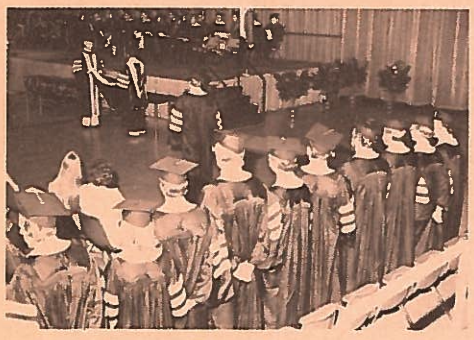
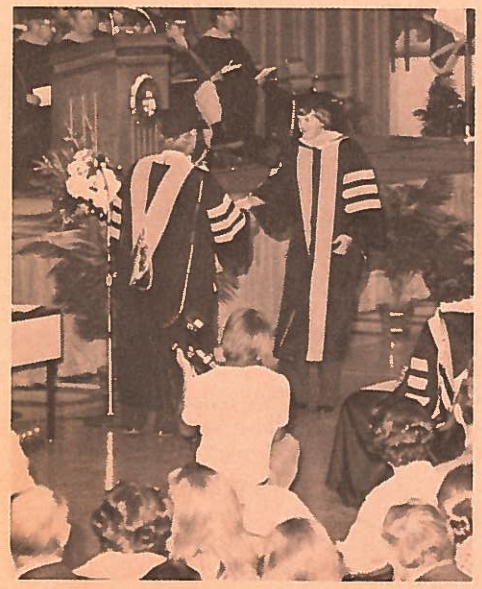
Veterinary Medical Review

College of Veterinary Medicine and UMC Extension Division





Precommencement Exercises

University of Missouri-Columbia

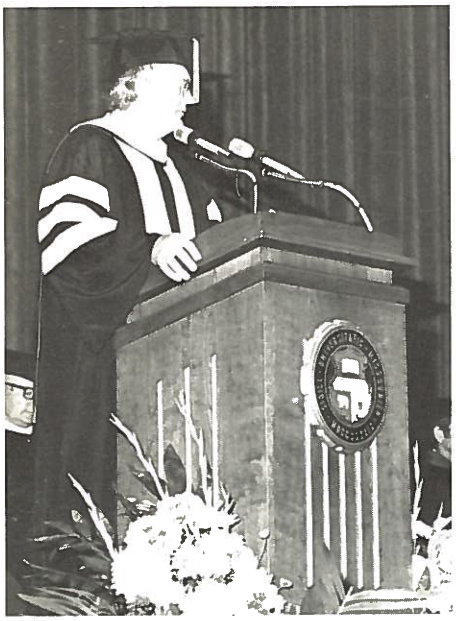
July, 1977, No. 101

Seventy graduates were conferred the title, "Doctor of Veterinary Medicine" at the College's 28th Precommencement Exercises, May 14, 1977.

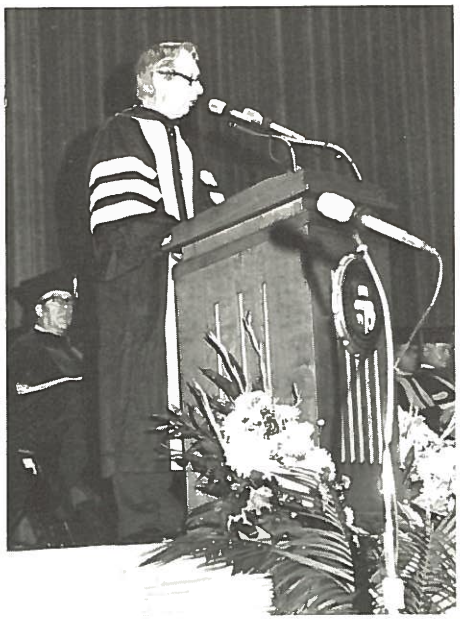
Since the graduation of 26 students at the College's first Precommencement Exercises in 1950, the College has produced an additional 1,041 Doctors of Veterinary Medicine.

See page 2 for more details and list of graduates.

See page 4 for well-researched discussions of two types of inherited problems of the canine eye: Glaucoma and Progressive Retinal Atrophy.



Dr. Kenneth D. Weide



Dr. Leo K. Bustad

Dr. William F. Bryson

College Graduates 28th Class

Speaking to the graduates and the audience before he administered the Veterinarian's Oath, Dr. William Bryson, President of the M.V.M.A., mentioned that an occasion such as this Precommencement may seem insignificant at the time but twenty years later it becomes a very important event in everyone's lives.

