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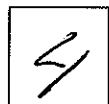
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HTLV-III-related RNA sequences detected in the spleen of the woman with transfusion-associated AIDS, to determine whether molecular changes occurred after transmission of the virus that might be related to the development of the syndrome. An understanding of host and virologic factors that determine different clinical outcomes after HTLV-III infection should be of value in the development of prophylactic (vaccine) and therapeutic strategies in AIDS.

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REFERENCES

- Gottlieb MS, Groopman JE, Weinstein WM, et al. The acquired immunodeficiency syndrome. *Ann Intern Med* 1983; 99:208-20.
- Fauci AS, Macher AM, Longo DL, et al. Acquired immunodeficiency syndrome: epidemiologic, clinical, immunologic, and therapeutic considerations. *Ann Intern Med* 1984; 100:92-106.
- Curran JW, Evatt BL, Lawrence DN. Acquired immune deficiency syndrome: the past as prologue. *Ann Intern Med* 1983; 98:401-3.
- Carney WP, Rubin RH, Hoffman RA, Hansen WP, Healey K, Hirsch MS. Analysis of T lymphocyte subsets in cytomegalovirus mononucleosis. *J Immunol* 1981; 126:2114-6.
- Tosato G, Magrath I, Koski I, Dooley N, Blaese M. Activation of suppressor T cells during Epstein-Barr-virus-induced infectious mononucleosis. *N Engl J Med* 1979; 301:1133-7.
- Gottlieb MS, Schroff R, Shanker H, et al. *Pneumocystis carinii* pneumonia and mucosal candidiasis in previously healthy homosexual men: evidence of a new acquired cellular immunodeficiency. *N Engl J Med* 1981; 305:1425-31.
- Siegal FP, Lopez C, Hammer GS, et al. Severe acquired immunodeficiency in male homosexuals, manifested by chronic perianal ulcerative herpes simplex lesions. *N Engl J Med* 1981; 305:1439-44.
- Friedman-Kien AE, Laubenstein LJ, Rubenstein PJ, et al. Disseminated Kaposi's sarcoma in homosexual men. *Ann Intern Med* 1982; 96:693-700.
- Drew WL, Conant MA, Miner RC, et al. Cytomegalovirus and Kaposi's sarcoma in young homosexual men. *Lancet* 1982; 2:125-7.
- Rogers MF, Morens DM, Stewart JA, et al. National case-control study of Kaposi's sarcoma and *Pneumocystis carinii* pneumonia in homosexual men. Part 2. Laboratory results. *Ann Intern Med* 1983; 99:151-8.
- Lederman MM, Ratnoff OD, Scillian JJ, Jones PK, Schacter B. Impaired cell-mediated immunity in patients with classic hemophilia. *N Engl J Med* 1983; 308:79-83.
- Popovic M, Sarngadharan MG, Read E, Gallo RC. Detection, isolation, and continuous production of cytopathic retroviruses (HTLV-III) from patients with AIDS and pre-AIDS. *Science* 1984; 224:497-500.
- Gallo RC, Salahuddin SZ, Popovic M, et al. Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. *Science* 1984; 224:500-2.
- Schüpbach J, Popovic M, Gliden RV, et al. Serological analysis of a subgroup of human T-lymphotropic retroviruses (HTLV-III) associated with AIDS. *Science* 1984; 224:503-5.
- Sarngadharan MG, Popovic M, Bruch L, Schüpbach J, Gallo RC. Antibodies reactive with human T-lymphotropic retroviruses (HTLV-III) in the serum of patients with AIDS. *Science* 1984; 224:506-8.
- Safai B, Sarngadharan M, Groopman JE, et al. Seroprevalence studies of human T-lymphotropic retrovirus type III in acquired immunodeficiency syndrome. *Lancet* 1984; 1:1438-40.
- Fahey JL, Prince H, Weaver M, et al. Quantitative changes in T helper or T suppressor/cytotoxic lymphocyte subsets that distinguish acquired immune deficiency syndrome from other immune subset disorders. *Am J Med* 1984; 76:95-100.
- Curran JW, Lawrence DN, Jaffe H, et al. Acquired immunodeficiency syndrome (AIDS) associated with transfusions. *N Engl J Med* 1984; 310:69-75.
- Cheseman SH, Sullivan JL, Brettler DB, Levine PH. Analysis of cytomegalovirus and Epstein-Barr virus antibody responses in treated hemophiliacs: implications for the study of acquired immunodeficiency syndrome. *JAMA* (in press).
- Preble OT, Black RT, Friedman RM, Klippel JH, Vilcek J. Systemic lupus erythematosus: presence in human serum of an unusual acid-labile leukocyte interferon. *Science* 1983; 216:429-31.
- Henle W, Henle G, Horowitz CA. Infectious mononucleosis and Epstein-Barr virus-associated malignancies. In: Lennette EH, Schmidt NJ, eds. Diagnostic procedures for viral, rickettsial and chlamydial infections. New York: American Public Health Association, 1979:470.
- Southern EM. Detection of specific sequences among DNA fragments separated by gel electrophoresis. *J Mol Biol* 1975; 98:503-17.
- Youssourian H, Hammer SM, Hirsch MS, Mulder C. Methylation of the viral genome in an *in vitro* model of herpes simplex virus latency. *Proc Natl Acad Sci USA* 1982; 79:2207-10.
- Seed B. Diazotizable arylamine cellulose papers for coupling and hybridization of nucleic acids. *Nucleic Acids Res* 1980; 1799-810.
- Beltz GA, Jacobs KA, Eichbush TH, Cherbas PT, Kafatos FC. Isolation of multigene families and determination of homologies by filter hybridization methods. *Methods Enzymol* 1983; 100:266-84.
- Groopman JE, Salahuddin SZ, Sarngadharan MG, et al. HTLV-III in saliva of people with AIDS-related complex and healthy homosexual men at risk for AIDS. *Science* 1984; 226:447-9.
- Essex M, McLane MF, Lee TH, et al. Antibodies to cell membrane antigens associated with human T-cell leukemia virus in patients with AIDS. *Science* 1983; 220:859-62.
- Jaffe HW, Francis DP, McLane MF, et al. Transfusion-associated AIDS: serologic evidence of human T-cell leukemia virus infection of donors. *Science* 1984; 223:1309-12.
- Barré-Sinoussi F, Chermann JC, Rey F, et al. Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS). *Science* 1983; 220:868-71.
- Vilmer E, Barré-Sinoussi F, Rouzioux C, et al. Isolation of a new lymphotropic retrovirus from two siblings with haemophilia B, one with AIDS. *Lancet* 1984; 1:753-7.
- Feorino PM, Kalyanaraman VS, Haverkos HW, et al. Lymphadenopathy associated virus infection of a blood donor-recipient pair with acquired immunodeficiency syndrome. *Science* 1984; 225:69-72.
- Shaw GM, Hahn BH, Arya SK, et al. Molecular characterization of human T-cell leukemia (lymphotropic) virus type III in the acquired immunodeficiency syndrome. *Science* (in press).

