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Chronic Effects of Fire Casualty on Pulmonary Function

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INTRODUCTION

Smoke inhalation on occasion of fire casualty causes a variety of symptoms which is of thermal, irritant or neuro/hemotoxic nature in acute stage. In addition to the acute effects of smoke inhalation, inhalation of various kinds of toxic gas such as CO, HCl and organic chloride gases (phosgene, chloroethane and acrolein) which are frequently produced by combustion of synthetic construction materials, adsorbed in smoke particles or carried in fumes and deposit mainly into small airway region, may result in a chronic and irreversible inflammation in the peripheral bronchopulmonary structures.

We had an opportunity to examine, in their chronic stage, 83 subjects out of more than 400 casualties who had suffered from acute smoke inhalation and had been rescued in one of the largest tunnel fire accident in Japan on November 6, 1972.

In an attempt to elucidate the chronic pulmonary function consequences in these victims we performed a series of medical examination including pulmonary function study.

MATERIALS AND METHODS

Eighty three casualties who gave informed consents to our medical examinations were studied over 8 months in the year 1979 and 1980.

The studies were performed in following sequence; questionaires on respiratory symptoms based on BMRC form modified by us adding to a few questions concerning situation of the casualty, examination of physical findings, chest roentgenograph, arterial blood sampling and a series of pulmonary function test using body plethysmograph (AUTO-BOX M 800, SRL MEDICAL INC.) which covered measurement of lung volume subdivisions, flow-volume curve and airway resistance (Raw). The measured values in pulmonary function test were expressed in percentage of predicted values according to formulas by Baldwin, Morris et al. and Briscoe et al. for vital capacity (VC), other lung subdivisions and airway resistance respectively. Maximal flow at 25% VC in flow-volume curve (Vs5) was standardized at body height (Vs5/Ht).

A-aDo2 was calculated using abbreviated alveolar equation, PAlO2 = PAlO2 - \( \frac{PaCO2}{R} \) assuming PAlO2 = 150 mmHg and R = 0.83.

Analysis of subjects’ background (Table 1) revealed that the study group consisted of 49
The study of questionnaire showed that 27.3% in group I subjects complained of cough or phlegm lasting for three weeks or more in the past three years, 57.9% in group II, and 57.1% in group III, and that 9.5% in group I, 18.8% in group II and 26.1% in group III complained of exertional dyspnea of 3rd grade or more according to Hugh Jones. (Fig. 1).

Percent vital capacity (%VC) was 114.7 ± 15.7, 109.9 ± 15.1 and 105.7 ± 13.0 in group I, II
Cough or expectoration for more than 3 months every year

<table>
<thead>
<tr>
<th>Group</th>
<th>%</th>
<th>Mean Age (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>27.3%</td>
<td>37.8 ± 0.4</td>
</tr>
<tr>
<td>II</td>
<td>57.9%</td>
<td>37.3 ± 26.3</td>
</tr>
<tr>
<td>III</td>
<td>57.1%</td>
<td>50.9 ± 15.0</td>
</tr>
</tbody>
</table>

Dyspnea on exertion (> grade II)

<table>
<thead>
<tr>
<th>Group</th>
<th>%</th>
<th>Mean Age (yr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>9.5%</td>
<td>36.6 ± 3.1</td>
</tr>
<tr>
<td>II</td>
<td>18.8%</td>
<td>40.3 ± 6.2</td>
</tr>
<tr>
<td>III</td>
<td>26.1%</td>
<td>49.8 ± 16.6</td>
</tr>
</tbody>
</table>

Hospitalization

1: for 0-1 W
2: for 1 W-1 Mo
3: for more than 1 Mo

Fig. 1 Results of Questionnaire

Fig. 2 % VC
and III respectively (p<0.05, group I vs III) (Fig. 2), forced vital capacity in one second (forced vital capacity \(FEV_{1.0}\)) was 80.7±5.6, 79.2±8.3 and 72.3±8.8 in group I, II and III (P<0.001, group I vs II, p<0.05 group II vs III) respectively (Fig. 3), both parameters showing deteriorating tendency in pulmonary function in accordance with the increased hospitalization length.