As the graduates of the Class of 1977 silently filed into Hearnes Auditorium on Saturday, May 14, some of them may have felt that this event was only a rather small eddy in the mainstream of their lives. But it was the one event which formally honored their years of work, study and personal change with the official investiture of the title of "Doctor".

Dr. Kenneth D. Weide, Dean of the College, presided at the ceremonies and he welcomed the 70 graduates, the nine doctors completing their internships and residencies, and the more than 850 family members, relatives and friends in the audience.

Dr. Weide's welcome was followed by greetings and introductions from Dr. Walter C. Daniel, Vice Chancellor of the University of Missouri-Columbia, Dr. John F. McGowan, Provost for Administration, and Dr. Theodore Higgins, President of the College of Veterinary Medicine Alumni Association.

Dr. Leo K. Bustad, Dean of the College of Veterinary Medicine, Washington State University, delivered the address. Rather than fall back on clichés, Dr. Bustad instead used examples taken from the writings of Loren Eiseley, *The Iliad* and the comic strip, "Peanuts," and emphatically stressed the never-ending development of the whole person in each of the graduates.

Following Dr. Bustad's address, Dr. Bryson gave the Veterinarian's Oath to the standing graduates. Then Dr. K. H. Niemeyer, Assistant Dean, and Dr. E. A. Corley, Associate Dean, invested each of the graduates with the hood as they stepped forward one by one. Following that, each graduate received a packet from Dr. Weide.

Judith Johnessee, the third graduate that day to be invested with the hood, was also the 1,000th graduate of the College. The 70 graduates that day brought the total number of graduates from the College to 1,067.

After the recognition of the completion of residencies and internships, and the benediction delivered by Dr. Alfred Illingworth, Dean of the Missouri School of Religion, everyone recessed to an informal reception.

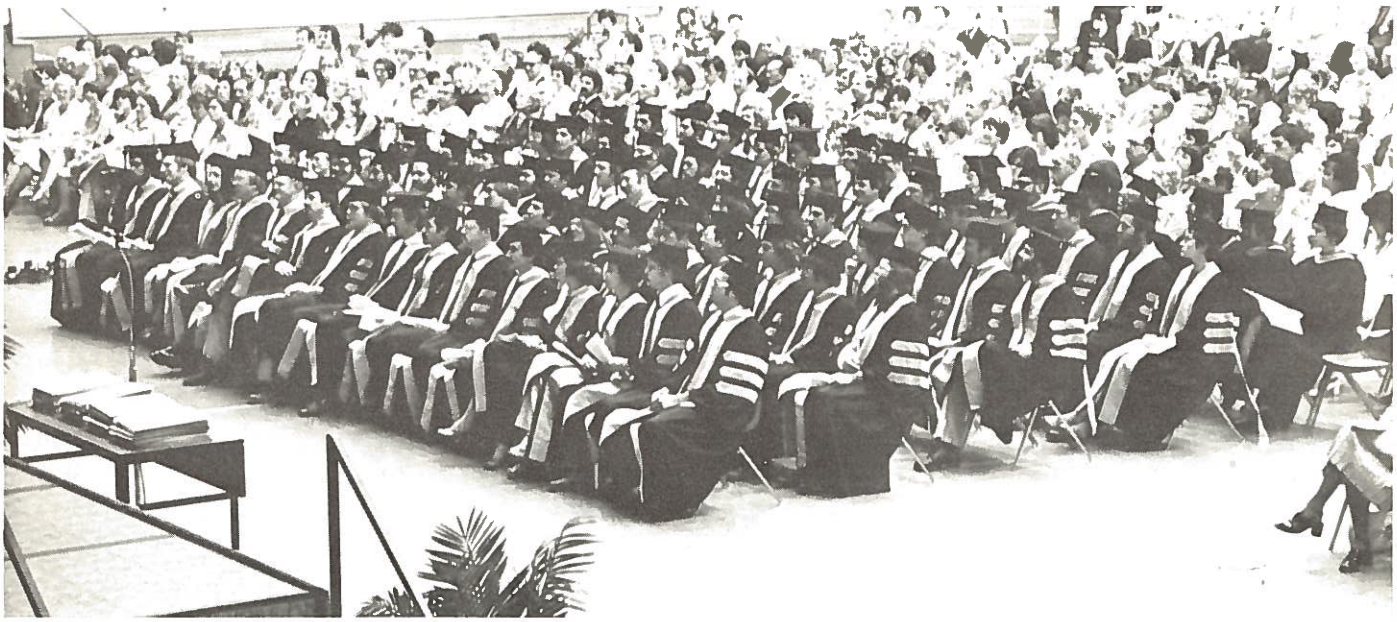
Dr. Kingrey Named Dean Emeritus

Dr. Burnell W. Kingrey, former Dean of the College, was awarded the title of Dean and Professor Emeritus (effective Oct. 1) during Commencement exercises, May 14, at the University of Missouri-Columbia.

