(receiving placebo or fentanyl) experienced a profound subjective drug effect. There is evidence that diazepam produces no sensory effects (*15*). The results of the second study were similar to the results of the first (*16*), in that the sensory intensity responses were reduced significantly in comparison to the responses with placebo. Unpleasantness responses were not significantly reduced, although a clear trend of greater reduction after placebo was noted. These results show that although subjective effects of fentanyl could be detected, they did not influence the results.

Our experiments demonstrate that the words chosen for a pain scale can influence significantly the outcome of experimental and clinical studies of the efficacy of pain control agents. They also indicate the need for independent measures of the multiple dimensions of the pain experience (*17*). In studies in which singular indices of pain are used, critical pain dimensions may not be assessed adequately.

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References and Notes

1. A. Goth, *Medical Pharmacology* (Mosby, St. Louis, 1972), p. 278; J. H. Jaffe, in *The Pharmacological Basis of Therapeutics*, L. S. Goodman and A. Gilman, Eds. (Macmillan, New York 1975), p. 248.
2. H. K. Beecher, *J. Chron. Dis.* **4**, 11 (1956); *Science* **130**, 267 (1959); *Measurement of Subjective Responses* (Oxford Univ. Press, New York, 1959); *Science* **151**, 840 (1966).
3. H. R. Moskowitz, R. A. Kluter, J. Westerling, H. L. Jacobs, *Science* **184**, 583 (1974); G. Mower, R. Mair, T. Engen, in *The Chemical Senses and Nutrition*, M. Kare and O. Maller, Eds. (Academic Press, New York, 1977), p. 104; D. A. Thompson, H. R. Moskowitz, R. G. Campbell; *J. Appl. Physiol.* **41**, 77 (1976); G. Mower, *J. Comp. Physiol. Psychol.* **90**, 1152 (1976); R. L. Doty, *Percept. Psychophys.* **17**, 492 (1975).
4. R. H. Gracely, P. McGrath, R. Dubner, *Pain* **5**, 5 (1978).
5. The ratio-scale values for these descriptors have been reliable within subjects from session to session (mean $r = .93$) and reliable between individual subjects and mean scales from groups of 15 subjects similar in age and education (mean $r = .93$). These equivalent correlations show that an individual scale is predicted equally well by another scale from either that individual or a similar group. This equivalence shows that the scales are highly objective and supports the use of group normative descriptor scales for the analysis of individual scaling data [J. P. Guilford, *Psychometric Methods* (McGraw-Hill, New York, 1974)].
6. R. H. Gracely, P. A. McGrath, R. Dubner, *Pain* **5**, 19 (1978); C. R. Chapman and B. W. Feather, *Psychosom. Med.* **35**, 330 (1973); K. Blum, *Psychopharmacology* **17**, 391 (1970); H. E. Shannon, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **33**, 474 (1974).
7. B. Tursky, in *Pain: New Perspectives in Therapy and Research*, M. Weisenberg and B. Tursky, Eds. (Plenum, New York, 1976), p. 171.
8. S. S. Stevens and H. B. Greenbaum, *Percept. Psychophys.* **1**, 439 (1966); S. S. Stevens, *Psychophysics: Introduction to Its Perceptual, Neural and Social Prospects* (Wiley, New York, 1975).
9. Stimuli from 6 to 100 μA were delivered by means of a hand-held nylon probe to the labial surface and incisal edge of intact healthy upper central incisors. The electrode was incorporated into the notched end of the probe, and contact was made with conductive paste. The metal handle of the probe served as an indifferent electrode. In a related study (M. W. Heft, R. H. Gracely, P. A. McGrath, R. Dubner, unpublished observations) we have shown that the sensations produced by these stimuli arise from pulpal nerve fibers rather than as a consequence of current spread to nonpulpal fibers in adjacent periodontal and gingival tissue. Dental patients undergoing endodontic treatment did not perceive stimuli up to 100 μA applied to saline-filled pulp chambers after the pulpal nerves were removed.
10. See D. J. Mayer and D. D. Price [*Pain* **2**, 379 (1976)] and Liebeskind and Paul (*11*) for reviews.
11. J. C. Liebeskind and L. A. Paul, *Annu. Rev. Psychol.* **28**, 41 (1977).
12. R. L. Hayes, R. D. Harris, P. J. Wolskee, R. Dubner, *Neurosci. Abstr.* **3**, 483 (1977).
13. D. D. Price and R. Dubner, *Pain* **3**, 307 (1977).
14. L. Lasagna, J. M. von Felsinger, H. K. Beecher, *J. Am. Med. Assoc.* **157**, 1006 (1955); G. M. Smith and H. K. Beecher, *J. Pharmacol. Exp. Ther.* **136**, 53 (1962).
15. C. R. Chapman and B. W. Feather, *Psychosom. Med.* **35**, 330 (1973).
16. R. H. Gracely, R. Dubner, P. A. McGrath, unpublished observations.
17. R. Melzack, *Pain* **1**, 277 (1975); _____ and K. L. Casey, in *The Skin Senses*, D. R. Kenshalo, Ed. (Thomas, Springfield, Ill., 1968), p. 423; R. Melzack and W. S. Torgerson, *Anesthesiology* **34**, 50 (1971).
18. We thank D. P. Butler, M. W. Heft, A. A. Macynski, and J. B. Sweet for assistance, and G. J. Bennett, R. L. Hayes, and D. D. Price for critical comments of the manuscript.

