Hind limb paralysis from electrical shock in three gilts

D. J. Steffen, DVM, PhD; D. A. Schoneweis, DVM, MS; J. L. Nelssen, PhD

Hind limb paralysis in swine can result from a variety of injuries to the spinal cord or musculoskeletal structures of the hind limbs. Trauma to the vertebral column with subsequent spinal cord damage often results in loss of caudal locomotor function and sensitization. Damage in sows may occur after minor trauma, such as breeding injuries or slipping on wet concrete. It usually can be related to preexisting skeletal changes, namely osteoporosis. Traumatic injury to the spinal cord in young growing swine is less common. Reported here is hind limb paralysis in 3 young pigs after nonfatal electrical shock.

A group of crossbred pigs, with average weight of approximately 85 kg, were being used in a study to evaluate a growth enhancer relative to various concentrations of lysine. The pigs had been observed earlier in the morning; all were alert and did not have observable gait problems. They were fed a milo and soybean meal ration with added synthetic lysine. The rations were balanced to meet or exceed the National Research Council requirements for calcium, phosphorus, and trace minerals. Later the same morning, the pigs were evaluated and again determined to be normal. After the caretaker finished checking the pigs, he noticed that a circuit breaker was thrown. He flipped the breaker on, heard several pigs squeal, and immediately turned the breaker off. The breaker was determined to control the current flow to the heater for the pen waters, and should have stayed in the off position for the summer season. Subsequent examination of the pens revealed that 3 pigs were down and unable to use their hind limbs.

The pigs were housed in an open-front building in pens 6 × 14 feet, with nipple waterers and self feeders. The concrete pen floors were wet because of a sprinkler system for combating high environmental temperatures (>26 C). The affected pigs were housed in separate pens, and all 3 may have been drinking when the breaker was turned on.

The 3 downed pigs were bright, alert, and aware of their surroundings. Evidence of burns was not found on the feet, limbs, snout, or in or around the oral cavity. They were able to remain sternal and dragged themselves around by their front feet; the hind limbs appeared to be nonfunctionable. Neurologic examination of the hind limbs revealed a withdrawal reflex when the area was pricked with a needle. Evidence of central pain recognition was not noticed in any of the 3 affected pigs. To confirm the absence of central pain recognition, an electrical livestock prod was applied to the hind limbs. This caused greater muscular contractions and withdrawal of the limbs; however, central pain recognition was not noticed. The line of cutaneous desensitization was near the level of the lumbosacral articulation. The pigs were acutely aware of any stimuli applied cranial to this region. Neurologic examination of the front limbs and cranial nerves revealed no abnormality. Because of the lack of perception of deep pain, a poor prognosis for recovery of locomotor function was given. Because the pigs were assigned to a research project, they were euthanatized IV 48 hours after the electrical shock, and necropsies were performed. Review of the records indicated that only 2 of the 3 pigs received a growth enhancer implant.

Pig 1 had bilateral sacroiliac luxations and a comminuted fracture of the seventh lumbar vertebra, with fragments being displaced into the spinal canal. Necrosis and hemorrhage were found in the muscles surrounding the fracture and luxation sites. Epidural hemorrhage extended cranial to the thoracolumbar articulation. Malacia and hematomyelia were in the spinal cord adjacent to the fracture. The urinary bladder was distended and filled much of the caudal portion of the abdomen. Ecchymotic and suffusion hemorrhages were in the bladder wall and extended into the lateral ligaments. Retroperitoneal hemorrhage was evident.

Pig 2 had lesions similar to those of pig 1 plus comminuted fractures of the sacrum and the sixth lumbar vertebra. Malacia was found over a wider

From the Departments of Pathology (Steffen) and Surgery and Medicine (Schoneweis), College of Veterinary Medicine, and Department of Animal Science and Industry (Nelssen), College of Agriculture, Kansas State University, Manhattan, KS 66506. Dr. Steffen's present address is Department of Veterinary and Microbiological Sciences, Van Es Hall, North Dakota State University, Fargo, ND 58105.

area of the spinal cord. The lungs were congested and had a rubbery texture.

