The health of poor Americans is getting worse. There is reason to suspect that health funding cuts already enacted are at least partly responsible for the declining health status of some poor Americans. Continued deprivation of access to care not only poses problems for those who bear the hardship of illness and disability, but also imposes unnecessary burdens on those who ultimately pay for their care. Careful study will be needed to determine whether these trends continue. If they do, we may soon face a major national problem that will require urgent remedial action.

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AN ANALYSIS OF SERUM SAMPLES POSITIVE FOR HTLV-III ANTIBODIES

To the Editor: During the investigational stage of the development of enzyme-linked immunosorbent assay (ELISA) kits for detection of antibodies to human T-cell lymphotropic virus Type III (HTLV-III), 15,368 plasma samples from random blood donors in the United States were tested by five companies (Abbott Laboratories, Dupont, Electronucleonics, Litton, and Travenol–Genentech Diagnostics), with approximately 3000 samples per company. Plasma samples were from units of blood that had been drawn as part of standard collecting procedures, and the linkage between the sample, the collection facility, and the individual donor was broken. Samples were collected during the month of October 1984, which was 18 months after blood-collecting facilities had begun special screening procedures relating to the acquired immunodeficiency syndrome (AIDS), including specific requests for members of groups at increased risk for AIDS to exclude themselves voluntarily from donating blood. A total of 236 samples were reported by the five companies to be repeatedly reactive for antibodies to HTLV-III. Few of the samples were sufficient in volume for repeat testing; however, we were able to find nine repeatedly reactive specimens with sufficient reserve volume to conduct the following additional tests: Western blot (performed by Dr. M.G. Sarngadharan),[1] reverse transcriptase activity,[2,3] and infectivity assay in H9 cells.[4] The results of these tests are summarized in Table 1. Because Western blot tests are not fully standardized, all laboratories did not detect the same bands. Table 1 presents the composite band data from four laboratories.

The reverse transcriptase activity was three times the background level or less in all samples except Sample 9. The finding of infective virus in Sample 7 without reverse transcriptase activity in the original sample was probably due to a small amount of infectious virus in the original sample, which could be detected only after amplification in H9 cells. On the other hand, the test results for Sample 9 suggest that HTLV-III may sometimes be present at a sufficiently high concentration in plasma to show detectable reverse transcriptase activity, that is clearly above background levels without amplification.

Of particular interest is the finding that Western blot analyses of the two virus-positive samples showed the presence of only band p24 in one case and bands p51 and p65 in the other. This suggests that bands other than gp41 in Western blots of ELISA-positive samples from blood-donor populations may be of more importance than previously thought. Even though these data are limited, the finding that two of nine antibody-positive samples had detectable HTLV-III supports the value of screening blood for antibodies to HTLV-III by the ELISA method, because units of blood were detected that not only had antibody to HTLV-III but also contained infectious virus in the plasma. It is also clear that even though special screening of donors for AIDS had been in place for 18 months before these samples were collected, persons with HTLV-III antibody and virus were still donating blood. Whether those

Table 1. Western Blot Bands and Reverse Transcriptase Activity in Nine Serum Samples That Were Positive for Antibodies to HTLV-III.

Sample No.	Western Blot Bands	Reverse Transcriptase Activity*	HTLV-III Recovered†
1	41, 56, 66	1.19	–
2	17, 51, 56, 66, 80	2.68	–
3	17, 24, 66	1.89	–
4	17, 24, 41, 51, 66	2.35	–
5	17, 24, 55	1.50	–
6	17, 24, 41	1.44	–
7	24	1.61	+
8	24	3.11	–
9	51, 65	6.08	+

*Particle-associated reverse transcriptase activity in the original sample is given as the ratio of sample counts per minute to background counts per minute. Background values ranged from 85 to 123 counts per minute (average, 102).

†Original sample absorbed into H9 cells with subsequent cultivation and periodic sampling for HTLV-III antigens by immunofluorescence and reverse transcriptase activity in the cell culture medium. Cultures were maintained for a maximum of six weeks.

donors were members of the well-defined groups at increased risk for AIDS was not possible to determine, because the linkage between the samples and donors had been broken.