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Possible Mechanism for Pressurized-Liquid Tank Explosions or BLEVE's

Abstract. *The hypothesis is made that rapid depressurization of hot, saturated liquids may result in an explosion. The temperature of the hot liquid must, however, be above the superheat limit temperature at 1 atmosphere, and the drop in tank pressure must be very rapid. Two examples of large-scale pressure-letdown explosions are cited and possible preventative measures suggested.*

Reports of explosions of tank trucks or railroad cars carrying pressurized liquids are frequently in the news. Investigation of such accidents usually reveals a common scenario. The tank truck or railroad car has been involved in an accident with a resultant fire. Energy transfer from the fire to the tank has two effects. First, the liquid contents are heated, and there is a concomitant rise in tank pressure as determined by the vapor pressure of the liquid. The tank metal wetted by the liquid increases only slightly in temperature, although the pressure can change significantly since the vapor pressure is an exponential function of temperature. Second, for areas of the tank not in contact with liquid, there is no efficient heat absorption mechanism and the fire can lead to rapid and large increases in the temperature of the metal. In time, the pressure rise causes the safety valves to operate and more fuel is released to exacerbate the local fire. Eventually, through a combination of high internal tank pressure and high metal wall temperatures in regions not covered by liquid, localized metal failure is initiated.

To this point, the mechanism described is well accepted and is normally invoked in explaining BLEVE's (boiling liquid expanding vapor explosions). The new element suggested in this report concerns events that immediately follow the initial failure of the tank metal. Soon after metal failure there is usually an explosion accompanied by a shock wave that destroys and often fragments the tank. The very rapid release of fuel is manifested in an impressive fire—even a fireball—and portions of the tank can be scattered over a wide area. In some cases there may be a secondary fuel-air explosion not dissimilar to those used by the military to produce damaging shock waves over wide areas. The question of primary interest is: What is the source of the initial powerful explosion?

The explanation suggested here is related to the behavior of the hot liquid as the tank is very rapidly depressurized after failure of its metal wall. Just before failure, the liquid was saturated—that is, the temperature and pressure were as expected for a system where the liquid was in equilibrium with the vapor phase. With a rapid decay in tank pressure due to failure of the metal wall, the bulk liquid should boil and reduce the temperature to a value compatible with the lower pressure. But initiation of the boiling process requires efficient nucleating sites. There are no such nucleating sites in the bulk of the liquid, and for a brief period after depressurization, this bulk liquid is superheated—that is, at a temperature higher than the boiling temperature predicted at the existing pressure. If the bulk temperature is sufficiently above the expected boiling point, a superheated liquid-vapor explosion would be expected. Such explosions are known to occur in the microsecond time domain with concomitant shock waves.

