OH$ | -- | -- | -- | -- | 0.49 | 0.02 | 1.51 | 0.06 |
| $CH_3CHO$ | Trace | | 16.05 | 0.71 | 42.37 | 1.86 | 126.40 | 4.75 |
| $CH_3CH_2OH$ | -- | -- | 1.13 | 0.05 | 2.52 | 0.11 | 4.57 | 0.17 |
| $CH_3CH_2CHO$ | Trace | | 10.73 | 0.47 | 24.05 | 5.46 | 279.50 | 10.40 |
| $CH_3CH_2CH_2OH$ | -- | -- | 1.75 | 0.08 | 4.39 | 0.19 | 10.31 | 0.38 |
| TDI[a] | 7.15 | 0.23 | 14.28 | 0.63 | 6.35 | 0.28 | 11.55 | 0.41 |

[a]Data obtained from a 3% Dexsil column.

TABLE V

PEAK IDENTIFICATION — THERMAL DECOMPOSITION PRODUCTS RESULTING FROM A FLEXIBLE FOAM ON CHROMOSORB-103

| Peak No. | Name | Method[a] |
|---|---|---|
| 1 and 2 | $CO/CH_4$ | GC/MS |
| 3 | $CO_2$ | GC/MS |
| 4 | Ethene | GC/MS |
| 5 | Ethane | GC/MS |
| 6 | Propene | GC/MS |
| 7 | Propyne | GC/MS |
| 8 | Allene | MS |
| 9 | 1-Butene | GC/MS |
| 10 | Ethanal + 1,3-butadiene | GC/MS |
| 11 | 1-Butene-3-yne | MS |
| 12 | N.I.[b] | |
| 13 | N.I. | |
| 14 | N.I. | |
| 15 | N.I. | |
| 16 | Propanal | GC/MS |
| 17 | Acetone | GC/MS |
| | +3 Pentene-1-yne | GC/MS |
| 18 | Propenitrile | GC/MS |
| 19 | Propanitrile | GC/MS |
| 20 | N.I. | |
| 21 | Hexatriene | GC/MS |
| 22 | Benzene | GC/MS |
| 23 | Toluene | GC/MS |
| 24 | N.I. | |
| 25 | Methylpyridine | GC/MS |
| 26 | N.I. | |
| 27 | *m*-Xylene | GC/MS |
| 28 | Styrene | GC/MS |
| 29 | Benzonitrile | GC/MS |
| 30 | Indene | MS |
| 31 | Dicyanobenzene | MS |

[a]GC - indicates confirmation using Kovats' Indices.
MS - indicates confirmation using mass spectral data.

[b]N.I. - no identification.