CONTROL AND ERADICATION OF METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS ON A SURGICAL UNIT

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AFTER the appearance of sporadic cases in 1961,¹ the incidence of methicillin-resistant *Staphylococcus aureus* infections in the United Kingdom,² Scandinavia,³ and Europe⁴ increased gradually throughout the 1960s, reaching a peak at the end of the decade before waning in the Old World. Because conventional procedures for source isolation and disinfection failed to contain their spread in most hospitals, strains of methicillin-resistant *S. aureus* are now endemic throughout the United States^{5,6} and eastern Australia.⁷

In the autumn of 1982 a methicillin-resistant *S. aureus*, phage type 29, introduced to our district general hospital from the community in the spring, caused a

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major explosive outbreak in a vascular surgical unit. The control of this epidemic through the eradication of the organism from carriage sites by regular whole-body antiseptic washing is reported here.

EPIDEMIOLOGY, CONTROL, AND BACTERIOLOGY

Our 800-bed hospital consists of 32 wards, each with 25 beds; each ward comprises two six-bed, two four-bed, and five single rooms. From the time the hospital was commissioned in 1978 until early 1982, an average of one infection with methicillin-resistant *S. aureus* was noted monthly, and the affected patients were conventionally isolated. Between April and June 1982, 10 cases emerged in four wards and, apart from two patients carrying phage type 29 of the staphylococcus, the affected patients also underwent containment isolation. Although these measures may have helped to prevent rapid dissemination during this period, no patient lost his or her carrier state.

