blood/air partition coefficients that are less than 0.44 (the value for nitrous oxide) (see Gargas *et al.*[69] for the correlation of olive oil and blood partition coefficients), and as such would be expected to induce any CNS effects quickly, *certainly in less than 15 minutes*, as cited above.[29]

Recommendations for a Workplace Exposure Limit

At present the listed TLVs for the light hydrocarbon gases are an 8-hour TWA for butane of 800 ppm, an 8-hour TWA for LPG of 1000 ppm, and a listing for all other C1 to C3 hydrocarbons that they be treated as asphyxiants (while acknowledging that they are flammable).

Based on the discussion above, the hydrocarbon gases (with the exception of pure methane) are in fact fast acting, mild narcotic agents at levels below that causing oxygen deficiency. They also have poor warning properties; the odor threshold of these materials is above the concentration causing physiological effects.

Therefore, it is appropriate to have maximum exposure limits for these materials in addition to a TWA limit. Because of the concern for flammability, and the wide availability of explosivity meters in the oil and gas industry, the recommendation is expressed in terms of percentage of the LEL.

It is recommended that exposure to all C1 to C3 hydrocarbon gases be kept below an 8-hour TWA of 1000 ppm on the basis of good industrial hygiene practice.

It is further recommended that a maximum exposure limit of 10 percent of the LEL be established.

The second recommendation recognizes the different potency of the hydrocarbons to induce narcosis, and takes advantage of the different sensitivity of explosivity meters to the different gases. While the relationship is not exact, a meter calibrated on any of the C1 to C4 hydrocarbons will provide a sufficiently accurate indication at 10 percent LEL of when to don respiratory protection. This avoids having to know the composition of the gas (e.g., in a gas processing plant where streams of variable composition are encountered) and allows an operator to make decisions quickly in the plant.

Implications for the Workplace

The levels of hydrocarbon gas causing narcosis which have been discussed here are very high when compared to the TLVs for most materials. Can such concentrations be encountered in the workplace?

In the production of oil and gas enormous quantities of fluids are handled under pressure in many thousands of items of equipment. In addition to the many opportunities for leaks, equipment which deliberately releases gas into the workplace is not uncommon, and work procedures sometime allow release of hydrocarbon gas into the workplace. These practices occur as there is a widely held perception that "sweet" gas (natural gas with no hydrogen sulfide present) is not harmful, a perception sometimes reinforced by treatment of the gas as a simple asphyxiant with the suggestion that as long as there is adequate oxygen present there is no health threat.

Every year there are a number of deaths and incidents in the Western Canadian oil fields as a result of exposure to sweet hydrocarbon gases. Leak detection and repair programs have improved conditions considerably, but places where gas levels exceeding 10 percent LEL (or 5000 ppm measured as methane) have been found are as follows:

In oil field facilities, pneumatic equipment operated by natural gas or by propane is sometimes vented inside buildings. Where it is necessary to use hydrocarbon gas as the power source (because of a remote location where electricity is not available), the equipment must be vented outside the building.

Certain equipment such as level controllers on test separators are prone to leak. In meter buildings, orifice plate changers are common leakage points. For gas compressors fueled by natural gas, the low-pressure fuel systems commonly leak (the vibration of the engine loosens the threaded fittings often used for the fuel system).

Several work practices which can cause high exposure to C1 to C3 gases have been encountered. One example was the purging of air from a compressor after repair using natural gas which was then released into the workplace. A less obvious example was draining the water from a gas dryer; the water was saturated with high pressure gas which "flashed," causing high worker exposure.

Finally, the most common occurrence where a worker will be exposed to high levels of hydrocarbon gas is in responding to a combustible gas alarm. As mentioned above, it is common practice to provide warning of potential fire and explosion hazard by providing permanent combustible gas alarms in buildings housing expensive equipment. There is often a two-level alarm system. When the lower level alarm sounds, an operator will enter the building to investigate and correct the problem. If the second level alarm trips, the equipment will perform an emergency shut-down including depressurizing the system and dumping the contents of the equipment to flare.

The above recommendation clearly suggests that the first level alarm be set at 10 percent of the LEL, and that the operator wear supplied air respiratory protection when entering the building to investigate the alarm.

In making this recommendation, the experience accumulated from nitrogen narcosis when diving again provides some lessons. Circumstances which increase the effects of nitrogen narcosis when diving are anxiety, inexperience, apprehension, and recent alcohol intake.[6-8] Presumably such factors will also influence the susceptibility of a person exposed to hydrocarbon gases. Surely some of these factors will be present when an operator must respond to an alarm announcing the approach of an explosive atmosphere, and it is at just such a time that clear judgment is required.

References

1. American Conference of Governmental Industrial Hygienists: Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. (4 annual updates included). ACGIH, Cincinnati, OH (1986).
2. Kennedy, S.K.; Longnecker, D.E.: History and Principles of Anesthesiology. In: Goodman and Gilman's The Pharmacological Basis of Therapeutics, 8th ed., Chap. 13. A.G. Gilman, T.W. Rall, A.S. Niles, and P. Taylor, Eds. Pergamon Press, New York (1990).
3. Brown, W.E.: Experiments with Anesthetic Gases Propylene, Methane, Dimethyl Ether. J. Pharmacol. Exp. Ther. 23:485–496 (1924).
4. Sato, A.; Nakajima, T.: Pharmacokinetics of Organic Solvent Vapors in Relation to their Toxicity. Scand. J. Work Environ. Health 13:81–93 (1987).
5. Winter, P.M.; Miller, J.N.: Anesthesiology. Sci. Am. 252(4):124–131 (1985).
6. Graves, D.; Wohlers, R.: PADI Advanced Diving Manual, pp. 180–181. PADI, Santa Anna, CA (1984).
7. Melamed, Y.; Shupak, A.; Bitterman, H.: Medical Problems Associated with Underwater Diving. New Engl. J. Med. 326(1):30–35 (1992).
8. Stone, W.: The Apprenticeship (A first-hand account of learning to do deep dives on air, well beyond the limits for onset of nitrogen narcosis). Caving Int. Mag. 12:44–51 (July 1981).
9. Fiserova-Bergerova, V.: Gases and their Solubility; A Review of Fundamentals. In: Modelling of Inhalation Exposure to Vapors; Uptake, Distribution, and Elimination, Chap. 1, pp. 3–28. CRC Press, Boca Raton, FL (1983).
10. American Conference of Governmental Industrial Hygiene: Notice of Intended Changes—Toluene, Trimethylamine, and Vinyl Acetate. Appl. Occup. Environ. Hyg. 6(11):966–977 (1991).
11. Larson, C.P.; Eger, E.I.; Severinghaus, J.W.: Ostwald Solubility Coefficients for Anesthetic Gases in Various Fluids and Tissues. Anesthesiology 23(5):686–689 (1962).
12. Fiserova-Bergerova, V.; Diaz, M.L.: Determination and Prediction of Tissue–Gas Partition Coefficients. Int. Arch. Occup. Environ. Health 58:75–87 (1986).
13. Gargas, M.L.; Burgess, R.J.; Voisard, D.E.; et al.: Partition Coefficients of Low Molecular Weight Volatile Chemicals in Various Liquids and Tissues. Toxicol. Appl. Pharmacol. 98:87–99 (1989).
14. Perbellini, L.; Brugnone, F.; Caretta, D.; Maranelli, G.: Partition Coefficients of Some Industrial Aliphatic Hydrocarbons (C5–C7) in Blood and Human Tissue. Br. J. Ind. Med. 42:162–167 (1985).
15. Weast, R.C.; Astle, M.J. (Eds.): CRC Handbook of Chemistry and Physics, 62nd ed., p. D-100. CRC Press, Boca Raton, FL (1982).