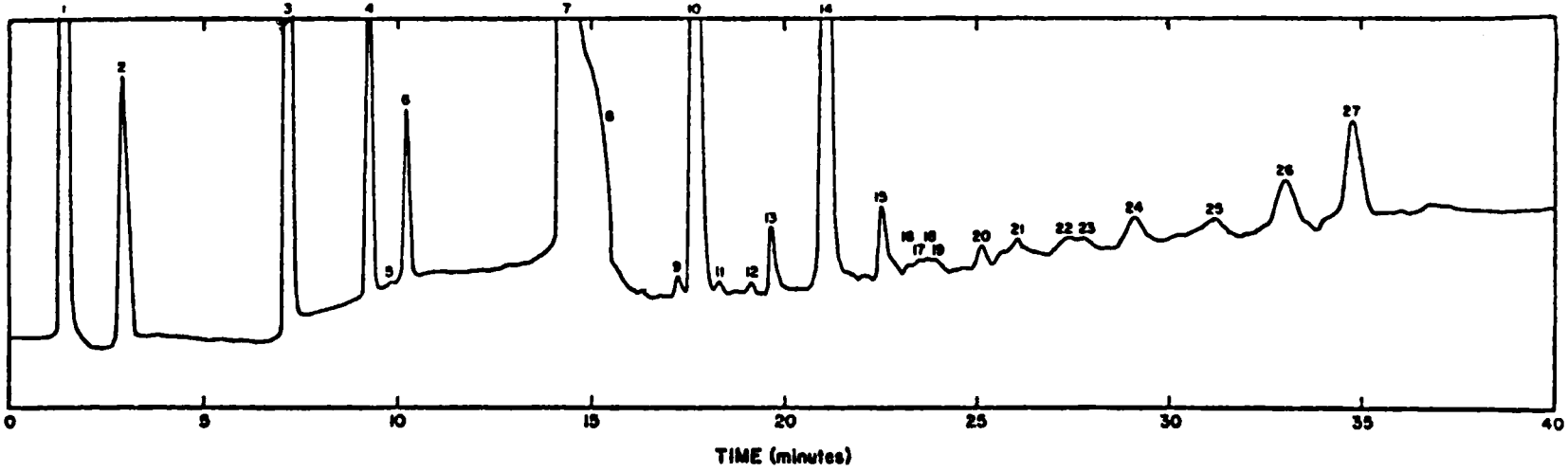


Figure 1a. Thermal Conductivity Detector (TC).

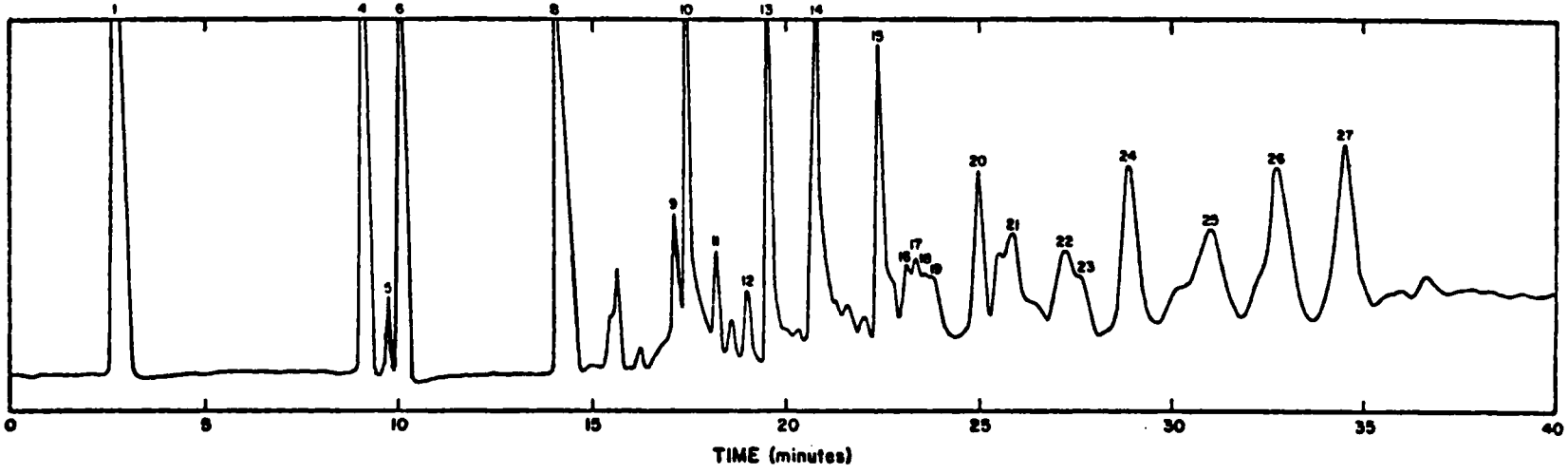


Figure 1b. Flame Ionization Detector (FID).

FIGURE 1. Chromatograms of the Products of Thermal Decomposition (1000°C) of a Flexible-Urethane Foam on Chromosorb-101.