Dr. Kingrey, a graduate of Iowa State University ('44), had been Dean of this college from 1963 to 1973. During Dr. Kingrey's administration the size of each incoming class increased from 30 students to 72 and plans were finalized for two new buildings on the College's campus.

Dr. Kingrey is known nationally and internationally as an educator in the field of veterinary medicine, and at the present time is consultant to several universities and firms interested in veterinary medicine. Dr. Kingrey also has an interest in international programs and has served as a consultant to several countries including Argentina, Chile and Guatemala.

Last fall the College presented him with the Distinguished Service Award.



". . .a person is a whole person only to the extent that he possesses compassion, reverence, and intellectual integrity."—Dr. Leo Bustad

Graduating Class of 1977

Doctor of Veterinary Medicine Summa Cum Laude

Danny Wayne Brown
James Carrol Vulgamott

Doctor of Veterinary Medicine Magna Cum Laude

Judith Sharon Johnessee
Brenda Davidson Politi
Raymond John Visco

Doctor of Veterinary Medicine Cum Laude

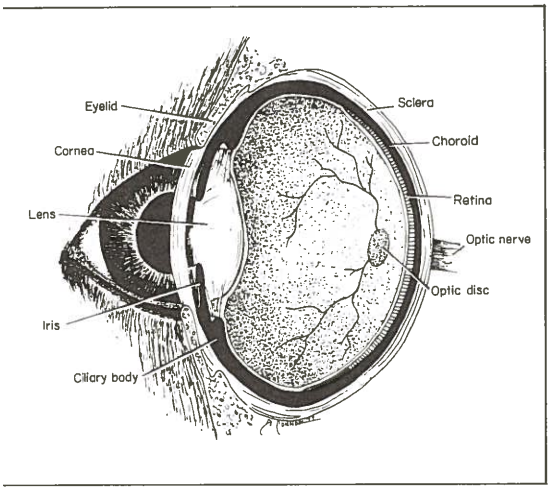
Steve Alan Nickell
Robert Max Thornsberry, Jr.

Doctor of Veterinary Medicine

Dennis Mical Arn
Ray Allen Acker, Jr.
Lance Lee Allen
Larry Don Berry
Christopher Lance Bratcher
Deborah Ruth Bruton
Alan Burns
Samuel P. Carson, III
Rodney Kent Chapman
Michael Kenneth Crecelius
Robert Eugene DeField
Ignacio T. dela Cruz
John Christopher Duemler, Jr.
Jerry Ray Eber
Karen Victoria Fenton
Clark Kenneth Fobian
Hires Walton Gage
Charles Jack Germeroth
Chester Earl Gleason, Jr.
Stephen Ray Goff
Bryce Marvin Goman
Jimmie Wade Greene
David Keith Hardin
John Robert Holden
David Edmond Hopson
Howard Russell Hutchins
Martha Witten Johnson
William Alfred Johnson
Barbara Jean Kingsborough
Rebecca Kirby
Donna Frances Kusewitt
Robert Rawleigh Link
Charles Antony Martin
Wiley Ray McVicker
Thomas Patrick Meehan
Daryl Glen Meyer
John Robert Middleton, Jr.
John Richard Miller
Margaret Allan Miller
Arlen Gene Mills
Terry H. Mitchel
Dwight Monroe Nash
Timothy Patrick O'Neill
Jack Randy Phillips
Jerry Lee Quance
Isaiah Daniel Russell
Philip Jay Shanker
Thomas Jerome Shumaker
Bradbury Bartlett Smith
Catherine Dahlem Smith
Glendell Lee Snider
Allan Richard Spector
Lynn Caryll Steele
Patricia Louise Stewart
Denis Edward Stuppy
Dennis Phillip Stuttgen
James Reid Turk
Charles Douglas Turmail
Thomas Walter Vorholt
Alan Ray Wessler
Gary Lee Wilson
Gary Wayne Witherwax
Gary Alan Yavitz

Glaucoma and Progressive Retinal Atrophy –

The following discussions were excerpted from a forthcoming book, *Inherited Abnormalities of Structure and Function in Companion Animals* by Drs. C. W. Foley, J. F. Lasley, and G. D. Osweiler.