Received 4/21/92; review decision 7/8/92; revision 8/10/92; accepted 10/5/92

Substance Use & Misuse, 32 (12&13), 1877–1882, 1997

Sudden Sniffing Death Following Inhalation of Butane and Propane: Changing Trends[1]

Earl Siegel, Pharm.D.,[2] and Suman Wason, M.D., M.B.A.

ABSTRACT

Toxicologic information and published data under-represents the toxicity of butane and propane as well as the number of fatalities related to their use. A review of the literature, databases, two index cases and several regional fatalities indicate that butane and propane use has become a serious problem and may represent the most common sudden sniffing death hazard in the U.S.

Key words. Inhalants; Death; Butane; Propane

SUMMARY AND CONCLUSIONS

In the United Kingdom, there have been over 1000 inhalant use deaths reported since 1971. Currently averaging over 100 deaths annually, the majority of these deaths resulted from butane and propane. Although recent U.S. surveys indicate approximately one fifth of adolescents have experimented with inhalant use, published U.S. death reports attributable to butane and propane are limited. Reports of the American Association of Poison Control Centers' annual data indicate that from 1987 to 1990 there were 10, 12, 25, and 20 inhalant-related deaths, respectively. The number caused by butane and propane for each of those years was 1, 7, 9 and 10, respectively. Published data underrepresent the true numbers of inhalant use fatalities related to butane and propane use for the following reasons: (a) The circumstances surrounding death may be misrepresented to coroners, (b) Volatiles are not easily or routinely screened for

[1] Summary and conclusions based on NIDA Research Monograph #129, pp. 193–201.

[2] Send requests for reprints to Earl Siegel, Pharm.D., The Drug and Poison Information Center, P.O. Box 670144, Cincinnati, OH 45267-0144.

in cases of sudden sniffing death, and (c) there is no formalized registry of this form of drug use.

In 1978, environmental concerns resulted in the replacement of most Freon propellants with butane and propane. In 1986, the first human case of ventricular tachycardia associated with inhalation of butane and propane was reported.

Two recent fatalities attributed to butane and propane serve to emphasize the potential for sudden death: (a) In March 1990, an 11-year-old boy collapsed in a movie theater bathroom. A butane cigarette lighter fuel container and a plastic bag were found next to him. Cardiopulmonary resuscitation proved unsuccessful. Toxicologic analysis confirmed the presence of butane in the patients blood and lung tissue. (b) In April 1990, a 15-year-old boy was found unconscious in a backyard. Three companions related that the four teenagers had taken a 20-gallon propane tank from the family gas grill, placed some of the gas in a plastic bag and were inhaling it in order to get high. The subject collapsed soon after inhaling the gas. Fumes, ignited by a match, resulted in a flash fire. The patient did not sustain any burns. He could not be resuscitated. Propane was detected in the blood and lung tissues.

Interviews with friends and school officials revealed that sniffing of butane lighter fuel is a common practice among many children at upper middle class schools in Cincinnati. The second subject discussed was avowedly antidrugs and did not consider sniffing to be "drug abuse."

Additionally, in 1991, the authors were aware of nine dramatic butane and propane use cases in their region involving seven sudden sniffing deaths, an explosion in a car full of huffers with multiple severe burns, a fatal auto accident and a case of a 6-year-old imitating an older sibling. Since the abandonment of fluorocarbons, butane and propane probably represent the most common sudden sniffing death hazard. This is in spite of the fact that many toxicology references list them as simple asphyxiants. Health care providers need to be aware that the profile of the teenager who inhales volatiles is broader than only the ethnic lower socioeconomic classes. An urgent need exists for preventative efforts directed at adolescents and pre-adolescents and their parents, with emphasis on the risk of sudden sniffing death.

Décès soudains dus à l'inhalation de butane ou de propane : tendances

E. Siegel, Pharm.D., et S. Wason, M.D., M.B.A.

L'information toxicologique et les données publiées sous-estiment la toxicité du butane et du propane, ainsi que le nombre d'accidents mortels liés à leur usage. Un passage en revue de ce qui a été publié, des bases de données, deux index de cas et plusieurs accidents mortels régionaux indiquent que l'utilisation du butane et du propane est devenue un sérieux problème susceptible de réprésenter le risque de mort soudaine par inhalation le plus fréquent aux Etats-Unis.

Plotselinge dood volgend op het inhaleren van butaan en propaan: Veranderende trends

E. Siegel, Pharm.D., en S. Wason, M.D., M.B.A.

Toxicologische informatie en gepubliceerde gegevens onderrepresenteren de toxiciteit van butaan en propaan, alsmede het aantal overlijdensgevallen als gevolg van het gebruik van deze middelen. Een literatuuroverzicht, gegevensbestanden, twee index gevallen en verschillende regionale overlijdensgevallen indiceren dat butaan en propaan een ernstig probleem is geworden, en het meest voorkomende risico vormt op plotselinge dood door snuiven in de Verenigde Staten.

Muerte Repentina Trás la Inhalación de Butano y Propano: Tendencias Cambiantes

E. Siegel, Pharm.D., y S. Wason, M.D., M.B.A.

Información toxicológica y los datos publicados subestiman la toxicidad y el número de fatalidades relacionadas con el uso de butano y propano. La revisión de literatura al respecto, bases de datos, dos casos índice y las circunstancias de varias fatalidades regionales indican que el uso de butano y propano se ha vuelto un problema serio; problema que puede incluso representar el más común peligro de muerte repentina por inhalación en los Estados Unidos.

