OCCUPATIONAL ASTHMA DUE TO OZONE

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ABSTRACT

Ozone increases bronchial reactivity in normal and atopic subjects. Ozone is produced by high voltage electric discharge. Persons with pre-existent bronchial hyperreactivity should be excluded from work where significant exposure to ozone can occur. We describe a case of occupational asthma due to ozone. The levels of ozone were about 0.04 ppm.

Keywords: ozone, asthma, bronchial hyperreactivity, occupational.

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INTRODUCTION

Ozone is a known respiratory irritant (1). It has been shown to increase bronchial reactivity in both atopic and non- atopic subjects (2-4). Significant decrements in lung function of healthy athletes following vigorous exercise with low concentration exposure to ozone have been demonstrated (5). Ozone has also been shown to potentiate asthma induced by known allergens in animals (6,7).

Occupational exposure to ozone occurs during inert gas-shielded arc welding (1,8,9) and in the cockpits of high flying aircraft (1,10). Potential occupational exposure to ozone can also occur in the treatment of water, sewage and industrial wastes, the bleaching of textiles, oil and wax and in the synthesis of organic chemicals (11). Significant environmental exposure can occur in a photochemical smog (11).

We describe a case of occupational asthma induced by ozone generated by high voltage electric discharge during charging of television tubes.

CASE REPORT

A 27-year old man developed frequent episodes of cough, breathlessness and wheezing about three months after starting work as a quality control inspector in a factory manufacturing television tubes. He was admitted to hospital on four occasions for bronchial asthma over a two-month period while working in the inspection section. His symptoms usually started at about 9.00 pm ie. after work. He worked a day shift from 8.00 a.m to 5.00 pm. Symptoms improved on weekends and holidays.

He had a past history of childhood asthma. As an adult his asthmatic attacks were infrequent. His last hospital admission for asthma was about four years prior to working in the inspection section.

His grandmother and brother also had a history of asthma.

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SERIAL MEASUREMENTS OF PEAK FLOW

Because of his symptoms he was transferred to another section (where there was no exposure to ozone). Peak expiratory flow rate was measured every three hours from waking to sleeping for about two weeks when he was working in the new section. He was then transferred back to the inspection section (i.e. exposed to ozone). The peak flow rates (maximum, mean and minimum) showed a pattern of progressive daily deterioration from the first day of exposure (figure 1). On the third day of exposure he developed an asthmatic attack and had to be hospitalised. He continued to measure his peak expiratory flow rate while he was in hospital and for another two weeks when he had returned to work in the new section (with no exposure to ozone). He had no asthmatic attacks when he was away from the inspection section.

OTHER INVESTIGATIONS

The patient had non specific bronchial hyperreactivity as assessed by histamine inhalation challenge, the PD_{20} FEVI being 0.13 μ mole. The histamine inhalation challenge was repeated two months later, after he had been transferred away from ozone exposure and the PD_{20} FEVI was 0.26 μ mole. Skin prick testing to common environmental allergens was positive to housedust, housedust mite, cat fur,dog hair, human hair, kapok and cotton flock. Prick tests to the metal oxide dust and to common allergenic metals like chromium, cobalt and nickel were negative. Chest x-ray examination was normal.

OCCUPATIONAL EXPOSURE

His job was to carry out electronic testing of television tubes in the inspection room. The test was the first of a series of quality control inspections. The tubes had been assembled in other sections of the factory. The assembled tubes arrived by an overhead conveyor system. He had to transfer the tubes to a testing panel, perform the testing and then return them to the conveyor system. No chemicals were used in the room. The possibility of contamination of the environment by processes from adjacent rooms was excluded.

High voltage electric charging of the television tubes took place while the tubes were being carried by the overhead conveyor system. Electric arc discharges could be seen as the tubes were moving along the conveyor system. Ozone is known to be generated by electrical discharges(9).

Environmental measurements showed that the concentration of ozone near the conveyor system was about 0.09 ppm and at the inspection station of the worker the concentration was 0.04ppm. The threshold limit value for ozone is 0.1ppm (12). Nitrogen oxides were not detected.

The electric discharge also produced metal oxide fumes from the electrodes. Measurement and analysis of the airborne particulates showed that the dust consisted mainly of copper oxide. The average copper-in-air concentration was 0.10mg/m^3. No known allergens such as nickel or chromium were detected in the airborne dust. Other known respiratory irritants (cadmium and beryllium) were also not detected.

