

Pedestrian Injury

Given the high likelihood of traumatic brain injury (TBI) or spinal cord injury (SCI), pedestrian injuries are among the most serious public health problems in developed societies - and also among the most preventable. The TBI and SCI cases described below demonstrate typical aspects of pedestrian injuries: people of both sexes, all ages, and all ethnic groups are at risk; alcohol is often a factor; and the injuries are usually devastating.

Case 1. On a Fall evening, a 3-year-old child was struck by a car as he ran across the street after his pet. He sustained femoral, humeral, and basilar skull fractures and experienced a decreased level of consciousness to the point of responding only to painful stimuli. Initially admitted to a small, rural hospital, the boy was subsequently transferred to a larger city hospital and then to a major trauma center as his condition worsened. Discharged home after a week in the hospital, he required home health services and out-patient rehabilitation.

Case 2. A 17-year-old girl was reportedly playing "chicken" with a friend when she jumped in front of a truck and was hit. At the scene of the crash, she was in respiratory distress, had multiple fractures, and had a severe, closed head injury. When she became more alert and awake, she was transferred to an in-patient rehabilitation hospital. After 49 days of therapy, she was discharged home to home health services and out-patient rehabilitation. The cost, billed to Medicaid, was \$85,507.

Case 3. At approximately 2:00 am, a 24-year-old man was struck by a motor vehicle going 40 miles per hour. He was thrown 25 feet and was unconscious at the scene. His blood alcohol concentration (BAC) was 376 mg/dL (nearly 4 times the legal limit for intoxication). He had a severe, closed head injury with a subdural hematoma, as well as rib and leg fractures, and was hospitalized for 6 days. Upon discharge he required home health services and out-patient rehabilitation.

Case 4. While crossing the street in a cross-walk one Friday evening, a 35-year-old woman was struck by a drunk driver who subsequently was arrested for his third drunk-driving offense. The woman's injuries, at the level of the sixth cervical vertebra, resulted in incomplete quadriplegia. After a 31-day acute hospital stay, she was admitted to a long-term rehabilitation facility.

An accurate profile of the extent and impact of fatal and nonfatal injuries is essential for the design, implementation, and evaluation of an effective prevention program.^{1,2} This profile relies on comprehensive morbidity and mortality data.

To this end, the Texas Department of Health goal is to receive accurate injury data from 100% of the prehospital firms (ie, ambulance and emergency medical service) and hospitals in Texas. For the state of Texas, mortality data are comprehensive and readily available, but the collection of injury morbidity data is in its infancy. A state trauma registry rule requires all health care entities to submit trauma data to the state trauma registry by August 31, 1996. As of October 1994, 1% of hospitals and 12% of prehospital firms had transmitted trauma data to the state and are to be applauded for their efforts.

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Obtained from several sources, the data in this report are for 1980 through 1993. National data are from the US Department of Transportation/National Highway Traffic Safety Administration and the Insurance Institute for Highway Safety. Mortality data were obtained from TDH Bureau of Vital Statistics death records using Epigram software.³ Traffic records data were obtained from the Department of Public Safety using Lanser software.⁴ Morbidity data are from the 1994 prehospital and hospital databases maintained by the Texas Trauma Registry. Patient data were electronically transmitted by 94 prehospital firms and 5 hospitals.

National Data

Pedestrian death, the second largest category of motor-vehicle-related fatalities (following vehicle-occupant deaths), accounts for 14% of all traffic-associated deaths and approximately 3% of all traffic-associated injuries.⁵ There were 5,638 pedestrians killed in motor vehicle collisions in the US in 1993. This was the second lowest annual number of pedestrian deaths since 1927, the first year for pedestrian fatality reporting.⁶

Alcohol consumption by pedestrians is an important contributing factor to pedestrian injuries.⁷ In 1993, 40% of pedestrians 16 years and older who were fatally injured had BACs of 100 mg/dL or more. Although the percentage of fatally injured drivers of passenger vehicles, large trucks, and motorcycles who had BACs of 100 mg/dL or more has decreased from 50% in 1980 to 36% in 1993, the percentage of fatally injured adult pedestrians with a BAC of at least 100 mg/dL has remained essentially the same.⁸

