Letters

Technological Hazard

"The nature of technological hazard" by C. Hohenemser *et al.* (22 Apr., p. 378) may provide a taxonomy for studying certain hazards, but it should not be taken as a guide for public policy decisions. It considers only a piece of the issue, and only part of that.

The taxonomy omits the entire subject of benefits and, in so doing, ignores the hazards to society and the environment of curtailing or being without the contributions of the technology itself.

While calculations of these downside risks (from energy shortages, lack of new medicines, loss of productivity, and so forth) are much harder to do, even rough estimates often show that such risks dwarf the environmental hazards. Predictions are uncertain, but in some areas, like energy shortages, the risks should be minimized: the consequences of such shortages would be much worse than those due to any combination of hazards from energy production.

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Hohenemser *et al.* provide a rich framework for a broadened conception of risk. However, a critical class of dangers is missing from their listing of 93 sample hazards—those posed by the accumulation of materials. The authors compare policy choices of triage, when dealing with the most hazardous individual items, with cost-effective reduction when trying to remedy those one can handle effectively. I suggest adding the disquieting choice of dealing with an accumulation of materials that are individually sanguine but deadly in sum.

Certain chemical accumulations appear somehow to overload otherwise healthy human immune or detoxification systems, or both. The resultant "environmental illness" is marked by varied, extreme allergic symptoms in some numbers of people. The critical question is whether many others will get sick as chemical varieties and concentrations increase and the total accumulation diffuses over large regions.

The Clark University group's framework shows the enormity of the danger if one just scans the dozen hazard descriptors with the wealth of commercial chemicals in mind. As the authors state, "we cannot make extraordinary efforts on each of the 100,000 chemicals." There may, however, be effective ways to minimize affronts within structures or to increase bodily resistance with individually tailored diets or genetic engineering. We need to research the effects of cumulative chemical hazards to learn the limits of the problem, the mechanisms involved, and corresponding courses of action.

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The letters from Rossin and Porter are helpful because each, in its own way, highlights the limitations of our analysis. Meeting their demands, however, will not be easy.

Comparing risk to benefit, as suggested by Rossin, is of course essential to all decisions about hazardous technology. But risk-benefit comparisons are not so much uncertain as afflicted by unresolved, and possibly unresolvable, value questions. How should benefits be measured? If in dollars, what is the value of human life? What value should be placed on more subtle "goods," such as wilderness, species diversity, or unlimited energy supply? How should society handle the typical case in which one group receives the benefits and another bears the risks?

In the light of such questions, I do not share Rossin's conclusion that "the consequences of energy shortages would be much worse than those due to any combination of hazards from energy production." It depends on who judges. In such value conflicts about hazards and benefits, it may therefore be useful for participants to share a relatively objective, commonly held analysis of hazards, such as that provided by our article. Separating the issues is often the first, best step toward useful decisions; in this sense, I believe our work does contribute to policy decisions, even though it cannot by itself resolve them.

The cumulative effects of many individually innocuous materials hazards is, as noted by Porter, an important issue; and it is, I admit, not handled well by our recent article. In earlier work (*1*), our group has discussed two possible approaches: (i) adding up the estimated consequences of individual materials exposures and (ii) estimating cumulative impact through the use of global measures of exposure, such as employment in industry. We concluded that approach (i) is at present hopeless, whereas approach (ii) yields answers with large error bounds. For example, we estimated that "10 to 30 percent of cancer is correlatively associated with technology." Because I find such necessarily vague conclusions unsatisfactory, I strongly support Porter's call for further detailed research on cumulative effects.

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Alcohol and Pregnancy

Mukherjee and Hodgen report (Reports, 12 Nov. 1982, p. 700) that a single intravenous dose of ethanol administered to pregnant monkeys produced a transient "collapse" of umbilical circulation and significant changes in umbilical vein blood gases (hypoxia and acidosis). This confirmation in primates of blood gas changes reported earlier in sheep fetuses (*1*) may indeed bring us closer to an understanding of mechanisms involved in the fetal alcohol syndrome (FAS), as Mukherjee and Hodgen suggest. However, they end their report with a recommendation that may be unwarranted.

Mukherjee and Hodgen suggest that "this striking interruption of feto-placental circulation may explain one of the mechanisms of mental retardation, a frequent manifestation in children afflicted with fetal alcohol syndrome." While there is evidence that a single injection of alcohol in pregnant mice with no previous exposure to it produces altered patterns of fetal brain and craniofacial morphogenesis (*2*), when alcohol was administered under conditions that more

closely approximate normal human consumption, there was little effect; when ethanol was ingested orally and regularly during pregnancy by female rats with some prior exposure to the substance, no effects on the development or behavior of their offspring were observed (*3*); in addition, available evidence indicates no adverse effects of moderate drinking in pregnant women (*4*). Either oral administration has less effect or tolerance develops with habitual alcohol consumption, so that fetal blood gases are not severely affected by moderate oral doses—or changes in feto-placental circulation are not a major mechanism responsible for FAS.

