THE UNIVERSITY OF MELBOURNE

Melbourne Veterinary School

Veterinary Bioscience: Digestive System

Subject Guide

SEMESTER 1, 2023

Dr Nicholas Bamford
Subject coordinator

n.bamford@unimelb.edu.au

VETS90120 VETERINARY BIOSCIENCE: DIGESTIVE SYSTEM

VETS30016 VETERINARY BIOSCIENCE: DIGESTIVE SYSTEM

Veterinary Bioscience: Digestive System

INTRODUCTION

Welcome to Veterinary Bioscience: Digestive System. Whilst there are different subject codes for the graduate (DVM) and undergraduate (BSc3) subjects, all lectures, practical classes and case studies are shared. Lectures will be pre-recorded and made available through the LMS to provide flexibility of study. Practical classes and case study sessions will be held on-campus with supporting online resources available through the LMS. Learning materials will be released weekly, to encourage deep learning and revision practices.

This is the first of the 'systems-based' subjects that will bring together the disciplines of anatomy (structure and physical relationships at the micro and macro levels), physiology (function at the cellular, organ, system and animal levels), pathology (mechanisms of disease) and pharmacology (modifying function for the treatment of disease) in our study of the digestive system. By combining these disciplines into an integrated course, we hope you will be able to make links between the different disciplines which will greatly help in constructing a strong framework on which to build your knowledge and experience.

The content of the Digestive System subject is arranged into themes such as 'motility' or 'secretion and digestion'. This enables the course to address the function and dysfunction of this system, while maintaining a coherent narrative. This will also enable a routine to develop, which will culminate in a case study at the end of each theme. Case studies are an important addition to the lectures and practical exercises, and they have been devised to illustrate the importance of the acquired knowledge in the practice of veterinary medicine and to foster your clinical problem-solving skills. Each case is an example of a condition which commonly occurs in veterinary practice, and these exercises will help students to understand these situations from fundamental principles; and to apply their knowledge to explain the disease mechanisms and how they might be resolved. Students will work through these case studies in small groups, with a moderated summary session at the end.

The major assessments in this subject include a 1-hour intra-semester test and a 2-hour end-of-semester examination. In addition, online quizzes related to each of the case studies will form part of the final mark. Students in the graduate cohort (VETS90120) will also be required to complete a group-work assignment towards the end of semester. Students are directed to the subject handbook for full details of assessments and mark allocations for each component.

Your feedback on this subject is greatly appreciated (and will be sought during semester), and all staff will be happy to help steer you in the right direction if anything is unclear, in terms of either academic content or subject coordination.

I hope that your studies are stimulating and rewarding.

Dr Nicholas Bamford
Subject Coordinator
n.bamford@unimelb.edu.au

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THEME 1: WHAT'S WHERE

Week 1

The first week of this subject introduces the approach to teaching and learning in the Veterinary Biosciences that will be taken across the first two years of the Doctor of Veterinary Medicine course. You will be introduced to the structure and function of the digestive system through lectures and practical classes, including an OHS induction and safe instrument handling exercise.

LECTURES

On-campus Introductory Lecture	• Subject overview • Approach to teaching and learning • Assessment
Introduction to the Digestive System	• General design of the digestive system • Regions of the abdomen • Boundaries of the abdominal cavity • The peritoneal cavity

PRACTICAL CLASSES

OHS Induction	Safe instrument handling sign-off	WEBS Dissection Lab (B104)
Skeleton of the Head and Neck	Components of the axial skeleton	WEBS Dry Lab 1 (127/128) and OBLA

LEARNING OUTCOMES

	**SCIENTIFIC BASIS OF CLINICAL PRACTICE** • Describe the gross structure, relationships and function of the organs of the digestive system of domestic animals. • Use appropriate anatomical terminology to describe bony features of the head and neck.
	**CLINICAL SKILL ACQUISITION** • Demonstrate how to correctly hold a scalpel, thumb forceps, scissors, needle holders and haemostats.

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LECTURE 1
INTRODUCTION TO THE DIGESTIVE SYSTEM AND ABDOMINAL CAVITY

LECTURER

DR NICHOLAS BAMFORD

Nick received his veterinary degree from the University of Melbourne and worked for several years in mixed-animal and equine practices in Australia and the UK. He then returned to Melbourne to complete a PhD in equine endocrinology, followed by a residency training program in large animal internal medicine. Nick is a Senior Lecturer in Veterinary Biosciences and a Diplomate of the American College of Veterinary Internal Medicine. His research interests cover various aspects of equine health, including endocrinology, metabolism and clinical nutrition.

n.bamford@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the general design of the digestive system.
- Identify the regions of the abdomen.
- Describe the external and internal surface features of the oral and abdominal cavities.
- Describe the structure and function of the peritoneum.

KEY WORDS

Digestive system; digestive tract; alimentary canal; mouth; pharynx; abdomen; peritoneal cavity; peritoneum; mesentery; omentum.

LECTURE OVERVIEW

The digestive system includes the digestive tract and the accessory organs and glands. The function of the digestive system is to turn food into fuel by performing the following functions: securing, conducting and storing food; digestion and absorption of nutrients; and storage and disposal of wastes. The design of the digestive system in different species is adapted to suit different diets (e.g. carnivore, herbivore, omnivore). This lecture will provide an overview of the digestive system and abdominal cavity in animals.

FURTHER READING

Singh: Dyce, Sack & Wensing's *Textbook of Veterinary Anatomy*, 5th Ed. Elsevier, 2018.

Boyd: *Color Atlas of Clinical Anatomy of the Dog and Cat*, 2nd Ed. Mosby, 2001.

Hermanson: *Miller and Evans' Anatomy of the Dog*, 5th Ed. Elsevier1964.

Smallwood: *A Guided Tour of Veterinary Anatomy*, Saunders, 1992.

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THEME 2: INGESTION AND SWALLOWING

Week 2 to Week 5

Studies in these weeks focus on ingestion and swallowing: the structure and function of the mouth and teeth, muscles of mastication and salivary glands; and how food passes through the oesophagus to the stomach, including the various forms that the stomach can take in different domestic animal species. The fundamental principles of dental examination will be covered in practical classes. The approach to clinical examination and imaging of the gastrointestinal tract will also be presented, which is important background for the case studies. Case studies are an important addition to the lectures and practical classes that will illustrate the importance of the acquired knowledge in the practice of veterinary medicine.

LECTURES

Teeth and Tooth Development	• Anatomy of the teeth and tooth development • Tooth structure
Comparative Dentition	• Comparative dentition of carnivores, herbivores and other mammals • Function and arrangement of teeth • Ageing animals by their teeth
Prehension and Taste	• Anatomy of the mouth, tongue and muscles of mastication • Gross anatomy of the oral cavity and related structures • Introduction to the histology of the oral cavity and related structures
From Mouth to Stomach	• Structure of the salivary glands and mechanisms of salivary secretion • Anatomy of the oropharynx and physiology of bolus formation • Anatomy of the oesophagus • Control of swallowing and bolus movement in the oesophagus
Compound Stomach of Ruminants	• Gross and microscopic anatomy of the reticulum, rumen and omasum • Relationship with other organs in the abdomen • Form and function of the oesophageal / reticular groove • Comparison between bovine and ovine forestomachs
Simple Stomach and Abomasum	• Gross anatomy of simple stomach of different species • Relationship with other organs including liver; surface anatomy • Layers of the stomach wall; blood supply and innervation
Clinical Examination of the GI Tract	• How do you assess if the gut is working properly? • History taking, clinical examination and clinical investigation
Clinical Imaging of the GI Tract	• What can I see with the following technologies? - Radiography, contrast radiography - Ultrasonography; endoscopy - CT scanning; fluoroscopy
Diseases of the Oral Cavity	• Pathology of the oral cavity • Stomatitis; epulis; vesicular disease; glossitis

Diseases of the Oesophagus	• Pathology of the oesophagus • Megaoesophagus; oesophagitis

PRACTICAL CLASSES AND CASE STUDIES

Introduction to Case Studies	Problem solving approach to clinical cases through collaborative group work	WEBS Collaborative Learning Centre (G04)
Dental Wetlab	Dentition of dog, cat, horse, sheep, rabbit	WEBS Dissection Lab (B104)
Introduction to Dissection	Basic dissection techniques	WEBS Dissection Lab (B104)
Case study: Misty the Pony	Application of knowledge to clinical case	WEBS Collaborative Learning Centre (G04)

LEARNING OUTCOMES

	**SCIENTIFIC BASIS OF CLINICAL PRACTICE** • Recognise different tooth types and their function in a range of domestic animal species. • Describe the structures involved in the ingestion, mastication, swallowing of food, and its passage to the stomach. • Explain the unique anatomical variations of the stomach among domestic animal species. • Describe different forms of upper gastrointestinal pathology in the domestic species. • Describe the possible consequences of poor mastication of food.
	**CLINICAL SKILL ACQUISITION** • Conduct a dental examination of different animal species and complete a dental chart. • Extract an incisor using correct techniques. • Predict the age of a dog, sheep and horse by examination of its teeth. • Undertake anatomic dissection using correct instrument handling.
	**PERSONAL AND PROFESSIONAL DEVELOPMENT** • Consider the imperative for developing excellent communication skills as a veterinarian. • Appreciate the importance of collaboration and teamwork in all professional endeavours. • Engage in a collaborative approach to solving clinical problems.