Glaucoma

Glaucoma refers to a group of ocular disorders which result in an increase in intraocular pressure. This increased pressure leads to structural damage and impairment of function. Glaucoma may result from an increased rate of aqueous humor production, but usually it is due to a narrowed iridocorneal angle i.e., inadequate aqueous drainage.

Glaucoma may be classified as primary, secondary, congenital or absolute. Congenital glaucoma may also be classifed as primary. Primary glaucoma refers to glaucoma in which the intraocular pressure increase is not the result of complications caused by previous ocular disease. Secondary glaucoma refers to an increase in intraocular pressure resulting from other known intraocular diseases or eye damage. Secondary glaucoma is more common than primary, and is often unilateral, whereas primary glaucoma is usually bilateral, although both eyes need not be affected simultaneously. One eye may be affected before the other by a few days or up to 2 years. Congenital glaucoma results from a rise in intraocular pressure due to an eye malformation, such as an abnormality of the angle of the anterior chamber which is present at birth. Absolute glaucoma or blindness is the final stage in any glaucoma.

Two theories have been suggested as to the cause of primary glaucoma, and either of these or the combination of the two may be the cause. The first theory, neurovascular, suggests abnormal ocular circulation, which might occur on a local basis within the eye or as a consequence of a disturbance in the general circulation of the body as a whole. Contributing factors include disorders of the hypothalamic region, autonomic nervous system or the endocrine system. The second, and perhaps most favored theory, is that it is mechanical. This refers to an actual blockage or inhibition of drainage. A common finding is an abnormal iridocorneal angle. The conversion of a narrow angle into one of complete closure is not well understood.

Primary glaucoma occurs for the most part in the American Cocker Spaniel, but is also found in the Basset Hound. More recently it has been reported in Beagles. In England, the condition has been reported in the Miniature Poodle, Wirehaired Terrier, Poodle, Samoyed, English Cocker, Basset Hound and English Springer Spaniel. Primary glaucoma is a disease of older dogs, usually occurring between the ages of 3½ to 13 years.

Early clinical signs may go unnoticed by the dog's owner. The general clinical signs may include (not all in one individual) the following: increased intraocular pressure, pain, cloudy cornea, insensitive cornea, shallow anterior chamber, dilatation of the pupil with no response to light, episcleral vascularization (congestion of the episcleral vessels), loss of vision (partial or complete), buphthalmos (hydrophtholmos), cupping of the optic disc, atrophy of the optic disc and retina, and atrophy of the iris. Acute glaucoma, at least in the Cocker, appears to be somewhat seasonal and occurs most often from October to May.

Pathophysiology

One observes an enlargement of the globe, a narrowed but not closed angle, atrophy of the ciliary body, choroid and retina. Pannus, vascularization of the cornea and congestion of the ciliary vessels may occur in an eye which has been subjected to numerous repeated attacks. Saggital sections of the eye reveal the open angles of the anterior chamber and cupping in the optic nerve.

An enlargement of the corneal epithelium reveals intra- and intercellular edema. Atrophy and degeneration of the retina, the uveal tract nerve fiber occurs, and the inner nuclear retinal layers are absent. Choroid is almost vascular with pigmentation. Lacunae in the optic nerve are visible in the cupped optic nerve.

The aqueous humor in both chambers is formed and drained from the eye in the ciliary region; the ciliary processes are split with a ciliary cleft between them. The ciliary process consists of a central core of stroma and blood vessels covered by epithelium and is supplied by blood from the ciliary arteries. The aqueous humor is formed from all the blood vessels in the anterior part of the eye, but largely from the vessels within the ciliary processes. Diffusion and filtration of nonelectrolytes and electrolytes are involved in the formation of aqueous, with substances from the blood initiated by an enzymatic activity of the cells of the ciliary process. The aqueous is drained by way of a network of capillaries and veins forming an intrascleral plexus which is associated with the ciliary cleft and drains into the ciliary veins. The processes which make and remove the aqueous must be in balance to maintain normal intraocular pressure (IOP), which is altered either by changes in the volume of aqueous within the eye or by some interference in outflow. Intraocular pressure may be greatly changed by differences in the rigidity of the sclera, by topical corticosteroids applied to the eye, and possible by the myxedema of hypothyroidism.

