THE COMPARATIVE CHARACTERISTICS OF RESPIRATORY TRACT LESIONS DUE TO VARIOUS THERMAL AGENTS AND FUMES

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V. N. Khrebtovich

Thermal Lesion Clinic (Head—Prof. T. Ya. Ar'ev) and Scientific Research Laboratory (Head—Doctor Med. Nauk. E. V. Gubler)
S. M. Kirov Order of Lenin Military Academy, Leningrad
Presented by Active Member AMN SSSR, I. R. Petrov
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Burns of the respiratory tract form one of the most frequent lesions in people who have been burned. They aggravate the course of burn sickness considerably and the secondary changes in the respiratory tract and lungs which develop at the same time are often the cause of the victim's death [1, 2, 3, 10].

Experimental work has been carried out mainly on models of respiratory tract burns caused by superheated steam [4, 5, 8].

Clinical observations indicate that burns of the respiratory tract caused by steam are seldom met with and are comparatively slight [3]. Most frequently the respiratory tract is damaged by various kinds of explosion, by fire or blazing clothes. Clinically, it is impossible to determine the role of the different factors concerned, such as flames, hot air and fumes, in causing burns of the respiratory tract. Experimental work was necessary to answer this question.

The role of hot air and flames in causing burns of the respiratory tract has been studied by only a few workers [6, 9], whose methods for inducing burns (flames and hot air introduced through the glottis into the trachea or through a tracheotomy opening by means of cannulae) differed considerably from the conditions under which burns usually occurred in man.

EXPERIMENTAL METHOD

One hundred and ninety-four experiments were made on cats. The burns were inflicted by a special method [7]. The conditions of the experiments were designed to approximate, as far as possible, those causing burns of the respiratory tract in man. To do this, a glass tube (length 6 cm, diameter 3 cm) was fixed in the oral cavity of the animal by means of damp bandages and sealed with a mixture of asbestos and fireproof clay. Through this tube the animal was connected to a source of flame, hot air or heated fume. The agents were inhaled by the animals themselves. A method used by other workers [4-6] was employed to inflict burns by steam. Superheated steam from an autoclave was led through a thin, rubber tube, at a pressure of 1-2 atm, into the nasal passages and pharyngeal cavity of the animal.

EXPERIMENTAL RESULTS

The main difference between the lesion-producing capacities of the various agents used in the experiments are shown in Table 1.

Thus, an exposure of 2 sec to superheated steam brought about serious lesions of the upper and middle respiratory tract. The inhalation of hot air at a temperature of 300° for 4 min did not injure the respiratory tract; inhalation of air at 400-450° caused burning of the upper parts of the tract and it was only when hot air at 500-550° was inhaled for a similar period that a lesion of the upper part of the trachea was caused in the majority of experiments. Tongues of fumeless flame (450-500° at the entrance to the mouth), inhaled for a considerably shorter time (25-30 sec), led to lesions of the upper and middle respiratory tract.

TABLE 1. Lesions of the Respiratory Tract in Relation to the Type of Lethal Agent

Agent	Temperature	Exposure period	No. of expts.	No. of expts. with lesions	
				Glottal region	Region below glottis
Steam	90-95	2 sec	10	10	10
Flames	450-500	25-30 "	60	60	60
	300	From 10 sec to 4 min	15	—	—
Hot air	400-450	4 min	11	—	—
	500-550	4 "	12	12	7
		20 "	7	7	7
	25	10 "	3	3	3
Fumes	150-200	5 "	2	—	—
		3 "	34	34	34

The causes for so many varied effects of steam, flames and hot air on the respiratory tract are the different physical properties of the agents and the methods used for inflicting the burns. Superheated steam has a high specific heat capacity. Led into the respiratory tract, it condenses and at the same time sets free a large quantity of heat which, combined with the action of the hot condensate settling on the mucous membrane, gives rise to severe lesions throughout the upper and middle parts of the tract.