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LECTURE 2
TEETH AND TOOTH DEVELOPMENT

LECTURER

DR CHRIS MURRAY

Following graduation from Sydney University, Chris worked in small animal practice for 10 years. She also was the Animal Welfare Officer at Macquarie University for several years. Since coming to Melbourne Veterinary School, Chris has taught first to third year veterinary students in a wide range of subjects, predominantly within the disciplines of veterinary anatomy and physiology.

cmmurray@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the general structure of a tooth and explain how the different tissues contribute to tooth function.
- Compare the development and general structure of brachydont and hypsodont teeth.
- Relate the different embryonic cell types to the dental structures they form.

KEY WORDS

Teeth, enamel, dentine, cementum, periodontal ligament, pulp cavity, gingiva, alveolar bone, vestibular, labial, buccal, lingual, palatine and occlusal surfaces, brachydont tooth, hypsodont tooth, deciduous teeth, dental lamina, tooth bud, enamel organ, dental papilla, dental sac, odontoblast, ameloblast, cementoblast, osteoblast.

LECTURE OVERVIEW

Structure of a simple tooth

The general structure of teeth will be explained using a simple brachydont (short-crowned) tooth as an example. The crown is the part of the tooth covered by enamel, and the root is embedded in the tooth socket in the jaw (alveolar) bone. Teeth are comprised of three different types of mineralized tissue, the shiny white enamel, and the dentine and cementum, which are both slightly yellowish. The dentine forms the framework of the tooth and is found in both the crown and the root, surrounding the central pulp cavity. The cementum surrounds the dentine of the root and may also contribute to the bulk of the crown in hypsodont (high

crowned) teeth. The periodontal ligament originates in the alveolar bone and inserts in the cementum, holding the tooth in place in the socket. The structure of hypsodont teeth will be compared to that of the brachydont.

Tooth development

In many animals, a temporary set of teeth develops first, and is then replaced by a permanent set. Teeth develop from oral epithelium (ectoderm) and neural crest-derived mesenchyme, which go on to form an enamel organ. The ectoderm gives rise to the enamel-forming cells, which remain on the outside of the tooth until they are worn off following eruption. The cementum and dentine are produced by mesenchyme-derived cells. The permanent tooth develops in the space vacated by the temporary tooth and exerts pressure on it, causing it to be shed. Brachydont teeth have a short period of growth and eruption and the root forms during eruption. Hypsodont teeth have a prolonged period of growth and eruption, and the root starts to develop at some period after eruption has commenced.

FURTHER READING

McGeady TA, Quinn PJ, Fitzpatrick ES, Ryan MT: *Veterinary Embryology.* (2006)

Singh B: *Dyce Sack and Wensing's Textbook of Veterinary Anatomy*, 5th edition (2018)

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LECTURE 3
COMPARATIVE DENTITION

LECTURER

DR CHRIS MURRAY

cmmurray@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the differences in dentition between domestic species.
- Relate tooth structure and arrangement to dietary and other functions.
- Explain how developmental and structural features of teeth are used for the ageing of animals and outline the factors that affect the accuracy of the different parameters used.

KEY WORDS

Teeth; carnivore; herbivore; omnivore; incisors; canines; premolars; molars; cheek teeth; dental pad; dental formula; diastema; brachydont; hypsodont; occlusion; infundibulum; dentition; ageing.

LECTURE OVERVIEW

Arrangement of teeth

The shape of teeth varies with the position in the oral cavity, and teeth are named according to their position in the oral cavity. In general, the most rostrally located teeth (incisors and canines) are used for cutting and tearing food, while the more caudally located teeth (premolars and molars) are used for grinding or crushing food.

Species differences

Different species have varying numbers of the different types of teeth. The teeth in carnivores are brachydont teeth, whereas the teeth in herbivores such as horses and ruminants are mainly hypsodont (high-crowned) teeth. Hypsodont teeth erupt continuously, and therefore the enamel extends below the gingival margin, and is surrounded by cementum, which wears off slowly after eruption. The enamel wears off the occlusal surface of the tooth soon after eruption, thus exposed dentine is visible on this surface.

Use of dentition for age estimation

The time of eruption and a variety of structural features of the different teeth in both the temporary and permanent sets of teeth can be used to estimate the age of animals, with levels of accuracy varying between species. The accuracy of age estimation also depends on the breed of animal, its individual genetics, whether there is malocclusion, and dietary, behavioural and environmental factors.

FURTHER READING

Easley J, Dixon, PM, Schumacher,J (eds): *Equine Dentistry*, 3rd ed, Edinburgh, Saunders (2011)

Singh B: *Dyce Sack and Wensing's Textbook of Veterinary Anatomy*, 5th edition (2018)

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LECTURE 4
PREHENSION AND TASTE

LECTURER

DR CHRIS MURRAY
cmmurray@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the structure and structural relationships of the oral cavity and pharynx in mammals.
- Describe the structure and actions of the lips, cheeks, tongue, palate, pharynx and temporomandibular joints, their associated muscles and their innervation, and relate these to their functions.
- Relate variations in oral and pharyngeal structure in different domestic species to functional requirements.

KEY WORDS

Oral cavity; vestibule; tongue; root, body, apex, frenulum, papillae, taste buds; prehension; mastication; swallowing; deglutition; palate; pharynx.

LECTURE OVERVIEW

The roof of the oral cavity is formed by the hard and soft palate, the walls are formed by the cheeks and lips, and the floor is formed by soft tissue extending between the two sides of the mandible. The tongue rests on the floor of the oral cavity. All of these structures, as well as the dental arcades, function in concert to bring food into the oral cavity, to prepare it for swallowing and to participate in the first stages of swallowing.

The hard palate is constructed from bone covered by mucosa. The lips surround the opening of the oral cavity and are continuous with the cheeks caudally. Both of these structures are composed of three layers, the outer layer being skin, the middle layer containing skeletal muscle, tendon and glands, and the layer facing the oral cavity consisting of mucosa.

The tongue is composed primarily of skeletal muscle with glands and adipose tissue scattered among the muscle bundles. The dorsal surface is covered in papillae, which assist in directing food towards the pharynx, and some of which contain taste buds, the structures responsible for the sense of taste.

The pharynx is the passage caudal to the oral and nasal cavities that is common to both the digestive and respiratory tracts. The skeletal muscles in its walls act in a coordinated manner to propel food from the oral cavity into the oesophagus.

FURTHER READING

Eurell, JA & Frappier BL *Dellmann's Textbook of Veterinary Histology*, 6th Ed. (2006)

Singh B, *Dyce Sack and Wensing's Textbook of Veterinary Anatomy*, 5th Ed. (2018)

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LECTURE 5
FROM MOUTH TO STOMACH

LECTURER

PROFESSOR SIMON BAILEY

After time spent in mixed veterinary practice in the UK, Simon undertook a PhD at the Royal Veterinary College, London. He then continued in research at the RVC, working in the fields of equine laminitis and inflammation. Simon then worked as a research scientist at the Heart and Lung Research Institute at the Ohio State University Medical Center, Columbus, Ohio. He then returned to the Royal Veterinary College as a lecturer and moved to the University of Melbourne in 2007. He is currently Professor of Pre-clinical Veterinary Sciences, and conducts research on inflammatory diseases, pharmacology and endocrinology in various species including horses.

bais@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Utilise your knowledge of the structure and location of the salivary glands in different species, in order to relate this to differences in diet and feeding.
- Explain the process of bolus formation and swallowing, in order to relate the structural features of the tongue and pharynx to their function.
- Apply your knowledge of the structure of the oesophagus and its relationships to other anatomical structures, in order to understand the potential physical and functional mechanisms by which obstruction may occur.

KEY WORDS

Oral cavity; salivary glands; oesophagus; mediastinum; neck; smooth muscle; skeletal muscle.

LECTURE OVERVIEW

When food is introduced into the oral cavity, it is moistened with the secretions of the salivary glands. The major salivary glands in most domestic species are the parotid, mandibular and sublingual glands; the carnivores have an additional salivary gland, the zygomatic gland. Most of these glands convey their secretions to the oral cavity through a single primary duct. The secretory cells within salivary glands are

arranged in acini, and can be either mucous-secreting or serous-secreting cells. The acini release their contents into a branching duct system, which conveys the secretions to the primary duct.

In preparation for swallowing (deglutition), the moistened bolus of food is shaped by the movement of the tongue, then moved into the pharynx where it initiates a series of reflex muscle actions, causing the pharynx to move rostrally to engulf the bolus and push it into the oesophagus; here, peristaltic contractions are initiated, resulting in the bolus being conveyed to the stomach.