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NYQUIL AND ACUTE HEPATIC NECROSIS

To the Editor: Nyquil, a common over-the-counter cold remedy, has not been recognized as a cause of hepatic injury. Nyquil contains a combination of ephedrine, doxylamine, promethazine, dextromethorphan, and 700 mg of acetaminophen per ounce (30 ml) of a 25 per cent alcohol elixir. This high level of alcohol led an alcoholic but otherwise healthy 45-year-old man to consume 12 oz (360 ml) of the elixir over a 12-hour period, causing massive hepatic necrosis.

When he had last been seen for inebriation in the emergency room, his laboratory values had been normal except for a slightly elevated level of aspartate aminotransferase (58 IU per deciliter). After six months of abstinence, he resumed his usual habit of consuming one to two cases of beer per day. Three weeks later, because of guilt and nausea, he tapered his drinking, and he stopped completely three days before admission. Needing sleep and feeling anxious, he purchased a 12-ounce (360-ml) bottle of Vicks Nyquil, noting the alcohol content. He drank all but two to three tablespoons over 12 hours. After breakfast the following day, he experienced intense nausea, vomiting, diaphoresis, cramping, and orthostatic dizziness. When he presented the next evening, he was full oriented, insisting he had been poisoned.

Physical examination was remarkable only for the patient tremulousness and icteric sclerae. He was afebrile, with a thread pulse of 120. Admission laboratory tests indicated severe liver in jury: total bilirubin was 9.6 mg per deciliter (indirect, 5.4), pro thrombin time 27.1 seconds, aspartate aminotransferase 10,100 I per deciliter, and albumin 3.8 g per 100 ml. Glucose was 54 mg pe deciliter, urea nitrogen 16 mg per deciliter, and creatinine 2.7 m per deciliter. Electrolytes and amylase and alkaline phosphatas values were normal. The leukocyte count was 19,000 per cubic mill meter, and the platelet count was depressed at 56,000 per cub millimeter. The sedimentation rate was 9 mm per hour. The urin showed numerous hyaline casts.

The patient was given N-acetylcysteine (Mucomyst) as treatmer for possible acetaminophen overdose and vitamin K on each of th first three days. His oliguric acute tubular necrosis resolved. H blood was sterile and negative on toxicologic screening. His urin showed high concentrations of Nyquil ingredients. His prothrombi time and aspartate aminotransferase level fell to normal within eigl days, when he was discharged with a total bilirubin level of 17 m per deciliter. He was last seen two weeks later and was still icter but declined follow-up because he felt well.

Although the patient survived, this case illustrates the deadl potential of over-the-counter polypharmacy. Attracted to the alco hol in the elixir, he consumed roughly 7 g of acetaminophen — n an amount usually associated with such profound hepatic necrosi It has been suggested that ethanol is "hepatoprotective" in cases acetaminophen overdose.[1,2] This case suggests that the protecti effect of ethanol may be negated by a recent binge of heavy drinkin Thus, although inclusion in the elixir of the sedative antihistamin doxylamine and promethazine may contribute to Nyquil's appeal a short-term antidote to alcohol withdrawal, the presence of acet minophen may contribute to a more harmful effect.

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PNEUMOPERICARDIUM FROM COCAINE INHALATIO

To the Editor: An unusual practice has developed among son users of "free-base" cocaine. These people inhale crystals or vapo of the drug, then have a partner apply positive ventilatory pressur This is usually done by direct mouth-to-mouth contact or through cardboard cylinder and is intended to enhance the effect of the dru Recently, we observed pneumopericardium in a patient who pra ticed this form of cocaine inhalation.

A 20-year-old, previously healthy man reported the sudden ons of retrosternal chest and neck pain as well as shortness of breat occurring several hours before he was seen in the emergency roon He had had symptoms of a viral upper respiratory tract infection fi several days before his admission, but no pain. There was no histo of trauma. Physical examination revealed subcutaneous emphys ma in the neck and a mediastinal crunch (Hamman's sign) in th precordial area. Blood pressure and oropharyngeal examinatio were normal. X-ray films disclosed the presence of pneumoperica dium and pneumomediastinum and subcutaneous emphysema i the neck. In response to careful questioning, he admitted havi practiced this form of cocaine inhalation with his girlfriend immed ately before the onset of chest pain. After a short period of bed res the patient's symptoms resolved. Subsequent echocardiogra (M-mode and two-dimensional) revealed essentially normal fin ings. The patient was later discharged to his home in good conditio and was doing well five months after discharge.

Previous reports have noted an association of pneumomedia tinum and pneumothorax with forceful inhalation of cocaine an other drugs.[1] Pneumopericardium is a known complication of pos