



## Pathology & Biology Section – 2008

### G30 Lethal Inhalation of Isomers of Butylene: A Case Report

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The goal of this presentation is to clarify the pathophysiology of butylene induced damage in humans by means of histological, histochemical and immunohistochemical investigation.

This presentation will impact the forensic community by serving as an educational resource on the pathophysiology and dangers of inhaling butylene isomers

There has been a steady increase in the number of deaths resulting from inhalation of volatile substances, which can be a suicide or an unintended consequence of “sniffing abuse”. Intentional inhalation of a volatile substance indeed is an under-recognized form of substance abuse in children and adolescents with a high morbidity and mortality.

Fatal outcome of inhalant abuse has been discussed due to several mechanisms: suffocation, trauma after dangerous behaviour, vagal inhibition, respiratory depression and the “sudden-sniffing death syndrome” following cardiac arrhythmia. However, the reason of sudden death related to volatile sniffing is rarely clear even after toxicological analysis. In most cases, reported aerosol propellants, n-propane or n-butane or mixtures of n-propane, n-butane, and isobutane are involved.

Sudden death due to the inhalation of butylene isomers has not yet been described in forensic literature.

There are four isomers of butylene ( $\alpha$ -butylene, cis- $\beta$ -butylene, trans- $\beta$ -butylene, isobutylene), which are all gases at room temperature and pressure, but can be liquefied by lowering the temperature or raising the pressure on them, in a manner similar to pressurized butane. These gases are colourless, but do have distinct odours, and are highly flammable. Although not naturally present in petroleum in high percentages, they can be produced from petrochemicals or by catalytic cracking of petroleum. There are few reports on the toxicology of these compounds in animals and humans; it is not clear if isomers of butylene can produce direct damage on lung endothelial cells or myocardial tissue like butane does, or if the injury is mediated by other mechanisms.

A 20-year-old male was found dead in his jail cell where a plastic bag and a portable cooking stove were present.

Forensic autopsy revealed cerebral edema, hemorrhagic edema of the lungs, and acute congestion of all inner organs. Histology (E&E) confirmed autopsy's results.

Toxicological analysis on the cooking stove gas and on biological specimens (blood and tissues) were performed. The cooking stove gas was formed by  $\alpha$ -butylene (71%), cis- $\beta$ -butylene (17%) and trans- $\beta$ -butylene (12%). Lormetazepam (85 ng/ml), GHB (800 ng/ml) and isomers of butylene ( $\alpha$ -butylene = 550 ng/ml; cis- $\beta$ -butylene = 130 ng/ml; trans- $\beta$ -butylene = 270 ng/ml) were determined in blood samples collected during autopsy.

The histochemical (Van Gieson and Azan Mallory) and immunohistochemical (myoglobin, actin, and desmin) investigations on myocardial samples showed interstitial fibrosis with acute necrosis and myocardial contraction bands.

The immunohistochemical examination (CD-34 and VIII factor) on lung specimens did not reveal endothelial damage.

These results suggest an acute electrical myocardial death due to adrenergic overdrive as a pathophysiologic mechanism of butylene induced sudden death.

To the authors knowledge, this is the first case study of sudden death due to the inhalation of isomers of butylene described in literature. The lack of knowledge of the exact biological effects of these compounds and the steady increase in the number of deaths resulting from inhalation of volatile substances need further investigations in toxicological and pathological fields.

**Isomers of Butylene, Lethal Inhalation, Toxicology**