Before causing burns of the respiratory tract in man, steam first of all escapes into the surrounding atmosphere. There it condenses and loses a considerable quantity of heat, thus reducing its capacity for tissue damage. It is evident that this fact might explain why burns of the respiratory tract in man are comparatively mild. In their experiments, research workers have always led the steam directly into the respiratory tract, often under pressure. This does not happen when the respiratory tract is burned in man. Such a method (also used in our work) should be acknowledged to be artificial and remote from the conditions leading to the burning of the tract by steam in man.

The resistance of the respiratory tract to the action of hot air may be accounted for by the low specific heat of air and also by the fact that superheated hot air entering the respiratory tract is saturated with water vapor arising from the evaporation of the moisture in the mucosa. At the same time, most of the heat is dispersed by evaporation. In experiments made to determine the temperature of jets of inhaled air in various parts of the respiratory tract, a rapid drop in temperature was observed from 500° at the entrance to the mouth to 80° in the upper part of the trachea and to 65° in the region where the trachea bifurcates (Table 2).

Moritz and co-authors [9] computed that inhaled air saturated with water vapor and heated to 142° must be cooled to body temperature in order that burns should not occur. It might be thought that injury to the mucosa of the respiratory tract sets in only after it has been dried. In order to verify this assumption we carried our experiments in which the mucosa was dried beforehand by means of atropine sulfate injected under the skin about 15 min before burning. For relieving the effects of the atropine after burning, proserine was injected. In experiments in which the mucous membrane was previously desiccated in this way, damage proved to be considerably more severe (Fig. 1). Experiments with models were also carried out to determine the effects of hot air jets at 150° on the dry and moistened fingers of man. The time elapsing before a painful sensation appeared in the moistened finger was 3-4 times greater than in dry finger and the painful feeling was felt at the place where desiccation had set in.

TABLE 2. Temperature of Jets of Inhaled Air and of the Mucous Membrane in Different Parts of the Respiratory Tract During Burning by Flames and Hot Air (Average Data)

Site of measurement	Hot air		Flames	
	Jets of inhaled air	Mucous membrane	Jets of inhaled air	Mucous membrane
	Temperature, deg			
At entrance to mouth cavity	500—550	—	450 – 500	—
Upper part of trachea	80	48 (near mucous membrane)	160	51 (near mucous membrane)
Region of tracheal bifurcation	65	—	85	—
Middle of bronchus (on mucous membrane)	—	43	—	45

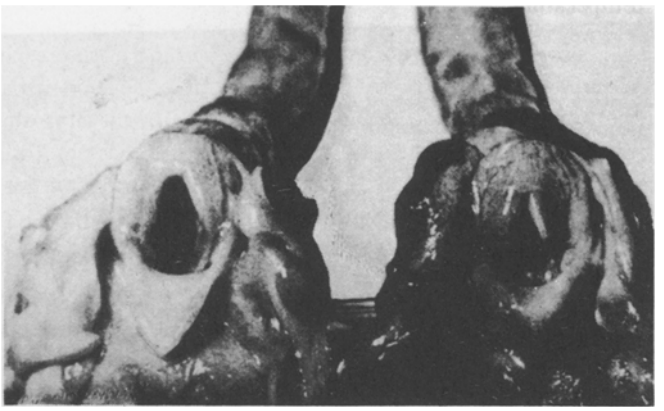

Fig. 1. Epiglottis and glottal region in the cat. Burn inflicted by hot air; temperature 400°; exposure 4 min. Right) Atropine injected before burning. An edematous epiglottis and vocal cords are seen. Entrance to larynx contracted. Left) No injection of atropine.