One of the two patients carrying methicillin-resistant *S. aureus*, phage type 29, was attended by surgeons from the vascular surgical unit. Early in August 1982 an infection with phage type 29 was detected for the first time in a patient in the vascular unit. By the first week in September five more patients were affected, and the unit was closed to new admissions. There were no septicemias, other major infective complications, or deaths in the unit during the period of the outbreak.

The patients, staff, and environment were screened for methicillin-resistant *S. aureus*, and 10 more patients and three staff members were identified as carriers; the environment was heavily contaminated with the organism. Conventional measures to control infection were introduced, and an antiseptic liquid skin cleanser (supplied by Hough Hoseason, Manchester) was prescribed for all patients and ward staff, regardless of their carrier status. This special antiseptic liquid was composed of 2 per cent triclosan (Irgasan DP 300, Ciba Geigy) in anionic/ampholytic surfactants with foam stabilizer, solubilizers, preservatives, dye, and perfume at pH 7.5. Each person was instructed to use this preparation exclusively for whole-body bathing (or showering) and handwashing initially for two weeks; applications three times a day were specified, with particular attention to the anterior nares, axillae, and perineum. After two weeks of treatment, the antiseptic wash was continued only for those whose cultures remained positive.

Screening of the vascular unit was performed as follows: Specimens were collected weekly from the anterior nares, axillae, and perineum of patients and staff and from 94 sampling points on horizontal surfaces. When routine investigations of a wound site revealed the presence of methicillin-resistant *S. aureus*, wound swabs were included in the subsequent weekly screenings. Specimens were inoculated on mannitol salt agar (Lab M, London) containing 10 mg of methicillin per liter. Staphylococcal isolates were tested for the production of DNase and β -lactamase and for susceptibility to methicillin⁸ at 30°C on direct sensitivity agar (Lab M, London) controlled with *S. aureus* (NCTC 6571). All methicillin-resistant *S. aureus* isolates were phage typed according to the methods of Parker.⁹ All but one isolate in the unit were phage type 29. This epidemic strain was resistant not only to methicillin, but also to cephalosporins, fusidic acid, penicillin, streptomycin, sulfonamides, and trimethoprim. It was susceptible to chloramphenicol, erythromycin, gentamicin, nitrofurantoin, rifampin, tetracyclines, and vancomycin. One patient was carrying a β -lactamase-negative strain that was otherwise indistinguishable from the principal strain. One member of the staff (a physiotherapist) was carrying a methicillin-resistant *S. aureus*, phage type 52/52A/80/83A/85, that differed from the main epidemic strain in being resistant to chloramphenicol but susceptible to trimethoprim.

RESULTS

Forty members of the staff and 25 patients were screened for methicillin-resistant *S. aureus*; 3 staff members (2 nurses and a physiotherapist) and 14 pa-

tients were identified as carriers. The two nurses were nasal carriers; the physiotherapist carried the organism in the nares, axillae, and perineum. Among the patients, the perineum was culture-positive in 11, the nares in 8, and the axillae in 2. Three patients were nasal carriers only, and five perineal carriers only. Four others carried methicillin-resistant *S. aureus* in the nares and perineum, one in the axillae and perineum, and one in all three sites. The affected patients were distributed throughout the ward, with no evidence of clustering.

Two patients were discharged from the hospital before antiseptic treatment began. Another was discharged after 27 days to continue treatment at home. When he was examined two months later his cultures were negative. The remaining 11 patients and 3 staff members were cleared of methicillin-resistant *S. aureus* within an average of 22 days (range, 6 to 44). Six patients (average age, 59) and the two nurses were cleared within two weeks. In five patients (average age, 75) carriage was eradicated within 44 days. The physiotherapist's cultures became negative by the 28th day of treatment. All the carriers remained culture-negative during subsequent screenings over a three-month period. No patient lost a limb, required removal of a vascular graft, or died as a result of the outbreak.

