BURNISHER'S ASTHMA - A CASE DUE TO AMMONIA FROM SILVERWARE POLISHING

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ABSTRACT
A 39-year-old man with no past or family history of asthma developed asthmatic symptoms five months after working as a burnisher in a hotel. He polished brass or silverware using "brasso" or "silvo" respectively. He noticed symptoms only when using "silvo". Specific bronchial provocation testing (BPT) to "brasso" was negative. Specific BPT to "silvo" produced a dual asthmatic reaction. Asthma was present in both polishes. The ammonia-in-air levels during polishing was 8-15 ppm with "silvo" and less than 1 ppm with "brasso". A specific BPT to 12 ppm of ammonia produced an immediate asthmatic reaction. Our opinion is that he had occupational asthma from the ammonia liberated while polishing silverware with "silvo". Ammonia has been reported to cause asthma. However, there have been no previous reports of occupational asthma among burnishers doing silver polishing.

Keywords: burnisher, polishing, silver, ammonia, occupational asthma

INTRODUCTION
Occupational asthma has been reported in a diamond polisher and nickel bumper polisher. However, as far as can be ascertained, there have been no reports of occupational asthma among workers polishing silverware. We report a case of occupational asthma in a burnisher due to ammonia exposure during polishing of silverware. We use the term "burnisher's asthma" as "burnisher" is an official job title in the hotel industry.

CASE REPORT
A 39-year-old man with no past or family history of asthma or atopy, started working in a hotel as a burnisher in September 1990. He was a non-smoker. He began to have symptoms of cough, breathlessness and wheezing five months later in February 1991. He was better on weekends and holidays.

Occupational Exposure
He worked in the basement of the hotel in a small, poorly ventilated room. His job was to clean and polish articles made of silver or brassware.

The smaller silverware eg cutlery were placed in a tank of boiling water with some cleaning solution added. Later they would be transferred into a burnishing machine for polishing.

However he noticed that his symptoms were related to the use of a silver polish "silvo" which he would manually apply with a sponge to polish the bigger silverware eg trays and bowls. He could sense a strong ammonia smell whenever he carried out this job.

Interestingly, he had no problems when he used a brass polish "brasso" to polish brassware manually. Both the silver and brass polish were manufactured by the same company and available for household use. He also noticed that there was no strong smell when using the brass polish.

Checking with the supplier, we ascertained that "silvo" contained isopropyl alcohol, clay, fatty acid, ammonia and water. "Brasso" contained tarax, fatty acid, clay and ammonia.

From the constituents of the silver polish only ammonia was known to cause occupational asthma. We carried out brass and silver polishing in the laboratory and measured the levels of ammonia in the breathing zone of the person doing it. Interestingly, the ammonia levels were about 8-15 ppm during silver polishing and less than 1 ppm during brass polishing.

Bronchial provocation testing
Bronchial provocation testing was done on an inpatient basis. On the first day he carried out polishing of a brassware using "brasso" for 15 minutes. His peak flow rate (PEFR) was monitored periodically. The highest of three PEFR recordings was taken each hour. He did not develop an asthmatic attack and his diurnal variation was 10.6%. On the second day he carried out polishing of silverware using "silvo". After about 15 minutes, he developed rhinitis, tearing and coughing. Rhonchi were detected in both lungs. His PEFR had fallen by 42% to 260 l/min from the baseline of 450 l/min. Nebulised ventolin was given and his PEFR returned to 450 l/min half an hour later. Six hours later his PEFR again fell by 18% to 370 l/min and ventolin was given (Fig 1).

He was readmitted three weeks later. On the first day (control) PEFR was monitored and showed a diurnal variation of 8.7%. On the second day he was exposed to a known concentration of ammonia gas. This was done by having the patient breathe through an oro-nasal respirator connected to a sampling bag filled with air containing 12 ppm of ammonia. Within two minutes he had an asthmatic attack and rhonchi were detected in both lungs. His PEFR fell by about 65% to 210 l/min. Nebulised ventolin was given and his PEFR returned to baseline levels within minutes (Fig 2).

Other Investigations
The patient had non-specific bronchial hyperreactivity as assessed by the provocation dose of inhaled histamine producing a
DISCUSSION
Our patient had an immediate and delayed reaction to silver polishing but had no reaction to brass polishing. This was consistent with the history he gave. He also had associated symptoms of rhinitis and tearing during silver polishing but none during brass polishing. Of the constituents of the silver and brass polish, only ammonia is a known and likely causative agent for occupational asthma. Although both polishes contained ammonia, it was interesting that the ammonia concentration at the breathing zone during silver polishing (8-15 ppm) was very much higher than that during brass polishing (<1 ppm). Based on these observations and the positive challenge test (immediate reaction) to ammonia (12 ppm for 2 minutes), we are of the opinion that our patient had occupational asthma due to the ammonia liberated during silver polishing. The ammonia concentration of 12 ppm used for the challenge test was well below the threshold limit value (TLV) of 25 ppm and the short term exposure limit (STEL) of 35 ppm recommended by the American Conference of Governmental Industrial Hygienists.

The TLV of 25 ppm is meant to protect against irritation of the eyes and respiratory tract and levels below the TLV should not be irritating to the majority of persons. The irritation threshold for ammonia was examined in 10 human volunteers by Industrial Bio-Test Laboratories. The subjects were exposed to four different concentrations of 32, 50, 72 and 134 ppm for five minutes. At 32 ppm, one subject complained of dryness of the nose; at 50 ppm, two subjects experienced nasal dryness; at 72 ppm, three subjects had eye irritation, two had nasal irritation and three had throat irritation. At 134 ppm, five had eye irritation, seven had nasal irritation, eight had throat irritation and one complained of chest irritation. On direct questioning, our patient also told us that he could get asthmatic symptoms from using window cleaning solutions which smell strongly of ammonia.

There remains a possibility that the patient’s symptoms may be caused by some other constituents eg isopropyl alcohol (IPA) in the silver polish which are not found in the brass polish. We do not know if he would develop an asthmatic reaction if challenged to IPA alone. However this is not likely as IPA has not been reported to cause asthma.

This case illustrates that levels of ammonia below its TLV or STEL may cause asthma in susceptible persons. It also reminds us that burnishers involved in silver polishing may be at risk of occupational asthma. They form another group of workers who are occupationally exposed to ammonia.

REFERENCES