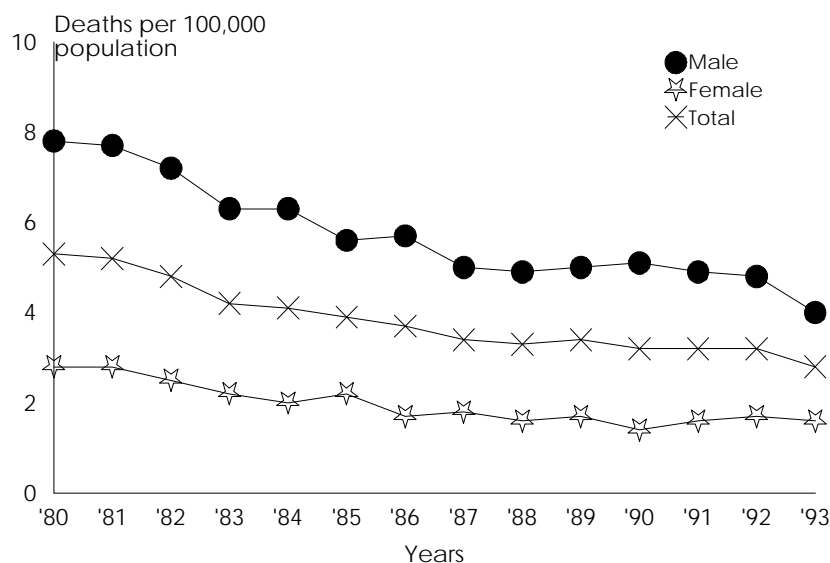
Mortality Data


Mortality data for the 14-year period through 1993 show that 8,648 pedestrians were killed in motor vehicle collisions in Texas. The pedestrian crude death rate has declined 47% from 5.3 per 100,000 population in 1980 (total: 750) to 2.8 per 100,000 population in 1993 (Figure 1). Pedestrian fatalities in 1992 numbered 562, compared with 502 in 1993.

Seventy-four percent of the total pedestrian fatalities during the 14-year period were among males. The male pedestrian crude death rate (5.7 per 100,000 population) was 3 times the rate for females (1.9 per 100,000 population). In 1993 the pedestrian crude death rates for both males and females in Texas (4.0 and 1.6 per 100,000 population, respectively) exceeded the national male and female pedestrian death rates (3.1 and 1.3 per 100,000 population, respectively).⁶

Younger pedestrians (ages 0 through 19) and older pedestrians (ages 60+) each accounted for 22% of all pedestrian fatalities in 1993. Thirty-six percent of the younger pedestrians who died were 4 years of age or younger. Those older than 75 years had the highest pedestrian death rate (6.8 per 100,000 population) (Table 1).

Figure 1. Pedestrian Crude Death Rates by Sex: Texas, 1980-1993



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During the 14-year period, Public Health Region (PHR) 10 had the highest pedestrian crude death rate (5.3 per 100,000 population); the lowest rate (2.7 per 100,000) was in PHR 2. In addition, Hispanics had the highest rate (5.3 per 100,000 population) followed by African Americans, whites, and other (5.2, 3.0, and 2.4 per 100,000 population, respectively).

Traffic Records Data

According to Department of Public Safety records, there were 393,104 motor vehicle collisions in Texas during 1993. Of these collisions, 6,029 (2%) involved pedestrians, of whom 4,048 (67%) were only injured and an additional 455 (8%) killed. The remaining 1,526 (25%) were reported as having possible injuries. Males accounted for 3,825 (68%) of the pedestrians involved in collisions.

Almost 2,628 (44%) of the pedestrians involved in collisions were aged 19 years of age or younger. Nearly one third (32%) of young pedestrians were between the ages of 5 and 9 years, followed by: 10 to 14 (28%), 15 to 19 (22%), and 0 to 4 (18%).

