

pecially in small capillaries of the organs and the skin. This may explain the high yield and the dissemination of cutaneous lepromas in some infected animals. The viability of *M. leprae* was not negatively influenced by ultrasonic vibration. The increase of the viable count of *M. leprae* might have been expected due to similar findings in other pathogenic and saprosaprophytic mycobacteria. Accordingly, mild ultrasonic vibration is recommended for experimental infection in armadillos and other animal models for leprosy.

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Acknowledgment. This work was supported by the German Leprosy Relief Association.

REFERENCES

1. BROWN, I. N. Animal models and immune mechanisms in mycobacterial infection. In: *The Biology of the Mycobacteria*. Ratledge, C. and Stanford, J., eds. London: Academic Press, 1983, vol. 2, p. 84.

Sudden Respiratory Collapse in an Armadillo (*Dasypus novemcinctus*, Linn.).

TO THE EDITOR:

Twenty armadillos (*Dasypus novemcinctus*, Linn.) were imported into the United Kingdom (U.K.) in late 1984 by air freight from the United States of America (USA) for inoculation with leprosy bacilli of human origin as part of the IMMLEP (Immunology of Leprosy) program within the Special Programme for Research and Training in Tropical Diseases. All of the animals were in excellent condition on arrival. Eight days later, before inoculation, one of the animals suddenly collapsed with rapid, labored breathing and a slightly blood-stained discharge from the nose and mouth. Treatment with a single intramuscular dose of 38 mg amoxicillin was ineffective, and the animal died before any further action could be taken. The acute respiratory symptoms lasted less than 12 hours before death ensued.

At necropsy, the only abnormal findings were pulmonary congestion and a blood-stained discharge in the mouth and upper respiratory tract. Histopathological examination of lung tissue revealed marked intra-

alveolar and interstitial edema, congestion, inflammation and hyaline membrane formation (Fig. 1). In many areas there was also collapse and consolidation with accumulation in the alveoli of an exudate containing desquamated pneumocytes, mononuclear cells, and relatively few polymorphonuclear leukocytes; siderophages were also present in some areas. There was extensive hyaline membrane formation affecting the alveolar ducts as well as the alveoli themselves. This stained positively for fibrin and was also PAS-positive.

No acid-fast bacilli were identified in Fite-Faraco stained sections, but large numbers of Gram-negative bacilli were visible in Gram- and in Giemsa-stained sections, often contained within macrophages (Fig. 2). Fungi and other organisms were not identified.

The appearances are suggestive of an overwhelming Gram-negative pneumonia. In the human subject and animals, hyaline membrane formation can be produced by similar pulmonary infections^(1,2). The extent and severity of the changes suggest that

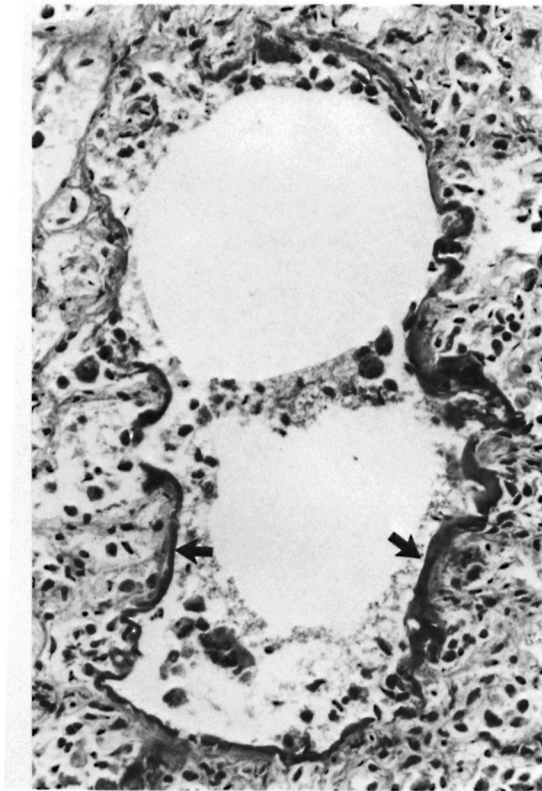


FIG. 1. A field showing hyaline membrane formation (arrows) and the widespread inflammatory infiltrate filling the alveolar spaces of the lung (PAS-hematoxylin $\times 400$).

the animal had had a respiratory illness for several days. Whether the Gram-negative infection present represents the initial disease or a complication of a pre-existing, possibly viral, infection cannot be determined from the material available.

Sudden respiratory collapse is a relatively common mode of death among laboratory animals in similar conditions of captivity. A nine-banded armadillo in the U.K. costs around US\$500 per annum to maintain, and it is obviously important to avoid such losses. Close observation for signs of respiratory infection or distress, bacteriological investigation, and early antibiotic treatment appropriate to Gram-negative bacillary infection may aid the prevention of further deaths of these valuable animals.

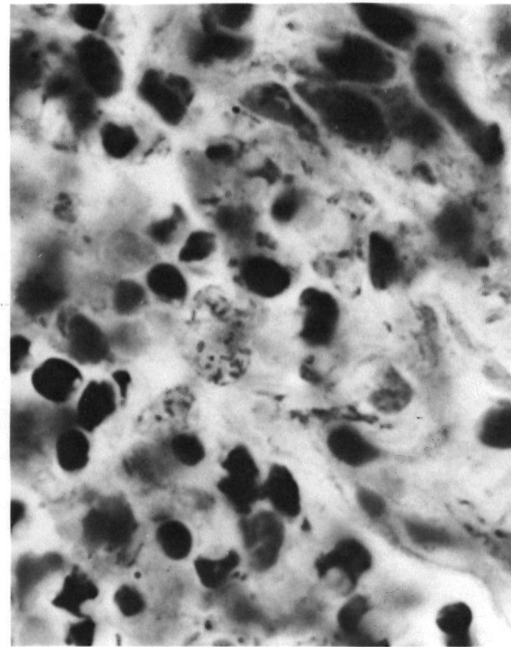


FIG. 2. High-power field showing numerous bacilli free and within macrophages (Giemsa $\times 2000$).

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REFERENCES

- HASLETON, P. S. Adult respiratory distress syndrome—a review. *Histopathology* 7 (1983) 307–332.
- JONES, T. C. and HUNT, R. D. Pneumonia of "Shipping fever." In: *Veterinary Pathology*. Philadelphia: Lea and Febiger, 5th ed., 1983, pp. 1231–1233.