Inheritance

The definite mode of inheritance for glaucoma in the Cocker Spaniel has not been reported. The high incidence of this condition in the Cocker, and perhaps the Bassets, indicates a hereditary predisposition. A narrow iris angle in the Cocker predisposes to acute congestive attacks. Glaucoma is reported to be inherited as a dominant or a recessive in man. Limited data for the Beagle suggests that glaucoma (with lens luxation) is inherited as an autosomal recessive trait.

Recommendation

Since the mode of inheritance is not well defined, specific recommendations cannot be made. The breeding of known affected animals and their close relatives should be discouraged. Elimination of this

trait should be attempted in the same manner as for any other recessive or dominant gene.

Progressive Retinal Atrophy

Progressive retinal atrophy is characterized by a progressive bilateral symmetrical degeneration of the visual elements of the retina. Two types or forms of progressive retinal atrophy have been reported. The first type, generalized progressive retinal atrophy (PRA) or peripheral is due to a simple autosomal recessive gene. The second type, central progressive retinal atrophy (CPRA) probably results from a dominant gene, with incomplete penetrance (penetrance 80%); however, in some breeds CPRA may be due to a recessive gene, or it is possible that both dominant and recessive genes may influence this trait in the same breed. Both types are progressive and cause blindness.

With PRA night vision is gradually lost and day vision remains normal. In the next stage the dog has complete night blindness, with day vision deteriorating. In the third stage the animal is completely blind and secondary cataracts often develop in some breeds. Also an early loss of peripheral vision may be noted, with resulting "tunnel vision"; i.e., the dog only sees objects directly in front of it.

With CPRA, night blindness is not frequently observed. Dogs may be able to see better in dull light than in bright light. Dogs with CPRA have poor near vision and have difficulty in seeing still objects but can see moving objects until the latter stages of the disease. Peripheral vision may be retained for some time. Blind spots in the dogs vision may occur.

Degeneration begins in the first few weeks of the pups life, and loss of vision is progressive with complete loss of vision occurring in early to mid adulthood, dependent somewhat upon breed. Progressive retinal atrophy has been identified in at least 46 breeds. PRA is reported in many breeds, for example, Irish Setters, Miniature and Toy Poodles, Cocker Spaniels, Miniature Pinschers, Labrador Retrievers, Golden Retrievers, Huskies, Norwegian Elkhounds, Samoyeds, English Cockers, Collies, Akitas, and Salukis. CPRA is common in Labrador Retrievers, Border Collies, English Springer Spaniels, Golden Retrievers, and Shetland Sheepdogs. Careful selection and good breeding practices have been extremely successful in reducing its frequency. Breeds differences are noted as to time of development and electrophysiology.

Eye examination, when (PRA) is present, reveals a dilated pupil with poor and slow pupillary light reflex, although some reflex is retained even in advanced cases. When present, a cataract follows the course of the retinal atrophy, beginning as a cortical cataract both posterior and anterior, and ending as a total cataract.

Examination of the fundus shows the most obvious signs of narrowing of the retinal blood vessels, both arteries and veins. This sign is most easily seen in the vessels crossing the tapetum, the venous circle on the disc remaining until the end. There is pallor of the optic disc and increased reflection from the tapetal region, which becomes silvery and mirror-like in its appearance. The tapetum nigrum become pale gray, sometimes with darker pigment scattered about. With CPRA the pupil is dilated but not in the early stages and there is less associated cataract. The retinal changes start at the area centralis or macular region, soon spreading to the rest of the tapetal region and therefore involving the central retina. The first change is an increased reflection and pigmentary disturbance, small clouds and irregular spots of brown pigment appearing scattered over the tapetum lucidum and between these pigment spots the surface is highly reflective. The tapetum nigrum is not affected until much later and whether the whole retina becomes finally affected is debatable, some normal peripheral retina remaining for a long time. Attenuation of the retinal vessels also occurs but late in the course of the disease.

Physiopathology

Histological studies, at least for some breeds, indicates that following birth the retina develops normally for 18 to 21 days, after which, spontaneous degeneration of the rods begin, until the final stage of degeneration occurs with complete disorganization of the retinal layers.

In stage 1 of (PRA), one observes atrophy of rods and their nuclei. In stage 2, final disintegration of the cones occur and the external limiting membrane now lies against the pigment epithelium. In stage 3, the outer nuclear layer is reduced to a layer of cone nuclei. The inner nuclear layer is almost normal, as well as the ganglion cells. Pigmented epithelium is devoid of pigment even in the non-tapetal fundus. Choriocapillaries are much reduced. Later in this stage the changes proceeded to sclerosis. An irregular single nuclear layer is formed and the whole retina is greatly reduced in thickness. Later still there is a definite thinning of the choroid with a reduction of its blood vessels. Different pathologic processes occur in different breeds. For example, rod dysplasia in the Elkhound, progressive rod-cone degeneration in Miniature Poodles, rod-cone dysplasia in Irish Setters, and cone degeneration in the Malamute.