Superheated liquid-vapor explosions occur frequently in some industrial operations. Usually, however, they occur when two liquids—one hot and nonvola-

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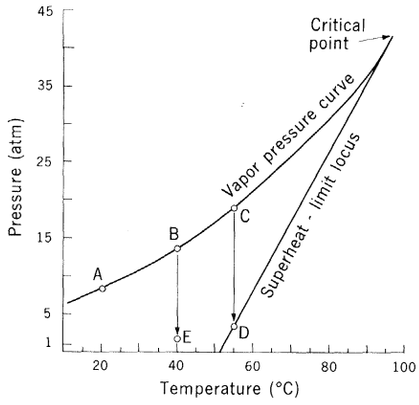

Fig. 1. Vapor pressure and superheat curves for propane.

tile and the other cold and volatile—come in contact under appropriate conditions. The cold, volatile liquid cannot boil on the hot liquid since there are no suitable nucleation sites, and it therefore superheats. There is a theoretical limit to the attainable degree of superheat, and if this limit is reached, spontaneous homogeneous nucleation occurs in the cold liquid with the formation of vapor in a very brief period of time. To an observer, this vapor formation resembles an explosion. Superheated liquid-vapor explosions are reviewed in (*1*, *2*).

Application of the superheated liquid-vapor explosion theory to the rapid depressurization of tanks containing a liquid may be illustrated by the following example. Consider a tank of liquid propane which, due to some accident, is engulfed in a fire. Before the accident, the liquid propane was at ambient temperature and the tank pressure was 8 to 9 atm. This initial equilibrium state is shown as point *A* in Fig. 1. Because of the fire, the propane warms and the pressure increase follows the vapor pressure curve (Fig. 1). (Although in an actual case there may be some stratification of the warmer liquid near the vapor-liquid interface, the assumption of a well-mixed liquid is not unreasonable.) Also shown in Fig. 1 is a line termed the superheat-limit locus. This represents the limits to which propane liquid may be heated before spontaneous nucleation occurs with a vapor explosion; it can be calculated from theory or measured experimentally in the laboratory.

Suppose the propane tank failed when the pressure and temperature were those corresponding to point *B* in Fig. 1. Rapid depressurization would occur, as depicted by a vertical line terminating at 1 atm (point *E*). Violent boiling would take place within a few seconds, but since the superheat-limit locus was not attained, no vapor explosion would be predicted. However, if failure occurred under the conditions denoted by point *C* (~55°C), rapid depressurization to point *D* (~3.3 atm) could lead to a vapor explosion. In fact, any tank temperature exeeding about 53°C (tank pressure >16.3 atm) could produce vapor explosions—but if failure occurred before this point, only rapid boiling would be expected. This definitive cutoff suggests that safety valves might be designed to prevent any pressure exceeding this critical limit.

Also, small-scale tests with single drops of superheated liquids indicate that the difference in pressure between the vapor pressure curve and the superheat-limit locus is related to the intensity of the initial explosion. Thus tank failure near the critical point may result in a lower explosive potential even though there is significant energy stored in the liquid.

Too few data are available at present to substantiate or disprove the hypothesis above. In one well-instrumented test (*3*), a 33,600-gallon railroad tank car filled with liquefied petroleum gas (LPG) containing ~ 98 percent propane was exposed to an intense kerosene fire, and catastrophic failure occurred in 24.5 minutes. Typical liquid temperatures and tank pressures measured in the test are shown in Table 1. Early in the test the tank pressure rose above the predicted vapor pressure, but in about 6 to 8 minutes it was close to the expected pressure (Fig. 1). At tank failure the liquid temperature exceeded the critical value of 53°C. I suggest that the bulk liquid attained the superheat-limit temperature when the internal pressure was about 16.5 atm, and an explosion occurred. (In Fig. 1, a vertical line at 69°C intersects the superheat-limit locus at 16.5 atm.)

Further support for the superheat-limit concept is provided by a different example. An industrial autoclave reactor containing vinyl chloride monomer recently exploded when the pressure was suddenly released. The resultant fireball was 300 feet in diameter, and a 2-ton section of the autoclave was blown 1600 feet. At the time of the pressure release the bulk liquid temperature exceeded the critical value of 110°C, the superheat-limit temperature for vinyl chloride at 1 atm. After a detailed analysis of the accident company engineers concluded that the explosion was due to rapid, homogeneous nucleation of the bulk liquid vinyl chloride as a result of the rapid pressure release.

Hess *et al.* (*4*) studied the bursting of small liquid propane tanks with the objective of clarifying the behavior of the cloud of propane vapor released. The tanks were 4 or 6 cm in diameter with a length-to-diameter ratio of 3.5. The liquid propane completely filled the tank at the moment the tank burst, and the pressure exceeded the saturation value. (On Fig. 1, a typical starting condition would be ~ 60°C and 60 atm.) Violent explosions were recorded, with the main event completed in about 3 msec. Hess *et al.* did not invoke superheated liquids as the cause of the explosion, but their data would support such a hypothesis.