Morte súbita suscedendo inalação de butano e e propano: tendências variáveis.

E. Siegel, Pharm.D., e S. Wason, M.D., M.B.A.

Informação tecnológica e dados publicados sub-representam a toxicidade do butano e do propano, bem como o número de mortes relacionadas com o seu uso. Uma revisão da literatura, bases de dados, dois casos arquivados e várias mortes na região indicam que o uso de butano e propano tornou-se um problema sério e pode representar o risco mais comum de morte súbita nos EUA.

Tod durch Inhalation von Butan und Propan: wechselnde Trends

E. Siegel, Pharm.D., en S. Wason, M.D., M.B.A.

Toxikologische Information und publizierte Daten unterschätzen die Toxizität von Butan und Propan sowie die Zahl der Todesfälle in Zusammenhang mit deren Gebrauch. Eine Literaturübersicht und Statistiken deuten darauf hin, dass Butan- und Propankonsum ein ernstes Problem geworden ist und die häufigste Ursache des sniffing death in USA sein könnte.

Sudden Sniffing Death Following Inhalation of Butane and Propane: Changing Trends

E. Siegel, Pharm.D., e S. Wason, M.D., M.B.A.

Le informazioni e le pubblicazioni tossicologiche, danno scarsa rilevanza sia alla tossicita' del butano e del propano che al numero di morti correlate al loro uso. Uno studio particolareggiato su molte morti in varie regioni, indica che butano e propano sono diventati un serio problema e possono diventare il piu' comune pericolo di morte improvvisa negli Stati Uniti.

الموت المفاجئ نتيجة للشم الذي يتبع استنشاق البيوتان والبروبين:
Butane and Progane
النزعات المتغيرة

E. Siegel, Pharm.D. and S. Wason, M.D., M.B.A.

ان المعلومات العلمية المتعلقة بالسموم والمعلومات المنشورة تقلل من مدى سمية البيوتان والبربين وتقلل من عدد الوفيات الناتج عن تعاطي السموم. ان مراجعة ما كتب وقائمة المعطيات وحالتين موثقتين ووفيات اقليمية تبين ان تعاطي البيوتان والبروبين قد اصبح مشكلة خطيرة ويمكن ان يؤدي للموت المفاجئ نتيجة الستنشاق والاخطر ما يكون في الولايات المتحدة نتيجة الشم.

鼻吸丁烷與丙烷後的突然死亡：
改變中的趨勢

毒藥學消息與發表了的資料低估了丁烷與丙烷的毒性和因使用而產生的死亡數字。一個關於文獻、資料庫、兩個目錄個案及數個地域性死亡的評論顯示，丁烷與丙烷的使用已變得嚴重，並可能代表著美國最普遍的鼻吸死亡危險。

Внезапная смерть при вдыхании ингаляторов бутана и пропана: изменения в направлениях

Д-р Э. Сигель (E. Siegel), д-р С. Вазон (S. Wason)

Информация о токсикологии и опубликованные данные не полностью представляют сведения о токсичности бутана и пропана, а также количество смертных случаев при их употреблении. Обзор литературы, базы данных, 2 отмеченных случая заболевания и несколько смертельных исходов указывают, что употребление бутана и пропана стало серьезной проблемой и является наиболее распространенным случаем внезапной смерти при употреблении токсических веществ в США.

The American Journal of Forensic Medicine and Pathology 18(3):299–302, 1997.

Sudden Death Due to Butane Inhalation

Timothy P. Rohrig, Ph.D.

Sudden death due to the inhalation of halogenated hydrocarbons is a well-documented phenomenon in the scientific and medical literature. Recently, there has been sporadic information suggesting that nonhalogenated hydrocarbons may cause potentially fatal cardiac arrhythmias. This report documents five sudden deaths due to inhalation of nonhalogenated hydroearbons: *n*-butane, isobutane and propane. The hydroearbons were identified by headspace gas chromatography in blood, brain, and lung tissue. Case histories, toxicologieal findings and analytical procedures are discussed.
Key Words: Sudden death—Hydrocarbons—Butane—Inhalation.

The practice of deliberately sniffing or inhaling the contents of aerosol cans in order to get "high" has become increasingly popular among today's youth. The propellants most commonly found in these cans are chloro-fluoro-hydrocarbons, commonly known by their trademark name, Freon. Sudden death resulting from this practice was reported on by Bass in 1970 (1). It is well documented in the scientific and medical literature that the mechanism of death is the induction of cardiac arrhythmias (2).

In the late 1970s, there was a call for the ban of halogenated hydrocarbons as propellants in aerosol products (3) becausc of the apparent damage caused by these compounds to the earth's ozone layer. These compounds were replaced, in these products, by propane, *n*-butane, isobutane, and isopcntane. In 1982 (4), aliphatic hydrocarbons were considered to be safe as aerosol propcllants, although, it was also noted that acute inhalation of high concentrations of these compounds could causc epinephrine-induced cardiac arrhythmias. In a limited human study reported in that same article, no cardiac abnormalities were observed.

In 1986 (5), there was a report of a 2-year-old girl who presented with seizures and ventricular tachycardia after "playing" with an aerosol can containing isobutane, *n*-butane, and propane. She required intensive care and survived without sequelae. Another episode of a nonfatal cardiac arrhythmia was reported in 1989 (6). A 15-year-old boy had a history of sniffing butane. On one such occasion, he experienced severe chest pain and suddenly collapsed. Cardiopulmonary resuscitation (CPR) was started, and when he presented at the hospital, he was in vcntricular fibrillation. He was treated and ultimately released without adverse effects. One of the earliest reported deaths associated with butane inhalation was reported in the Japanese literature in 1985 (7). A 13-year-old Japanese boy, after several inhalations from a bag containing lighter refill gas, suddenly collapsed and died. Pro-

Received February 7, 1996; accepted March 21, 1996.
From Osborn Laboratories, Shawnee Mission, Kansas, U.S.A. Director of Toxicology.
Address correspondence and reprint requests to Dr. Timothy P. Rohrig at Osborn Laboratories, P.O. Box 2920, Shawnee Mission, KA 66201, U.S.A.
Presented, in part, at the 1993 Fall Meeting of the Southwestern Association of Toxicologists, Arlington, TX, U.S.A.

