

Short term respiratory effects of acute exposure to chlorine due to a swimming pool accident

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Abstract

Objective—Acute exposure to chlorine causes lung damage, and recovery may proceed slowly for several weeks. The short term respiratory effects of acute chlorine inhalation during a swimming pool accident were examined.

Methods—A total of 282 subjects (134 children, aged <14 years) inhaled hydrogen chloride and sodium hypochlorite during an accident caused by a malfunction of the water chlorinating system in a community pool in Rome in 1998. Most people received bronchodilators and cortisone at the emergency room; five children were admitted to hospital. A total of 260 subjects (92.2%) were interviewed about duration of exposure (<3, 3–5, >5 minutes), intensity of exposure (not at all or a little, a moderate amount, a lot), and respiratory symptoms. Lung function was measured in 184 people (82 children) after 15–30 days. The effects of exposure to chlorine were analysed through multiple linear regression, separately in adults and in children.

Results—Acute respiratory symptoms occurred among 66.7% of adults and 71.6% of children. The incidences were highest among those who had chronic respiratory disease and had a longer duration of exposure. In about 30% of the subjects, respiratory symptoms persisted for 15–30 days after the accident. Lung function levels were lower in those who reported a high intensity of exposure than in those who reported low exposure, both in children and in adults (mean (95% confidence interval (95% CI)) differences in forced expiratory volume in 1 second (FEV₁) were -109 (-310 to 93) ml, and -275 (-510 to -40) ml, respectively).

Conclusion—Persistent symptoms and lung function impairment were found up to 1 month after the incident. Although community pool accidents happen rarely, the medical community needs to be alerted to the possible clinical and physiological sequelae, especially among susceptible people.

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Keywords: accidental exposure; chlorine gas; lung function

Chlorine is a highly irritating gas that, when inhaled, can damage larger airways as well as distal lung structure. After massive acute chlorine inhalation, pulmonary oedema,

respiratory failure, and death have been described both in laboratory animals and in humans.^{1 2} Acute accidental inhalation of sub-lethal doses can be responsible for symptoms ranging from upper airway irritation to more serious respiratory effects, such as intense coughing, wheezing, and dyspnoea,³⁻⁸ together with lung function decrements.^{5 6 9 10} Although most studies have shown that pulmonary deficit after acute exposure to chlorine tends to disappear after a few weeks,^{5 6 10} recent reports have documented long term effects, such as asthmatic reactions, bronchial hyperresponsiveness, and reduced lung function among exposed people both in the general^{8 11 12} and in the work environment.^{13 14} Brooks *et al* described the reactive airway dysfunction syndrome as an asthma-like occupational illness after an acute exposure to highly concentrated respiratory irritants.¹⁵ Moreover, it has been suggested that a history of asthma, bronchial hyperreactivity, and atopy can be associated with a greater acute decrement in pulmonary function and more serious long term pulmonary sequelae.^{12 16 17}

The potential sources of exposure to chlorine are multiple and include industrial operations¹⁸⁻²⁰ inappropriate mixing of household cleaning agents,¹² emissions during transport,¹¹ and inhalation of fumes from solid chlorine products.⁷ Due to the common use of chlorine and its compounds for water disinfection, mishaps in the chlorinating of swimming pools represent an important risk of exposure. However, the health effects of chlorine inhalation after swimming pool accidents have been documented in only few studies, and have concentrated on pathophysiology, clinical presentation, and types of treatment.^{3 4} Early physiological and clinical pulmonary alterations have rarely been reported in accidents involving a few people.^{5 6 10}

We have assessed the short term impact of an accidental acute exposure to chlorine, which occurred in a swimming pool. The respiratory health of 260 people has been studied and followed up with spirometry within 15–30 days from the accident.