For the majority of pedestrians (78%), whether they had been drinking alcohol was recorded as unknown. However, of those pedestrians reported as drinking, 60% suffered from incapacitating or fatal injuries, compared with 31% of those reported not drinking. Males made up 81% of those reported drinking.

Table 2. 1994 Pedestrian Injury - Hospital Sample: Amount Billed to Each Payer

Payment Type	No. Cases	Total Amt.	Avg. Amt.
Other	24	422,698	17,612
Medicaid	11	99,283	9,026
Self-pay	10	53,839	5,384
Worker's Comp	6	320,713	53,452
Medicare	5	48,567	9,713
Other Group	4	50,406	12,602
HMO	2	9,480	4,740
Blue Cross/Shield	1	56,232	56,232

Total number of cases: 63
 Total amount billed all payors: \$1,061,218

Table 1. Pedestrian Deaths by Age Group: Texas, 1993

Age Group (Years)	Male		Female		Total	
	Rate*	Deaths	Rate*	Deaths	Rate*	Deaths
Birth-4	3.5	27	1.6	12	2.6	39
5-9	1.3	9	1.7	12	1.5	21
10-14	2.5	18	0.7	5	1.6	23
15-19	2.4	16	1.4	9	1.9	25
20-24	4.3	31	1.2	8	2.7	39
25-29	4.2	31	1.1	8	2.7	39
30-34	5.5	45	1.0	8	3.3	53
35-39	5.1	39	1.2	9	3.2	48
40-44	5.1	33	2.3	15	3.7	48
45-49	4.2	22	1.3	7	2.7	29
50-54	2.3	9	0.7	3	1.5	12
55-59	2.4	8	1.1	4	1.8	12
60-64	5.1	15	2.1	7	3.5	22
65-69	4.5	12	2.2	7	3.3	19
70-74	5.3	11	2.6	7	3.8	18
75-99	10.9	29	4.5	22	6.8	51
Total	4.0	357	1.6	145	2.8	502

* Crude death rates are per 100,000 population

The most commonly noted factors related to pedestrian behavior were "crossing at a non-intersection" (38%) and "crossing at an intersection" (17%). Sixty-eight percent of the pedestrians involved in collisions were reported as having committed a violation.

Prehospital Data

There were 43,700 prehospital patient records in the 1994 non-random sample file. Of these patients, 142 (<1%) were pedestrians injured as a result of a motor vehicle crash. Pedestrians were injured in 109 (77%) traffic and 33 (23%) non-traffic motor vehicle crashes. A non-traffic motor vehicle crash is one which occurs entirely in any place other than a public highway.

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Seven (5%) of the 142 patients had Revised Trauma Scores (RTS) at the scene that were less than 11. Patients with an RTS less than 11 at the emergency department are considered to have suffered major trauma.

The payor category for 35 (25%) patients was either "Self Pay" or "No Pay." These two categories typically comprise uncompensated trauma care. The average (mean) charges for prehospital care for pedestrians was \$245.47.

As many as 3 injuries can be recorded for each injured pedestrian. The most frequently reported injury type was "Fracture/Dislocation," of which there were 96 (37%). There were 28 (11%) injuries coded as "Spine/Brain" which can be one of the more severe and costly types of injury.

Hospital Data

There were 1,358 hospital patient records in the 1994 non-random sample file. Sixty-four (5%) of these patients were injured as pedestrians in motor vehicle collisions; of these patients, 7 died.

Alcohol. Although there is no statutory level of intoxication for pedestrians, the statutory level of intoxication for drivers is a BAC equal to or greater than 100 mg/dL.⁷ For this report, a pedestrian with a BAC of at least 100 mg/dL is considered "intoxicated."

Thirty-three (52%) of the 64 injured pedestrians were tested for BAC; of these, 20 (61%) were intoxicated. Eighteen (90%) of the intoxicated pedestrians were male. Of the 34 injured patients who were 21 through 49 years of age, 18 (53%) were intoxicated; 2 of the 11 (18%) aged 50 years and older were intoxicated.