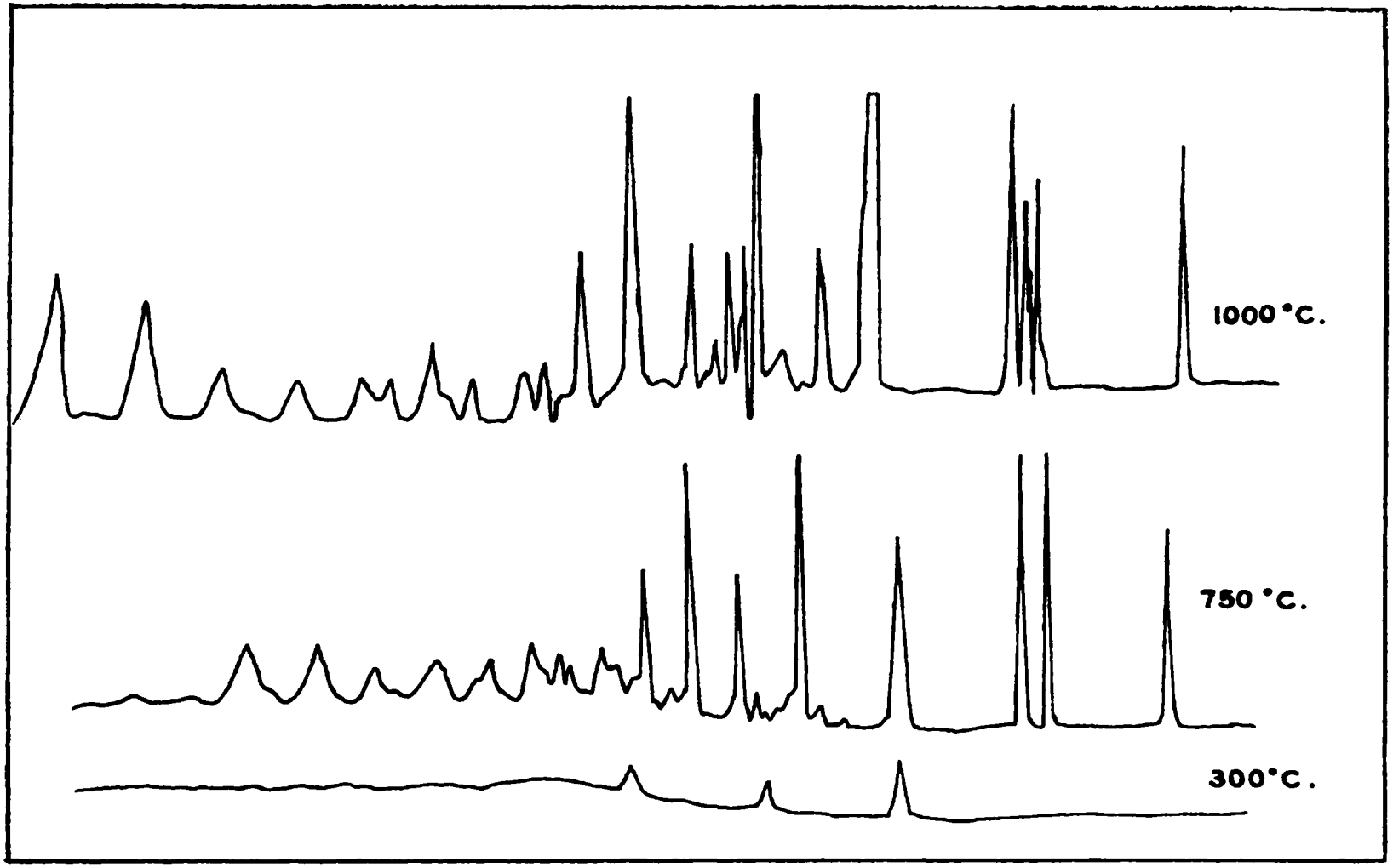


FIGURE 2. Effect of Temperature on Pyrolysis Products. Chromosorb-101 Chromatograms of Flexible-Urethane Decomposition Products (FID).