Methods

SWIMMING POOL ACCIDENT

On 22 October 1998 an emission of chlorine vapours occurred in the chlorinating maintenance procedures room of a recreational centre in Rome during the afternoon swimming courses. This room was 12 m² in size and situated two floors below the ground. It was

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reported that the windows were closed when the accident happened. The water disinfection system used sodium hypochlorite (NaClO) at a concentration of 14%, and hydrochloric acid (HCl) at a concentration of 33%, for the microbiological water purification and for the pH control, respectively. The chemicals were placed in separate tanks and injected into a container, where they were mixed to circulate in the pool in controlled concentrations. The accident happened as the tube carrying the hydrochloric acid to the mixing container was broken causing the acid to flow onto the floor with a subsequent emission of chlorine fumes. The exact amount of liquid released was unknown. The chlorine vapours naturally spread through the entire environment of the sports centre, exposing the swimmers and others in the centre to high concentrations of chlorine gas. The chlorine concentration in the swimming pool water remained in a normal range as proved by specific tests.

A total of 116 people, aged 4–65, were referred to the emergency department of different hospitals in Rome, most often complaining of eye and throat irritation, cough, shortness of breath, and anxiety. Five children (range age 4–12) were admitted to the hospital because of the severity of clinical and physiological findings and were discharged after a few days. All these children reported a history of asthma, wheezing, or atopy. Most people were initially treated with bronchodilators and aerosol cortisone. In some cases, in particular for those admitted to hospital, intravenous cortisone and humidified oxygen were given. No patients received sodium bicarbonate inhalation treatment.

TELEPHONE FOLLOW UP

Demographic information on people attending the pool at the time of the accident was obtained from the pool managers. After 2 weeks, all 282 exposed people were invited to answer a telephone questionnaire administered by trained operators. It was not possible to find seven people and 15 refused to participate in the survey. Questionnaires included items on demographic data, acute symptoms (eye irritation, nose and throat problems, shortness of breath, wheezing, cough) on the day of the accident (no or yes), history of chronic respiratory disease (including asthma, bronchitis, emphysema) (no or yes), and smoking among adults (never, ex, or current). People were also asked whether they were engaged in physical exercise at the time of the accident (no or yes). Two questions were asked to define the exposure levels: (a) How long did it take from the alarm warning to escape outside? (<3, 3–5, >5 minutes) (time to vacate the pool), and (b) How much do you think you have been exposed? (not at all or a little, a moderate amount, a lot) (perception of exposure). Parents reported information about their children. At the end of the telephone interview, people were invited to and scheduled for the clinical examination.

CLINICAL EXAMINATION

A total of 236 (90.1% of the target) people underwent clinical examinations, which took place 15–30 days after the accident. Two trained physicians specialised in respiratory medicine worked with the help of two nurses, alternating daily in the clinic. People were asked to complete a self administered questionnaire about the presence of current respiratory symptoms (including shortness of breath, wheezing, and cough) (yes or no), and about pharmacological treatment either at the emergency department or at home (yes or no), and to provide a written consent for the medical examination and spirometry. A medical examination was performed, together with a measurement of weight (kg) and standing height (cm). A total of 13 people refused spirometry, 14 were excluded because they were less than 5 years old, and 25 people were excluded for technical reasons, leaving a final sample of 184 lung function tests (79 children and 105 adults) available for the analysis. Pulmonary function measurements were taken with two pneumotachographs (Vitalograph). The performance of the instruments agreed with the standard suggested by the American Thoracic Society.²¹ At least three successful forced expiratory manoeuvres, with a nose clip, out of a maximum of eight trials, were required for each subject. The operators accepted only forced expirations which met the criteria recommended by the American Thoracic Society.²¹ The following spirometric indices were calculated from each expiration: forced vital capacity (FVC), forced expiratory volume in one second (FEV₁), and forced expiratory flow at 25%–75% (FEF_{25–75}). Blood oxygen saturation was also measured by a pulse oximeter (NONIN 8500 MA).