Length of Stay. The mean length of stay (LOS) was 7.9 days for all pedestrian patients, excluding the 7 who died; the longest was 54 days. The mean LOS for surviving intoxicated patients was 12.9 days compared with 9.5 days for surviving patients with a BAC equal to 0 mg/dL. Of the 7 patients whose LOS was 16 days or greater, 6 were reported as having a BAC greater than 100 mg/dL.

Cost. The total amount billed by hospitals for the care of these pedestrian patients was \$1,061,218. (The amount billed for

one patient was not reported.) The mean amount billed per pedestrian patient was \$16,844.73. The amount billed ranged from \$1,193 to \$127,998.

The most common payer category was "Other" (38% of cases) followed by "Medicaid" (17%). The large number for "Other" indicates that payers falling within this category should be closely examined to determine if a major payer has been overlooked and should have a category of its

Prevention - Safety Tips for Individuals

1. Supervise young children.
2. Always walk on a sidewalk when one is provided.
3. When there is no sidewalk, walk on the **left** side of the road, facing traffic.
4. Cross streets safely:
 - ◆ Stop at the curb or edge.
 - ◆ Look left.
 - ◆ Look right.
 - ◆ Look left again.
 - ◆ At an intersection, look behind you and watch out for vehicles turning right.
 - ◆ Keep on looking as you cross the street.
5. Avoid walking at night. If you must walk after dark, carry a flashlight and wear light-colored, reflective clothing. You need to see and be seen.
6. Cross streets at intersections or pedestrian crosswalks. Don't enter a street in front of visual barriers such as bushes or parked cars.
7. Obey traffic control signals. A flashing "Don't Walk" signal means that you should not begin to cross the street. Wait for the next "Walk" signal.
8. If you have been drinking alcoholic beverages, avoid walking near traffic.
9. Be alert. Avoid wearing headphones while walking or running near traffic.
10. Be predictable - avoid sudden changes in directions; don't dart out into a street.

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own. It is possible that "No Pay" makes up a large portion of "Other" as it does not have its own category (Table 2).

By August 31, 1996, all hospitals and prehospital firms in Texas will be required to report data electronically on traumatic injury to the state. The purpose of this legislation is not to create "just another rule" but rather to provide a solid, comprehensive trauma reporting and analysis system on which preventive action can be based.

For information about pedestrian injury, call (512) 458-7266. For information about traffic safety, call Safe Riders (800) 252-8255



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Prevention - The Community

The National Committee for Injury Prevention and Control suggestions on how to prevent pedestrian-related injuries and deaths include the following:²

- ◆ One-way street networks and the conversion of two-way to one-way streets
- ◆ Adequate roadway lighting
- ◆ Requirement of sidewalks in new housing subdivisions (Although universal in urban areas, sidewalks are not normally constructed in rural areas and often are not provided in suburban neighborhoods.)
- ◆ Roadway barriers, including chains, fences, and other devices to physically separate pedestrians from vehicles
- ◆ Pedestrian crossing signs in unusually hazardous locations
- ◆ Use of special devices and materials by all night-time pedestrians to make them more visible

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In the News:

Cows . . .

Speculation that "mad cow disease" was responsible for the recent death of an East Texas man is unfounded. He had been hospitalized and treated for Creutzfeld-Jakob Disease (CJD) before he died at home. Despite international publicity, debate, and research - a solid scientific link has never been established

between bovine spongiform encephalopathy (BSE) and CJD.

Typically, CJD occurs in about 1 of every million humans (approximately 9 cases in Texas per year). A few more are possibly misdiagnosed as Alzheimer's disease or a similar dementing illness.

Monkeys . . .

...laboratory tests have shown no evidence of Ebola infection in any of the remaining monkeys.

On March 30, a cynomolgus monkey (*Macaca fascicularis*) died in a South Texas primate quarantine facility following a 3-day illness. A second monkey became ill on April 9. Serologic test results for both were positive for Ebola. The second monkey was euthanized. Following further testing that confirmed Ebola-Reston in both monkeys, 48 others were euthanized to minimize potential exposure of staff and prevent additional transmission among the monkeys.