The oesophagus is a muscular tube that runs from the pharynx to the stomach. It is found in close proximity to the trachea within the neck, and within the mediastinum within the thorax; it penetrates the diaphragm before joining the stomach within the abdomen. The oesophagus is lined with a mucosa, which includes a muscle layer and is surrounded by the submucosa, which contains glands. An external muscle layer surrounds the submucosa; this layer is composed of skeletal muscle at the cranial end, and in most species changes to smooth muscle at the caudal end. A connective tissue layer surrounds the muscle layer.

FURTHER READING

Dyce, KM & Wensing, CJG (2010) *Textbook of Veterinary Anatomy*, 4th ed, St Louis, Saunders/Elsevier

Eurell, JA & Frappier BL (2006) *Dellmann's Textbook of Veterinary Histology*, 6th ed, Ames: Blackwell

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LECTURE 6
THE COMPOUND STOMACH OF RUMINANTS

LECTURER

PROFESSOR SIMON BAILEY

bais@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Apply an understanding of the position of the forestomachs within the abdominal cavity, in order to integrate the structural features of the different chambers with their function.
- Utilise your knowledge of the anatomy of the ruminant forestomachs in order to determine the nature of displacements or obstructions when they occur.
- Apply an understanding of the relationship of the forestomachs with other abdominal organs and structures, in order to relate this to the potential for the development of disease in neighbouring structures.

KEY WORDS

Stomach; reticulum; rumen; omasum; abomasum; digestion.

LECTURE OVERVIEW

The compound stomach functions to act as a fermentation chamber where fodder (composed of complex carbohydrates) is broken down into useable metabolites by symbiotic micro-organisms. It has four compartments, the relative sizes of which vary with age, in correlation with the nature of the food ingested. It occupies almost three quarters of the abdominal cavity and fills almost the entire left half of the abdominal cavity in the adult animal, extending from diaphragm to pelvic inlet and extending considerably over the median plane into the right half.

It has four compartments which are grouped into the fore-stomach (or proventriculus) which is non-glandular, and consists of the **rumen**, **reticulum** and **omasum**; and the **abomasum** which is glandular.

At birth, the abomasum is the largest compartment, but in the adult, the rumen is the largest. In cattle, the reticulum is the smallest compartment and Lies on the left side cranial to the rumen. The omasum lies on the right side of the rumen and reticulum, and in sheep, this is the smallest compartment. The abomasums is somewhat elongated and lies on the abdominal floor.

Reticulum

The reticulum (or ‘honeycomb’) is the most cranial compartment and lies on the left side. It serves as a sieve for coarse material, also absorbs fatty acids, water, salts, etc. It passes coarse material back to the rumen and fine material to the omasum by very forceful contractions.

Rumen

The rumen fills the left half of the abdominal cavity and contributes ~80% of total stomach capacity. It is divided into several compartments (sacs) by thickened pillars, corresponding to grooves on the outside. Ruminal contractions mix the ingesta and furthers the process of microbial fermentation releasing volatile fatty acids and gases. The lining of the rumen is also adapted to absorb volatile fatty acids, sodium and water. Papillae increase the surface area for absorption and assist mixing of ingesta.

Omasum

The omasum (the ‘butcher's bible’) contributes ~ 7-8% of total stomach capacity in the cow (smallest compartment in the sheep and goat), and lies mainly on the right of the midline. It lies between the rumen and reticulum on its left and the liver and body wall on its right. It is spherical to ellipsoidal in shape, but somewhat compressed, having greater and lesser curvatures. The omasum serves as a two-stage pump for transfer of ingesta from the reticulum to the abomasum and as a sieve for quality control separation. It also absorbs fatty acids, water, salts.

Abomasum

The abomasum is the 'true stomach', equivalent to the simple stomach of carnivores. It is rich in glands that produce digestive enzymes and HCl for digestion and mucus for protection. It is a somewhat elongated sac which chiefly lies on the abdominal floor - insinuated between the ventral sac of the rumen and the reticulum; the caudal end is flexed around the lower pole of the omasum. The abomasum also contributes ~ 7-8 % of total stomach capacity in the cow.

FURTHER READING

Ashdown and Done: *Color Atlas of Veterinary Anatomy*: The Ruminants, 1984.

Eurell JA, Frappier BL. *Dellman’s Textbook of veterinary histology*. 6th Edn. 2006.

McCracken TO, Kainer RA, Spurgeon TL. *Spurgeon’s color atlas of large animal anatomy*, 2006.

Dyce, Sack and Wensing: *Textbook of Veterinary Anatomy*, 3rd edition, 2002.

Getty: *Sisson and Grossman's The Anatomy of the Domestic Animals*, Volumes I and II, 5th edition, 1975.

Smallwood: *A Guided Tour of Veterinary Anatomy*, 1992.

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LECTURE 7
THE SIMPLE STOMACH AND ABOMASUM

LECTURER

PROFESSOR SIMON BAILEY

bais@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Utilise your knowledge of the anatomy of the stomach in order to integrate its structural features with their function.
- Apply an understanding of the position of the stomach within the abdominal cavity, in order to determine the nature of displacements or obstructions when they occur.
- Apply an understanding of the relationship of the stomach with other abdominal organs and structures, in order to relate this to the potential for their involvement in disease or displacement.
- Utilise your knowledge of species differences in stomach anatomy in order to integrate these structural differences with differences in digestive function.

KEY WORDS

Simple stomach; abomasum; fundus; body; pylorus; glandular regions; saccus caecus; comparative anatomy.

LECTURE OVERVIEW

The simple stomach is the dilated part of the alimentary canal in which the processes of digestion are initiated. The structure of the stomach is determined by the feeding habits of the species: Horses, pigs and carnivores have a simple stomach, while ruminants have a compound stomach – the abomasum has a similar glandular structure and fulfils a similar role to the simple stomach.

The stomach is a musculo-glandular organ interposed between the oesophagus and the small intestine. It consists of two distinct parts that converge and join at a ventral angle forming a C- or J-shape.

The cardiac, fundic and body regions form the larger part - into which the oesophagus opens. This part of the stomach lies mainly to the left of the median plane, well forward under cover of the ribs and is in direct contact with the liver and diaphragm. It is relatively distensible.

The pyloric region is the second part which is narrower, has thicker walls and is more constant in appearance. It passes to the right to continue into the duodenum at the pylorus.

The stomach has two surfaces - both variably convex. The cranial or parietal surface is mainly in contact with the liver and diaphragm, and the caudal or visceral surface is in contact with the intestinal mass, the left kidney, the pancreas and the greater omentum.

The stomach also has two curvatures (the borders between the two surfaces). Both run between the cardiac and pyloric openings. The lesser curvature is very short and sharply concave and is connected with the liver by the lesser omentum. The greater curvature is very long and convex and gives attachment to the greater omentum and the gastrosplenic ligament which attaches the spleen with the stomach. The two extremities of the stomach are the cardia (the oesophageal orifice into the stomach) and the pylorus (the opening of the stomach into the intestine).

The stomach wall

As with most other parts of the gastrointestinal tract, there are 4 main layers in the wall of the stomach:

The **mucosa** forms gastric pits -lined by mucus secreting simple columnar surface epithelium. The function of the mucus is to protect the gastric mucosa. Tubular glands are present in the laminar propria. These extend to the muscularis mucosa and open into the base of the gastric pits.

The **submucosa** contains no glands, but is rich in blood vessels and nerves, and contains mainly collagen fibres.

The **muscularis externa** is very thick and churning of the ingesta enables further mechanical breakdown. It has 3 incomplete layers of smooth muscle: the inner layer which is oblique; the middle circular layer (which helps form the thick ring of the pyloric sphincter and the weaker ring of the cardiac sphincter) and the thinner outer longitudinal layer.

The outer **serosa** consists of a serous membrane (simple squamous epithelium) which reduces friction, overlying connective tissue conducting nerves and blood vessels.

Glandular regions within the stomach

Within the stomach there are different zones or regions, based on the distribution of the types of glands in the walls. In the dog (and human) these glandular regions correspond to the major parts of the stomach, i.e. cardiac glands in the cardia, fundic glands in the fundus and body, and pyloric glands in the pylorus. However, in the other species, the glandular regions to not correlate with the gross anatomical regions of the stomach, which can be confusing.

The true fundic glands secrete HCl and pepsin, as well as mucus, and this glandular region is generally the largest. The cardiac and pyloric regions are mainly mucus secreting.

Comparative aspects

In the dog, the stomach is C-shaped and is relatively large (2.25 litres in an average sized dog). When empty the stomach does not contact the abdominal wall and lies cranial to the last rib, but when distended it may extend caudally quite considerably. There are three distinct regions to the mucosa. Cardiac glands are found in a very narrow pale zone around the cardiac opening and scattered along the lesser curvature. The fundic gland region has a thick reddish-brown mucosa which lines about 2/3 of the stomach and has prominent longitudinally orientated rugae. The pyloric mucosa is thin and pale. The greater omentum is very extensive and may contain much fat. When the abdomen is opened ventrally it covers the entire intestinal mass, extending from the greater curvature of the stomach to the pelvic inlet.