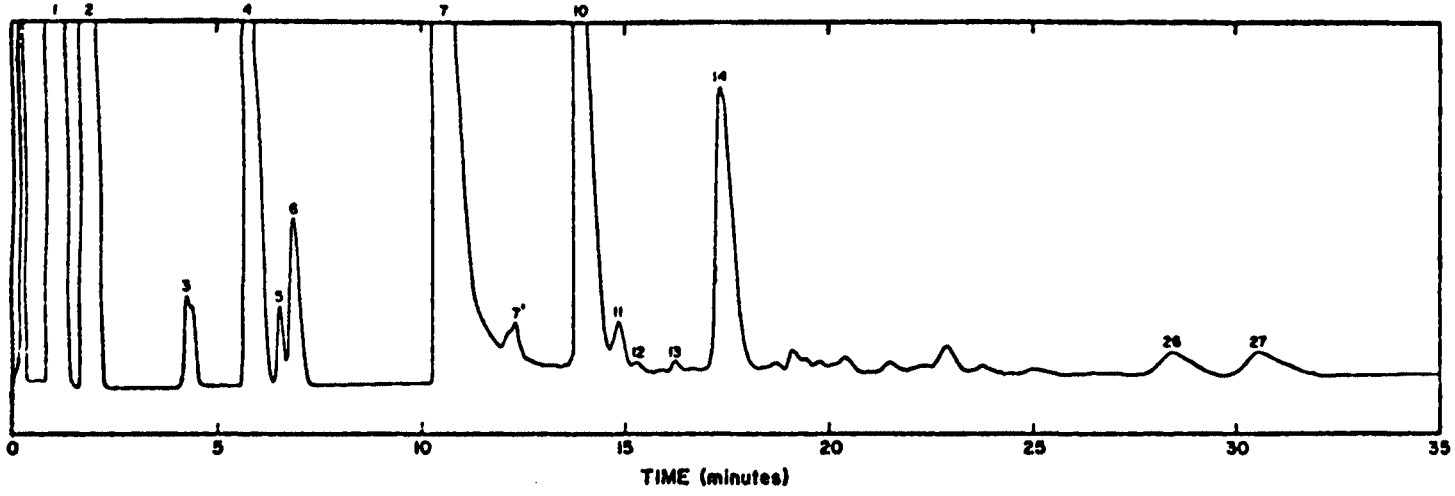


FIGURE 3. Pyrolysis (1000°C) Products of 3000 M.W. Polyol on Chromosorb-101 Column (TC).

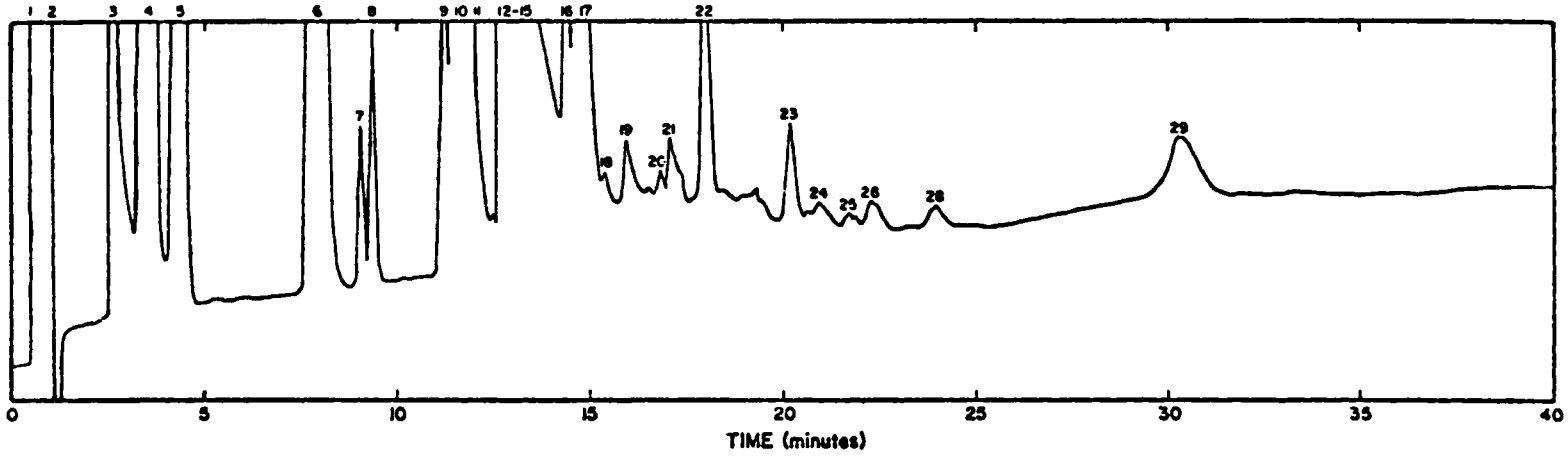


Figure 4a. Thermal Conductivity Detector.