Pig 3 had lesions similar to those of pig 1 but did not have an enlarged bladder. However, it did have a fractured sacrum. The lungs were congested and firm. The rectum was prolapsed.

Histologic examination of the spinal cord confirmed severe malacia. Congestion, alveolar edema, and multifocal areas of hemorrhage were found in the lungs of pigs 2 and 3. Endochondral ossification was normal in the ribs, thoracic vertebra, and long bones. Cortices of all bones appeared normal, and evidence of preexisting skeletal lesions was not found.

The 3 affected gilts apparently received electrical shocks from a malfunctioning heating system to the nipple waterers. The resulting muscular contractions caused fractures of lumbar or sacral vertebrae, spinal cord trauma, and hind limb paralysis. Gross lesions of electrocution were not found on clinical examination or at necropsy. To our knowledge, hind limb paralysis caused by electrocution without death has not been reported. Giles and Simmons[1] reported fractures of the pelvis and vertebrae in the lumbosacral area in pigs coming in contact with electrically charged fencing. Best[2] reported similar lesions in pigs struck by lightning, and DeBowes and Roden[3] reported vertebral fracture in a foal after electrical shock. It is probable that all 3 gilts were standing on wet concrete and drinking when the electrical shock occurred. Each received a short burst of electricity that caused sudden and severe muscular contractions, resulting in fractures. Evidence of burns was not found on the feet, limbs, snout, or in or around the oral cavity.

Complete history aided in determining the cause of the fractures and resulting hind limb paralysis. If it had not been established that the pigs received an electrical shock, the cause might not have been determined because death or morphologic lesions suggestive of electrocution did not occur. If fractures of the pelvic or lumbosacral area are found in pigs, in the absence of trauma or preexisting skeletal lesions, electrical shock should be considered as a possible cause, and the electrical wiring and service to the pens or waterers should be checked.

1. Giles N, Simmons JR. Electrocution of pigs. *Vet Rec* 1975;97:305–306.

2. Best RH. Lightning stroke in swine. *Can Vet J* 1967;8:23–24.

3. DeBowes RM, Roden PH. Vertebral compression fracture in a foal following electrical shock. *J Vet Orthop* 1981;2:14–19.

Book Review: Veterinary Ophthalmology, Second Edition

When the first edition of this text was published ten years ago, it marked a watershed in the development of veterinary ophthalmology. For the first time, in order to cover the subject comprehensively, a multi-authored effort was necessary and possible. *Veterinary Ophthalmology* instantly became the specialty's standard reference text. A variety of other texts, earlier and more recent, address specific aspects of veterinary ophthalmology, but none approach this publication's simultaneous depth and breadth of coverage.

As in the previous edition, the current volume concentrates largely on canine ophthalmology. This reflects the relative attention that dogs have received in the scientific literature and clinically. Approximately a third of the book, however, deals with other species and includes chapters on feline, equine, food animal, laboratory animal, and, new for this edition, exotic animal ophthalmology.

The publishers expended considerable effort to ensure the quality and durability of this compendium. It is hardbound and is printed on glossy paper, with generally excellent illustrations, including line art and black and white and color photographs. The only fault detected was a misprinted figure on page 494. The book deserves pride of place on the bookshelf.

This new edition will continue as the primary reference in its field. For students or general veterinary practitioners who choose to have only one text in this specialty, *Veterinary Ophthalmology* is without question the one to have.—[*Veterinary Ophthalmology, Second Edition. Edited by Kirk N. Gelatt. 750 pages; illustrated. Lea & Febiger, 200 Chester Field Parkway, Malvern, PA 19355-9725. 1991. Price $125.00.*]—GREGORY M. ACLAND