In the horse, the stomach is relatively small (5-15 litres) and has a sharply curved J-shape. The fundus is extensive, forming a large non-glandular sac (the saccus caecus). Internally, the saccus caecus is separated from the rest of the stomach by a mucosal fold-the margo plicatus, which marks the boundary between the non-glandular and glandular regions of the gastric mucosa.

The pig has a relatively large stomach (approximately 9 litres), with an irregular J-shape. It has a large region of cardiac glands in the fundus.

Ruminants have a complex stomach that was covered in a separate lecture.

Birds have two distinct parts to the stomach, separated by a constriction (the isthmus). The first part is the proventriculus, a small cranial glandular stomach which is elongated and spindle-shaped. Ducts of the mucosal glands open on visible papilla which project into the lumen. The second part is the Gizzard (Ventriculus) which is the large caudal muscular stomach.

FURTHER READING

Dyce, Sack and Wensing: *Textbook of Veterinary Anatomy* 3rd edition 2002.

Boyd: *Color Atlas of Clinical Anatomy of the Dog and Cat* 2nd edition 2001.

Eurell JA, Frappier BL. *Dellman's Textbook of veterinary histology.* 6th Edn. 2006.

Constantinescu: *Clinical Dissection Guide for Large Animals*, 1991.

Getty: *Sisson and Grossman's The Anatomy of the Domestic Animals*, Volumes I and II, 5th edition, 1975.

Miller, Christensen and Evans: *Anatomy of the Dog* 1964.

Smallwood: *A Guided Tour of Veterinary Anatomy*, 1992.

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LECTURE 8
CLINICAL EXAMINATION OF THE GASTROINTESTINAL TRACT

LECTURER

DR NICHOLAS BAMFORD
n.bamford@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Take a thorough history to establish the likelihood of digestive system dysfunction in animals.
- Differentiate normal and abnormal digestive system function based on physical examination findings.
- Apply an understanding of the physiology of the digestive system to localise disease process.
- Describe some of the diagnostic aids that will enable further investigation of the digestive system.

KEY WORDS

Clinical examination; clinical investigation; history taking; auscultation; palpation; endoscopy.

LECTURE OVERVIEW

This lecture will introduce the principles of how a veterinarian can assess the digestive system in animals, when in reality the entire system remains hidden and difficult to evaluate.

Issues with the digestive system can present as an emergency - for example, gastric dilation-volvulus (GDV) in a dog or ruminal tympany ('bloat') in a cow. In those situations, triage and stabilisation will occur before history taking, and diagnostics are generally done to assist in prognostication rather than diagnosis. However, in less acute cases, taking a detailed history will save time in the long run. A holistic (whole animal/whole herd) approach should be taken, since many different factors and systems may be interrelated. A thorough history should include questions about appetite; how well the animal can eat; diet (full); any episodes of vomiting/description of the vomiting and how long after eating it may occur; nature of the faeces; precipitating events; weight loss and many other questions.

The digestive system is always assessed during a routine clinical examination of any animal. This includes examining the mouth, the throat, and palpating the abdomen for any signs of pain, bloating, etc. A stethoscope can be used (auscultation) over the abdomen to listen for sounds associated with intestinal motility (particularly in large animals). A knowledge of anatomy helps to identify structures that are being palpated or auscultated.

More detailed investigations may then be carried out if a problem with this system is suspected. This can include endoscopy and other types of imaging such as radiography and ultrasound (imaging will be covered in another lecture). Examination of the faeces can provide a lot of information but is often overperformed in small animal practice. Blood samples can also be useful to rule out non-GI disease, and if necessary, tissue samples (biopsies) can be obtained from different parts of the gastrointestinal tract.

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LECTURE 9
CLINICAL IMAGING OF THE GASTROINTESTINAL TRACT

LECTURER

DR MARJORIE MILNE

Dr Milne will not be available this semester to answer questions directly, but please post any questions on this topic to the subject Discussion Board, and they will be answered.

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- List the different methods available for imaging the gastrointestinal tract.
- Identify the abdominal organs on a lateral and ventrodorsal radiograph of the abdomen of a dog.
- Recall the radiographic signs of gastrointestinal obstruction.

KEY WORDS

Clinical investigation; imaging; radiography; radiology; ultrasonography; ileus; obstruction.

LECTURE OVERVIEW

This seminar will provide an introduction to the different technologies available for imaging the internal organs of the body, including those of the gastrointestinal tract. Each different method provides different information and levels of detail which can be useful when trying to diagnose or assess a particular disease or condition. Understanding some of the principles of how these techniques work will help in the understanding and interpretation of the images that they provide.

The normal radiographic anatomy of the abdomen of the dog is discussed followed by some principles of interpretation of the radiographic appearance of the gastrointestinal tract. This is followed by a number of case examples of gastrointestinal obstruction to illustrate these principles.

OPTIONAL FURTHER READING

Thrall, D.E. (ed), (2017). *Textbook of Veterinary Diagnostic Radiology*. 7th edition Elsevier.

Chapter 46 – Stomach; Chapter 47 – Small Bowel; Chapter 48 – Large Bowel

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LECTURE 10
DISEASES OF THE ORAL CAVITY

LECTURER

DR SMITHA GEORGY

Dr Smitha Georgy graduated from Kerala Agricultural University, India and proceeded to work in mixed animal practice under Government. She moved to Australia in 2006 and undertook PhD from The University of Melbourne. Smitha then worked as a research scientist in the Department of Medicine, Monash University. In 2015, she completed the National Veterinary Examination and started small animal practice in Melbourne. Smitha is currently Lecturer in Veterinary Pathology and a Diplomate of the American College of Veterinary Pathologists. Her research interests include epithelial cancers affecting skin, oral cavity and oesophagus.

s.georgy@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Analyse the developmental anomalies of the oral cavity and its consequences.
- Classify the inflammatory lesions in the oral cavity of domestic animals based on aetiology and pathogenesis.
- Identify and differentiate the proliferative lesions of the oral cavity of domestic animals.

KEY WORDS

Palatoschisis, brachygnathia, stomatitis, vesicle, papule, erosion, ulcer, glossitis, epulis.

LECTURE OVERVIEW

In this lecture, we will overview various diseases of the upper gastrointestinal tract focusing on the oral cavity. The protective mechanisms of the oral cavity and basic reactions of the gastrointestinal tract to injury will be reviewed. Developmental anomalies affecting palate, lips, mandible/maxilla as well as inflammatory and neoplastic conditions of the oral cavity will be examined in detail. Oral inflammatory lesions are classified into vesicular, erosive and ulcerative, papular, idiopathic, and deep stomatitis. The etiology and pathogenesis of these conditions are reviewed in detail. The common proliferative conditions of the oral

cavity including gingival hyperplasia, epulis, papillomatosis, squamous cell carcinoma, melanoma, and fibrosarcoma are examined in detail in this lecture.

FURTHER READING

McGavin *General Veterinary Pathology*, 2nd edition, Chapter 7, p 301-392.

Jubb, Kennedy and Palmer, *Pathology of domestic animals*, 5th edition, Ed M Grant Maxie, volume 2, chapter 1, p 1-296.

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LECTURE 11
DISEASES OF THE OESOPHAGUS

LECTURER

DR SMITHA GEORGY

s.georgy@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Analyse the pathogenesis and consequence of megaoesophgus, 'choke' and oesophagitis.
- Identify the common parasitic and neoplastic diseases affecting the oesophagus of domestic animals.
- Identify some important causes of upper gastrointestinal diseases.

KEY WORDS

Oesophagitis, megaesophagus, choke, Sarcocystis, Spirocerca, leiomyoma.

LECTURE OVERVIEW

In this lecture, we will overview various diseases of the upper gastrointestinal tract focusing on the oesophagus. Pathology of tonsils and salivary glands are briefly reviewed. Some of the inflammatory lesions in the oesophagus are caused by extension of the inflammation from the oral cavity. Congenital and acquired megaoesophagus will be reviewed in detail in this seminar. Other important and common conditions affecting the oesophagus of domestic animals include choke, oesophageal rupture, reflux oesophagitis, parasitic infections and neoplasia.

FURTHER READING

McGavin *General Veterinary Pathology*, 2nd edition, Chapter 7, p 301-392.

Jubb, Kennedy and Palmer, *Pathology of domestic animals*, 5th edition, Ed M Grant Maxie, volume 2, chapter 1, p 1-296.

Veterinary Bioscience: Digestive System

THEME 3: MOTILITY

Week 6

The theme for this week is *motility*. We will consider in detail the patterns of motility seen in the gastrointestinal tract, and the mechanisms that control motility in the monogastric and ruminant stomachs at a cellular, organ and whole-body level. Abnormal patterns of motility (vomiting and regurgitation) will also be considered, as well as classes of drugs used to modify motility and prevent vomiting. Studies of the intestines of the dog and cat will culminate in an abdominal dissection class in which students will have the opportunity to explore the abdominal cavity of a dog or cat cadaver with the gastrointestinal tract in situ.