The histopathological changes of (CPRA) are hypertrophy and migration of the pigment epithelium cells and loss of the layer of rods and cones. The large pigment-laden, cells migrate through the retina and form cell nests on its inner surface. By this time the retina is disorganized. The changes occur in the central region and there is a sharp division between the affected and the unaffected peripheral retina.

Inheritance

Indications are, as previously mentioned, that generalized progressive retinal atrophy is inherited as a simple autosomal recessive gene (Toy and Miniature Poodle, Norwegian Elkhound, Irish Setter, Alaskan Malamute). Central progressive retinal atrophy is inherited as a dominant in some breeds (Labrador), but in at least one breed, a dominant as well as a recessive mode of inheritance may be involved.

Recommendation

For PRA (recessive) any individual animals exhibiting this trait should not be used for breeding. A breeding test could be established to detect carrier individuals. Selection has been extremely successful in Irish Setters. Test mating is less practical in breeders where the defect develops later in life.

For CPRA all one would need to do is to not use the dog expressing this trait for breeding.

Apparently there is no treatment for the condition.

Antifreeze Poisoning Treatment Methods Investigated

A dog that has ingested 10 milliliters of ethylene glycol per each kilogram of body weight will die if allowed to wait just one hour before initiating current therapeutic methods. Another dog, having ingested the same ratio of ethylene glycol to body weight and allowed to wait for six hours, has been saved using a technique being investigated by Drs. V. V. St. Omer, M. J. Dallman, R. A. Green, and R. W. Zumwalt.

And, as Dr. St. Omer stressed, an animal can be saved with only four treatments with the new technique versus eight or more with older methods.

Currently, dogs suffering from ethylene glycol toxicosis are treated with a mild diuretic such as Mannitol (while kidney function is still normal), intravenous injections of ethanol, and intraperitoneal injections of sodium bicarbonate.

The technique being investigated by Dr. St. Omer and co-workers involves intravenous injections of ethanol like the old method but the sodium bicarbonate is also injected intravenously. A more powerful diuretic such as hydrochlorothiazide is used while the kidneys are functioning. At the onset of treatment, 350 - 500 ml of an isotonic saline solution is given subcutaneously just behind the neck. This method of fluid hydration is repeated throughout therapy.

Dr. St. Omer emphasized the importance of keeping the poisoned dogs well hydrated. He has found through experiments that dogs will last longer with only hydration therapy than with current methods of treatment. He believes the subcutaneous injection of fluid is more practical than giving the fluid intravenously in that the animals' bodies can absorb the fluid as it is needed without the danger of overhydration. More fluid can be injected as necessary.

When considering ethylene glycol toxicosis, many veterinarians have believed that dogs were killed only by calcium oxalate crystals blocking the kidney nephrons. This terminal renal failure (and uremia) attributed to calcium oxalate crystalization in the kidneys occurs only in animals that survive the acute stage of the toxicosis. According to Dr. St. Omer, in acute cases the animals are actually killed by metabolic acidosis. He has noted that the blood pH drops from 7.4 to 7.2 (with a corresponding but more marked drop in urine pH). To successfully treat an afflicted dog, the veterinarian must quickly correct the blood pH. Dr. St. Omer added that by monitering the urine pH, a veterinarian can determine when to stop treating the dog (stopping when the urine reaches a pH of 7.5 or 8).

Death following long-term toxicity will be due to calcium oxalate crystals in the kidneys.

Drs. St. Omer and co-workers are continuing to investigate ways of coping with delayed treatment. Experiments are being conducted on the incorporation of dimethyl sulfoxide (DMSO) as part of the antidotal regimen.

The inclusion of DMSO allows the systematic therapeutic regimen of ethanol and sodium bicarbonate to be effective in chronic ethylene glycol toxicosis or late diagnosed cases of acute toxicosis. Presently, such animals can only be treated by peritoneal dialysis and supportive drug treatment with the exclusion of ethanol. The prognosis is usually very poor.

The rationale for DMSO as part of the antidotal regimen in ethylene glycol poisoning is based on the following pharmacologic actions: (1) DMSO is a highly polar solvent and is effective in preventing experimentally induced urolithiasis; (2) it is an anti-inflammatory agent and will reduce edema and scarring; (3) it is a potent diuretic; (4) it causes vasodialation and aids in the diffusion of oxygen into the tissues thus affording protection from anoxia; (5) it causes enhancement of the action of a concommitantly administered drug.