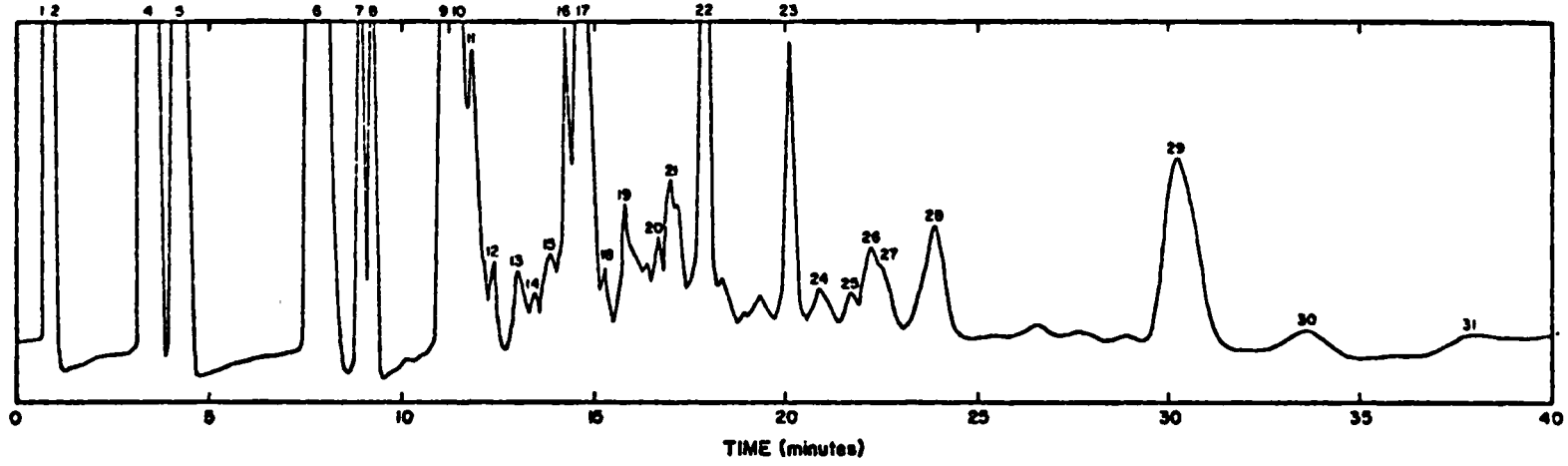


Figure 4b. Flame Ionization Detector.

FIGURE 4. Chromatograms of the Pyrolysis (1000°C) Products of a Flexible-Urethane Foam on Chromosorb-103.

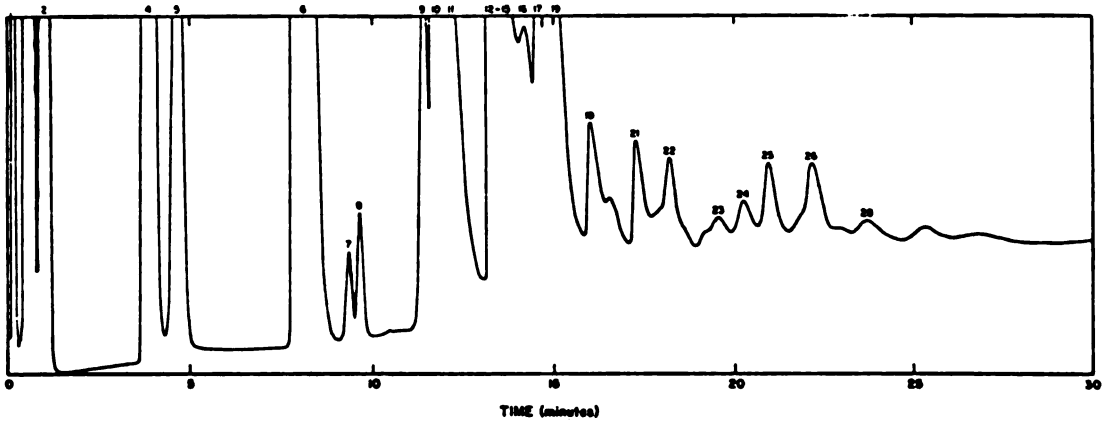


Figure 5a. Polyol.

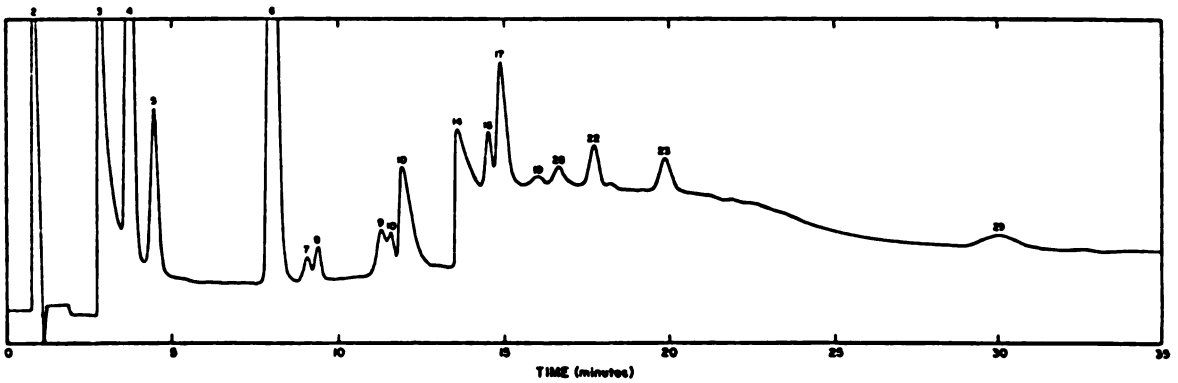


Figure 5b. TDI.