Table 1. Typical liquid LPG temperatures and pressures measured in a tank car engulfed in flame.

Time* (min)	Temperature (°C)	Pressure (atm)
0	21	7.5
4	32	16.0
8	52	18.1
10	60	19.1
12	66	20.4
14	67	21.5
16	69	22.1
18	70	22.5
20	69	22.5
22	69	22.1
24	69	21.8
24.5	Explosion	

*Note: The safety valve began to lift between 2 and 4 minutes.

In a study reported by Ogiso *et al.* (*5*), rapid depressurization of superheated liquids was cited as the cause of a number of industrial accidents in Japan. No specific incidents were described, but limited experimental data were given for water that was mildly superheated before depressurization (108° to 155°C). Under these conditions no superheat-limit explosion would be predicted and none was found. After the initial rapid pressure drop in the water tank, the pressure rose to several times the initial saturation value. But the time scale for these pressure changes ranged from 20 to 100 msec—much longer than would be expected for true superheat-limit explosions (~ 1 msec). What Ogiso *et al.* observed was the violent boiling of a superheated liquid with some water hammer as slugs of liquid were accelerated and decelerated within the tank and exit vent.

The hypothesis presented here to explain pressurized-liquid explosions and BLEVE's has not been proved and well-conceived experiments are needed. However, if the basic concept is valid, several preventive measures are immediately suggested. One has already been noted—proper design of the safety valves or burst disks, so that the liquid temperature is never allowed to exceed the critical value. Another method would

be to "load" the liquid with solid particles to provide nucleation sites and induce rapid boiling in the bulk should a pressure decay occur. Apart from the obvious practical difficulties, however, recent experiments by Buivid and Sussman (*6*) appear to show that this idea is not viable. They showed that the experimental superheat-limit temperature for liquids was not greatly affected even when the liquids were loaded with suspended hydrophobic or hydrophilic particles. Another concept worth further study would be the use of "gelled" liquids—liquids modified to resemble gels by use of very small quantities of frozen water or methyl alcohol. The solid phase is composed of very fine particles (< 1 μm) dispersed homogeneously throughout the liquid (*7*). In this case, the spacing between nucleation sites might be sufficiently small to allow effective nucleation in the event of a pressure drop. Finally, safety valves and rupture disks might be redesigned to prevent very rapid pressure decays even in the event of overpressurization.

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References and Notes

1. R. C. Reid, *Am. Sci.* **64**, 146 (1976).
2. ———, *J. Chem. Eng. Educ.* **12** (No. 2), 60 (1978); *ibid.* (No. 3), p. 108; *ibid.* (No. 4), p. 194.
3. C. Anderson, W. Townsend, J. Zook, G. Cowgill, *The Effects of a Fire Environment on a Rail Tank Car Filled with LPG* (FRA-ORED 75-31, U.S. Department of Transportation, Washington, D.C., 1974).
4. K. Hess, W. Hoffmann, A. Stoeckel, paper presented at the First International Loss Prevention Symposium, The Hague, Netherlands, 1974.
5. C. Ogiso, N. Takagi, T. Kitagawa, paper presented at the Loss Prevention and Safety Symposium PACHEC, Japan, 1972.
6. M. G. Buivid and M. V. Sussman, *Nature (London)* **275**, 203 (1978).
7. L. M. Shanes, Ph.D. thesis, Massachusetts Institute of Technology (1977).

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Noradrenaline and Seizures

Tabakoff *et al.* (*1*) presented evidence on the role of brain noradrenaline (NA) in the development of tolerance to barbiturates: animals that suffered a 50 percent depletion of brain NA through intraventricular injection of 50 μg of 6-hydroxydopamine (6-OHDA) failed to develop tolerance to long-term barbiturate treatment, as measured by sleep time and hypothermia after a subsequent challenge dose of barbiturate, or by potentiation of seizures induced by pentylenetetrazol (Metrazol). Tabakoff *et al.* (*1*) also compared the susceptibility of control and 6-OHDA-treated animals to Metrazol-induced seizures even in the absence of chronic barbiturate treatment and found no effect of the 6-OHDA.