Upcoming Continuing Education Programs

Listed below are some of the Continuing Education seminars and workshops which will be offered this coming school year. Of special interest to practitioners is the seminar on "Client Service and Practice Growth," to be presented on February 25, 1978. For more information, contact the Continuing Education And Extension Office, 234 Veterinary Medicine Bldg., University of Missouri-Columbia.

Lower Limb Lameness (in horses) — Sept. 21 - 22, 1977; fee $150. Instructors: Drs. Johnson, Garner, Tritschler, and Coffman. Registration deadline: Aug. 22, 1977.

Use of Pins and Wires in Fracture Repair — Sept. 30 - Oct. 1, 1977; fee $160. Instructors: Dr. Stoll and residents. Registration deadline: Aug. 31, 1977.

Comparative Internal Medicine of Horses and Cattle — Oct. 28 - 29, 1977; fee $50. Instructors: Drs. Coffman and Vestweber. Registration deadline: Sept. 29, 1977.

Conditions of the Equine Respiratory Systems — Nov. 15 - 16, 1977; fee $150. Instructors: Drs. Johnson and Garner. Registration deadline: Oct. 14, 1977.

Ophthalmic Surgery Workshop — Nov. 19 - 20, 1977; fee $185. Instructors: Dr. Jensen and anesthesiologist. Registration deadline: Oct. 20, 1977.

Client Service and Practice Growth — Feb. 25, 1978; fee $35 (sponsored jointly with the Student Chapter of the AVMA). Instructor: Mr. Robert P. Levoy, Director, Professional Practice Consultants, Great Neck, N.Y. Mr. Levoy has conducted more than 2500 management seminars, and is the author of two books and more than 300 articles on practice management. This seminar will be held at Columbia's Ramada Inn. Registration deadline: Jan. 25, 1978.

Unique Program in the Visiting Lecturers Series This Fall

On Thursday, October 27, Dr. Pedro Acha, Chief of the Division of Disease Control, Pan American Health Organization, will speak on, "Epidemiology - Economical and Social Implications of Foot and Mouth Disease Control Programs in the Americas." The program will be given at 4:00 p.m., place to be announced. For more information, contact the Continuing Education Office, 234 Veterinary Medicine Bldg.

Alumni Notes

Beginning this fall, most news items concerning the College's alumni will appear in the *Missouri Veterinarian* instead of this publication.

Nevertheless, please continue to send news about *you* or your colleagues to Dr. K. H. Niemeyer, Assistant Dean.

Grant Given for Research Animal Diagnostic Laboratory

Dr. Joseph E. Wagner, Professor of Veterinary Pathology, has been awarded a five year grant with $85,576 to be utilized the first year to continue the Research Animal Diagnostic and Investigative Laboratory at the College of Veterinary Medicine, University of Missouri-Columbia.

The grant was made by the Division of Animal Resources of the U.S. Department of Health, Education and Welfare. The laboratory which Dr. Wagner directs was started in 1968 and has been continually funded by HEW.

This laboratory provides investigators at the University of Missouri and other regional institutions the service of making definitive diagnoses of naturally occuring diseases of laboratory animals used in human health-related research.

In addition to that, the laboratory does research on naturally occuring diseases of laboratory animals with regard to improving the efficiency and accuracy of identifying diseases. Because animal models of disease are important in the study of human disease, the laboratory continually surveys animal disease cases and specimens for evidence of potential as animal models of similar human diseases.

Volunteers Move Veterinary Library

More than 150 faculty, staff and students moved the estimated 22,000 volumes of books and bound periodicals in the College's library to their new home on the second floor of the recently completed Veterinary Medical Building. Starting shortly after 8 a.m., Saturday, April 23, the volunteers carried the books by hand from the old facility despite intermittant rain showers. The move was completed before 2 p.m. that same afternoon.

The new home for the College's library has approximately 6,725 square feet of floor space, nearly three times the floor space of the old location, and has more than twice the amount of shelving. The seating capacity has been increased from 52 to 106. Mr. Trenton Boyd, the College's librarian, said that for the first time an entire veterinary class will be able to use the library at the same time.

Dr. Loren Kintner receiving a standing ovation after being presented his second Norden Distinguished Teacher Award by Dr. E. A. Corley.

