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Editorial

The odorless, the colorless, the tasteless and the complacent

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In this issue of the Acta Anaesthesiologica Belgica, Chakupurakal et al describe a case series of acute CO_2 overexposure in patients undergoing general anesthesia, caused by CO_2 delivery instead of N_2O[1]. Because the thread of the fittings between the N_2O and CO_2 cylinders in Belgium differ by only 0.2 mm, N_2O and CO_2 cylinders could easily be misconnected. The case series illustrates that despite all existing precautions (reviewed in some detail by the authors) deadly mixtures of odorless, colorless, and tasteless gases can still be delivered to the lungs of our patients.

CO_2 can kill. It is used to provide euthanasia in animals[2]. Death is caused by its toxic effects, not necessarily by hypoxic mixtures per se (although hypoxemia may contribute). In humans, low concentrations have little, if any, toxicological effects. At higher concentrations (>5%), hypercapnia and respiratory acidosis ensue. Concentrations above 10% may cause convulsions, coma, and death[3]. CO_2 levels of more than 30% rapidly lead to loss of consciousness within seconds. The effects in humans are illustrated in Table 1[4-6].

In the reports by Chakupural et al., the dialed gas mixture was 50% O_2 and 50% "N_2O". The CO_2 concentration at the common gas outlet was 297 mmHg or approximately 40% because CO_2 was delivered via a N_2O flow meter. While the viscosity of CO_2 and N_2O are identical at room temperature, the density of CO_2 (1.87 kg/m^3) is higher than that of N_2O (1.977 kg/m^3), causing the flow of CO_2 to be lower than dialed. Even so, this was a potentially lethal CO_2 concentration. The only line of defense left between the wrong connection and the patient was, fortunately, a properly functioning multigas analyzer with active alarm and discoloration of the sodalime which rapidly alerted the anesthesiologists to the problem. Swift action prevented patient harm: administering 100% O_2, increasing fresh gas flows, and confirming delivery of O_2.

But what about other lines of defense for other gases we co-administer with O_2? Even when the correct gas cylinder is attached, hypoxic mixtures can form when O_2/N_2O and O_2/air are administered in a circle breathing system. N_2O is not acutely toxic per se but can kill by becoming part of an inspired hypoxic mixture. This risk is supposed to be minimized not only by gas analysis but also by the obligatory O_2/N_2O proportioning device. This device increases the relative proportion of O_2 in the O_2/N_2O mixture as the total FGF is lowered. Halas, the system does not work: inspired hypoxic mixtures can still form, especially within the 1 – 2 L/min FGF range[7]. The system will fail altogether when a CO_2 cylinder is wrongly attached instead of the N_2O cylinder because even non-hypoxic CO_2 mixtures can be toxic! End-expired gas analysis with properly set alarm limits remains essential.

And what about the plain good air we breath? Air/O_2 mixtures can also cause the formation of inspired hypoxic mixtures when an improper mixture of O_2/air is delivered into a circle breathing system. In a sense, N_2 is worse than N_2O. First, we do not measure N_2 concentrations because it is a symmetrical molecule (and thus cannot be measured by infrared gas analysis) and because it has no paramagnetic properties (and thus

cannot be measured by a paramagnetic analyzer, used to measure O_2). Second, there is no proportioning system such as the O_2/N_2O proportioning device. Third, N_2 is far less soluble than N_2O (total body stores are only 3 L in the average adult). When air is delivered at a FGF below minute ventilation hypoxic inspired mixtures will form[7-9]. The low FiO_2 alarm will go off, but if no appropriate action is taken, the FiO_2 will decrease to lethal levels. Even though an active inspired hypoxic guard exists that automatically increases O_2 delivery if this should occur, the response by the anesthesia community to its availability has been lackluster: its adaptation in modern anesthesia workstations is rather slow. An active inspired hypoxic guard would also be activated when inspired hypoxic mixtures form when the wrong O_2/N_2O mixture is dialed. Target controlled delivery (where the clinician would demand a target inspired or end-expired O_2 concentration) would minimize the risk of improper carrier gas mixing by the anesthesia provider.

To summarize, it is appalling that no uniform standard exists for cylinder fittings in Europe. And it is even more appalling that two nearly identical fittings can be used to connect both N_2O and CO_2 cylinders. N_2O and N_2 can kill. It is dreadful that active inspired hypoxic guards are not becoming an obligatory requirement for future anesthesia workstations. Why are we, the anesthesia community, not more aggressive at closing these safety loopholes? Complacency reigns, it seems. Complacency is defined as overconfidence, self-satisfaction, or smugness that's bred from success and makes us unaware of potential dangers or threats. The success is patient safety. Anesthesiology has obtained a very commendable track record when it comes to reducing anesthesia related mortality and morbidity. But, as this case series illustrates, loopholes still exist. Therefore, we should not become complacent but instead continue actively closing as many as we can. There is no room for complacency when we are delivering odorless, colorless, and invisible gases and drugs to our patients.

Table I. — Health effects in function of concentration and duration of carbon dioxide (CO_2) exposure. The lowest published lethal concentration is 9% for 5 min.
min. = minutes; BP = blood pressure; HR = heart rate. Figure adapted from text references[4-6].

EXPOSURE			HEALTH EFFECTS
CO_2 concentration		maximal exposure limit (min.	exposure duration and symptoms
partial pressure CO_2			
mm Hg	%		
3.8	0.5	unlimited	no symptoms
7.5	1.0	unlimited	
11	1.5	480	
15	2.0	60	several hours → headache, dyspnea with mild exertion
23	3.0	20	1 hour → headache, sweating, dyspnea at rest
30	4.0	10	
38	5.0	7	few min. → headache, dizziness, increased BP, dyspnea
45	6.0	5	1 – 2 min. → auditive and visual disturbances ≤ 16 min. → headache, dyspnea several hours → tremors
53	7.0	<3	few min. → progressive loss of consciousness several hours → symptoms described above, increased HR, tachpnea
68	9.0	-	dizziness → drowsiness → severe muscle twitching → loss of controlled activity → unconsciousness → convulsions → coma → death
75	10	-	
113	15	-	
128	17	-	

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