We have recently obtained evidence that brain NA is indeed involved in Metrazol-induced seizures and that a marked alteration in these seizures occurs if NA is depleted (*2*). Animals that received 4 μg of 6-OHDA injected into the fibers of the dorsal noradrenergic bundle, a procedure that depleted forebrain NA to less than 5 percent of control values, showed a marked potentiation in the duration, number, and type of seizure elicited by subcutaneous administration of 70 mg of Metrazol per kilogram of body weight (Table 1). Thus, the seizures lasted longer, occurred more frequently, and were tonic rather than clonic. These data support our earlier report that depletion of both NA and dopamine (DA) in varying proportions also potentiates Metrazol-induced seizures (*3*). They also support data indicating that catecholamines are significantly involved in seizures induced by other means (*4*). An alteration in brain NA may therefore be important in the pathogenesis of human conditions such as epilepsy. We suggest that the failure of Tabakoff *et al.* (*1*) to observe a potentiation of Metrazol-induced seizures in rats treated with 6-OHDA reflects the modest (no more than 50 percent) depletion achieved by their manipulation. In other catecholaminergic systems, such a small loss would be without behavioral effect (*5*), and the effectiveness of this loss in altering the development of tolerance to barbiturates testifies to the pervasive role of brain NA in this phenomenon.

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References and Notes

1. B. Tabakoff, J. Yanai, R. F. Ritzmann, *Science* **200**, 449 (1978).
2. S. T. Mason and M. E. Corcoran, *Life Sci.*, in press.
3. M. E. Corcoran, H. C. Fibiger, E. G. McGeer, J. A. Wada, *J. Pharm. Pharmacol.* **25**, 497 (1973); M. E. Corcoran, H. C. Fibiger, J. A. McCaughran, J. A. Wada, *Exp. Neurol.* **45**, 118 (1974).
4. E. W. Maynert, *Epilepsia* **10**, 145 (1969); P. S. Arnold, R. J. Racine, R. A. Wise, *Exp. Neurol.* **40**, 457 (1973); I. H. Ayhan, *Arch. Int. Pharmacodyn.* **223**, 282 (1976); R. A. Browning and E. W. Maynert, *Fed. Proc. Fed. Am. Soc. Exp. Biol.* **29**, 417 (1970); A. Quattrone and R. Samanin, *Eur. J. Pharmacol.* **41**, 333 (1977); A. Lehmann, *Life Sci.* **20**, 2047 (1977).
5. I. Creese and S. D. Iversen, *Nature (London) New Biol.* **238**, 247 (1972); *Brain Res.* **83**, 419 (1975).

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Mason and Corcoran have presented evidence that a profound depletion of brain norepinephrine (NE) results in a potentiation of pentylenetetrazol-induced seizures. They suggest that the lack of such effects in our studies (*1*) was due to a less extensive destruction of brain NE neurons. We totally agree with their assessment and would like to reiterate certain points that appeared in (*1*) as well as in our previous publications on this subject (*2, 3*). Adrenergic receptor supersensitivity develops during the 2-week interval between injection of 6-hydroxydopamine (6-OHDA) and the testing of the animals in our studies and we have stated: "the development of recep-

Table 1. Seizure response to subcutaneous injection of Metrazol (70 mg/kg) in NA-depleted rats (4 μg of 6-OHDA was injected into the fibers of the dorsal NA bundle); N.S., not significant.

Group	Duration of first seizure (sec)	Number of rats		Noradrenaline content of tissue*				Dopamine content of striatum
		With multiple seizures	With tonic seizures	Hippocampus-cortex	Hypothalamus	Cerebellum	Spinal cord	
Control (N = 7)	32.9 ± 3.0	0/7	0/7	246 ± 6	2,230 ± 77	219 ± 12	255 ± 6	13,170 ± 570
6-OHDA (N = 9)	113.2 ± 11.7	5/9	5/9	6 ± 1	590 ± 87	271 ± 8	307 ± 12	11,570 ± 1,190
P	.01	.05	.05	.001	.001	N.S.	N.S.	N.S.

*Percentages for the 6-OHDA-treated rats were, for NA: hippocampus-cortex, 2; hypothalamus, 26; cerebellum, 124; and spinal cord, 120; and for DA: 88.

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