LECTURES

Gastrointestinal Motility	• Special features of gut smooth muscle • The hierarchy of control in gastrointestinal smooth muscle • Contractile patterns in gastric and intestinal motility • Pharmacological modification of gastrointestinal motility
Control of Motility in the Ruminant Forestomach and Salivary Secretion	• Rumen function, patterns of motility: eructation and rumination • Function of the oesophageal groove • Control of rumen, reticulum and omasum motility • Ruminant salivary secretion and fluid balance
Vomiting Reflex and Antiemetic Therapy	• Mechanics of vomiting; control of the vomiting reflex • Drugs that stimulate vomiting; classes of drugs that inhibit vomiting • Drugs that modify gastrointestinal motility
Intestines of the Dog and Cat	• Structure of the small and large intestines • Vascular supply to the intestines • Neural supply to the intestines

PRACTICAL CLASSES AND CASE STUDIES

Sheep Head Dissection	Structures of the cheek and mouth, including tongue and salivary glands	WEBS Dissection Lab (B104)
Histology of the Upper GI Tract	Microscopic anatomy of the mouth, oesophagus and rumen	Online
Case Study: Jack's Night Out	Application of knowledge to clinical case	WEBS Collaborative Learning Centre (G04)

LEARNING OUTCOMES

	**SCIENTIFIC BASIS OF CLINICAL PRACTICE** • Explain the mechanisms by which digestive motility and secretion are controlled and coordinated in different domestic animal species. • Predict the outcome of alterations in gut motility or secretion for digestive function and wellbeing of the animal. • Explain how different classes of drugs can be used to modify gastrointestinal motility or to alleviate vomiting. • Describe the microscopic structure of the upper alimentary tract and relate this to function.
	**CLINICAL SKILL ACQUISITION** • Use dissection skills to identify structures associated with the cheek and mouth.
	**PERSONAL AND PROFESSIONAL DEVELOPMENT** • Engage in a collaborative approach to solving clinical problems.

Veterinary Bioscience: Digestive System

LECTURE 12
GASTROINTESTINAL MOTILITY

LECTURER

PROFESSOR ELIZABETH TUDOR

Liz Tudor is Professor in Veterinary Biosciences. Prof. Tudor completed a PhD in the Faculty of Medicine at Monash University. She has worked in private small animal practice in Melbourne. Liz was for ten years the Associate Dean Curriculum in FVAS, and in this role oversaw the development and delivery of the Melbourne DVM. She is deeply committed to Aboriginal reconciliation, and for the past 16 years has led dog health programs in remote Aboriginal communities in Arnhem Land in the Northern Territory, Australia.

etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Explain the features of gastrointestinal smooth muscle that provide for its autonomous activity and describe the hierarchy of neural and hormonal pathways that control secretory and motor activity in the gastrointestinal tract.
- Describe the contractile patterns characteristic of gastric motility and explain their purpose, and explain how the structural and functional properties of gastric smooth muscle permit changes in volume with minimal changes in intra-luminal pressure.
- Explain the mechanisms responsible for gastric emptying, the complex mechanisms that control this process and describe the characteristic motility forms of the small intestine.
- Explain how different classes of drugs, including pro kinetics, laxatives and antispasmodics, can be used to either increase or decrease gut motility.

KEY WORDS

Chemoreceptor, mechanoreceptor, intrinsic nerves, myenteric plexus, submucosal plexus, extrinsic nerves, autonomic nervous system, vagus nerve, sympathetic nervous system, parasympathetic nervous system, enterochromaffin cell, enterogastrone, cardiac sphincter, gastric cardia, gastric fundus, pylorus, pyloric antrum, pyloric sphincter, gastric filling, receptive relaxation, gastric emptying, chyme, gastrin, secretin, cholecystokinin (CCK), gastric inhibitory peptide (GIP), hydrochloric acid, entero-gastric reflex, prokinetics, anti spasmodics, metaclopramide, cisapride.

LECTURE OVERVIEW

The digestive system performs four basic functions- motility, secretion, digestion and absorption of ingested food. Of these secretion and motility exhibit the highest degree of control.

Motility of the gut is provided for by layers of smooth muscle- circular and longitudinal, along the length of the gut. The sequenced contraction and relaxation of these muscle layers allow for mixing of ingested food with secretions, mechanical breakdown of food particles, propulsion of food along the gut tube and periodic release of chyme through sphincters from one region of the gut to the next. This lecture will describe the range of motility forms characteristic of gut smooth muscle and introduce you to the control mechanisms that modulate motility, particularly in the stomach.

FURTHER READING

Sherwood, L. *Human Physiology from Cells to Systems* 8th Edition 2013 Ch 16

Cunningham's *Textbook of Veterinary Physiology* 6th Ed Elsevier 2020 Ch 27 & 28

Sjaastad OV, Sand O & Hove K *Physiology of Domestic Animals* Scandinavian Veterinary Press 2010 Ch 15

Berne RM, Levy MN, Koeppen BM & Stanton BA *Physiology*. (ebook) 6th Edn 2010 Section Six

Veterinary Bioscience: Digestive System

LECTURE 13
RUMINANT PHYSIOLOGY

LECTURER

DR CHRISTINA MARTH

Christina is a Lecturer in Veterinary Biosciences in the Melbourne Veterinary School at The University of Melbourne. Christina completed a PhD exploring how innate immune factors influence the way horses respond to breeding and the ability of healthy horses to clear all traces of inflammation from their uterus efficiently. Prior to coming to the University of Melbourne, Christina has completed a veterinary degree at the University of Veterinary Medicine in Hannover, Germany. Her research interests are in reproductive physiology, immunology and microbiology and their impact on fertility in horses, cattle and dogs.

christina.marth@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Discuss the main functions of the forestomachs in ruminants and how they relate to their anatomical design.
- Explain the main patterns of motility of the reticulo-rumen in order to interpret their respective functions and control mechanisms.
- Explain the anatomy of the gastric groove in order to relate it to function in juvenile animals.
- Compare the ruminant salivary gland physiology to that of monogastric animals in order to evaluate the main differences and their relevance for rumen function and water balance.

KEY WORDS

Rumen fermentation and absorption, rumen motility patterns, rumen motility control, smooth muscle, rumination, eructation, gastric groove, vagus nerve, salivary glands, water balance.

LECTURE OVERVIEW

The digestive system of ruminants is much more complicated than that of monogastric animals. For effective fermentation of ingesta by the microbiota residing in the reticulo-rumen, this environment needs to be carefully controlled regarding acid-base homeostasis and motility patterns.

Some of the main functions of the ruminant forestomachs are:

- To serve as a fermentation chamber for microbes.
- To mix and separate ingesta appropriately.
- To allow built-up gas to escape.
- To buffer the rumen content to maintain an optimal pH for the microbes while conserving water and electrolytes.

FURTHER READING

Reece. *Dukes' Physiology of Domestic Animals*. 13th edition (2015). Wiley Blackwell Publishing.

Cunningham and Klein. *Textbook of Veterinary Physiology*. (2007) 4th edition. Saunders.

The ruminant animal digestive physiology and nutrition. Church DC Ed. (1988). Eaglewood Cliffs, Prentice Hall.

Veterinary Bioscience: Digestive System

LECTURE 14
THE VOMITING REFLEX AND APPROACH TO ANTIEMETIC THERAPY

LECTURER

PROFESSOR LIZ TUDOR
etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the sequence of events in the vomiting reflex, the nerve pathways, neurotransmitters and receptors involved in the vomiting reflex.
- Explain how vomiting differs from regurgitation.
- Describe the mechanism of action of classes of peripherally acting and centrally acting emetics, in particular dopamine antagonists, neurokinin-1 antagonists and 5HT3 antagonists, antihistamines, phenothiazines and anticholinergics and explain how understanding of their mechanism of action and the cause of vomiting can assist in rational selection of an appropriate anti-emetic.

KEY WORDS

Vomiting centre, vomitus, brain stem, chemoreceptor trigger zone, CTZ, vestibular apparatus, semi-circular canals, emetic, anti-emetic, apomorphine, metoclopramide, cisapride, phenothiazine, anti histamine, H1 receptor, dopamine receptor antagonist, neurokinin-1 receptor antagonist, 5HT3 antagonist, acepromazine, maropitant, ondansetron, prokinetic, anti-spasmodic.

LECTURE OVERVIEW

The mechanics of vomiting

Vomiting is a complex reflex event, coordinated by the vomiting centre in the brain stem (medulla). It is not reverse peristalsis; the stomach, oesophagus, gastro-oesophageal sphincter and pyloric sphincter are all relaxed. Not all mammals can vomit: dogs and cats vomit readily.

Once stimulated, the vomiting reflex involves a predictable sequence of mechanical events. Preceding vomiting there is salivation, increased heart rate, and pallor. Vomiting commences with deep inspiration and closure of glottis, that is followed by contraction of the diaphragm and abdominal muscles leading to

increased intra-abdominal pressure. With concomitant relaxation of the stomach and its sphincters, gastric content is forced into the oesophagus. Increased pressure leads to relaxation of the pharyngo-oesophageal sphincter and passage of vomitus into the mouth.