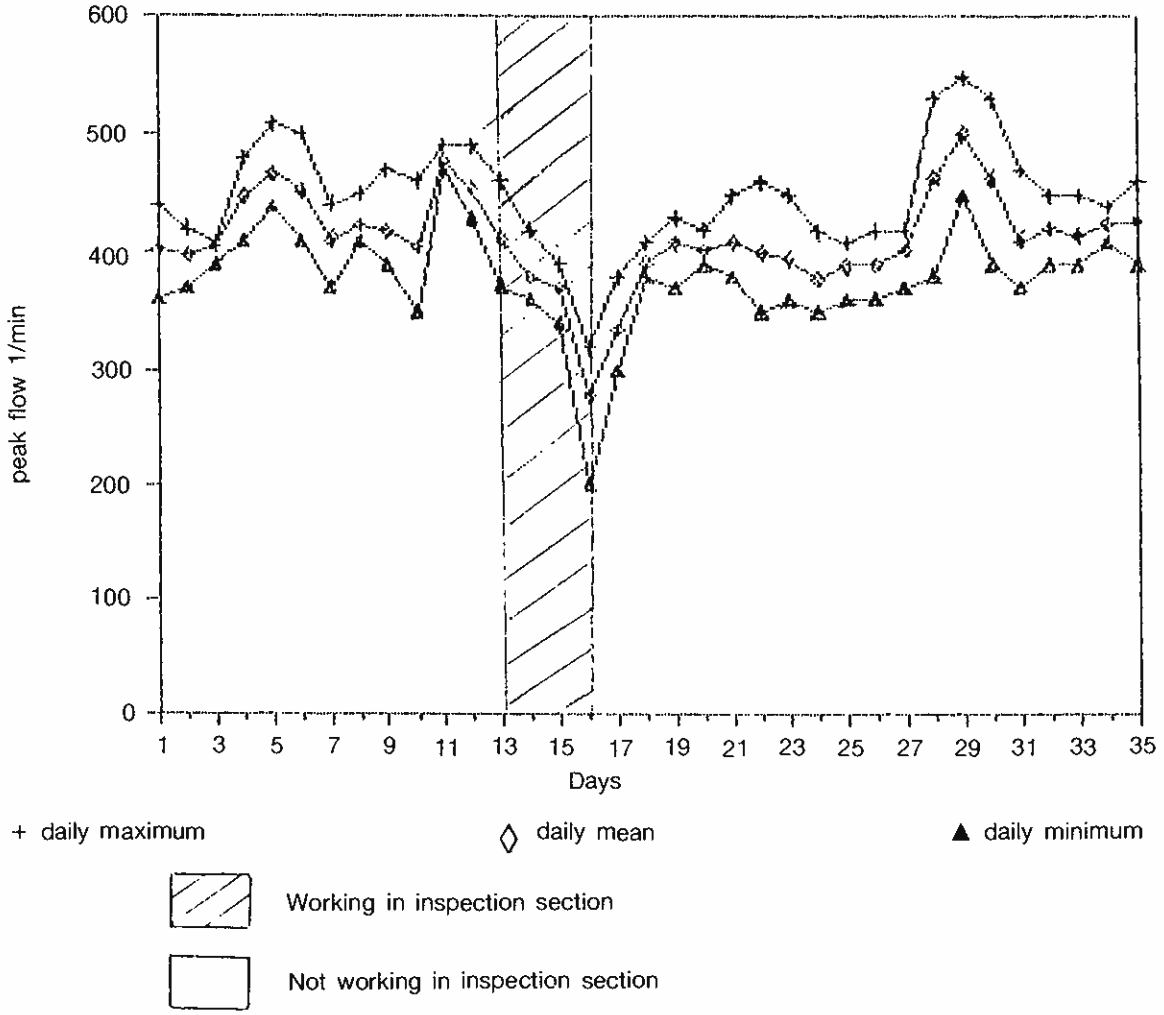

Figure 1:
Daily maximum (top line), mean (middle line) and minimum (bottom line) peak expiratory flow.

DISCUSSION

The peakflow recordings and the history support the diagnosis of occupational asthma. Although he had a history of asthma, there was an increase in the frequency and severity of his asthmatic attacks since working in the inspection section of the factory. His asthma and peakflow readings improved when he was transferred to another section of the factory.

An environmental assessment of his workplace showed that he was exposed to ozone and metal oxide dust. It is highly unlikely that the metal oxide dust was the cause of his asthma. The dust was essentially copper oxide which is not a known or likely cause of asthma. On the other hand, ozone is a known respiratory irritant and shown to increase bronchial reactivity in both atopic and normal individuals. Given the asthmatic background of our patient it was not surprising that he was the only one affected in the section. We are of the opinion that the asthmatic attacks he had while working in the inspection section were due to his exposure to ozone.

. Our patient was exposed to ozone concentrations of about 0.04 ppm. McDonnell reported that ozone

concentration as low as 0.12 ppm induced coughing and small changes in forced vital capacity and forced expiratory volume in normal subjects without asthma or respiratory disease (13). Lebowitz observed that ozone had a negative effect on lung volume at concentrations of about 0.08 ppm (14). Although the threshold limit value for ozone is 0.1 ppm it appears that effects on the airways can occur even at levels below this, particularly in persons with pre-existent bronchial hyperractivity.

Our patient has been permanently transferred from the inspection section. It is prudent to exclude persons with an asthmatic background from working environments where exposure to ozone is possible.

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