The epidemic strain was recovered from superficial postoperative wound infections in five patients. The eradication of methicillin-resistant *S. aureus* from their wounds coincided with its disappearance from their skin. Six other patients had colonization in their groin wounds but with no signs of inflammation. These patients also stopped carrying the organism in their wounds and on their skin simultaneously. The wounds of these 11 patients healed satisfactorily.

Methicillin-resistant *S. aureus* was found in the dust in every patient room and all but 2 (the office and the treatment room) of the 20 support rooms. The epidemic strain was widespread and abundant, particularly around the beds, in the toilets, showers, and bathroom, and even on the bathroom scales and the instrument trolley. Despite such heavy contamination of the unit, the other three wards on the same floor were free from methicillin-resistant *S. aureus*. After a week's vigorous and methodical cleansing, only eight sampling sites still had the organism, though in markedly reduced numbers. At the end of two weeks, scanty amounts were detected in only two sampling sites. The environment in the unit remained free from methicillin-resistant *S. aureus* during all subsequent screenings over three months.

After five days of treatment with erythromycin, the groin wound of the patient who had been superficially infected with the only β -lactamase-negative strain of methicillin-resistant *S. aureus* was found to be colonized by a β -lactamase-producing strain of the same phage type but distinct from the epidemic strain in being resistant to erythromycin. This erythromycin-

resistant strain was found only in the single room where the affected patient was treated.

Concurrently with this major outbreak, four cases of methicillin-resistant *S. aureus*, phage type 29, emerged in four other wards. One of the patients died before and two during the first week of triclosan treatment, from causes unrelated to infection. The skin of the fourth patient became permanently free of the organism after two weeks of treatment. At the same time, a minor outbreak with a different phage type (29/79) occurred in a general surgical ward, involving two surgeons and three of their patients. One of the surgeons, who had Hailey-Hailey disease (benign familial chronic pemphigus) with secondary infection with methicillin-resistant *S. aureus*, was the source of this outbreak. Whereas the three patients were discharged before triclosan treatment, both surgeons were cleared of the organism within a week.

DISCUSSION

Several factors may have contributed to this explosive outbreak of methicillin-resistant *S. aureus*. Phage type 29 was probably introduced into the vascular unit by surgeons who had attended a patient with this phage type in another ward eight weeks earlier. Since none of the medical staff involved were carriers, the introduction of this strain into the vascular unit was possibly effected through clothing or transient hand contamination. The isolation procedures, applied only intermittently for the first three cases, failed to contain staphylococcal dissemination. Inadequate cleansing of the ward environment may have facilitated its rapid spread to an additional 13 patients within the unit.

Although containment isolation of affected patients is accepted as the single most important measure to control infection, in practice the procedures that are usually feasible in an acute-care hospital do not contain the spread of methicillin-resistant *S. aureus* indefinitely. Since it is the pressure of antibiotics on endogenous staphylococcal populations that selects for resistance to methicillin, further systemic administration of antibiotics is normally reserved for life-threatening infections.^{6,10} Systemic or local antibiotic therapy can temporarily reduce the incidence of nasal carriage of methicillin-susceptible *S. aureus*, but permanent eradication is extremely difficult; in all the studies published the staphylococci have recurred in 63 to 100 per cent of the subjects after therapy was discontinued.¹¹

Whole-body treatment with antiseptics should be more effective than treatment of selected carriage sites only. Agents with a high skin substantivity (affinity) and prolonged remanent¹² antibacterial effect are preferable. Hexachlorophene, chlorhexidine, and triclosan have such antibacterial activity, but not the iodophors or alcohols.¹³⁻¹⁸ Triclosan preparations have been evaluated in surgical¹⁹⁻²¹ and hygienic hand disinfection,²² as bath additives for the treatment of staphylococcal carriers,²³ and combined with corticosteroids, in secondarily infected eczema and epider-

momycosis.²⁴ When whole-body bathing twice daily with a soap bar containing triclosan was prescribed for patients with leukemia, they were cleared of *S. aureus* within two weeks.²⁵ Our preliminary in vitro experiments have confirmed that 2 per cent triclosan in detergent is active against *S. aureus* whether it is susceptible or resistant to methicillin, and the remanent effect of a similar preparation of the skin of volunteers was quantified.¹⁸