TABLE 1. *Gas chromatographic conditions for butane analysis*

Column	6' porapak Q 80/100 mesh	30 m GSQ megabore
Column temp	110°C isothermal	125 (7)–200 (5) C @ 5°C/min
Injector temp	150°C	150°C
Detector	FID	FID
Detector temp	300°C	300°C

FID; flame ionization detector.

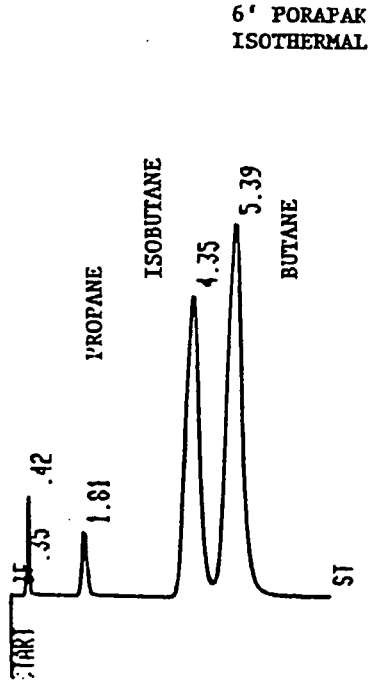
pane, *n*-butane, and isobutane were detected in his blood and tissues. A limited number of other deaths due to butane inhalation have since been reported in the literature (8,9).

The present report describes the postmortem toxicologic findings and case histories in five fatalities resulting from inhalation of butane.

CASE REPORTS

Case 1

A 15-year-old white boy was riding in a car with two friends. They all were purportedly inhaling the contents of a Ronson butane lighter refill canister when the decedent suddenly lost consciousness. He was taken to a nearby police department where resuscitation was begun; he was subsequently taken to a hospital via ambulance where resuscitation efforts were continued for ~1.5 h before he was pronounced dead.



GAS CHROMATOGRAPHIC SEPARATION
OF
LIGHT HYDROCARBONS

FIG. 1. Gas chromatographic separation of light hydrocarbons.

Case 2

A 17-year-old white man was inhaling "Scotchguard" from a glove. According to witnesses, he apparently went into respiratory arrest immediately after inhaling. The two friends he was with summoned his father who attempted CPR while awaiting the arrival of an ambulance. He was taken to a local Emergency Department where resuscitation was continued, but no response was obtained.

Case 3

A 15-year-old white boy had a history of "huffing" butane. On the night of his death, the decedent's brother and a friend stated that he took "four large hits of butane". After he took his last "hit", he collapsed onto the floor and began having a seizure. Emergency medical services (EMS) was immediately notified and transported the boy to a nearby hospital where he was pronounced dead.

Case 4

A 14-year-old white boy was found by his mother lying on his bedroom floor gasping for breath; shortly thereafter he went into respiratory arrest. A partially empty can of butane was found on the bed and four or five additional cans of butane were in the bedroom. EMS was called, and the boy was dead-on-arrival (DOA) at the hospital. According to his parents, the decedent had not used drugs and had only consumed alcohol on occasion. However, 1 week prior to his death, his friends had brought him home in a "semiconscious state". It was never determined what caused the stupor. It was also noted that the morning before his death, the decedent had been in a deep sleep from which his mother had been unable to arouse him.

Case 5

A 15-year-old white boy died 13 h after inhaling the contents of a "Zippo" butane lighter refill canister. The decedent and three other friends had reportedly purchased three cans of the butane and driven about while inhaling the fumes. The teens

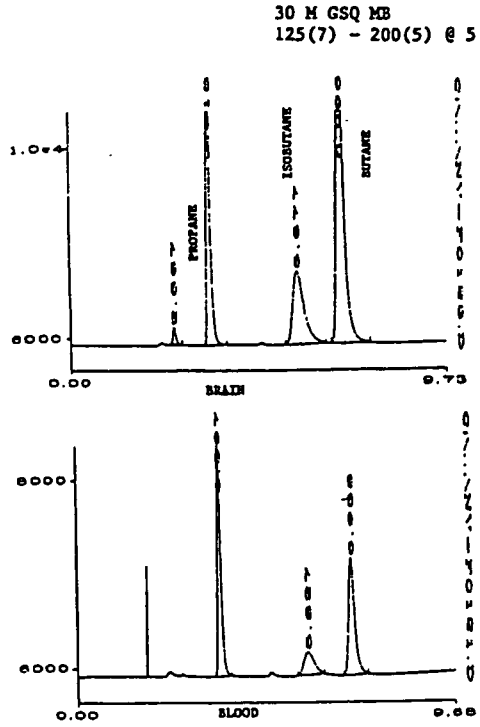


FIG. 2. Gas chromatographic analysis of blood, brain, and tissue.

arrived at the decedent's home, where he then stepped out of the automobile and immediately collapsed. EMS was notified and, while awaiting the arrival of the ambulance, two neighbors, who were registered nurses, administered CPR. He was taken to a hospital, placed on a ventilator, and given supportive care until he died. According to the decedent's friends, he had a history of butane inhalation.

ANALYTICAL PROCEDURES

Propane, *n*-butane, and isobutane were identified by headspace gas chromatography. A 2–5 ml aliquot of blood (vial filled half way for tissue) was placed in a 20-ml headspace vial and heated at 60°C for a minimum of 30 min. A 1-ml headspace sample was injected into the gas chromatograph. Gas chromatographic conditions are given in Table 1. The chromatographic performance of propane, *n*-butane, and isobutane under the prescribed conditions is illustrated in Figs. 1 and 2. All cases were screened for ethyl alcohol by gas chromatography and acidic, basic and neutral drugs by a combination of thin-layer chromatography, gas chromatography, and immunoassay.

RESULTS

One or more light aliphatic hydrocarbons (*n*-butane, isobutane, and propane) were detected in blood, brain, and lung tissue in Cases 1–4. The specific compounds detected in each case are presented in Table 2. Given the extensive medical intervention and time of onset of symptoms to death, specimens were not collected for analysis in Case 5.

Brain tissue generally had the highest amount of the hydrocarbons detected. The number of plus signs in Table 2 indicates the relative concentration of each compound.

Other toxicological testing in these cases was negative.

In consideration of the circumstances surrounding the deaths and the absence of any anatomical or histological evidence as to the cause of death, deaths were certified as toxic effects of butane inhalation (Cases 1, 3–5) and toxic effects of inhalation of volatile substances (Case 2); death in all cases was considered to be accidental.

