Cerebral imaging showed no lesions of the cortex or the brain stem [4]. There was no hypoglycemia contrary to reports of propranolol intoxication in children [2]. This is the first report, to our knowledge, of such a pure form without dream-like features [3].

Propranolol-related low cardiac output could be responsible for transient hypoxic consequences in the peduncular areas [1–3]. In our case a direct pharmacological effect of this lipid-soluble beta-blocker that crosses the brain-blood barrier would explain the hallucinosis because of the history, the effectiveness of the antidote with the disappearance of hallucinations before the haemodynamic improvement and the membrane stabilizing effect of propranolol [1–3]. A serum level of 1,324 nmol/l two hours after exposure accounted for major propranolol intoxication [1]. The direct neurologic effectiveness of glucagon might be explained by the presence of glucagon receptors in some region of the brain and glucagon crossing the brain-blood barrier [5].

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Ether suicide poisoning by intravenous injection

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Sir: We read with interest the case report of Lambermont and colleagues [1] regarding intravenous ether-induced acute respiratory distress syndrome (ARDS). We would like to present another case of intravenous ether injection and to discuss several points in connection with this report.

Case report. A 24-year-old male was admitted in our ICU after a suicide attempt through an intravenous diethyl ether injection, in the same way he used this for killing animals in a veterinary clinic where he was an employee. Just before he injected 5 ml of this pure substance into his left cubital vein, he had called his relatives, who immediately ordered an ambulance, and the patient was transported to hospital in a few minutes. At arrival in the emergency room he appeared severely comatous, his eyes remained closed and he only uttered sounds as verbal response, he showed alternating flexion and extension movements, presenting a remarkable hypertonicity which led at times to opisthotonos. He was immediately intubated and connected to mechanical ventilation. His initial temperature was 35 °C, and it increased to 38.5 °C in less than 1 h. Initially, the laboratory tests showed increased plasmatic levels of ionic calcium (iCa^{2+} 3.79 mEq/l; normal values 2.24–2.60 mEq/l), which was followed by relative hypocalcemia (iCa^{2+} 1.04 mEq/l) in the next hours. Arterial blood gases on ICU admission were 7.28/53.4/534 with FIO_2 1.0 and they normalized later to modify the ventilator setting. Chest roentgenogram and ECG were considered normal. Screening for other toxic substances was found unremarkable. Gas chromatography confirmed the nature of the ether as diethyl ether. No alterations were detected on the liver function test and the remaining laboratory values were normal. The patient remained unconscious during the next 24 h, and finally he recovered consciousness. He could then be successfully extubated and discharged from the ICU on the next day, and was referred to psychiatric care.

Ether is the common name of diethylether (CH3-CH2-O-CH2-CH3), a solvent and, in the past, a widely used anesthetic agent. According to Lambermont and colleagues, pulmonary insult due to large doses of intravenous ether have never been reported [1]. In this journal these authors reported a 36-year-old woman who injected herself with 30 ml of ether intravenously, but, surprisingly, the physical examination on admission was normal with the exception of tachypnea and crackles on auscultation leading to pulmonary edema in the next hours. No mention of neurological signs was made by these authors. With a lesser amount of intravenous ether, deep coma was present in our patient for a long time. We believe that the compound reported by Lambermont was possibly not common ether (diethylether).

However, a large number of chemical compounds are ethers, with a general molecular formula R-O-R', where R and R' are organic -C radicals. This group includes thousands of possible structures, such as the well-known inhalational ethers (enflurane, isoflurane, methoxyflurane), and other compounds such as ethylene glycol butyl ether (EGBE) which is widely used as solvent and diluent in resins, inks, perfumes and cleaning fluids. Cases of pulmonary edema following inhalation, injection or ingestion of some of these compounds have been reported. Thus, in a fatal case of voluntary inhalation of enflurane, reported in 1989 by Jacob and colleagues, the lungs showed moderate to severe congestion at autopsy [2]. Previously, in 1972, Hohman had published another fatal case of intravenous methoxyflurane injection (5 ml), where the cause of the death was hemolysis, although pulmonary symptoms were also present [3]. In 1992 Bauer and co-workers reported a non-fatal case of a 53-year-old man with transient non-cardiogenic pulmonary edema following massive ingestion of EGBE [4]. In a case of suicide with Taxus baccata leaves, phloroglucindimethylether (3,5-dimethoxyphenol) could be identified as the main substance by Musshoff and Coworkers. At forensic autopsy, the bronchial epithelium was found markedly inflamed without signs of aspiration [5].