Residual volume as percent of predicted value (\%RV) was 119.5±37.4\% in group I, 129.3±15.8\% in group II and 149.7±40.4\% in group III (p<0.025, group I vs III, p<0.005, group II vs III) (Fig. 4), and residual volume/total lung capacity (RV/TLC) was 31±7.9\% in group I, 33.9±4.4\% in group II and 41.5±6.6\% in group III (p<0.001, group I vs III) (Fig. 5).

Raw in each group calculated as percent of predicted value was 102.8±38.2\% in group I, 126.7±47.7\% in group II, and 152.4±67.0\% in group III (Fig. 6).

\(\dot{V}_{25}/Ht\) was 1.10±0.32 l/sec/m in group I, 0.94±0.43 l/sec/m in group II, and 0.62±0.38
l/sec/m in group III (Fig. 7). The differences in Raw and $\dot{V}_{25}/Ht$ between 3 groups indicated an increase in both upper and peripheral airway resistance among longer hospitalized casualties.

In order to evaluate the effect of subjects' smoking history on measured airway resistance, especially on lower airway one, measured values of $\dot{V}_{25}/Ht$ and Raw were reassorted according to their smoking index (Fig. 8). $\dot{V}_{25}/Ht$ showed again significant difference between groups with graded S. I. 0 vs 0–250, 0–250 vs 250–750 and 0–250 vs 750–, reflecting the effect of cigarette
Fig. 7 \( \dot{V}_{\text{ss}}/Ht \):

- **I**: Hospitalized for 0-1W (mean age 37.0±9.7 Yrs)
- **II**: Hospitalized for 1W-1Mo (mean age 40.9±15.0 Yrs)
- **III**: Hospitalized for over 1Mo (mean age 51.6±15.3 Yrs)

- I vs II N.S.
- I vs III \( P < 0.025 \)
- II vs III N.S.

Fig. 8 \( \dot{V}_{\text{ss}}/Ht \) and Raw (% of prediction) according to smoking habit

- O vs 250 \( P < 0.001 \)
- ~250 vs 250~750 \( P < 0.005 \)
- 250~750 vs 750~ N.S.
- 250 vs 750~ \( P < 0.005 \)
smoking on peripheral airway resistance in each groups. On the contrary, %Raw showed no dependency on subjects' smoking history (146.6±61.4% in 0 S. I. group, 111.7±22.9% in 0–250 S. I. group, 124.2±60.8% in 250–750 S. I. group and 116.3±50.2% in 750– S. I. group).

A-aDO₂ calculated from measured PaO₂ and PaCO₂ also showed increasing tendency in accordance with longer hospitalization but no significant differences between each group were demonstrated (8.5±7.0 mmHg in group I, 10.4±6.6 mmHg in group II and 10.8±6.8 mmHg in group III) (Fig. 9).

DISCUSSION

According to the report from MAC,³) the upholster inside the burned dining car in the accident was made up of various kinds of synthetic materials such as polyester synthetic resin, vinyl chloride and vinylidene chloride (saran). Gases produced by combustion of these new synthetic materials are known to be CO, HCl, organic chloride gases (phosgene, chlorethane and acrolein). Inhalation of these gases may result in severe respiratory injuries including tracheobronchitis, laryngeal edema, and non-cardiogenic pulmonary edema.

On the other hand, severe obstructive lung disease in chronic stage after inhalation of smoke in fire was reported infrequently.⁴) The prominent findings in these victims have been bronchectasis, bronchial stenosis and obliterative bronchiolitis.