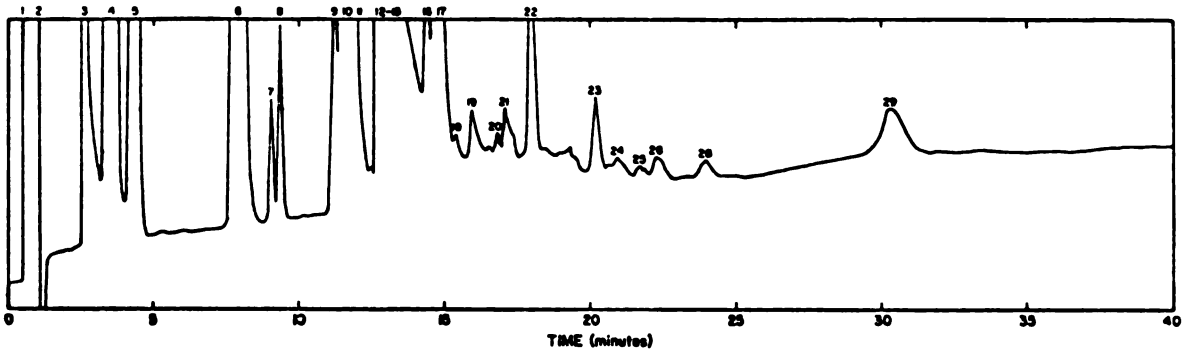


Figure 5c. Flexible-Urethane Foam.

FIGURE 5. Comparison of 1000°C Pyrolysis of TDI, Polyol and Flexible-Urethane Foam on Chromosorb-103.