Four additional episodes of Ebola-Reston infection among monkeys imported from the Phillippines have occurred in the US and Italy. One of these was in Texas in 1990. Following the earliest episodes, the Centers for Disease Control and Prevention (CDC) updated and modified the mandatory disease-control procedures for transportation and quarantine of non-human primates. The purpose of quarantine is to allow an infected animal to show signs of disease before it is sent elsewhere in the country. Quarantine protocol in the US requires strict infection control precautions that include protective clothing and equipment for staff and strict contact guidelines.

As of May 3 all blood test results have been negative for all facility employees.

These 50 monkeys were among 100 received from the Phillippines on March 21. All newly-arrived monkeys are placed in individual cages in 2 different quarantine rooms (50 monkeys per room) and observed for a 31-day quarantine period. The monkey that died and the 49 that were euthanized were all housed in the same quarantine room. No monkeys from this shipment ever left the facility.

The recent episode in South Texas underscores the purpose and effectiveness of current quarantine process. Both cases of infection were detected while the animals were in quarantine; the potential for transmission to employees was minimal; and transmission outside the facility was prevented entirely. The facility was in compliance with all guidelines and the staff quick, cooperative, and professional in handling the problem and assisting with the investigation.

Although infection with Ebola-Sudan or Ebola-Zaire subtypes often is fatal in humans, the Reston strain has never caused human illness. Ebola-Reston infections confirmed in humans (4 cases) have all been asymptomatic. Nevertheless, local, state, and federal health officials began an immediate investigation of this recent Ebola-Reston outbreak in Texas and continue to monitor the situation.

Surveillance has been enhanced for the remaining 50 monkeys housed in the other quarantine room, and the quarantine period has been extended a

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minimum of 21 days. As of mid-April, laboratory tests have shown no evidence of Ebola infection in any of the remaining monkeys.

All animals will continue to be tested extensively, and the possibility of any present or future Ebola infection will be ruled out before lifting of the quarantine is even considered.

Mice . . .

A 25-year-old Amarillo man died April 17 of hantavirus pulmonary syndrome (HPS). Previously healthy, the patient became ill on April 12 (5 days before his death) with severe headache for which he took acetaminophen. On April 15 he developed intense myalgias, fatigue, and prostration. Nausea, vomiting, diarrhea, cough, and dyspnea began April 16. On the morning of April 17, he experienced postural syncope, and was taken to a Potter County hospital.

On arrival the man had a blood pressure of 90/60 mm Hg; pulse rate of 132/min; respiratory rate of 28/min; and temperature of 101.2°F. He was mottled, cyanotic, had dry mucous membranes, and was wheezing. Laboratory studies revealed a leucocytosis (white blood count of 22,000/cu mm) with a marked left shift, hemoconcentration (hemoglobin level of 20.5 gm/dL and hematocrit of 61%), and thrombocytopenia (platelet count of 38 10³/cu mm). Room air blood gases revealed significant hypoxemia (PaO₂ of 46). A chest roentgenogram revealed bilateral fibro nodular infiltrates. Other laboratory findings are listed in Table 1.

The patient was admitted and treated with fluids, broad-spectrum antibiotics, and vasopressors. He developed refractory hypoxemia, hypotension, and acidosis, and died 3 hours after arrival at the hospital. Autopsy disclosed diffuse alveolar pulmonary edema, bilateral

Monitoring of potentially exposed staff also will continue. As of May 3 all blood test results have been negative for all facility employees.

Extensive additional information regarding ebola infection is available on the TDH Web Page. <http://www.tdh.state.tx.us/>

pleural effusions, congestive hepatosplenomegaly, gastritis, and thyroiditis. Premorbid blood cultures were sterile.

