JAMA®
The Journal of the American Medical Association
October 24/31, 1986

Letters

Death and Injury by Firearms: Who Cares?

To the Editor.—Who cares about death and injury by firearms? Ask the families of firearms victims. And ask about the repeated, defeated efforts of these people to convince legislators to enact a satisfactory gun-control law. Our present session of Congress has avoided such legislation, presumably due to intense lobbying by the National Rifle Association.

The creation of a special center to study injuries from firearms is reminiscent of the "Crusade Against Cancer" that was legislated into being in 1970. The allocation of great sums of money has not brought us much closer to successful therapy for the neoplastic diseases.

Jagger and Dietz[1] have suggested a method to gather information that, in the end, would generate some interesting articles but would not really help to solve our problem. We know that the manufacture, distribution, and sale of handguns must be curtailed. Why not get on with the job and write a satisfactory law without creating another expensive agency?

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1. Jagger J, Dietz PE: Death and injury by firearms: Who cares? *JAMA* 1986;255:3143-3144.

In Reply.—Dr Miller's letter strikes a familiar note. The frustration he conveys is shared by many others who attribute the firearm wound epidemic to the repeated failures of legislators to restrict gun availability. We ask Dr Miller and those who share this prevalent outlook to consider the following points:

1. The significance of a health problem cannot be fully assessed without adequate documentation. Currently, our knowledge of firearm injuries as a national public health problem is based solely on fatalities representing fewer than 20% of all cases. Without data on nonfatal cases, we will remain in the dark about the distributions of disabling and disfiguring sequelae, costs of acute care and long-term rehabilitation, and psychological and societal consequences of firearm trauma. Only information about nonfatal shootings can tell us how they differ from fatal shootings, which is crucial to the development of prevention efforts.

2. Legislative curtailment of lawful manufacture, distribution, and sale of firearms is not the only way to reduce firearm injuries. Moreover, there is dispute among scholars as to the likely effects of such curtailment. It is argued, for example, that because American homes are already saturated with firearms, because the half-life of firearms is so long, and because recycling of spent bullet casings is so common, even an absolute ban on the sale of new guns and ammunition would not produce significant reductions in shooting frequencies for many years. Even if curtailment were to occur and reduce injuries, the public could still benefit from a creative search for other potential strategies. Many approaches are required, and they may be as varied as the circumstances under which the injuries occur.

Only if we knew more about the epidemiology of firearm injuries could we even guess at the likely benefits of untried countermeasures. How many productive years of life could be saved by distributing free trigger locks, improving lighting in alleys, confiscating guns from homes lodging domestic violence complaints, or magnetometer screening of bank patrons? None of these or many other potential countermeasures can even be theoretically assessed without better data than are now available.

3. We do not propose the creation of a special center to study firearm injuries. Rather, we support the recommendation of the National Academy of Science's Committee on Trauma Research that a center for injury control (dealing with all types of trauma) be established as part of the Centers of Disease Control. The study of firearm injuries should be integral to the center's program and should be allocated resources in proportion to the magnitude of this problem. Health professionals have before them the opportunity to make an immense contribution to this neglected health problem. We must not allow the apparent difficulty of the task to dissuade us from taking the first step toward a solution.

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Borrelia in the Brains of Patients Dying With Dementia

To the Editor.—I have identified spirochetes in serial subculture of autopsy brain tissues from two patients with dementia. Indirect immunofluorescence, using monoclonal antibodies specific for *Borrelia* species, resulted in fluorescence of spirochetes that had previously been identified by darkfield microscope examination (Figs 1 through 4).

Case 1 was a 74-year-old woman with mild dementia of less than one year's duration. She had resided in New York and Florida. Case 2 was a 69-year-old man who died in a nursing home in Texas after a four- to five-year history of progressive dementia. Parkinsonian symptoms were noted during his last year of life. Neither patient had symptoms of the skin, joint, or cardiac disorders described in *Borrelia burgdorferi* infection.

Recent reports have described patients with various chronic degenerative neurological disorders whose blood or cerebrospinal fluid contains antibodies against *B burgdorferi*.[1] Individuals with cognitive impairment or memory disorders are found within the larger group of patients with chronic borreliosis.[2,3] *Borrelia* species appear to share with *Treponema pallidum* the potential to survive in host tissues for prolonged periods of time and to cause disease in various organ systems after months or years of clinical latency. Pachner and Steere[4] have classified the neurological manifestations of *B burgdorferi* infection into primary, secondary, and tertiary forms. If *Borrelia*

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Edited by Drummond Rennie, MD, Senior Contributing Editor; Sheron Iverson, Assistant Editor.

Fig 1.—Case 2. Spirochetes in subculture (BSK medium). Darkfield microscope image (×400).

Fig 2.—Mouse antibody H9724 (axial filament); goat antimouse IgG fluorescein. Indirect immunofluorescence image (×1000).

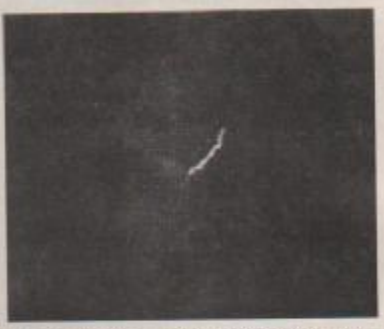

Fig 3.—Mouse antibody H5332 (outer envelope); goat antimouse IgG fluorescein. Indirect immunofluorescence image (×1000).