Regurgitation is different from vomiting

Regurgitation involves a reverse peristaltic wave in the oesophagus. It generally occurs shortly after eating and is not preceded by salivation, increased HR and so on. In contrast to vomitus, regurgitated material is generally undigested or poorly digested.

Control mechanisms in the vomiting reflex

There are many and diverse stimuli to the vomiting reflex- unpleasant sights and smells, processed at the level of the cerebral cortex may stimulate vomiting. Abnormal motion, sensed by the semicircular canals of the vestibular apparatus, as well as a range of stimuli from visceral organs- distension, inflammation, intense pain, can all stimulate vomiting. In addition, blood borne toxins and chemicals can directly stimulate vomiting.

The vomiting centre in the brain stem (medulla) receives afferent input from cortical, visceral and vestibular receptors. In addition, in close proximity to and in communication with the vomiting centre in the medulla is a discrete area known as the chemoreceptor trigger zone (CTZ). In this region, the normally tight blood-brain barrier is more permeable, allowing contact between blood borne toxins and neurons. Blood borne toxins thereby stimulate vomiting via activation of the CTZ and hence the vomiting centre.

A number of different neurotransmitters are involved in the different elements of the vomiting reflex. In the vomiting centre, the predominant neuroreceptors are for acetylcholine, neurokinin1 and 5 hydroxytryptamine (5HT), in the vestibular apparatus for histamine. In the CTZ dopamine, 5HT and neurokinin 1 receptors are important and, in the periphery, 5HT and acetylcholine.

Why is this important? Because when the neurotransmitters and their receptors in the vomiting pathway are identified, it is possible to develop drugs that target and block these receptors, thereby blocking the vomiting reflex.

Drugs that stimulate vomiting - centrally and peripherally acting emetics

Sometimes veterinarians actually want to stimulate vomiting- for example when the stomach needs to be emptied of toxic or irritant ingesta. Two types of drugs are used for this purpose- peripherally acting compounds, such as washing soda, that stimulate the vomiting reflex via receptors and afferent pathways from the gastric mucosa, and centrally acting compounds such as apomorphine, that is absorbed from the stomach to the bloodstream, and stimulates the CTZ and hence the vomiting centre.

Classes of drugs that inhibit vomiting - anti-emetics

A class of drugs is a group of drugs that shares a common mechanism of action. Because there are multiple causes (stimuli) for vomiting, involving various neural pathways and neurotransmitters and receptors, a number of different classes of drugs are used to treat vomiting. It is important to recognize too, that when anti-emetics are used, they are generally being used to alleviate a sign of disease - not the disease itself. (for example, if vomiting is due to increased blood urea levels as a consequence of kidney disease, the anti-emetic has no effect on blood urea levels or on the progress of the renal disease. The following are the classes of anti-emetics most commonly used in veterinary medicine; a prototypical drug in each class has been included.

Neurokinin-1 receptor antagonists for example maropitant are the newest class of veterinary anti-emetic. These drugs target NK1 receptors in both the CTZ and the vomiting centre and are effective antiemetics.

Dopamine receptor antagonists for example metoclopramide, domperidone. These drugs act by antagonizing dopamine at the CTZ. Metoclopramide also promotes the release of and increases the sensitivity of visceral smooth muscle to acetyl choline, which results in: increased tone of the lower oesophageal sphincter, increased strength of oesophageal contraction, increased gastric antral contraction, relaxation of pylorus, increased smooth muscle contraction in the duodenum. Often nausea and vomiting are related to delayed gastric emptying. These actions increase gastric emptying and small intestinal activity without altering gastric or intestinal secretion or absorption. Significant side effects relate to dopamine's other roles as a CNS neurotransmitter, and range from hyperactivity to depression, disorientation and frenzy.

5HT3 antagonists for example ondansetron (three times daily); dolasetron (once daily). These are very effective antiemetics, that have been used extensively in cancer chemotherapy in humans. They are thought to have a central action on the CTZ, probably also act through antagonism of peripheral 5HT-3 receptors.

Anti-histamines for example promethazine. These are effective in treatment of vomiting associated with middle ear infections or motion sickness (there are histamine receptors in semicircular canals). Their most significant side effect is sedation (which is not necessarily a bad thing for a car sick puppy!)

Phenothiazines for example prochlorperazine. We will meet this class of drugs again as pre-anaesthetic sedatives. These drugs act at multiple receptors, blocking the CTZ at low doses, through dopamine receptors, and the vomiting centre at higher doses, through muscarinic acetylcholine (ACh) receptors. They also block peripheral dopamine receptors in the stomach. AS they have a number of side effects, there are better classes of drugs available.

Drugs that modify gastrointestinal motility

Prokinetics, that normalize gastrointestinal activity and antispasmodics that reduce activity are sometimes used in the treatment of vomiting. Drugs that are prokinetics include metoclopramide (discussed above) and cisapride, that increase levels of the parasympathetic neurotransmitter acetylcholine in the synaptic cleft, and myenteric plexus.

Most antispasmodics are antagonists of acetylcholine at parasympathetic receptors, for example hyoscine. Anticholinergics reduce gastrointestinal motility, which can act as stimulus to further vomiting. Overuse can result in atony and predispose to absorption of endotoxins through damaged mucosa. Whilst they are included in a number of formulations for treatment of vomiting, their use is not recommended.

FURTHER READING

Sherwood, L. *Human Physiology from Cells to Systems* 8th Edition 2013 Ch 16

Cunningham's *Textbook of Veterinary Physiology* 6th Ed Elsevier 2020 Ch 27 & 28

Maddison J, Page S and Church D *Small Animal Clinical Pharmacology* 2nd Ed 2008

Riviere JE, & Papich MG *Veterinary Pharmacology & Therapeutics* 10th Edn 2018 Wiley Blackwell (ebook)

section 46

Veterinary Bioscience: Digestive System

LECTURE 15
INTESTINES OF THE DOG AND CAT

LECTURER

DR NICHOLAS BAMFORD
n.bamford@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the gross anatomy of the small and large intestines of the dog and cat, including their course and position in the body and relationships to other organs.
- Describe the gross anatomy of the anal canal and the clinical importance of the anal sacs.
- Describe the gross anatomy of the pancreas of the dog and cat.

KEY WORDS

Intestines; duodenum; jejunum; ileum; caecum; colon; rectum; anus; anal sphincter; anal sacs; mesenteric arteries.

LECTURE OVERVIEW

This lecture will examine the gross anatomy of the small and large intestines of the dog and cat. The different segments that will be discussed include the duodenum, jejunum, ileum, caecum, colon and rectum. Their course and position within the abdominal cavity and relationships to other organs, as well as their vascular and nervous supply will be outlined. The clinical importance of the anal sacs in these species will be discussed.

FURTHER READING

Singh: *Dyce, Sack & Wensing's Textbook of Veterinary Anatomy*, 5th Ed. Elsevier, 2018.

Boyd: *Color Atlas of Clinical Anatomy of the Dog and Cat*, 2nd Ed. Mosby, 2001.

Hermanson & de Lahunta: *Miller and Evans' Anatomy of the Dog*, 5th Ed. Elsevier, 2020.

Smallwood: A Guided Tour of Veterinary Anatomy, Saunders, 1992.

Veterinary Bioscience: Digestive System

THEME 4: SECRETION AND DIGESTION

Week 7 and Week 8

Studies in these two weeks begin by considering the mechanisms of gastric acid secretion. Hydrochloric acid secretion by gastric parietal cells is used to demonstrate the hierarchy of control of secretory activity. The importance of this understanding is highlighted by introduction to the mechanisms of action of classes of drugs that can be used to modify acid release. The exocrine secretory activity of the liver and pancreas are introduced, including the role of enterogastrones in the regulation of these secretions. Diseases of the simple and compound stomachs, exocrine pancreas and peritoneal cavity will be considered. Studies of the gross and histological anatomy of the gastrointestinal tract of the dog and cat will be reinforced through a dissection class in which students will have the opportunity to explore the abdominal cavity of a dog or cat cadaver with the gastrointestinal tract *in situ.*

LECTURES

Mechanisms of Gastric Acid Secretion and its Control	• Digestive secretions of the stomach • Gastric parietal cell and control mechanisms of gastric acid secretion • Drugs used to inhibit gastric acid secretion • Other anti-ulcer drugs
Pancreatic and Biliary Contributions to Digestion	• The role of the pancreas in digestion • Control of pancreatic secretion • Bile secretion and the biliary tree, control of bile secretion
Diseases of the Ruminant Forestomach	• Dietary adaptation of forestomachs • Primary and secondary bloat; vagus indigestion • Hardware disease; rumenitis; lactic acidosis; tumours
Diseases of the Stomach and Abomasum	• Pathology of the stomach • Gastritis; gastric ulceration
Diseases of the Exocrine Pancreas	• Pathology of the exocrine pancreas • Acute and chronic pancreatitis; exocrine pancreatic insufficiency
Diseases of the Peritoneal Cavity	• Drainage of peritoneal fluid • Mesothelial cell function; response of peritoneum to injury • Internal and external peritoneal hernias • Abnormal peritoneal contents; peritonitis

PRACTICAL CLASSES AND CASE STUDIES

Abdomen Dissection	Gastrointestinal tract *in situ*	WEBS Dissection Lab (B104)
Histology of the Stomach and Pancreas	Microscopic anatomy of the stomach and pancreas	Online
Case Study: Benji the Dog	Application of knowledge to clinical case	WEBS Collaborative Learning Centre (G04)

LEARNING OUTCOMES

	**SCIENTIFIC BASIS OF CLINICAL PRACTICE** • Explain the mechanisms by which gastric acid secretion is regulated in domestic animals and how different classes of drugs can be used to modify acid secretion. • Describe the movement of acid, enzymes, electrolytes and fluid into the lumen of the GI tract. • Describe different forms of gastrointestinal pathology in the domestic species. • Identify the different parts of the intestinal tract of dogs and cats.
	**CLINICAL SKILL ACQUISITION** • Perform a surgical approach to midline celiotomy in a dog or cat cadaver and correctly identify the structures encountered. • Describe the signs associated with exocrine pancreatic dysfunction.
	**PERSONAL AND PROFESSIONAL DEVELOPMENT** • Engage in a collaborative approach to solving clinical problems.