DATA ANALYSIS

Separate analyses were performed for children (<14 years old) and adults (≥14 years old). The occurrence of acute symptoms (eye irritation, nose and throat problems, shortness of breath, wheezing, cough) was described relative to levels of exposure. Both time to vacate the pool and perception of exposure were tested separately. Pearson's χ^2 test was used to compare percentages of respiratory symptoms at 15–30 days across different categories. The effects on lung function were analysed through a multiple linear regression separately in the two age groups, as there may have been an age specific size of the effect. Lung function measures (natural logarithm (ln) transformed) were regressed on sex, age (ln_{age}), height (ln_{height}), body mass index (BMI, weight/height²; ln_{BMI}), history of chronic respiratory disease, instrument, physician performing the examination, and smoking among adults. Moreover, a variable indicating the number of days since the accident was calculated for each person and added in the model to take into account the potential recovery of lung function over time. We calculated the ln of all lung function values because of their skewed distribution in our sample and the heteroscedastic relation between lung function and its predictors. In a

Table 1 Characteristics of the study population exposed at the community pool accident in Rome, 1998

	Children n=134		Adults n=126		Total n=260	
	n	%	n	%	n	%
Sex:						
Male	62	46.3	33	26.2	95	36.5
Female	72	53.7	93	73.8	165	63.5
Smoking habit:						
Never	—	—	77	61.1	77	61.1
Ex	—	—	8	6.3	8	6.3
Current	—	—	40	31.7	40	31.7
History of chronic respiratory disease:*						
No	116	86.6	112	88.9	228	87.7
Yes	18	13.4	14	11.1	32	12.3
Present at the pool as:						
Staff	—	—	11	8.7	12	4.6
Swimmer	116	86.6	3	2.4	119	45.8
Accompanying person	16	11.9	111	88.1	127	48.8
Engaged in physical exercise at the time of the accident:						
No	56	41.8	101	80.2	157	60.4
Yes	69	51.5	4	3.2	73	28.1
Time to vacate the pool (minutes):						
<3	45	33.6	36	28.6	81	31.1
3–5	44	32.8	40	31.7	84	32.3
>5	34	25.4	49	38.9	83	31.9
Perception of exposure:						
Not at all/a little	81	60.4	65	51.6	146	56.2
A moderate amount	39	29.1	42	33.3	81	31.2
A lot	10	7.5	19	15.1	29	11.2

Totals may vary because of missing values.

*Chronic respiratory disease includes asthma, bronchitis, emphysema.

second step, the exponential residuals from the two linear regression models were calculated and then regressed on the exposure variables so as to derive age specific and combined effect estimates. Dummy variables for the exposure variable (the lowest level being the reference group) were generated. Slightly higher effect estimates were obtained for models including perception of exposure, which was then preferred as the exposure variable. The results were expressed as the mean adjusted ml differences between the lung function values in the exposed people and the values in the reference group.

Stratified analyses were then performed: (a) between those who went to the emergency department and those who did not; (b)

between those who received pharmacological treatment either at the emergency department or at home and those who did not; (c) between those who had a history of chronic respiratory disease and those who had not; (d) between smokers, ex-smokers, and non-smokers. All analyses were conducted with the statistical program STATA 6.0.²²

Results

CHARACTERISTICS OF THE STUDY POPULATION

Table 1 shows the characteristics of the study population (134 children, 126 adults). Among children, 86.6% were swimmers and 51.5% were engaged in physical exercise during the accident; a history of chronic respiratory disease was present in 12.3%. Most adults were parents or pool personnel; 31.7% were current smokers. A total of 63.4% vacated the pool in a few minutes, and 56.2% reported having been exposed not at all or a little.