Consequently, the evidence available does not justify advising even monkeys to abstain from alcohol entirely during pregnancy, which is Mukherjee and Hodgen's "prudent recommendation" for pregnant women. The danger of recommending total abstention on the basis of available data appears twofold:

1) With specific reference to FAS, the recommendation may result in more unsuccessful than successful attempts at total abstention. This may produce just the reverse of the intended benefits, an intake pattern that mimics what appear to be dangerous conditions—acute ingestion of ethanol in those who have not drunk for an extended period beforehand. Those who attempt abstinence but relapse at some point during pregnancy may lose the tolerance that may protect the fetus in the case of those who continue to drink moderately.

2) More generally, detrimental effects of anxieties that such warnings induce may well outweigh the benefits—if any—that compliance produces. And, further, recommendations that urge changes in life-styles on the basis of tenuous evidence may result in less compliance with warnings that are more solidly supported by findings.

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In their report describing the effect of ethanol on pregnant monkeys, Mukherjee and Hodgen acknowledge several limitations in their investigation. Not made clear to the reader, however, is that the ethanol dose they applied is the human equivalent of a bolus amount of 530 milliliters of 80 proof whiskey injected into the femoral vein of a pregnant woman weighing 60 kilograms (including fetus) in her 32nd week of gestation. Because this and similar reports usually have received nationwide press coverage, it would seem judicious for authors of such reports to present human dose equivalents so that the relevance of such research to realistic human exposures can be easily evaluated.

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We note the concern expressed by J. M. Joffe and A. W. Kimball regarding the recommendation in our report that "women consider total abstinence from ethanol throughout pregnancy." Kimball's point is well taken that we used high dosages of ethanol in our experiments. Our primary purpose was to delineate the kinetics of transfer of maternally administered ethanol to the developing fetus. The collapse of the umbilical cord was an unexpected but very important observation. Clearly, it will be necessary to determine the lowest orally administered dose of ethanol that can provoke similar umbilical cord changes. Altura *et al.* (*1*) have now independently reported very similar effects of ethanol on isolated human umbilical cord blood vessels with lower concentrations of ethanol than those we used. In fact, the low concentrations of alcohol (52 mg/dl) reported, "which can induce threshold spasms of human umbilical arteries and veins, can be found in the blood of pregnant women within 30 minutes after ingestion of 1–1.5 drinks of 100 proof whisky" (*1*).

There is compelling evidence in the scientific literature to suggest that ethanol is teratogenic to the developing fetus (*2*), contrary to Joffe's interpretation. We note that he refers to an article in the *New England Journal of Medicine* (his reference 4) as stating, "available evidence indicates no adverse effects of moderate drinking in pregnant women," when in fact the authors clearly state, "thirty-two percent of infants born to heavy drinkers demonstrated congenital anomalies, as compared to nine percent in the abstinent and fourteen percent in the moderate group ($P < 0.001$)." Moreover, other reports have shown that drinking by a pregnant woman may affect a newborn's suckling ability (*3*) and the ability to learn (*4*). When children of moderate drinkers were evaluated at 8 months of age, they had small but significant delays in their mental and motor development (*5*). Another follow-up study found that drinking by pregnant women was related to altered attention span and fidgetiness among their children when examined at 4 years of age (*6*). Kline *et al.* (*7*) reported that alcohol consumption on two to six occasions per week of amounts as low as 30 ml (two tablespoons of absolute ethanol) per occasion increased the likelihood of spontaneous abortion. Harlap and Shiono (*8*) reported statistically significant increased risk for second trimester spontaneous abortions from one to two drinks (two tablespoons of absolute ethanol) per day. A primate model for fetal alcohol syndrome has been developed in which moderate levels of ethanol are implicated (*9*). There are numerous other reports as well (*10*).

Clearly, we should not ignore the voluminous data on the teratogenic effects of ethanol when educating our patients during their prenatal care. The National Academy of Science's Committee on Nutrition of the Mother and Preschool Child (*11*) called alcohol a "dangerous drug" that may be "one of the most frequently recognizable causes of mental deficiency and developmental delay." The Surgeon General of the U.S. Public Health Service has issued a similar statement regarding ethanol consumption during pregnancy (*12*). Accordingly, we remain confident in our original recommendation that, until additional evidence clarifies "safe" levels of ethanol consumption during pregnancy, expectant mothers should consider avoiding alcohol consumption.

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