DISCUSSION

Inhaling the contents of canned aerosol products to get "high" has been a common 20th century

TABLE 2. *Details of sudden death due to "butane" inhalation*

Age (yr)	Race/sex	Product	Compounds detected*	Signs/symptoms
15	W/M	Ronson butane lighter refill	Butane +++ Isobutane ++ Propane +	Sudden collapse
17	W/M	Scotch Guard	Butane	Respiratory arrest
15	W/M	Ronson butane lighter refill	Butane + Isobutane +++	Four "hits" seizures
14	W/M	Ronson butane lighter refill	Isobutane	Found down
15	W/M	Zippo butane lighter refill	Not determined	Sudden collapse

*The number of plus signs indicates the relative amount of each compound.

practice among certain groups. The common propellants in these cans, up until the late 1970s, were halogenated hydrocarbons. Resulting from the abuse of these aerosol products, numerous deaths occurred, described by Bass (1) and others (2,8,9) as "sudden sniffing deaths".

With the change of propellants to nonhalogenated compounds along with the known cardiac toxicity of such chemicals at high concentrations in animals, one could expect an increase in mortality due to these compounds. This report documents the sudden death, presumably through a cardiac dysrhythmic mechanism (given the history), of five individuals who were abusing these light aliphatic nonhalogenated hydrocarbons.

Acknowledgments: The author is grateful to the Office of the Chief Medical Examiner (State of Oklahoma), and Toxicology Laboratory staff for their analytical analyses. The author would also like to thank Warren L. Kleinsasser, M.D. (Medical Director, Osborn Laboratories) for his helpful comments and critical review.

REFERENCES

1. Bass M. Sudden sniffing death. *J Am Med Assoc* 1970;212: 2075–79.
2. Flanagan R, Ruprah M, Meredith T, Ramsey J. An introduction to the clinical toxicology of volatile substances. *Drug Safety* 1990;5:359–83.
3. Federal Register 1978;43:11301–26.
4. Final report of the safety assessment of isobutane, isopentane, *n*-butane, and propane. *J Am Coll Toxicol* 1982;1:127–42.
5. Wason S, Gibler W, Hassan M. Ventricular tachycardia associated with non-freon aerosol propellants. *JAMA* 1986; 256:78–80.
6. Gunn J, Wilson J, Mackintosh A. Butane sniffing causing ventricular fibrillation. *Lancet* 1989;1:617.
7. Nishi K, Ito N, Mizumoto J et al. Death associated with butane inhalation: report of a case. *Jpn J Legal Med* 1985;39:214–6.
8. Segal E, Wason S. Sudden death caused by inhalation of butane and propane. *N Eng J Med* 1990;323:1638.
9. Segal E, Wason S. Sudden sniffing death following inhalation of butane and propane: changing trends. In: Sharp C, Beauvais F, Spence R, eds. *Inhalant Abuse: A Volatile Research Agenda.* NIDA Research Monograph 129, National Institute on Drug Abuse, Rockville, MD, 1992.

Available online at www.sciencedirect.com
SCIENCE DIRECT
Forensic Science International 143 (2004) 211–214
Forensic Science International
www.elsevier.com/locate/forsciint

Case report

Three cases of sudden death due to butane or propane gas inhalation: analysis of tissues for gas components

Hideaki Sugie[a,*], Chizuko Sasaki[a], Chikako Hashimoto[a], Hiroshi Takeshita[a], Tomonori Nagai[a], Shigeki Nakamura[a], Masataka Furukawa[a], Takashi Nishikawa[b], Katsuyoshi Kurihara[a]

[a]*Department of Legal Medicine, Kitasato University School of Medicine, 1-15-1 Kitasato, Sagamihara, Kanagawa 228-8555, Japan*
[b]*Division of Medico-chemical Analysis, Kyoritsu College of Pharmacy, 1-5-30 Shiba-koen, Minato-ku, Tokyo 105-8512, Japan*

Received 20 November 2003; accepted 17 February 2004

Available online 19 June 2004

Abstract

We report three cases of sudden death due to inhalation of portable cooking stove fuel (case 1), cigarette lighter fuel (case 2), and liquefied petroleum gas (LPG) (case 3). Specimens of blood, urine, stomach contents, brain, heart, lung, liver, kidney, and fat were collected and analyzed for propylene, propane, isobutane, and *n*-butane by headspace gas chromatography. *n*-Butane was the major substance among the volatiles found in the tissues of cases 1 and 2, and propane was the major substance in case 3. A combination of the autopsy findings and the gas analysis results revealed that the cause of death was ventricular fibrillation induced by hard muscle exercise after gas inhalation in cases 1 and 2, and that the cause of death in case 3 might be hypoxia. It is possible that the victim in case 3 was under anesthetic toxicity of accumulated isobutane which is a minor component of liquefied petroleum gas.

Keywords: Portable cooking stove fuel; Cigarette lighter fuel; Liquefied petroleum gas (LPG); Butane abuse; Propane abuse; Volatile substance abuse

1. Introduction

Abuse of gas fuel is spreading among the youth in Japan. Cigarette lighter refills containing butane are the most frequently abused, and other frequently abused includes butane-containing cans for portable cooking stoves. Abuse of liquefied petroleum gas (LPG) which contains propane as a major component is rare and usually not fatal.

Three cases of sudden death after inhalation of these gas fuels were investigated. Body fluids and tissues of the three decedents were analyzed for propylene, propane, isobutane, and *n*-butane for the investigation by head space gas chromatography.

2. Case reports

- Case 1: A 24-year-old man rushed out of his room on the third floor in the apartment, ran down the staircase at his full speed, and collapsed. He was taken to an emergency room but died. Investigators found 24 empty cans with a capacity of 250 g volatile liquid for portable cooking stove on the floor in his room.

The victim was 166 cm in height and weighed 54.7 kg. The rigor mortis was strong at the time of autopsy. The heart weighed 308 g and showed no fibrosis or evidence of myocardial infarction. The left and right lungs weighed 276 and 308 g, respectively, showing slight congestion. The brain weighed 1350 g and showed no anatomical abnormalities. The other organs showed no anatomical abnormalities but showed evidence of congestion.

* Corresponding author.

0379-0738/$ – see front matter
doi:10.1016/j.forsciint.2004.02.038

- Case 2: A 14-year-old boy and his four friends put cigarette lighter oil in vinyl bags and inhaled the gas. He kicked one of the friends in the abdomen and suddenly fell down. He was taken to a hospital by an ambulance but died despite resuscitation. Police officers found 13 butane lighter refill canisters at the scene.