ACUTE SYMPTOMS

Tables 2 and 3 show the incidences of acute symptoms among children and adults respectively. Among children, eye irritation occurred in 50.0%, nose and throat problems in 54.5%, any respiratory symptoms (shortness of breath, wheezing, cough) in 71.6%. The corresponding values among adults were 61.9, 73.0, and 66.7%. Among children, the incidences of all symptoms tended to be higher among those who had a history of chronic respiratory disease than among healthy people (nose and throat problems: 61.1 v 53.5%), among those who were engaged in physical exercise at the moment of the accident in comparison with those who were not (for respiratory symptoms: 84.1 v 57.1%), among those who spent more time to vacate the pool (any respiratory symptoms: 91.2% for >5 minutes v 60.0% for “<3 minutes”), among those who reported having been exposed a lot compared with not at all or a little (eye irritation: 80.0% v 38.3%). Similar

Table 2 Incidences of acute symptoms for selected characteristics among children (n=134), Rome, 1998

	Eye irritation	Nose/throat problems	Respiratory symptoms			Total
			Shortness of breath	Wheezing*	Cough	
Total	50.0	54.5	7.5	2.2	70.2	71.6
Sex:						
Male	56.5	56.5	6.5	3.2	67.7	71.0
Female	44.4	52.8	8.3	1.4	72.2	72.2
History of chronic respiratory disease:†						
No	50.9	53.5	6.9	1.7	68.1	69.8
Yes	44.4	61.1	11.1	5.6	83.3	83.3
Present at the pool as:						
Swimmer	52.6	56.9	65.5	71.6	3.5	71.6
Accompanying person	31.3	37.5	68.8	68.8	—	75.0
Engaged in physical exercise at the time of the accident:						
No	42.9	41.1	5.4	1.8	57.1	57.1
Yes	57.9	66.7	10.1	2.9	81.2	84.1
Time to vacate the pool (minutes):						
<3	31.1	31.1	2.2	—	60.0	60.0
3–5	52.3	65.9	9.1	2.3	65.9	70.5
>5	67.7	73.5	11.8	5.9	91.2	91.2
Perception of exposure:						
Not at all/a little	38.3	39.5	3.7	—	66.7	67.9
A moderate amount	69.2	79.5	7.7	5.1	71.8	74.4
A lot	80.0	90.0	40.0	10.0	100.0	100.0

Totals may vary because of missing values.

*Wheezing includes shortness of breath with wheezing.

†Chronic respiratory disease includes asthma and bronchitis.

Table 3 Incidences of acute symptoms for selected characteristics among adults (n=126), Rome, 1998

	Eye irritation	Nose/throat problems	Respiratory symptoms			Total
			Shortness of breath	Wheezing*	Cough	
Total	61.9	73.0	17.5	7.1	61.1	66.7
Sex:						
Male	66.7	63.6	18.2	12.1	60.6	66.7
Female	60.2	76.3	17.2	5.4	61.3	66.7
History of chronic respiratory disease:†						
No	61.6	71.4	17.9	7.1	62.5	67.0
Yes	64.3	85.7	14.3	7.1	50.0	64.3
Smoking habit:						
Never	57.1	70.1	15.6	2.6	58.4	64.9
Ex	100.0	87.5	37.5	12.5	62.5	62.5
Current	62.5	75.0	17.5	15.0	65.0	70.0
Present at the pool as:						
Staff	66.7	75.0	33.3	33.3	83.3	91.7
Swimmer	100.0	100.0	33.3	—	33.3	33.3
Accompanying person	60.4	72.1	15.3	4.5	59.5	64.9
Engaged in physical exercise at the time of the accident:						
No	61.4	72.3	17.8	6.9	63.4	68.3
Yes	50.0	75.0	50.0	25.0	100.0	100.0
Time to vacate the pool (minutes):						
<3	44.0	58.3	5.5	2.8	41.7	47.2
3–5	60.0	70.0	17.5	5.0	62.5	67.5
>5	77.6	87.8	26.5	12.2	75.5	81.6
Perception of exposure:						
Not at all/a little	41.5	56.9	7.7	—	47.7	53.8
A moderate amount	78.6	85.7	19.1	14.3	66.7	71.4
A lot	94.7	100.0	47.4	15.8	94.7	100.0

Totals may vary because of missing values.