All the patients in this outbreak were treated similarly. There were no controls. The inclusion as controls of patients treated under alternative regimens, all of which have previously been found ineffective, would have prejudiced the interests of the entire hospital community. In a hospital that had been free from entrenched infection, the overriding considerations in dealing with this explosive but localized outbreak were to prevent the methicillin-resistant *S. aureus* from spreading and to eradicate its sources totally. The two staff members and six younger ambulant patients who complied with our recommendation of frequent and energetic body washings were free of methicillin-resistant *S. aureus* on their skin within two weeks, whereas in the elderly and mostly non-ambulant patients eradication took longer, perhaps because of the less vigorous application of the antiseptic preparation during passive bed bathing. In all the patients, however, the skin and wounds were free from the organism within an average of three weeks, and cultures remained negative during three months of follow-up. Despite the considerable risk of sepsis in these patients who had undergone vascular surgery, serious infection was prevented by the rapid control of this epidemic.

We are indebted to the control-of-infection nurse, Miss A.M. Buckles, and the nursing staff of the vascular unit, without whose active participation and joint efforts the control of this outbreak would have been impossible; and to Dr. C. Taylor-Robinson for his opinions and comments during the preparation of this paper.

REFERENCES

1. Jevons MP. "Celbenin"-resistant staphylococci. *Br Med J* 1961; 1:124-5.
2. Benner EJ, Kayser FH. Growing clinical significance of methicillin-resistant *Staphylococcus aureus*. *Lancet* 1968; 2:741-4.
3. Rosendal K, Jessen O, Bentzon MW, Bülow P. Antibiotic policy and spread of *Staphylococcus aureus* in Danish hospitals. 1969-1974. *Acta Pathol Microbiol Scand [B]* 1977; 85:143-52.
4. Kayser FH. Methicillin-resistant staphylococci 1965-75. *Lancet* 1975; 2:650-3.
5. Haley RW, Hightower AW, Khabbaz RF, et al. The emergency of methicillin-resistant *Staphylococcus aureus* infections in United States hospitals: possible role of the house staff-patient transfer circuit. *Ann Intern Med* 1982; 97:297-308.
6. Thompson RL, Wenzel RP. International recognition of methicillin-resistant strains of *Staphylococcus aureus*. *Ann Intern Med* 1982; 97:925-6.
7. McDonald PJ. Methicillin-resistant staphylococci: a sign of the times? *Med J Aust* 1982; 1:445-6.
8. Pearson CH, Whitehead JEM. Antibiotic sensitivity testing: A modification of the Stokes method using a rotary plater. *J Clin Pathol* 1974; 27:430-1.
9. Parker MT. Phage-typing of *Staphylococcus aureus*. In: Norris JR, Ribbons DW, eds. *Methods in microbiology*. Vol. 7B. New York: Academic Press, 1972:1-28.
10. Watanakunakorn C. Treatment of infections due to methicillin-resistant *Staphylococcus aureus*. *Ann Intern Med* 1982; 97:376-8.
11. Wheat LJ, Kohler RB, White A. Treatment of nasal carriers of coagulase-positive staphylococci. In: Maibach H, Aly R, eds. *Skin microbiology: relevance to clinical infection*. New York: Springer-Verlag, 1981:50-8.