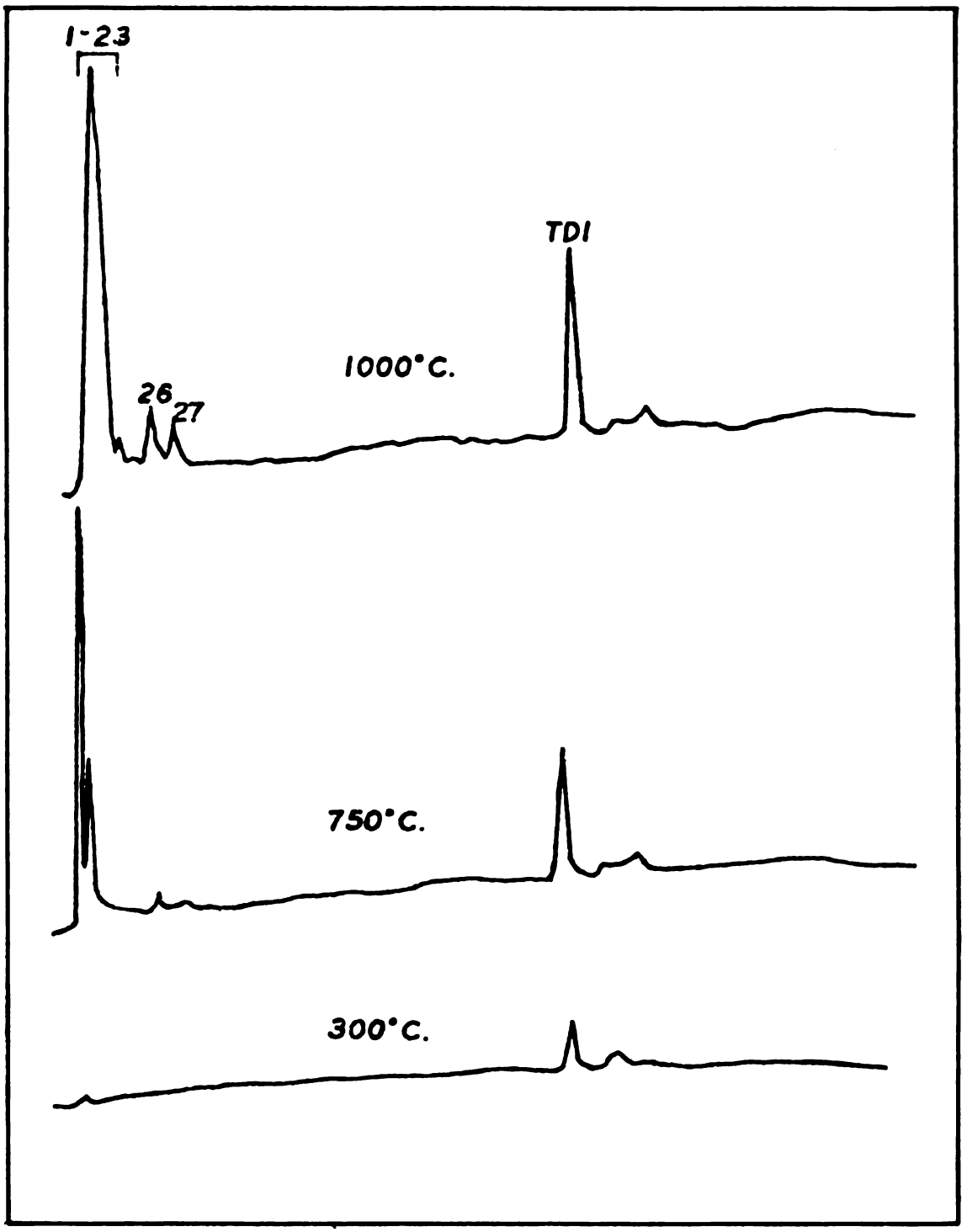


FIGURE 6. Effect of Temperature on Pyrolysis Products from Flexible-Urethane Foam - Dexsil Chromatograms (TC).

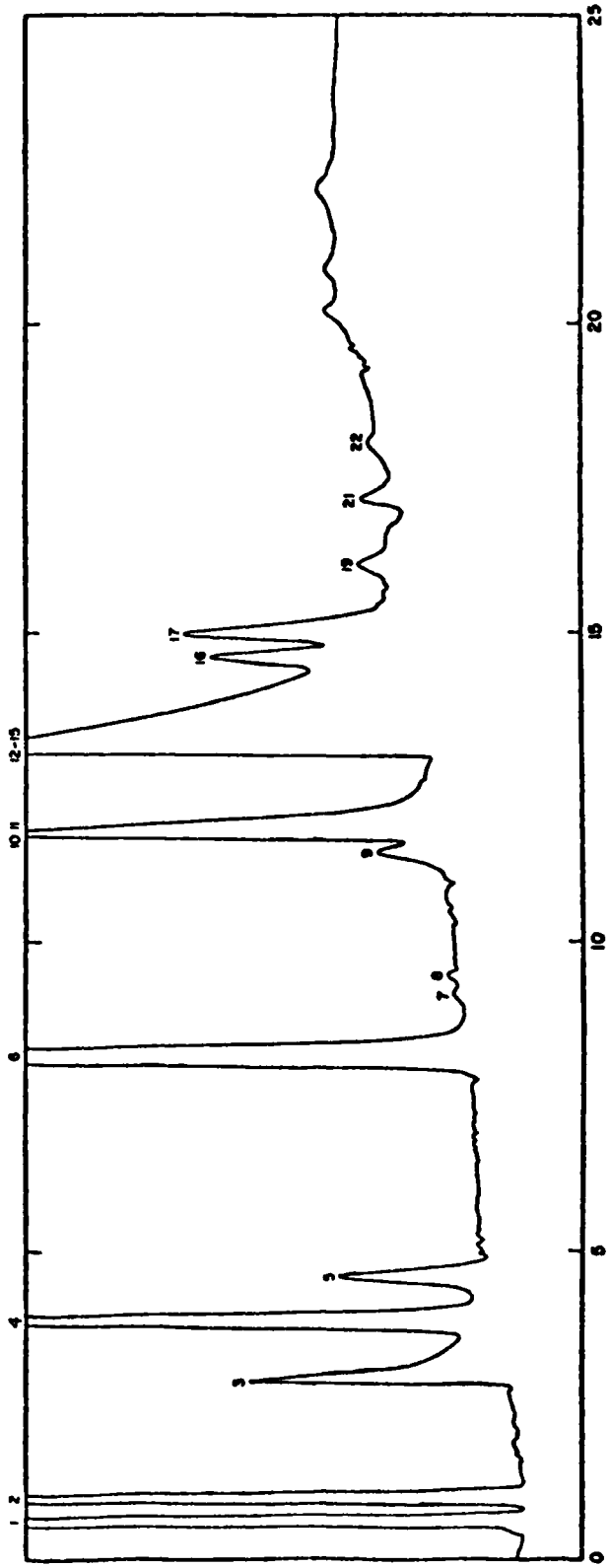


FIGURE 7. Residue Pyrolysis at 1000°C on Chromosorb-103 (TC).