  The victim was 170 cm in height and weighed 87.8 kg. The rigor mortis was strong at the time of autopsy. The heart weighed 336 g and showed no fibrosis or evidence of myocardial infarction. The left and right lungs weighed 491 and 776 g, respectively, showing marked congestion. The brain weighed 1400 g and showed no anatomical abnormalities. The liver weighed 2090 g and showed mild fatty degenerations. The other organs showed no abnormalities but showed evidence of congestion.
- Case 3: A 19-year-old man who lived alone was found dead in his room, holding a hose for LPG in his hand pressing it against his right cheek.

  The victim was 168 cm in height and weighed 50.9 kg. The rigor mortis passed off at the time of autopsy. The heart weighed 247 g and showed no fibrosis or evidence of myocardial infarction. The left and right lungs weighed 392 and 349 g, respectively, showing marked congestion. The brain weighed 1440 g and showed no anatomical abnormalities. The liver weighed 1070 g and showed mild fatty degenerations. The other organs showed no abnormalities but showed evidence of congestion.

3. Toxicological findings

3.1. Materials and methods

The specimens of blood, urine, stomach contents, brain, heart, lung, liver, kidney, and fat tissues were collected and analyzed for propylene, propane, isobutane, and *n*-butane by the method of Terada et al. [1] and Ago et al. [2] using headspace gas chromatography.

One milliliter or 1.0 g of the specimens was mixed with 2.0 mL of water in a 10 mL vial. The vial was sealed immediately with a Teflon cap and kept warm in a water bath at 55 °C for 30 min. After equilibrating to room temperature, 1.0 mL head space sample from the vial was injected into the gas chromatographic instrument.

The gas contents were determined on a Shimadzu GC-17A gas chromatograph equipped with a flame ionization detector. Separation was achieved on a capillary column (GS-Q, 30 m × 0.53 mm i.d.). The column temperature was raised after injection from 125 to 150 °C at a 5 °C/min rate. The temperature of injection port and of the detector was 150 and 200 °C, respectively. The flow-rate of helium carrier gas was adjusted to 5.8 mL/min. The chromatogram of the standard gases (1.0% each of ethylene, ethane, propylene, propane, isobutane, and *n*-butane; GL Sciences, Tokyo) is shown in Fig. 1. The concentrations in the sample fluids or tissues were determined as reported by Terada et al. [1] and Ago et al. [2]. The absolute calibration curve was constructed by plotting the peak areas of 0, 0.1, 0.5, 1, 5, 10, and 50 μL/mL, which was linear in this range. The Triage® plate (Biosite Diagnostics, San Diego, CA) was used for the detection of the metabolites of eight classes of drugs (phencyclidines, benzodiazepines, cocaine, amphetamines, tetrahydrocannabinol, opiates, barbiturates, and tricyclic antidepressants) in urine.

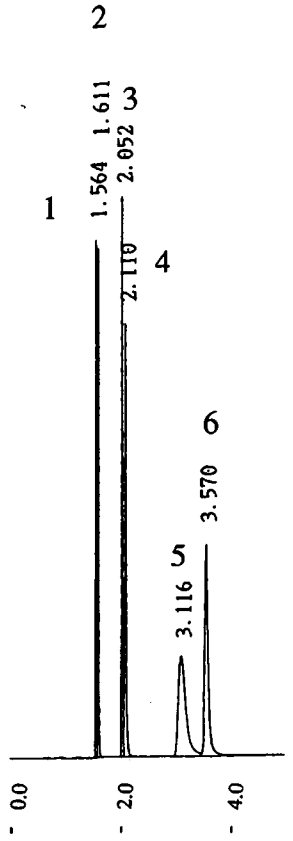


Fig. 1. Gas chromatogram of a standard mixture of six gases. Peaks: 1, ethylene; 2, ethane; 3, propylene; 4, propane; 5, isobutane; and 6, *n*-butane.

3.2. Results

As Fig. 2 shows, each peak of the volatiles from the tissue specimens was sharp with no interfering peaks. Table 1 shows the concentrations of propylene, propane, isobutane, and *n*-butane in all the specimens analyzed. *n*-Butane was the major species among the volatiles found in the tissues of cases 1 and 2, and propane was the major in case 3.

The *n*-butane concentration was high in fat, heart, blood, stomach contents, and brain in this order in case 1; in case 2, that was high in liver, brain, heart, and kidney in this order. In

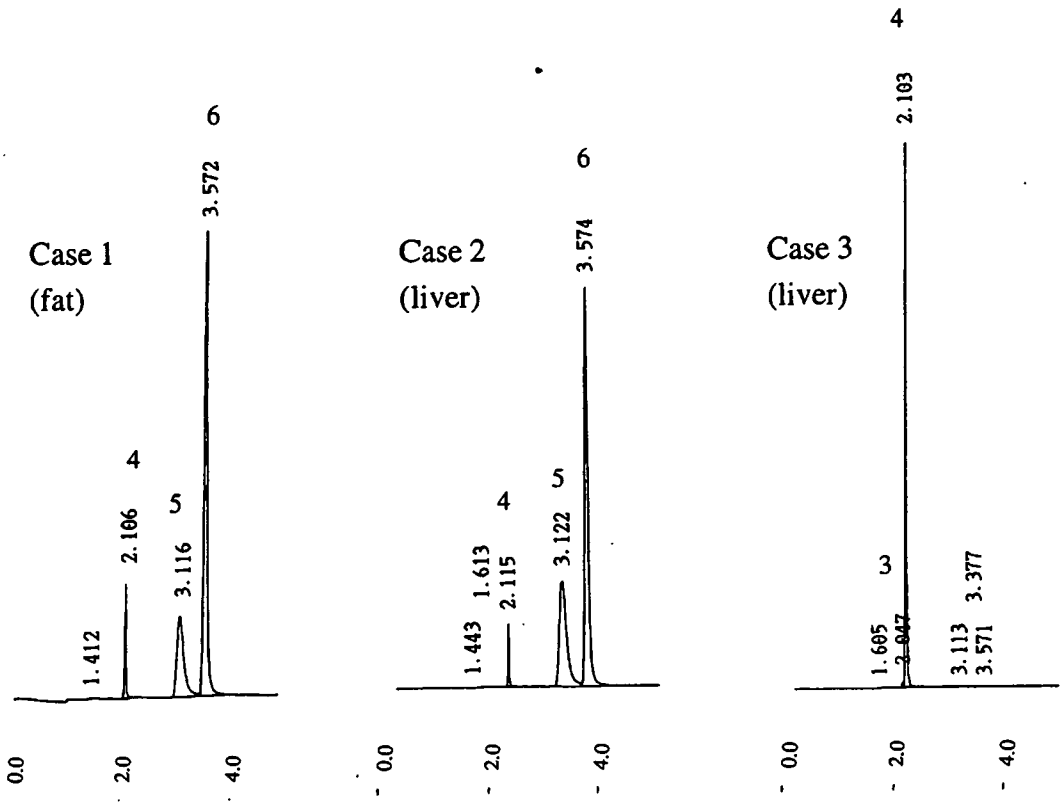


Fig. 2. Gas chromatograms of the tissue samples from case 1(left), case 2 (middle), and case 3 (right). Peaks: 3, propylene; 4, propane; 5, isobutane; and 6, *n*-butane.

case 3, the propane concentration was high in liver, fat, brain, and heart in this order. A certain amount of isobutane was found in various tissues in the three cases. Trace amounts of propylene was detected in some tissues of case 3 but was not detected in cases 1 and 2.

