

ISOCYANATE EXPOSURE IN BATHTUB REFINISHING: À PROPOS A CASE OF OCCUPATIONAL ASTHMA

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Abstract

Work-related asthma in a bathtub refinishing technician prompted measurements of isocyanate exposure in this operation. Very high levels of hexamethylene diisocyanate (HDI) oligomer, up to 8500 µg/m³ NCO, were found during top varnish spray-painting, whereas the levels of HDI monomer were substantially lower. The results suggest that only full-piece, self-contained respirators would suffice to protect technicians from serious pulmonary disease.

Key words:

Irritant-induced asthma, Isocyanates, Polyisocyanates, Spray painting, Lacquer, Occupational exposure

INTRODUCTION

Volatile isocyanates, like toluene diisocyanate (TDI) and hexamethylene diisocyanate (HDI), are widely used as curing agents for paints and varnishes and recognized to induce respiratory disorders like asthma, rhinitis and, occasionally, hypersensitivity pneumonitis, HP [1]. Advanced polymer technology has produced so-called polyisocyanates, e.g. larger molecules such as trimers, whose toxicity, however, is similar to that of the more conventional diisocyanates [2].

“Bathtub refinisher’s lung”, an isocyanate-related HP variant associated with bathtub renovation, originally described by Fink and Schlueter, seems to have remained an isolated observation in the scientific literature [3]. This report included a detailed outline of the refinishing process but no isocyanate exposure data were provided.

We have encountered a case of bronchial asthma in a bathtub-refinishing technician and measured the isocyanate exposure

in this procedure. This communication describes the case and summarizes the results of exposure measurements.

CASE REPORT

A 47-year-old man had been self-employed for 18 months as a bathtub refinisher when he was hospitalized in August 1998 with chest pain and dyspnea, soon recognized as an acute attack of asthma. On the day of admittance, he had become dyspnoic one hour after spray-painting a bathtub. His illness was preceded by a month of cough; this was relieved during two weeks of summer holiday only to recur upon his return to work.

At the hospital, he was afebrile but presented with dyspnea and wheezing; his peak expiratory flow (PEF) was 175 l/min (reference 560 l/min) but no spirometry was performed at the emergency department. His electrocardiogram was normal and so was a pulmonary radiograph as well as acute phase laboratory tests including blood

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leukocyte count, erythrocyte sedimentation rate and C-reactive protein. He was treated overnight with bronchodilators, yielding a PEF of 250 l/min, and dismissed the following day with a diagnosis of asthma along with prescriptions for systemic and inhalation steroids as well as terbutalin. On admittance for a follow-up at the pulmonary department of another hospital one month later, his PEF had improved to 460 l/min. A spirogram showed an FEV₁ (forced expiratory volume in one second) of 2.4 l (63% of expected [4]) and vital capacity (VC) of 3.6 l (69% of expected), in comparison with FEV₁ 3.8 l and VC 4.7 l (86% and 85%, respectively, of expected [4]) in a similar test 16 years earlier, conducted as a part of routine examinations at the time he was a construction worker. A reversibility test with 1.2 mg salbutamol by inhalation revealed FEV₁ increase of 11%. His total leukocyte count was again normal at $4.8 \times 10^9/l$ but he had a slight eosinophilia of 5% (normal 1–3%). A standard prick test panel and serum IgE tests for isocyanates (TDI, HDI and methylene bisphenyldiisocyanate, MDI) were negative; moreover, an exercise cardiogram was normal. After his acute admission to hospital, the patient never returned to his job as a bathtub refinisher, this precluding further occupational isocyanate exposure. He had no specific inhalation challenge to isocyanates performed (rarely or never done in Sweden), nor was he subject to a bronchial hyperreactivity test.

The patient had no history of allergic or respiratory diseases and he had quit smoking in 1972 after a short period of moderate cigarette consumption. He was trained to use a half-piece respirator, equipped with a charcoal filter, during the mixing of varnish, spraying paint and cleaning of his instruments, but his compliance with this routine decreased over time. The diagnosis was irritant-induced occupational asthma, probably caused by massive isocyanate exposure. The asthma subsided and three months after the incident, the patient spontaneously and permanently discontinued his asthma pharmacotherapy.

EXPOSURE MEASUREMENTS

In the late 1990's, bathtub refinishing in Sweden was conducted using a similar technology as reported in the USA by Fink and Schlueter [3], but prepolymerized HDI

was substituted for TDI as a hardener. In short, the process included degreasing and cleaning of the tub with a phosphoric acid/paraformaldehyde formula, a styrene-based filler for surface defects, followed by grinding and acetone cleansing before spray-painting the isocyanate-based varnish. Normally, two to three layers of varnish were applied with infrared radiation to finish the curing process.

The varnish contained pigments and solvents, whereas the curing agent was based on 60–70% HDI oligomers (mainly isocyanurate and biuret), 0.1–1% HDI monomer and solvents. Two different measurements focused on isocyanate exposure were performed, in July 1999 and in May 2000. On both occasions, two layers of varnish were applied, and for each layer, four to five consecutive short-term breathing zone samples were collected. For each spraying cycle, a stationary area sample was collected in the area adjacent to the bathroom, separated from the exposure site by a plastic curtain. A mobile exhaust ventilation system, as normally applied by the technician, was connected to the bathroom with the outlet through the nearest available window.