Diagnostic testing for HPS was performed at the University of New Mexico Hantavirus Diagnostic Unit. Serum reacted to all 4 antigens of Sin Nombre Virus (SNV), and Western blot showed IgG and IgM antibodies to SNV antigens. Polymerase chain reaction was used to amplify viral RNA from blood monocytes; nucleotide sequencing of the amplification product verified SNV as the etiologic agent.

Table 1. Other Test Result Values

Sodium (mEq/L)	135
Potassium (mEq/L)	4.7
Chloride (mEq/L)	105
Bicarbonate (mEq/L)	24
Urea nitrogen (mg/dL)	20
Creatinine (mg/dL)	2.1
Bilirubin (mg/dL)	1.5
Albumin (g/dL)	3.5
Aspartate aminotransferase (U/L)	449
Alanine aminotransferase (U/L)	248
Lactate dehydrogenase (U/L)	957

Discussion

This patient's prodromal symptoms and the rapid development of shock and adult respiratory distress syndrome are typical for HPS. Gastrointestinal symptoms are prominent in many cases, and

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hemoconcentration, modest elevations of creatinine, and thrombocytopenia are typical.

This patient had no known exposure to rodents and lived in town. He was a truck driver who delivered auto parts to retail stores and warehouses in a number of rural West Texas communities. As part of the case investigation, the Rapid Response Team of the TDH Zoonosis Control Division (ZCD) trapped rodents in Amarillo, in Potter County, and in Randall County. The TDH Laboratory in Austin is testing serum from these rodents.

Prepared by J Rush Pierce Jr, MD, Health Authority, Amarillo Bi-City-County Health Department

Summary of Previous HPD Cases

As of May 1, 1996, 134 cases of HPS from 24 states have been reported in the US. Ninety percent of these cases have been caused by SNV, which is 1 of 4 hantaviruses known to cause HPS.

The most recent Texas case is the 5th for Texas and the 2nd from the Panhandle. The 1st case in the Panhandle was confirmed in a 15-year-old Deaf Smith County adolescent who died in May 1995. Serologic testing of rodents collected during the case investigation indicates that *Peromyscus leucopus* (white-footed mouse), *P. maniculatus* (deer mouse), *Reithrodontomys megalotis* (harvest mouse), and *Simodon hispidis* (cotton rat) are hantavirus reservoirs in West Texas.

The 1st reported case of HPS in Texas was in a 57-year-old nurse from Zavala (Angelina County) who died in June 1993. The 2nd reported case was in Kingsville (Kleberg County) in 1994; a 29-year-old woman became ill in March and survived. Thorough TDH investigation was not able to establish a definite source of exposure for these 3 cases.

The 4th patient to contract HPS was a 23-year-old male who became infected last October but survived. This case was in Jefferson County. As part of an ongoing investigation into the source of the man's infection, the ZCD Rapid Response Team trapped 143 rodents. The TDH Laboratory has completed blood tests on these specimens, representing 11 species of rats and mice.

These test results show that 9 of the 143 rodent specimens (6.3%) had antibodies for hantavirus. Additional laboratory testing will be conducted at the University of New Mexico.

(Additional information on hantavirus infection, as well as comprehensive reports on the above cases, can be found in DPN Vol. 54, Nos. 9&15; Vol. 55, Nos. 4,12,&15; and Vol. 56. N.3.)

For further information regarding these investigations contact Beverly Ray, RN, CIC, or Julie Rawlings, MPH, of the TDH Infectious Disease Epidemiology and Surveillance (IDEAS) Division at (512) 458-7676 or Jane Mahlow, DVM, TDH Zoonosis Division, at (512) 458-7255.

The Hottest Zone: Mercury Poisoning

While mice, cows, and monkeys - the reservoirs for exciting exotic diseases - are currently in the limelight, a very serious health risk is hiding in a plastic jar. The Texas Department of Health (TDH) issued a news release on April 19, warning that Manning Beauty Cream, sold over the counter in Mexico as "Crema de Belleza Manning[®]," has been linked with 2 cases of mercury poisoning in border residents.