*Wheezing includes shortness of breath with wheezing.

†Chronic respiratory disease includes asthma, bronchitis, emphysema.

results were found among adults. Furthermore, incidences were higher among smokers and ex-smokers than among never smokers, and among the swimming pool personnel who spent more time in the exposed area to help people.

RESPIRATORY SYMPTOMS AT 15–30 DAYS

A total of 63 people (27% of the total) reported some respiratory symptoms at 15–30 days after the accident (21.1% of children and 32.7% of adults). Proportions of people with respiratory symptoms at 15–30 days and some personal characteristics of children and adults are shown

in table 4. People who reported having been exposed a lot were more likely to have respiratory symptoms at 15–30 days than those exposed not at all or a little (42.9% v 18.1% in the entire sample). Adults who went to the emergency department, as well as those who received pharmacological treatment either at the emergency department or at home, were more likely to have respiratory symptoms at 15–30 days than those who did not (44.7 v 24.2%, and 45.5 v 24.6%), but no significant differences were found among children. In both age groups, those with a history of chronic respiratory disease had slightly higher rates of respiratory symptoms at 15–30 days than healthy people (34.5 v 25.6%). Similarly, smokers and ex-smokers had a higher probability of having respiratory symptoms at 15–30 days than never smokers (40.6, 37.5, and 27.8% respectively).

Table 4 Personal characteristics of children and adults who underwent clinical examination after 2 weeks and proportion (%) of respiratory symptoms at 15–30 days, Rome, 1998

	Respiratory symptoms† at 15–30 days					
	Children n=123		Adults n=113		Total n=236	
	n	%	n	%	n	%
Time to vacate the pool (minutes):						
<3	37	13.5	29	24.1	66	18.2
3–5	44	15.9	38	28.9	82	21.9
>5	31	41.9*	45	42.2	76	42.1*
Perception of exposure:						
Not at all/a little	73	12.3	54	25.9	127	18.1
A moderate amount	38	36.8	41	34.1	79	35.4
A lot	10	30.0*	18	50.0	28	42.9**
Visit to the emergency department:						
No	55	23.6	66	24.2	121	23.9
Yes	68	19.1	47	44.7*	115	29.6
Treatment‡:						
No	68	20.6	69	24.6	137	22.6
Yes	55	21.8	44	45.5*	99	32.3
History of chronic respiratory disease:						
No	107	20.6	100	31.0	207	25.6
Yes	16	25.0	13	46.1	29	34.5
Smoking habit:						
Never			72	27.8		
Ex			8	37.5		
Current			32	40.6		

*p<0.05; **p<0.005.

†Respiratory symptoms include shortness of breath, wheezing, and cough.

‡Treatment=pharmacological treatment either at the emergency department or at home.

Totals may vary because of missing values.

LUNG FUNCTION MEASUREMENTS AT 15–30 DAYS

The relation between exposure (perception of exposure) and spirometric indices at 15–30 days after the accident is shown in table 5. In both age groups, FVC, FEV₁, and FEF_{25–75} tended to be lower among those people with the highest exposure, although the results from linear regression analysis were significant only for FEV₁. For this variable, in the entire sample, we found a reduction of 178 ml (95% confidence interval (95% CI) –334 to –22) for people who reported having been exposed a lot versus not at all or a little. The corresponding values were –109 (–310 to 93) ml among children and –275 (–510 to –40) ml among adults. In both age groups there was no effect modification of the relation between exposure and lung function either by the visit to the emergency department, by pharmacological treatment, or by history of chronic respiratory

Table 5 Association between exposure to chlorine and lung function (adjusted ml difference, 95% CI) at 15–30 days, Rome, 1998