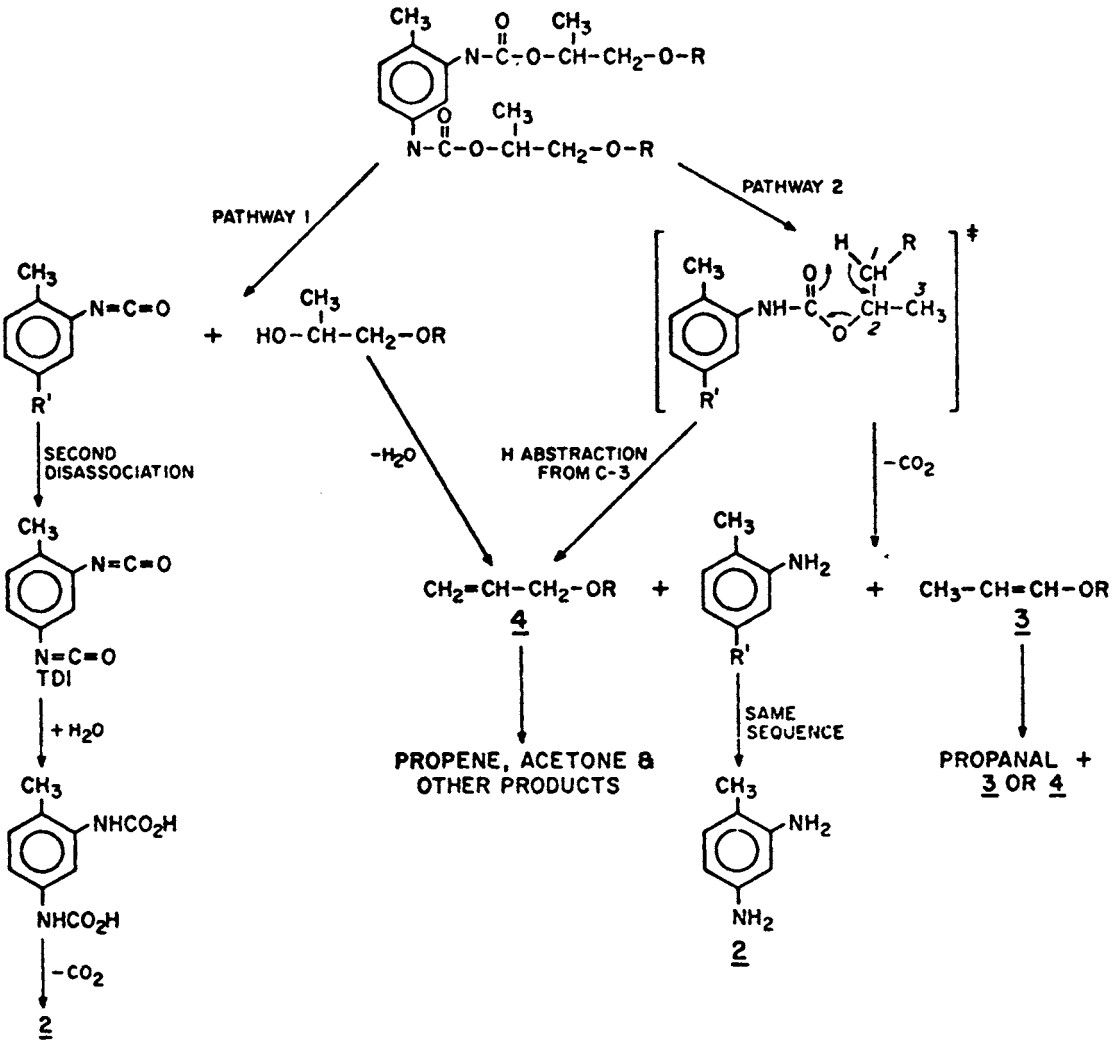


FIGURE 8. Reaction Scheme for Pyrolysis of Flexible-Urethane Foam.