Fig 4.—Case 1. Spirochetes in subculture (BSK medium). Darkfield microscope image (×400).

infection can be linked to cases of dementia by serology or by culture, a group of patients will be candidates for intensive parenteral antimicrobial therapy analogous to the treatment now used for neurosyphilis.

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1. Kohler J, Kaeper J, Kern U, et al: *Borrelia* encephalomyelitis. *Lancet* 1986;2:35.
2. Reik L Jr, Smith L, Khan A, et al: Demyelinating encephalopathy in lyme disease. *Neurology* 1985;35:267-269.
3. Hansen K, Rechnitzer C, Pedersen NS, et al: *Borrelia* meningitis and demyelinating CNS disease. *Int J Microbiol Hyg* 1986, in press.
4. Pachner AR, Steere AC: Tertiary lyme disease: Central nervous system manifestations of long standing infection with *Borrelia burgdorferi*. *Int J Microbiol Hyg* 1986, in press.

Leukocytoclastic Vasculitis Associated With Positive HTLV-III Serological Findings

To the Editor.—The Henoch-Schönlein type of vasculitis is one acute variety of leukocytoclastic vasculitis that manifests as "palpable purpura" and is frequently associated with systemic manifestations.[1] To our knowledge it has been seen in one previous homosexual male patient who had no clinical or serological manifestations of acute acquired immunodeficiency syndrome or its variants.[2] I report here the case of a homosexual man with Henoch-Schönlein purpura and positive human T-cell lymphotropic virus type III serological findings with massive gastrointestinal tract hemorrhage and disseminated intravascular coagulopathy (DIC). The adverse outcome was atypical of the general course seen in this disease, as most patients have resolution and recurrence with ultimate spontaneous resolution.

Report of a Case.—A 43-year-old homosexual male patient was admitted to the hospital for lower-extremity skin lesions, joint pains, swollen feet, severe abdominal pain, hematuria, and bloody diarrhea. He had had hepatitis A virus, hepatitis B virus, varicella-zoster virus, cytomegalovirus, and gonorrhea. He reported 500 random homosexual contacts in the preceding ten years. On examination, his temperature was 37°C; heart rate, 100 beats per minute; and blood pressure, 122/80 mm Hg. The abdomen was distended with decreased bowel sounds; palpable, nonblanching purpura was present on the lower extremities. The right wrist and elbow were tender. The stool sample was positive for fresh blood.

At admission, the hemoglobin level was 13.1 g/dL (131 g/L); hematocrit, 40.1% (0.40); leukocyte count, 9700/mm^3 (9.7×10^9/L) with 77.1% (0.77) neutrophils and 15.9% (0.16) lymphocytes; and platelet count, 400 000/mm^3 (400×10^9/L). Urinalysis showed eight leukocytes, 100 erythrocytes, 3+ hemoglobin, and 4+ protein. Serum values were as follows: total protein, 4.3 g/dL (43 g/L); albumin, 1.5 g/dL (15 g/L); γ-globulin, 1.4 g/dL; C3 complement, 50 mg/dL (0.5 g/L) (normal range, 83 to 177 mg/dL [0.83 to 1.77 g/L]); and total complement, 40 mg/dL (0.4 g/L) (normal range, 70 to 199 mg/dL [0.7 to 1.9 g/dL]). Cryoglobulins were not present. Results of tests for antinuclear antigen, rheumatoid factor, and rapid plasma reagin were negative. The prothrombin and partial thromboplastin times were normal. The acute-phase antistreptolysin titer was 330 (normal range, 85 to 100). The T-helper (T_H) lymphocyte/T-suppressor (T_S) lymphocyte ratio was greatly reduced: 18 T_H/66 T_S cells (mean, 44 T_H/30 T_S cells; range, 33 to 63 T_H/ten to 46 T_S cells). Serological test findings were positive for HTLV-III, antibodies to hepatitis B core and hepatitis B surface antigen, antibody to hepatitis A virus, and cytomegalovirus and negative for hepatitis B surface antigen. A mesenteric angiogram showed "evidence of probable intramural bleeding or edema based on thumbprinting seen." Sigmoidoscopic examination revealed edematous hyperemic areas at 13 cm without active bleeding. A punch biopsy specimen of the palpable purpuric lesions on the lower limbs was consistent with the diagnosis of "leukocytoclastic vasculitis."

High-dose prednisone treatment was started. The patient initially improved with this regimen. On the ninth day after admission, bilateral pleural effusions developed (transudates). The bloody diarrhea worsened; renal failure ensued, and hemodialysis was started. On the 20th day, signs of DIC developed. The patient did not respond to multiple transfusions with packed cells, fresh-frozen plasma, or cryoprecipitate and died the next day. Permission for autopsy was not granted.

Comment.—The cause of leukocytoclastic vasculitis is unknown. It occurs in association with streptococcal tonsillitis, hepatitis B, drug allergy, systemic lupus erythematosus, mixed cryoglobulinemia, and some viral infection.[3,4] The mechanism of vascular damage may include replication of the virus within vascular endothelium or immune complex deposition within vessel walls. In neonatal herpes infection, DIC is a frequent fatal complication. Mural vascular damage may result in exposure of free collagen leading to DIC and hemorrhage.[5]

An intriguing possibility is that HTLV-III virus may also be similarly