Parameter	Perception of exposure	Children			Adults			Total		
		n	ml difference†	95% CI	n	ml difference†	95% CI	n	ml difference†	95% CI
FVC*	Not at all/a little	44	Reference		47	Reference		91	Reference	
	A fair amount	27	56	-65 to 177	38	-204	-434 to 26	65	-49	-175 to 76
	A lot	8	-110	-300 to 80	17	-204	-502 to 95	25	-140	-315 to 35
FEV ₁ *	Not at all/a little	44	Reference		47	Reference		91	Reference	
	A moderate amount	27	79	-49 to 207	38	-156	-336 to 25	65	-17	-130 to 95
	A lot	8	-109	-310 to 93	17	-275	-510 to -40	25	-178	-334 to -22
FEF ₂₅₋₇₅ *	Not at all/a little	44	Reference		47	Reference		91	Reference	
	A moderate amount	27	23	-310 to 356	38	24	-495 to 543	65	27	-272 to 326
	A lot	8	-78	-594 to 438	17	-245	-904 to 415	25	-165	-574 to 243

*The mean (SD) FVC, FEV₁, and FEF₂₅₋₇₅ in the reference groups were respectively: 1902 (624) ml, 1654 (524) ml, 2037 (826) ml among children; 3662 (938) ml, 2978 (727) ml, 2948 (1320) ml among adults; 2819 (1191) ml, 2344 (919) ml, 2503 (1191) ml in the whole sample.

†Difference (ml) in lung function variables between exposed people and the reference groups after adjustment for age, sex, height, BMI, history of chronic respiratory disease, instrument, physician, number of days since the accident and smoking among adults.

disease. On the contrary, the effect of exposure on lung function tended to be higher among smokers and ex-smokers than among never smokers (mean (95% CI) FEV₁ ml adjusted difference between exposed and unexposed people was -499 (-1049 to 51) for smokers, -338 (-1528 to 849) for ex-smokers, -165 (-449 to 117) for never smokers). Oxyhaemoglobin saturation was normal in all people (SatO₂ ≥96%).

Discussion

The present study documents that short term exposure to chlorine due to a swimming pool accident can cause significant immediate morbidity in most exposed people and potential lung damage after 15–30 days. Although we did not know the exact concentration of the inhaled chlorine vapours, a mild degree of exposure can be hypothesised as the accident happened in a closed space and most people escaped outside in a few minutes. The immediate clinical manifestation in most people was predominantly due to the irritant effect of chlorine gas on the lachrymal, nasal, oral, and tracheobronchial tree.

Inhalation of chlorine gas can damage both the airways and the alveolar-capillary structures because of its solubility.²³ At physiological pH on most surfaces, chlorine gas combines with tissue water to form hydrochloric and hypochlorous acids which diffuse into cells to react with the amino groups of cytoplasm proteins, forming N-chloral derivatives.²⁴ During these chemical reactions, nascent oxygen is released. Oxidative effects are responsible for the main cytotoxic damage, and the acid production for secondary irritation. The variations in the type and amount of injury are related to the concentration of chlorine gas, the duration of exposure, the treatment given, and the individual susceptibility.

Short term symptoms after acute exposure to chlorine have been previously documented especially in occupational environments but also during community accidents. Decker *et al* described two accidents in the same swimming pool, the first involving 30 people in 1986 and the second 11 people in 1988 in Texas; those exposed to heavy concentrations of chlorine gas developed classic early signs and symptoms of acute exposure.³ Two children, after brief

exposure to fumes from solid chlorine products used in residential pools in Nebraska in 1986, developed lethargy, productive cough, severe hypoxaemia, tachypnoea, cyanosis, retraction, and expiratory wheezes and crackles in the base of the lungs.⁷ After acute exposure to chlorine from two separate accidents in community pools in Pennsylvania in 1996, all 13 children involved showed not only classic signs and symptoms, but also a fall in blood oxygenation saturation.⁴