Ethanol was not detected in their blood and urine specimens. The abused drug screening test of Triage® on the three urine specimens gave negative results.

4. Discussion

The components of the gas samples are as follows according to the manufacturers: refill can for oven (71% *n*-butane, 28% isobutane, and 1% propane), a lighter refill (54% *n*-butane, 20% isobutane, and 26% propane), and LPG (97.8% propane, 1.5% isobutane, 0.1% *n*-butane, 0.2% propylene, and 0.4% other gases).

Table 1
Concentrations of propylene, propane, isobutane, and *n*-butane in body fluids or tissues of the three cases

	Propylene			Propane			Isobutane			*n*-Butane		
	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3	Case 1	Case 2	Case 3
Blood	n.d.	n.d.	0.11	0.01	0.07	10.19	0.10	0.03	n.d.	0.52	0.11	n.d.
Urine	n.d.	n.d.	n.d.	n.d.	n.d.	0.19	n.d.	n.d.	n.d.	0.01	0.01	n.d.
Sto.con.	n.d.	n.d.	0.06	0.01	0.03	6.73	0.14	0.02	0.10	0.47	0.05	n.d.
Brain	n.d.	n.d.	0.30	0.01	0.14	43.54	0.14	0.10	0.42	0.39	0.21	0.11
Heart	n.d.	n.d.	0.15	0.01	0.06	28.39	0.22	0.02	0.32	0.79	0.15	0.08
Lung	n.d,	n.d.	0.04	0.01	0.09	4.15	0.06	0.04	n.d.	0.29	0.13	n.d.
Liver	n.d.	n.d.	0.76	0.01	0.28	70.63	0.05	0.17	0.54	0.14	0.38	0.19
Kidney	n.d.	n.d.	0.05	0.01	0.07	5.85	0.08	0.07	n.d.	0.27	0.14	n.d.
Fat	n.d.	n.d.	0.56	0.13	0.05	68.25	1.96	0.03	0.60	6.30	0.07	0.14

Sto.con.: stomach contents; n.d.: not detected; units: μL/mL or g.

As Table 1 shows, the ratios of the gas components in these gas samples were consistent with those determined in their tissues, as *n*-butane was the major species in cases 1 and 2 while propane was the major in case 3. These substances are lipophilic so that after being taken up from the lungs into blood, they distributed at high concentrations in lipid-rich tissues such as brain and fat tissues, and also in liver, heart, and kidney [2–5].

The mechanism of sudden death directly related to volatile abuse is seldom clear but suggested to include cardiac arrhythmia, hypoxia, and respiratory depression. *n*-Butane and isobutane have an anesthetic or narcotic effect on the central nervous system, and induce fatal arrhythmia at 0.5–15% in the air [6]. According to the reports, many victims of *n*-butane or isobutane inhalation collapsed due to fatal ventricular fibrillation immediately after sudden fear or hard muscular exercise such as fright, running, and sexual activity [2,6]. The victims of cases 1 and 2 collapsed immediately after running or kicking, so ventricular fibrillation appeared to be their cause of death.

Propane is less toxic than *n*-butane or isobutane having a weaker anesthetic effect and a negligible effect on heart. The LD_{50} value of propane is over 80% in the air whereas that of *n*-butane or isobutane is about 50% in experimental animal, also indicating propane is less lethal [2,6]. The cause of death after propane gas inhalation is reported to be usually hypoxia [5,6] so that the cause of death in case 3 might be hypoxia. However, LPG contains isobutane in addition to propane, and the isobutane concentration in brain, heart, and liver in case 3 was 0.32–0.54 μL/g, which was higher than that in cases 1 and 2. It is possible that the victim in case 3 had been breathing LPG for a long time so that isobutane accumulated as well as propane in his brain to a toxic level resulting in a possible anesthetic effect of isobutane rather than direct hypoxia.

References

[1] M. Terada, T. Watanabe, H. Kashiwade, S. Yoshimura, An autopsy case of a sudden death due to inhalation on butane gas, Jpn. J. Legal Med. 37 (1983) 696.
[2] M. Ago, K. Ago, M. Ogata, A fatal case of *n*-butane poisoning after inhaling anti-perspiration aerosol deodorant, Legal Med. 4 (2002) 113–118.
[3] C. Fuke, T. Miyazaki, T. Arao, Y. Morinaga, H. Takaesu, T. Takeda, T. Iwamasa, A fatal case considered to be due to cardiac arrhythmia associated with butane inhalation, Legal Med. 4 (2002) 134–138.
[4] T.P. Rohrig, Sudden death due to butane inhalation, Am. J. Forensic Med. Pathol. 18 (1997) 299–302.
[5] T. Kojima, T. Takeoka, H. Kobayashi, K. Hara, M. Kageura, T. Nagata, Determination of liquefied petroleum gas (mainly propane) in biological materials by gas chromatography and gas chromatography–mass spectrometry, a case report, Jpn. J. Legal Med. 33 (1979) 80–85.
[6] H. Naitou, Chudoku Hyakka, second ed., Nanzando, Tokyo, 2001, pp. 164–166.

British Journal of Addiction (1989) 84, 563-564

CASE REPORT

Commercial Butane Abuse: a disturbing case

B. MATHEW[1], ELINOR KAPP & T. RYLAND JONES[2]

[1]*The Maidstone Hospital, Psychiatric Wing, Hermitage Lane, Maidstone, Kent, ME16 9QQ,* & [2]*St Cadocs Hospital, Caerleon, Gwent, NP6 1XQ, United Kingdom*

Summary

The case report discusses a 16-year-old girl with a history of commercial butane gas abuse over a year. As the patient had collected the empty canisters, it was possible to calculate the quantity abused which is not usually possible due to lack of co-operation and lack of evidence. Other aspects discussed are the mode of inhalation, accessibility, and psychological consequences.