Dr. Kintner Honored at Banquet

Dr. Loren D. Kintner, Professor of Veterinary Pathology, was presented the Norden Distinguished Teacher Award by Dr. E. A. Corley, Associate Dean, at the Honors Convocation Banquet which was given on May 12 at Columbia's Ramada Inn. This is the second time Dr. Kintner has received this award. Dr. Kintner has also received the AMOCO Good Teaching Award this spring.

Dr. Roger E. Brown, Professor of Veterinary Medicine and Surgery, received a standing ovation when Dean Kenneth D. Weide surprised him with a plaque as a token of appreciation for Dr. Brown's efforts as coordinator between the College, and the architects and contractors of the new buildings.

More than 25 awards and honors were announced that night.

The banquet was sponsored by both the Upjohn Company and the College.

At the banquet, the Upjohn Company presented to the College a limited edition print of "Up and Running", now hanging in the Dean's office area.

College Appoints Anesthesiologist

Dr. Cynthia Trim has been appointed Associate Professor for the College; she began work on April 25 and her specialty is anesthesiology.

Dr. Trim received the Bachelor of Veterinary Science degree from Liverpool University in 1970. She went on to Cambridge University to study anesthesiology and received the Diploma in Veterinary Anesthesiology in 1973.

Dr. Trim was on the faculty at the veterinary school at the University of Guelph in Ontario, Canada from 1974 to 1976. She then served briefly on the faculty at the University of Illinois before coming to UMC.

Donors to the College of Veterinary Medicine

January 1, 1975 through December 31, 1976

The list below is a continuation of the list of donors in the May/June issue of the *V.M.R.*

Northwest Animal Hospital
Bridgeton, Missouri

Mr. & Mrs. Jean O'Dell
Iowa City, Iowa

Mr. William J. Oetting
Clayton, Missouri

Ms. Patricia M. Ogle
East Alton, Illinois

Cont. on back page

A typographical error slipped by in the last issue of the *V.M.R.* Gifts donated to the College ranged from $5.00 to $50,000 and *not* from $5,000 to $15,000. Any gift the College receives is sincerely appreciated.

Dr. Thomas Omohundro
Jackson, Missouri

Ms. Joanne Orth
Kirkwood, Missouri

Dr. & Mrs. Brent M. Parker
Columbia, Missouri

Dr. & Mrs. Darrell C. Payne
Sedalia, Missouri

Mr. & Mrs. Eugene Pearline
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Professional Veterinary Pharmaceuticals
Princeton, New Jersey

Mr. & Mrs. Walter Prugh
Salem, Missouri

Ralston Purina Company
St. Louis, Missouri

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St. Louis, Missouri

Dr. & Mrs. John Rhoades
Columbia, Missouri

Dr. Richard T. Riegel
St. Louis, Missouri

Mr. Bernard A. Ross
St. Louis, Missouri

Mr. & Mrs. Milton Sager
Stanberry, Missouri

Ms. Geraldine Sapp
Columbia, Missouri

Ms. Nancy A. Sayers
Kirkwood, Missouri

Mr. & Mrs. Gordon Scherck, Jr.
St. Louis, Missouri

Dr. & Mrs. Warren Schilb
Oklahoma City, Oklahoma

Mr. & Mrs. Martin Schneider & Sons
Creve Coeur, Missouri

Mr. David E. Schoeffel
Kirkwood, Missouri

Dr. & Mrs. S. S. Schwartzbrott
West Babylon, New York

Mr. & Mrs. Robert J. Senkosky
St. Louis, Missouri

Mrs. Courtney Shands
Kirkwood, Missouri

Mr. & Mrs. John A. Shaw
Glendale, Missouri

Dr. & Mrs. Robert W. Shelby
Avon, Ohio

Mrs. Myrna Sherrill
Jefferson City, Missouri

Dr. F. Harel Shimp
Raytown, Missouri

Sight Hound Club of Greater St. Louis
St. Louis, Missouri

Dr. John B. Simpson
LaPlata, Missouri

Mr. & Mrs. George J. Smith
Harrisonville, Missouri

Southeast Missouri Kennel Club, Inc.
Jackson, Missouri

Dr. Gary Spragg
Rogersville, Missouri

Saint Louis Boxer Club
Affton, Missouri

St. Louis, Dog Breeders, Assn., Inc.
St. Louis, Missouri

St. Louis Southeast Arabian Youth Group
St. Louis, Missouri

Mr. & Mrs. William A. Stark
St. Louis, Missouri

Dr. Norden Stefanides
Hermiston, Oregon

Dr. Tim Stout
Florissant, Missouri

Mr. & Mrs. Dave Strudell
Kirkwood, Missouri

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