Fig. 10 shows a microphotograph from open lung biopsy specimen from a patient who is also one of casualties in the Hokuriku Tunnel accident but was not included in the present study. This 44-year-old electrician was admitted in our institution two years after the accident with complaints of shortness of breath and recurrent constricting chest tightness. Although the results of pulmonary function test gave no gross abnormalities except for maximal flow-

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**Fig. 9** A-aDΟ₂

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volume curve and Raw which had not been done at that time, his biopsy specimen showed
distinctive findings of bronchiolitis obliterans as shown in Fig. 10, which completely coincide with
those of the burning automobile victim reported by Arora et al.\(^5\)

Although our original intent in the present study was to detect these pathological lesions in
peripheral airways by means of pulmonary function testing, it was revealed that measured values
of maximal flow at 25% VC which should be one of the most sensitive parameters detecting the
lesion in small airway region was much interfered with by subjects' smoking status.

Recently Loke et al.\(^6\) reported higher incidence of non-responder in non-smoking fire fighters
using He-O\(_2\) flow volume curve but he did not reach the conclusion that these methods could
distinguish the relative contribution of cigarette smoking and chronic fire fighting to abnormalities
of the small airway in smoking fire fighters.

According to our experience, He-O\(_2\) maximal flow-volume study conducted in a limited
number of subjects selected from the present study group also showed more dependency on
smoking index. If one utilizes the more sensitive methods such as frequency dependency of
dynamic compliance and so on for the purpose of detecting the small airway lesions in fire casulty
victims, the more the results would be interfered with by subjects' smoking status.

Concerning our results showing increased Raw in longer hospitalized group a few points
must be made clear. Since aging effect has not been taken into consideration in Briscoe's predic-
tion formula for Raw, our result might reflect the difference in age of subjects constituting each
group as shown in Fig. 6. However, our measurement performed on 23 control subjects covering
age of 18 to 56 years old could not detect any significant differences in % Raw among different age

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**Fig. 10** Open lung biopsy specimen from 44-year-old electrician, one of sufferers in
Hokuriku Tunnel casualty 2 years after his involvement in the accident.
(Magnification ×100)
groups. In addition, the fact that more impaired pulmonary function consequences in older age group in the present study coincide with the report from MAC having observed more frequent incidence of reduced vital capacity in older age group in acute stage. The report also gave laryngologic findings on vocal cord indicating the presence of pathologic findings on vocal cord in 82 of 97 cases (84.5%). Our questionnaire also revealed the high incidence of complaints such as long lasting hoarseness or soar throat. In evaluating the increased Raw which reflects the increased upper airway resistance the laryngeal lesion must be taken into consideration as well as those in tracheobronchial tree.

In conclusion, these results indicate that while \( \dot{V}_{25} \) correlated both to cigarette smoking status and hospitalization length of subjects, Raw correlated only to the latter, that is \( \dot{V}_{25} \) failed to discriminate the effects of smoke or toxic gas inhalation from that of cigarette smoking. The increased Raw in subjects with longer hospitalization is supposed to reflect the severity of acute smoke or heat injury to the upper airways including laryngeal region.

**SUMMARY**

Eighty three of train passengers who had been rescued from tunnel fire casualty in 1972 were examined for the study of the chronic respiratory function consequences. The interval between the outbreak of casualty and the study was 7 years. Dyspnea on exertion and FEV\(_{1.0}\%\) less than 70 were found in 19.70/0 and 15.9% of studied subjects respectively. According to the hospitalization length in acute stage, more lowered maximal flow at 25% VC (\(\dot{V}_{25}\)) and raised airway resistance (Raw) were found among longer hospitalized victims. While \(\dot{V}_{25}\) was shown to correlate both to smoking index and hospitalization length, Raw correlated only to the latter, that is \(\dot{V}_{25}\) failed to discriminate the effects of smoke or toxic gas inhalation from that of cigarette smoking. The increased Raw in subjects with longer hospitalization is supposed to reflect the severity of acute injury to the upper airways on occasion of the fire accident.

**REFERENCES**