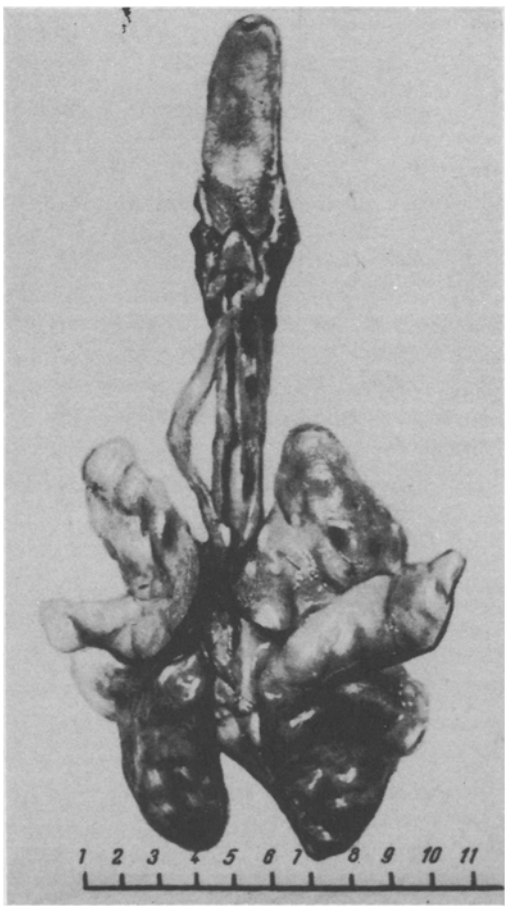

Fig. 2. The respiratory tract and lungs of a cat. Trachea opened, a fibrin film in the form of a "cast" displaced laterally. Inhalation of fumes at 150-200° for 3 min. Cat died in 3 days.

Flames are burning gases and the incandescent portions have a high calorific capacity. The heat emission of the incandescent portions evidently plays an important role in causing burns. The drop in temperature of the jets of inhaled air in these experiments was less marked than in the inhalation of hot air (see Table 2).

We also studied the effects on the respiratory tract due to inhaling "cold" fumes and fumes heated to 150-200°. Smoldering cotton fabric served as a source of fumes. When these fumes were inhaled for 20 min serious changes developed in the respiratory tract. If the fumes were inhaled for 10 min the changes were less evident and if inhaled for 5 min the respiratory tract only became irritated and hyperemic and these symptoms disappeared in 3-6 h. Inhaling fumes, heated to 150-200°, for a period of 3 min also caused severe lesions of the respiratory organs similar to those arising from the inhaling of "cold" fumes for 20 min. It was interesting to find that neither fumes nor hot air at a temperature of 150-200°, inhaled separately for 3 min, caused disturbances to the respiratory organs.

The action by which fumes inflict injury on the respiratory tract does not seem to be quite clear. Probably it can be linked with the chemical action of the products of combustion and to the irritant action of particles of incompletely burnt substances. The following hypothesis may be put forward to account for the action of fumes in jets of hot air. Firstly, it is possible that the rise in temperature increases the chemical action of the combustion products. The resemblance of the morphological changes in the respiratory tract and lungs due to the action of "cold" and hot fumes is evidence for this idea. Secondly, there may be an increase in the thermal action of hot air combined with fumes consisting of hard particles which, heating up to the temperature of the air and settling on

the mucosa, extend the damage. Evidently, the specific heat capacity of such a jet is considerably greater than that of air at the same temperature.

A primary necrosis of the mucous membrane, distinct from the burning of the respiratory tract by fume action, was not observed. An acute hyperemia with copious secretion was seen an hour or two after inhaling the fumes. After 3-6 h the mucous membrane lost its luster due to the formation of a fibrin film which had already grown to a considerable thickness within 24 h (Fig. 2). This film was easily peeled off and sometimes, at autopsy, it was possible to obtain a "cast" of the trachea and larger bronchi. It was only when fumes in a jet of hot air were inhaled that the film was completely fused to the epiglottis, vocal cords and larynx where, evidently, a necrosis of the mucosa had set in.

The results of the investigation made it possible to separate the lesions of the respiratory tract into two types: 1) true burns, 2) lesions resulting from the action of combustion products. In the first instance the subject concerns the burning of the tract by flames or steam. Hot air at 300° does not itself cause burning of the respiratory tract, as our experiments demonstrate. It is difficult to produce a situation in which hot air at a higher temperature could have acted on the respiratory tract without flame and fumes. The fact is that air jets above 300° themselves set fire to clothing. In this instance, lesions of the respiratory tract will be associated with the combined action of hot air, fumes and the products of combustion. In the second instance we are concerned with the action of fumes and other products of combustion.

The comparative ease of causing damage to the respiratory tract in experiments on the combined action of hot air and the products of combustion gives grounds for assuming that this type of action plays a very important part in lesions of the respiratory tract in man.

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