12. Taylor J, Harris C. Terminology and skin disinfection. *J Hosp Infect* 1983; 4:323-4.
13. van der Hoeven E, Hinton NA. An assessment of the prolonged effect of antiseptic scrubs on the bacterial flora of the hands. *Can Med Assoc J* 1968; 99:402-7.
14. Müntener M, Schwarz H, Reber H. Zur chirurgischen Händedesinfektion mit einem Iodophor (Betadine). *Schweiz Med Wochenschr* 1972; 102:699-706.
15. Kundsinn RB, Walter CW. The surgical scrub — Practical consideration. *Arch Surg* 1973; 107:75-7.
16. Lowbury EJJ, Lilly HA. Use of 4 per cent chlorhexidine detergent solution (Hibiscrub) and other methods of skin disinfection. *Br Med J* 1973; 1: 510-5.
17. Peterson AF, Rosenberg A, Alatary SD. Comparative evaluation of surgical scrub preparations. *Surg Gynecol Obstet* 1978; 146:63-5.
18. Bartzokas CA, Corkill JE, Makin T, Pinder DC. Assessment of the remanent antibacterial effect of a 2 per cent triclosan-detergent preparation on the skin. *J Hyg (Lond)* 1983; 91:521-8.
19. Lilly HA, Lowbury EJJ. Disinfection of the skin with detergent preparations of Irgasan DP 300 and other antiseptics. *Br Med J* 1974; 4:372-4.
20. Eitzen HE, Ritter MA, French MLV, Gioe TJ. A microbiological in-use comparison of surgical hand-washing agents. *J Bone Joint Surg (Am)* 1979; 61:403-6.
21. Cremieux A, Guiraud-Dauriac H, Duménil G. Activité d'une mousse antiseptique sur la flore normale des mains. *Med Mal Infect* 1980; 10:4-7.
22. Bartzokas CA, Gibson MF, Graham R, Pinder DC. A comparison of triclosan and chlorhexidine preparations with 60 per cent isopropyl alcohol for hygienic hand disinfection. *J Hosp Infect* 1983; 4:245-55.
23. Ayliffe GAJ, Babb JR, Collins BJ, Davies J, Deverill C, Varney J. Disinfection of baths and bathwater. *Nurs Times* 1975; 3:22-3.
24. Reiffers J. Essai d'une nouvelle crème antifongique et antibactérienne à base de triclosan (CGP 433-Logamel). *Schweiz Rundschau Med (Praxis)* 1981; 70:1050-3.
25. Bodey GP, Rosenbaum B. Evaluation of a bacteriostatic soap, P-300, on skin flora of patients in protected environments. *Curr Ther Res* 1973; 15:253-60.

**CASE RECORDS
OF THE
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CASE 48-1984

PRESENTATION OF CASE

A 43-year-old right-handed man was admitted to the hospital because of headache and visual disturbances.

He was well until three days earlier, when he experienced the sudden onset of a "flashing" light in the left visual field, followed by a severe, throbbing right-sided headache and pain in the right eye; his left visual field was "cloudy." The visual symptoms soon cleared, the headache subsided after 16 hours, and he returned to his work as a dishwasher. On the following day his symptoms recurred in a milder form for four hours. One day before entry another bout of symptoms persisted for several hours. On the day of admission the visual symptoms recurred, followed by a headache that was intense behind both eyes, especially the right. He noticed diplopia and diminished vision to the left side and observed that all objects in the visual field appeared magnified and inverted. He experienced weakness in all the limbs, especially the left

arm, and complained of spinning dizziness and bilateral tinnitus. He was admitted to the hospital.

The patient was a native of Haiti and spoke little English. There was a history of headaches and tearing of his eyes in his 20s. During the 5 to 10 years before entry he experienced bouts of ocular pain with headaches that were more prominent in the right posterior part of the cranium, accompanied by visual problems of an uncertain nature; the symptoms were treated with topical drops and pills of unknown type. The symptoms had increased in frequency in recent months. One year before admission he immigrated to this country. He reported a positive tuberculin skin test approximately 12 years previously; he had no information about BCG vaccination. He recalled pain in a right upper molar tooth during the month before entry. He had ingested raw milk and uncooked beef in the past but had not eaten pork in recent years. There was a family history of headaches in a grandfather. There was no history of fever, vomiting, cardiovascular disease, stroke, seizures, symptoms of otitis media or sinusitis, use of tobacco, medications, or illicit drugs, surgical procedures, head trauma, known exposure to tuberculosis or to domestic animals, homosexual activity, or travel other than in Haiti and the northeastern United States.

The temperature was 36.2°C, the pulse was 84, and the respirations were 18. The blood pressure was 140/95 mm Hg.

The patient appeared muscular and well except for evident pain about the head, with tearing of both eyes; there was no evidence of head trauma. The optic fundi and disks appeared normal. Dental hygiene was poor. The neck was supple; the carotid pulses were full and without bruits. No lymphadenopathy was found. The lungs were clear. The heart was normal except for a Grade I systolic ejection murmur along the left sternal border. Abdominal and rectal examinations were negative. Neurologic examination disclosed that the patient was oriented and spoke apparently fluent Haitian French. A left partial homonymous hemianopia was present. Extraocular movements were full, with-