We found some lung function impairment 15–30 days after the accident as a result of injury to lung parenchyma and small airways. Our findings of a short term effect (within 1 month) can be compared with other studies, in most of which lung function decrements tended to disappear after a few weeks. Ploysongsang *et al* found restrictive ventilatory function with reduced diffusing capacity and some obstruction in small but not in large airways 14–16 hours after the exposure of four healthy men in a swimming pool in Ohio in 1982; all impairment of lung function cleared within 1 month without residual lung damage.¹⁰ Hasan *et al* described lung function deficit in 18 asymptomatic subjects accidentally exposed to chlorine in a dormitory in Lebanon in 1983 at 1, 7, and 14 days after the exposure; a reduction of the flow rates was found, which cleared at 15 days in 12 subjects.⁵ Both obstructive and restrictive impairment have been documented which could possibly be related to different degrees and durations of exposure as well as different individual susceptibility and reaction to a noxious agent.^{6 11}

Although most studies did not have long term sequelae of a single high dose exposure, the debate on this issue is still open. On the one hand, Jones *et al* did not find any evidence of an exposure effect on the annual rates of change over 2 years among the 84 victims of a train derailment with chlorine gas diffusion which occurred in Florida in 1978.¹¹ Abhyankar *et al* described an accidental exposure to chlorine in an open space among eight people in India in 1985 and found that at 6 months all the spirometric measures had returned to normal values.⁶ On the other hand, Brooks *et al* documented that in some people acute high level irritant exposure may cause an asthma-like

syndrome, reactive airway dysfunction syndrome, which is different from typical occupational asthma, and can lead to chronic airway disease.¹⁵ Although the exact pathological change is not known, bronchiolitis obliterans has been proposed as a lesion occurring after some toxic gas inhalation. A typical feature of reactive airway dysfunction syndrome has been experienced by three German policemen without history of respiratory disease 4 months after an accidental exposure to chlorine,⁸ but more consistent evidence of long term lung damage after acute accidental exposure to chlorine has been documented in occupational settings.^{13 14 19 20} It has been hypothesised that low background levels of the respiratory irritants among workers cause a chronic inflammatory response in the small airways that may enhance the effects of a single acute gassing episode with a higher possibility of long term sequelae than in other settings.²⁰

We found greater effects of exposure to chlorine among those with a history of respiratory and atopic disorders. A pre-existing airway hyperresponsiveness could be responsible for an exaggerated pulmonary response to chlorine gas inhalation.¹⁷ Subjects who had a history of asthma or wheezing had the lowest FEV₁ and FVC together with a slower resolution of symptoms after the accidental exposure in a dormitory.³ A case of asthma persisting 2 years after the inhalation of a mixture of sodium hypochlorite and hydrochloric acid was documented in a woman with a familial history of atopic disease.¹² Finally, we found that smokers tended to have persistent respiratory symptoms at 15–30 days and greater lung function decrements than non-smokers. Smoking might act in conjunction with irritant gas to cause an inflammatory reaction in the small airways and reduce ventilatory function.¹³

Some limitations of the present study should be noted. As in other studies, we could not obtain information about the concentration of chlorine gas, so we used reports on perception of exposure as a proxy. Information bias might have resulted from the self reported occurrence of accidental exposure as well as symptoms. However, the fact that objective indices of respiratory function were related to the levels of exposure argues against this bias. We did not know the criteria for or doses of pharmacological treatment given to the victims, so we could not measure its influence on the lung function variables. Finally, given that pre-exposure lung function variables were not available, the association between acute chlorine inhalation and lung function decrements cannot be attributed with certainty to exposure. A longer follow up design could be more sensitive in establishing the relation between initial lung damage and long term sequelae, especially among susceptible people.

In summary, we have presented the acute consequences of a disaster in a community

pool and the persistence of lung impairment at 15–30 days. Although uncommon, our experience cannot be an isolated one and the medical community needs to be alert. Extreme caution must be used in maintenance of pool chlorinating equipment.

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