Case 1. In September 1995, a 15-year-old boy in Eagle Pass began complaining of extreme fatigue, weakness, insomnia, myalgias of his extremities, severe headache, sore throat, cough, constipation, and paresthesias of his feet and hands. Prior to this illness, he had no significant medical history. He received symptomatic treatment from a physician in Piedras Negras, Mexico, for the paresthesias and cough. A week later, the teenager complained that he could no longer taste his food; this symptom lasted 2 weeks. None of the other family members, including his mother, father, an 8-year-old brother, and a 19-year-old brother, complained of any similar symptoms.

In October 1995 the mother gave her son several injections of B-complex vitamins which she acquired in Piedras Negras, Mexico. She also gave him Cat's Claw root (an herbal preparation containing an alkaloid purported to have anti-inflammatory effects). The teenager reported that he felt better after taking the Cat's Claw root. However, by the end of the month, he had lost 10-15 pounds, and his weakness was progressing.

A physician in Piedras Negras performed an electromyogram and measured nerve conduction velocities, which were felt to be consistent with a polyneuropathy with demyelination. Blood tests for creatine phosphokinase (CPK) and sedimentation rate were normal. Guillain-Barre syndrome was suspected.

In early November the boy was seen in a San Antonio emergency room; a magnetic resonance imaging (MRI) of his brain was normal. At that point, he was referred to a local pediatric neurologist with a chief complaint of progressive weakness of the arms and legs that was particularly noticeable when he attempted to climb stairs. On physical exam, his cranial nerves were intact, his deep tendon reflexes were decreased diffusely, and he had some mild weakness of the lower extremities. On November 3, 1995, his blood lead level and urine arsenic level were normal, but his urine mercury level was 178 $\mu\text{g}/\text{L}$. Urinary mercury concentrations greater than 20 $\mu\text{g}/\text{L}$ have been associated with signs and symptoms of mercury intoxication.

December 9 the patient began a 1-month course of oral chelation therapy with penicillamine, 250 mg every 8 hours. Two weeks later, after completing this regimen, he started a second course of chelation therapy, this time with dimercaptosuccinic acid (Succimer[®]) for 1 month.

The TDH Office of Border Health initiated a public health investigation at the request of a nurse in Eagle Pass on December 11, 1995. A family exposure history was elicited. Family members reported that they ate fish from Mexico only once or twice a year and had no hobbies at home or school known to be related to mercury exposure. In addition, a portable mercury vapor tester did not detect mercury in indoor air. Laboratory tests for mercury were conducted on urine from the rest of the family, soil



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samples, indoor paint chips, and a sample from a container of facial cream the teenager used for treatment of acne.

Urine mercury results for the parents and the older brother were 1 - 5 $\mu\text{g}/\text{L}$ (well below the 20 $\mu\text{g}/\text{L}$ reference level). However, the younger brother, who shared a bedroom with the index patient, had a slightly elevated urine mercury level of 27 $\mu\text{g}/\text{L}$. Test results on soil and paint samples were negative. The skin cream contained extremely high levels of mercury: 50,000 $\mu\text{g}/\text{L}$ (approximately 5% by weight mercury).

The product label indicates that "Crema de Belleza - Manning®" is manufactured in Tampico, Tamaulipas, Mexico. A second unopened sample of the facial cream was obtained from Piedras Negras, Mexico, to eliminate the possibility of tampering. Elevated mercury levels (approximately 6% by weight) were confirmed in the second bottle of skin cream. The "Crema de Belleza" label instructions indicate use for greasy skin, blackheads, and acne; the listed ingredients are water, glycerine, benzoin, and calomel.

Case #2. In April 1996 a neurologist at Texas Tech Health Science Center in El Paso discovered a 35-year-old woman with urinary mercury levels of 355 $\mu\text{g}/\text{g}$ creatinine. Urinary mercury concentrations greater than 25 $\mu\text{g}/\text{g}$ creatinine have been associated with signs and symptoms of mercury intoxication. The patient's primary complaints at the time of diagnosis were irritability and insomnia, tingling in the medial aspects of the left forearm, numbness in the right leg, and numbness or tingling in the external ear. The symptoms had developed over a period of several months.