During the 1999 measurements, liquid chemisorption with impinger bottles containing 9-(*N*-methylaminomethyl)anthracene (MAMA) in toluene and an air flow rate of 1 l/min was used [5]. This technique, however, proved inadequate to absorb the high levels of HDI oligomer ($> 1200 \mu\text{g}/\text{m}^3 \text{NCO}$) in the bathroom and this prompted a second attempt to measure the isocyanate exposure. On this occasion, the sampling time was reduced from five to three minutes as an extra precaution against isocyanate saturation, and dibutylamine (DBA) was substituted for MAMA in the impingers [6]. Analysis was conducted with high-pressure liquid chromatography coupled to a mass selective detector. Isocyanurate trimer (Bayer Desmodur N3300; molecular weight, MW 505) and biuret trimer (Bayer Desmodur N100; MW 478) were used as isocyanate oligomer standards. Isocyanate results are reported in $\mu\text{g}/\text{m}^3 \text{NCO}$, calculated as the concentration of the compound divided by its MW times the number of NCO groups times the MW of NCO (42) [7].

Table 1. Levels of HDI monomer and oligomer ($\mu\text{g}/\text{m}^3$ NCO) in spray painting of a bathtub

Sampling Site and Type	Sampling Time (Min)	HDI Monomer	HDI Oligomer
1 st spray cycle			
Bathroom, personal sampling			
P 1	3	3.5	2900
P 2	3	7	1600
P 3	3	7	5200
P 4	3	11	8500
P 5 (immediately after spraying)	3	3.5	1200
Outside bathroom, area sampling			
A 1	17	< 3.5	5
2 nd spray cycle			
Bathroom, personal sampling			
P 6	3	7	3700
P 7	3	11	6500
P 8	3	18	8500
P 9 (immediately after spraying)	3	3.5	1400
Outside bathroom, area sampling			
A 2	12	< 3.5	15

The second round of measurements (Table 1) confirmed the initial observation of very high levels of HDI oligomer, ranging from 1600 to 8500 $\mu\text{g}/\text{m}^3$ NCO during two separate spraying sessions with parallel but much lower levels of HDI monomer (maximum 18 $\mu\text{g}/\text{m}^3$ NCO). The isocyanate levels in the stationary samples outside the bathroom were very low.

DISCUSSION

Polyisocyanates may elicit four major types of reactions in the lower respiratory tract:

1. Toxic bronchitis with asthma, also called reactive airways dysfunction syndrome (RADS), caused by very high exposure levels.

2. Bronchial asthma caused by sensitization to diisocyanates, where very low exposure levels may provoke symptoms.
3. Accelerated decline in FEV_1 .
4. Extrinsic allergic alveolitis like e.g. in "bathtub refinisher's lung" [1,2].

The case reported here showed no characteristics of sensitization or alveolitis but was considered an example of the first type, a toxic, irritant bronchial reaction to a particularly high polyisocyanate exposure.

Our study showed that under the given circumstances, bathtub refinishing appeared to be a serious health hazard. The HDI prepolymer levels recorded were exceedingly high and would pose a threat of acute, severe respiratory reactions in any unprotected subject even after a short period of exposure (minutes). We do not know, however, if our data are representative of a bathtub refinisher's day, be it a worst-case or even a best-case scenario, since additional options for investigation were hard to identify by our contact in the refinishing business. The two measurements reported here were awaited for six and ten months, respectively, and further measurements were thus considered unfeasible.

Whatever their external validity for regular bathtub refinishing, our data represent some rather unique findings in contemporary isocyanate spray-painting. For comparison, a recent major study of Dutch spray-painting environments [7] reported maximum isocyanurate and biuret concentrations, expressed as $\mu\text{g}/\text{m}^3$ NCO, of 1900 and 550 $\mu\text{g}/\text{m}^3$, respectively, and in another study of spray painting operations in the US Air Force, the oligomer levels did not exceed 1400 $\mu\text{g}/\text{m}^3$ NCO [8].

For HDI monomer, a 10-minute short-term exposure limit (STEL) of 70 $\mu\text{g}/\text{m}^3$ NCO is used in the Netherlands. For the sum of isocyanate oligomers, no such exposure limit is generally accepted, but a STEL of 220 $\mu\text{g}/\text{m}^3$ NCO has been suggested [7]. Taking our short (3 minutes') sampling time into account, most of our HDI oligomer concentration levels still considerably exceeded this value.

Given our findings, "bathtub refinisher's lung" or other respiratory reactions caused by isocyanates in this process appear to be surprisingly rare events. Besides the

original HP case reported by Fink and Schlueter [3], we are aware of only one similar case, locally reported in Sweden but again without exposure measurements [9]. The number of exposed subjects in the refinishing business worldwide is unknown and would be expected to vary according to market demands. Even so, instances of overexposure to isocyanates (including polyisocyanates), both by accident and negligence (like in our case) could be anticipated. However, bathtub refinishing is typically conducted as a franchise with self-employed workers, a system which may discourage reporting of occupationally related disorders.

In conclusion, due to massive exposure to polyisocyanates, bathtub refinishing is a potentially dangerous job, and from a preventive point of view, only full-piece, self-contained respirators provide a safe respiratory work environment, given their correct handling and maintenance. Moreover, disposable gloves and coveralls would be necessary to prevent any skin contamination from the highly reactive chemicals involved in the process.

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