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LECTURE 16
MECHANISMS OF GASTRIC ACID SECRETION AND THEIR CONTROL

LECTURER

PROFESSOR LIZ TUDOR
etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the structure of the gastric mucosa, recognise the layers of the stomach wall and the cell types of the gastric gland in tissue specimens, and explain how the structure and ultrastructure of gastric mucosal cells relates to their function.
- List the secretory components of gastric juice and describe the cellular and biochemical mechanism of HCl secretion from gastric parietal cells, and of pepsinogen from chief cells.
- Describe the mechanisms that control gastric secretion during the cephalic, gastric and duodenal phases of gastric secretion.
- Describe the mechanism of action of different classes of drugs that modulate gastric acid secretion and explain the circumstances in which they may be used.

KEY WORDS

Gastric gland, gastric pit, surface mucous cell, mucous neck cell, chief cell, parietal cell, enterochromaffin cell, hydrochloric acid, pepsinogen, pepsin, secretory canaliculus, gastrin, histamine, acetylcholine, prostaglandin, vagus nerve, carbonic anhydrase, hydrochloric acid, proton pump, H/K ATPase, cephalic phase, gastric phase, intestinal phase, H2 receptor antagonist, proton pump inhibitor, cytoprotective drug, cimetidine, omeprazole, misoprostol, sucralfate, antacid.

LECTURE OVERVIEW

Digestive secretions of the stomach

Along with mechanical digestion that is the consequence of gastric motility, chemical digestion continues in the stomach as a result of secretions from gastric mucosal cells.

The stomach wall is comprised of the same tissue layers found throughout the gut tube - mucosa, submucosa, circular and longitudinal muscle layers and peritoneum. With the exception of the region around the cardiac sphincter of some species, the gastric mucosa is a single layer of cells thick, that is folded into glands that empty their secretions into gastric pits and from there into the lumen of the stomach. Secretion of digestive enzymes is from specialized cells located in the fundic region of the mucosa: pepsinogen secreted by chief cells, that is cleaved to the active protease pepsin in the presence of hydrochloric acid (HCl), HCl secreted from gastric parietal cells, and mucus released from both surface mucous and mucous neck cells. The structure of each of these cell types reflects its specialized function.

The gastric parietal cell: a cell specialised for secretion of HCl

The gastric parietal cell is a wonderful example of a cell specialized for its function- the secretion of HCl by active transport against a concentration gradient. The active transport mechanism- a H/K ATPase, is located membrane bound on the luminal border of the cell. The surface area of this luminal border is greatly expanded by a deep infolding of the surface to form an intracellular secretory canaliculus. During periods of active secretion this surface is further expanded by translocation of intra cellular tubules and vesicles to form microvilli on the canalicular border. The energy for active transport and also for membrane cycling is provided by large numbers of mitochondria in parietal cells. Generation of hydrogen ions in parietal cells is catalysed by the enzyme carbonic anhydrase. Chloride secretion is also active, by a separate membrane bound mechanism. Chloride ions enter the cell in exchange for bicarbonate ions generated in the carbonic anhydrase reaction.

Control mechanisms in HCl secretion

The secretion of HCl is a rate-limiting step in gastric digestion- without HCl pepsinogen remains inactive. As a consequence, understanding the factors controlling acid release is fundamental to managing gastric digestion, and in particular to managing medical situations such as gastric ulceration.

Control of acid release from parietal cells can be considered at a macro or at a cellular level. Acid release is observed in response to the sight or smell of a meal, and when food is chewed in the mouth even before swallowing. This is the so-called cephalic phase of secretion and involves extrinsic nerves. Acetylcholine released from vagal efferents binds to muscarinic receptors on the basal surface of parietal cells. Vagal efferents also stimulate the release of the enterogastrone gastrin from G cells in the pylorus, that circulates to the fundic region and binds to its receptor on parietal cells. Finally, vagal stimulation of enterochromaffin cells, stimulates local release of histamine that binds to H2 histamine receptors on parietal cells in close proximity, stimulating acid release.

Once food enters the stomach (in the gastric phase of secretion), mechano- and chemo- receptors (sensitive in particular to intra gastric protein levels) activate local intrinsic neural and neuro-endocrine pathways to further augment both acid and pepsinogen release. Binding of neurotransmitter or hormone to the parietal cell activates an intracellular pathway involving the second messenger cAMP. Acid secretion is inhibited by prostaglandins binding to cell surface PG receptors, that results in decreased intracellular cAMP levels.

Just as gastric motility is inhibited by the presence of increasingly fatty, acidic or hyperosmotic chyme in the duodenum, gastric secretion is similarly inhibited. This is the intestinal phase of secretion and involves local intrinsic (gastro-enteric) nerve reflexes, enterogastrones- secretin, CCK and GIP.

Anti-ulcer drugs: drugs used to inhibit gastric acid secretion

Understanding the cellular mechanism of acid secretion provides receptor targets for inhibiting acid release. Classes of drugs used to reduce acid secretion include:

Histamine (H2) receptor antagonists: for example, cimetidine, ranitidine. As the histamine receptor is the dominant receptor on parietal cells, these drugs can reduce acid release by up to 70%. Whilst they are

relatively free of side effects, they share metabolic pathways with several other classes of drugs, so drug interactions can be important.

Proton pump inhibitors: for example, omeprazole. These drugs are very potent inhibitors of acid release because they irreversibly bind and block the active transport protein. Their effect persists even after drug is detectable in the blood stream. They are used in the treatment of unresponsive ulcer.

Anti-ulcer drugs: cytoprotective drugs

Prostaglandin analogues: for example, misoprostol. These drugs mimic the effect of endogenous prostaglandins (PG's) to decrease acid release. In addition, PG's protect against gastric ulceration and aid ulcer repair by increasing mucosal blood flow, stimulating mucosal cell turnover, and stimulating mucus secretion. Their role in the maintenance of the gastric mucosa will be discussed further in the upcoming lecture on 'mucosal barrier'.

Sucralfate has sometimes been described as an ulcer bandaid- it dissociates in the gastric lumen to octosulfate, that forms a sticky sucrose gel, and aluminium hydroxide, that neutralizes gastric acid.

Anti-ulcer drugs: antacids

Act to neutralise hydrochloric acid, bind bile acids and decrease pepsin activity. They provide only symptomatic relief. Antacids generally contain AlOH, $CaCO_3$, $Mg(OH)_2$, $NaHCO_3$. Magnesium salts lead to increase bowel motility, aluminium salts to a decrease in bowel activity. As a consequence, antacids often include both Mg and Al salts. Antacids are used only in human medicine.

FURTHER READING

Sherwood, L. *Human Physiology from Cells to Systems* 8th Edition 2013 Ch 16

Cunningham's *Textbook of Veterinary Physiology* 6th Ed Elsevier 2020 Ch 27 & 28

Maddison J, Page S and Church D *Small Animal Clinical Pharmacology* 2nd Ed 2008

Riviere JE, & Papich MG *Veterinary Pharmacology & Therapeutics* 10th Edn 2018 Wiley Blackwell (ebook)

section 46

Rang and Dale's *Pharmacology* 9th edn Ch 31 (ebook)

Young B *Wheater's Functional Histology : A text and colour atlas* 4th Edn 2014 Churchill Livingstone (ebook)

Bacha WJ & Bacha LM *Color Atlas of Veterinary Histology* 2nd Edn 2012 Wiley-Blackwell

Veterinary Bioscience: Digestive System

LECTURE 17
PANCREATIC AND BILIARY CONTRIBUTIONS TO DIGESTION

LECTURER

PROFESSOR LIZ TUDOR
etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the histology of the pancreas and identify the exocrine and endocrine elements of the pancreas in tissue specimens prepared for light microscopy.
- Identify the exocrine glandular cells of the pancreas and explain how the structure and ultrastructure of the different cell types relates to their secretions.
- Describe the structure of the biliary tree, the secretory components of bile, and the path of bile from the hepatocyte to the intestinal lumen.
- Describe the cellular and biochemical mechanism of bicarbonate secretion from pancreatic ductal cells, and the mechanisms that control both binary and pancreatic secretions.