The case was referred to TDH by the New Mexico Department of Health. A TDH investigation revealed that the woman had been using "Crema de Belleza" for an extended period of time. The physician is reportedly initiating chelation therapy with Succimer®.

Discussion

Mercury exists in three forms: elemental (metallic), inorganic (mercuric and mercurous salts), and organic (methyl-, ethyl-, and phenyl mercury). The sources of exposure, pathways of exposure, pharmacokinetics, and biological effects of mercury vary among the different forms. The main active ingredient listed on the "Crema de Belleza" container is calomel, which is mercurous chloride.

In the past, calomel was widely used in a variety of medicinal products such as laxatives, worming medications, and teething powders. However, as more instances of toxicity were reported over the years, most of these uses have been replaced by safer and more effective agents.

Approximately 1 - 2% of a solution of mercuric mercury can be absorbed through intact skin. Although mercurous mercury (calomel) is considerably less soluble than mercuric mercury (and, hence, less readily absorbed), long-term, repeated skin applications can result in significant accumulation in the body and produce toxic effects. Also, the presence of pimples and blackheads is likely to enhance dermal absorption.

In the body, inorganic mercury has a half-life of 30 - 60 days and is excreted primarily through the urine and feces. Shortly after exposure to mercury salts, an exposed person may experience a metallic taste, nausea, vomiting, bloody diarrhea, severe abdominal pain, and tenesmus. Signs of kidney damage (eg, excessive protein, casts, and red blood cells in the urine) may be noted 1 day - 2 weeks after ingestion. Urinary output may decrease significantly due to acute tubular necrosis, and death from uremia may result. The acute lethal dose for most mercury salts is approximately 1 - 4g for adults.

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Inorganic salts of mercury, such as calomel, generally do not cross the blood-brain barrier or the placenta and, hence, are less toxic to the central nervous system (CNS) and to the developing fetus than are metallic or organic mercury compounds. However, mercurous forms of mercury, such as calomel, are decomposed in the body into the metallic and mercuric forms. Consequently, calomel possesses the toxic characteristics of both of these forms of mercury. Chronic exposure to calomel can result in various additional signs and symptoms of CNS toxicity: personality changes, nervousness, irritability, tremors, weakness, fatigue, loss of memory, and changes in or loss of hearing, vision, or taste. Gingivitis, stomatitis, and excessive salivation are also classically seen in cases of toxicity from mercury salts.

Children exposed to elemental mercury, mercury salts, or phenyl mercury may develop acrodynia - a rare syndrome characterized by severe leg cramps, irritability, paresthesia, excessive perspiration, pruritus, and painful redness and peeling of the palms of the hands and soles of the feet. It is not known why adults are not affected by acrodynia or why only some children exposed to mercury develop this syndrome.

"Creme de Belleza" is readily available over the counter in Mexico, and border residents are warned of the dangers of this product. Many concerned Texans who have used the cream are contacting TDH and are being advised to immediately stop using it and have a physician evaluate the possibility of toxic exposure right away. A medical alert has been sent to over 600 border area physicians.

On April 30 the State of Tamaulipas Secretary of Health informed TDH Region 8 staff that his department had confiscated 35,000 bottles of "Crema de Belleza" for laboratory testing. On May 1 the Consul

General of Mexico informed TDH officials in Austin that Mexican authorities are conducting an investigation of this product.

Health professionals are asked to report any cases of mercury poisoning to Richard Beauchamp, MD, or John Villanacci, PhD, at (521) 458-7268, FAX (512) 458-7689. (Authority for reporting is found in Texas Health and Safety Code, Chapter 161.0211.)

Health professionals may obtain additional information regarding medical management by contacting the TDH staff listed above or the Texas Poison Center Network at (800) POISON1.

General information for the public is available from the TDH Communications and Special Health Initiatives. Contact Emily Palmer at (512) 458-7400.

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