In our patient, where the exact nature of the ether – dietylether – could be demonstrated by gas chromatography, pulmonary edema or alterations of gas exchange were not present during the clinical course. Mechanical ventilatory support was only necessary due to neurological impairment. In addition to the neurological symptoms in our patient, the tetanic spasms we described can be explained by calcium derangement due to the effect of diethyl ether upon the function of the vesicles of the sarcoplasmic reticulum. This subject has been widely reported in several experimental studies [6–8].

In conclusion, we present a case of intravenous diethyl ether injection with features different from those reported by Lambermont in this journal. These differences suggest that an ether compound other than diethyl ether could have been the causative agent in the report of these authors.

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Reply

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Sir: The case reported by Serrano et al. is a very well documented and interesting observation of ether intravenous injection. As well as ours [1], this report probably represents a particularly rare event in emergency medicine, since most patients admitted after suicidal attempts use rather sedative and/or neuroleptic drugs. However Serrano et al. are concerned about our presentation and suggest that a derived ether compound rather than ether itself could be the cause of acute respiratory distress syndrome.

As a matter of fact, blood toxicologic screening revealed no particular drug overdose in our patient. Which ether was responsible was not proven in our report by gas chromatography, as was the case in the report of Serrano et al. In essence, the hypothesis that a significant amount of ether was injected intravenously by the patient was based upon the history provided by the patient and her relatives. This spontaneous declaration was in agreement with a pronounced smell of ether on her breath. She used "ether", identified as diethyl ether by her usual pharmacist provider, to dry clean cutaneous areas.

Assuredly, the clinical course of our patient was quite different from the ether-related case report of Serrano et al. Due to the lack of chemical identification of the solvent on admission, any a posteriori interpretation to explain the difference in the observations remains purely speculative.

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The use of fibrinolytics in purulent pericarditis

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Sir: I read with great interest the three case reports by Defouilloy et al. [1] and Maynar et al. [2] over the last year in *Intensive Care Medicine* regarding the use of fibrinolytics in purulent pericarditis (PP).

I feel it important to add an historical perspective to these cases in light of the fact that the unfortunate hallmark of this most promising treatment, for over forty years, has been ignorance of previous work and resultant repeated empiric trials in isolation from the published literature. Defouilloy's erroneous claim of there only being one previous documented case serves to highlight this point in addition to referencing difficulties in both current and past medical indexing services.

Intrapericardial fibrinolysis with streptokinase for the treatment of PP was first instituted by Wright and co-workers in 1951 [3]. Over the next four years four further case reports appeared, with no reference made to earlier work [4]. Despite universal success and the absence of side effects, the treatment did not draw attention.

Bennett published two cases in 1984 [5] but contemporaneous respondents were not aware of the earlier work of the 1950s. As such, the technique was considered promising but anecdotal, requiring further documentation. Maynar and Defouilloy's cases when reviewed in the context of the 1950s work, Winkler's 1994 experience with urokinase [6] and the case I reported in 1996 [4] fulfill this requirement. Intrapericardial fibrinolysis appears to be a safe and relatively noninvasive treatment option.

The theoretical risks and benefits of differing fibrinolytic regimes with or without local antibiotic lavage have been previously documented, as has the potential role of fibrinolytic lavage in the loculated form of the disease [7, 4]. Recent individual case reports have played their role in increasing awareness of fibrinolytic therapy for PP. It is now time to move ahead and institute a multicentered prospective randomized trial in order to determine the most efficacious treatment regime.