Introduction

The abuse of popular commercially available volatile hydrocarbon by inhalation is well documented in literature. Although the exact incidence and prevalence is not know, it is now accepted that it is an adolescent phenomenon, predominantly in males aged between 8 and 19 years of age.[1] The common constituents of the solvents are hydrocarbons which are highly lipid soluble and therefore have increased affinity to the CNS. Grabski in 1961 reported a case of cerebral atrophy following regular toluene inhalation.[2] Others have also reported disabling peripheral neuropathy[3,4] encephalopathy and cerebellar dysfunction.[5] However this case reported is concerned with the quantity of N-butane abused, the mode of inhalation and the psychological consequences.

Case

Miss X a 16-year-old girl was seen for N-butane gas abuse. She had been inhaling it for a year, and it was supplied by her boy-friend. She had collected over 22 cannisters each with a capacity of 232 ml which she had inhaled during the previous 3 months. The purpose of collecting them was to use the small quantity of gas left in them when she did not have a new supply. She suffered from visual hallucinations during initial abuse. During the period of abuse her school attendance became very irregular and she subsequently dropped out without taking any examinations. She was in trouble with the police for assaulting a police officer and was on probation. There was a gradual deterioration in social functioning leading to social isolation with very little contact with her peer group. The parents had noticed her becoming increasingly irritable and easily provocative and there was little communication with family members.

The family and personal history was unremarkable. Although she had experienced difficulties with her studies, the parents believe that her performance deteriorated rapidly with butane abuse. Physical examination was unremarkable. The patient did not co-operate with further haemotological an psychological investigations.

Discussion

While checking with the manufacturers, we found that commercial butane contains a mixture of propane, N-butane, and isobutane in varying pro-

Correspondence should be addressed to Dr B. Mathew.

portions to achieve a vapour pressure of 3.05–3.35 bar at which it is stored in the cannister. It is illegal to sell solvents to persons under the age of 18 years in the U.K. Commercial butane is used as a fuel in cigarette lighters.

Quantity

The most disturbing aspect of this case was the quantity of the solvent abused by the patient. She had abused about 22 cannisters each with a capacity of 232 mls, i.e. a total quantity of 5 litres in 3 months. As she had admitted to abusing a similar quantity over the past 12 months it is quite possible that she had abused about 20 litres in 1 year. This is an important finding because in most cases a reliable history is not forthcoming and it cannot be corroborated as most evidence is destroyed by the abusers.

Mode of Inhalation

The patient used the cover of the cannister as the mask for the abuse. It acted in a way similar to an oxygen mask, ensuring prompt delivery of the solvent from the cannister into the lungs. This method was certainly dangerous as the mask fitted firmly around the mouth and the nostils leading to a lowering of the partial pressure of inhaled oxygen causing hypoxia and other related disturbances. It is quite possible that prolonged abuse by this mechanism alone could cause death.

Accessibility

Although the patient could not obtain the cannisters over the counter because of her age she had regular supplies through her boy-friend. This illustrates the fact that legislation in sales alone is not enough[6] in combating solvent abuse.

Psychological Consequences

In this case the deterioration in academic performance during the period of N-butane abuse was leading to discontinuation of schooling was disturbing. Although the literature mentions that solvent abuse is a group activity, we noted this case to be an individual one with very little influence from the peer group. Another interesting finding was the rejection of the patient by her peers mainly due to her aggressive behaviour towards them, leading to social isolation. This was associated with irritability, impulsivity and deterioration in communication with the parents.

References

1. Barns, G. E. (1979) Solvent abuse. A review, *International Journal of Addiction*, 14, pp. 1–26.
2. Grebiski, D. A. (1961) Toluene sniffing producing cerebellar degeneration, *American Journal of Psychiatry*, 118, pp. 461–462.
3. Lewis, J. D., Moritz, D. J. & Mellin, L. P. (1981) Long term toluene abuse, *American Journal of Psychiatry*, 138, pp. 368–370.
4. Korobkin, *et al.* (1975) Glue sniffing neuropathy, *Archives of Neurology*, 32, pp. 158–62.
5. Boor, J. W. & Hurtig, W. I. (1977) Persistent cerebella ataxia after exposure to toluene, *Annals of Neurology*, 2, pp. 440–42.
6. Solvent Abuse (1982) Editorial, *Lancet*, pp. 1939–40.

文献 10

Health Effects of Selected Chemicals
- Volume 3

Nord 1995:28

Health Effects of Selected Chemicals

- Volume 3

Nord 1995:28
ISBN 92 9120 716 0
ISSN 0903-7004

Cover illustration: Kjeld Brandt, Copenhagen
Print: AKA-PRINT A/S, Århus 1995
Copies: 600
Printed on paper approved by the Nordic Environmental Labelling.
Distribution: This publication may be purchased from any of the agents listed on the last page.

Nordic Council of Ministers
Store Strandstræde 18
DK-1255 Copenhagen K
Telephone: +45 33 96 02 00

The Nordic Council
P.O. Box 19506
S-104 32 Stockholm
Telephone: +46 8 453 47 00

Nordic Environmental Cooperation
Environmental cooperation is aimed at contributing to the improvement of the environment and forestall problems in the Nordic countris as well as on the international scene. The cooperation is conducted by the Nordic Committee of Senior Officials for Environmental Affairs. The cooperation endeavours to advance joint aims for Action Plans and joint projects exchange of information and assistance, e.g. to Eastern Europe, through the Nordic Environmental Finance Corporation (NEFCO).

Nordic Council of Ministers
was established in 1971. It submits proposals on co-operation between the governments of the five Nordic countries to the Nordic Council, implements the Council's recommendations and reports on results, while directing the work carried out in the targeted areas. The Prime Ministers of the five Nordic countries assume overall responsibility for the co-operation measures, which are co-ordinated by the ministers for co-operation and the Nordic Co-operation Committee. The composition of the Council of Ministers varies, depending on the nature of the issue to be treated.

The Nordic Council
was formed in 1952 to promote co-operation between the parliaments and governments of Denmark, Iceland, Norway and Sweden. Finland joined in 1955. At the sessions held by the Council, representatives from the Faroe Islands and greenland form part of the Danish delega tion, while Åland is represented on the Finnish delegation. The Council consists of 87 elected members - all of whom are members of parliament. The Nordic Council takes initiatives, acts a consultative capacity and monitors co-operation measures. The Council operates via its insti