KEY WORDS

Acinar cell, Islet of Langerhans, ductal cell, hepatocyte, trypsinogen, chymotrypsinogen, procarboxypeptidase, pancreatic amylase, pancreatic lipase, bile salts, enterogastrone, vagus nerve, secretin, cholecystokinin, gastrin, bile salts, biliary tree, gall bladder, micelle, fat digestion, emulsification, bile salt dependent bile flow, enterohepatic circulation, bile canaliculus, auto-digestion, steatorrhea.

LECTURE OVERVIEW

Chyme that enters the duodenum is subjected to further chemical digestion due to the activity of secretions of the pancreas, the liver and intestinal mucosal cells.

The role of the pancreas in digestion

The pancreas functions both as an exocrine gland-secreting enzymes and electrolytes that enter the intestinal lumen through the pancreatic duct, and as an endocrine gland-releasing the hormones insulin and glucagon from islets of Langerhans into the blood stream. The exocrine secretions of the pancreas include

the proteolytic enzymes trypsinogen, chymotrypsinogen and pro-carboxypeptidase, as well as pancreatic amylase, lipase and bicarbonate.

Proteolytic enzymes are secreted in an inactive form and are auto-catalytically converted to their active form on entry to the small intestine- trypsinogen by activity of enterokinase secreted by cells of the intestinal mucosa, and the other cells by the activity of trypsin. These enzymes catalyse the breakdown of proteins to amino acids and small peptide chains. Complex intracellular mechanisms stabilize proteolytic enzymes within the pancreatic acinar cell to prevent auto-digestion of the pancreas.

The pancreas is the only site of production of lipase, the enzyme that can accomplish fat digestion, by hydrolysing dietary triglycerides to the absorbable units of monoglycerides and free fatty acids. As a consequence, excessive fat in the faeces (steatorrhea) may be result from insufficient pancreatic secretion.

Chyme entering the duodenum is highly acidic. Electrolytes (bicarbonate) secreted by ductal pancreatic cells, in a process that is catalysed by carbonic anhydrase, buffers this acidic chyme, to prevent damage to mucosal cells and to provide a pH optimal for the activity of pancreatic enzymes. Ductal secretion of an electrolyte rich solution contributes greatly to the volume of pancreatic fluid.

Control of pancreatic secretion

Pancreatic secretion is subject to a similar hierarchy of control mechanisms as exists for gastric secretion. Pancreatic secretion can be observed in all phases of digestion- cephalic, gastric and intestinal- and both neural and neuro-endocrine pathways are involved.

The cephalic phase- in response to the sight, smell or ingestion of food is mediated via vagal stimulation of acinar and ductal cells. The presence of food in the stomach, and particularly gastric distension and the presence of protein, increase pancreatic via two pathways- a vago-vagal reflex, and via release of gastrin that is a potent stimulus to pancreatic secretion. The intestinal phase of pancreatic secretion is the most significant, stimulated by the presence of chyme in the duodenum. An acidic (or fat laden) chyme stimulates release of the enterogastrone secretin from duodenal S cells, and subsequent secretion of bicarbonate rich aqueous fluid from ductal cells. A protein or fat rich chyme stimulates release of cholecystokinin (CCK) and subsequent release of enzyme containing zymogen granules from acinar cells with little change in fluid volume.

Bile secretion and the biliary tree

The liver is a complex metabolic organ with a multiplicity of functions. Amongst these is the synthesis and secretion of bile salts, that aid in the emulsification and subsequent digestion of fats by pancreatic lipase.

Bile salts are formed in hepatocytes by conjugation of cholesterol with amino acids. The steroid backbone of the bile acid is lipophilic; the amino acid conjugate is hydrophilic. Conjugated bile salts are thus able to function as detergents and serve to keep fats in solution in an aqueous environment and hence accessible to lipase. Bile salts aggregate spontaneously with fats to form micelles.

Bile salts are secreted across the lateral border of hepatocytes into the bile canaliculus, and then through a network of ductules and ducts to the bile duct and to storage in the gall bladder. The gall bladder stores and concentrates bile but has no secretory role.

Control mechanisms in secretion of bile

The vagus nerve plays a minor part in control of bile secretion. The enterogastrones secretin and CCK are significant regulators of bile secretion and release. As it does with pancreatic $NaHCO_3$ secretion, secretin stimulates an increased aqueous alkaline bile secretion by duct cells without a corresponding increase in bile salt secretion, i.e. secretin stimulated release of alkaline biliary secretion helps to neutralise gastric acid

entering duodenum. The presence of fat in duodenal chyme stimulates release of CCK, which as its name suggests, stimulates contraction of the gall bladder and delivery of bile to the duodenum.

Secretion of bile by hepatocytes is also regulated by chemical means, the so called 'bile salt dependent bile flow'. During a meal, bile is emptied from the gall bladder to duodenum, bile salts participate in fat digestion, then pass to the ileum where they are reabsorbed and returned by entero-hepatic circulation to the hepatocytes, where they serve to increase bile secretion. As bile is secreted and reabsorbed, its secretion is increased. Between meals, when bile salts are being stored in the gall bladder, secretion is low. Entero-hepatic circulation allows for a very high degree of conservation of bile salts (up to 98%), with the remainder synthesized de novo.

FURTHER READING

Sherwood, L. *Human Physiology from Cells to Systems* 8th Edition 2013 Ch 16

Cunningham's *Textbook of Veterinary Physiology* 6th Ed Elsevier 2020 Ch 27 & 28

Young B *Wheater's Functional Histology: A text and colour atlas* 4th Edn 2014 Churchill Livingstone (ebook)

Bacha WJ & Bacha LM *Color Atlas of Veterinary Histology* 2nd Edn 2012 Wiley-Blackwell

Veterinary Bioscience: Digestive System

LECTURE 18
DISEASES OF THE RUMINANT FORESTOMACH

LECTURER

ASSOCIATE PROFESSOR JENNY CHARLES

As a veterinary graduate of the University of Sydney, Jenny Charles undertook specialist training in veterinary anatomic pathology at the University of Melbourne and the University of Guelph, and also worked in the United Kingdom on the clinical diagnosis and eradication of bovine spongiform encephalopathy. She is a Diplomate of the American College of Veterinary Pathologists and a member of the international WSAVA multi-disciplinary team responsible for refining diagnostic criteria for hepatobiliary disorders of dogs and cats. Jenny's research interests include disorders of the liver, pancreas and cardiovascular and reproductive systems of domestic animals, diseases of New World camelids, causes of wastage in the horse-racing industry, and applied aspects of clinical pathology.

charlesj@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- describe the aetiopathogenesis of the common disorders of the ruminant forestomachs.
- explain the potential consequences of injury to a forestomach compartment that disturbs its motility, reduces the mucosal absorptive surface area and/or increases mucosal permeability.
- identify the characteristic gross and/or microscopic lesions that are used to reach a specific diagnosis.

KEY WORDS

Bloat, traumatic reticuloperitonitis, hardware disease, vagus indigestion, rumenitis, reticulitis, omasitis, lactic acidosis.

LECTURE OVERVIEW

Several diseases commonly afflict the forestomach compartments, particularly the rumen. Many lesions of the reticulum, rumen and/or omasum are subclinical but others can be responsible for significant morbidity and mortality through compromising feed digestion and gastrointestinal motility, disturbing hydration

and/or acid-base balance, or by extension of the disease process to secondarily involve other organs and body systems.

In this lecture, we will review the important disorders of the forestomachs (including diet-induced mucosal dystrophic changes, luminal foreign bodies, motility disturbances, traumatic injuries, inflammatory and infectious conditions, and common neoplasms). Knowledge of the normal structure and function of these compartments and of their anatomic relations to other structures will be applied to predict the potential consequences of injury. The causes and characteristic diagnostic features of the disorders will be outlined, together with the gross features of the forestomach compartments that should be evaluated during any necropsy examination of a ruminant.

FURTHER READING

C.A. Brown, D. C. Baker and I. K. Barker. Alimentary system. In: *Jubb, Kennedy and Palmer's Pathology of Domestic Animals*. 5th ed., Volume 2. Ed. M. G. Maxie. Elsevier Saunders, Philadelphia, USA (2007). pp. 41-51.

H. B. Gelberg. Alimentary system. In: *Pathologic Basis of Veterinary Disease*. 4th edition. Ed. M. D. McGavin and J. F. Zachary. Mosby Elsevier, St Louis, USA (2007). pp. 326-330.

Veterinary Bioscience: Digestive System

LECTURE 19
DISEASES OF THE STOMACH AND ABOMASUM

LECTURER

DR LIZ DOBSON

Liz completed her veterinary degree at Murdoch University and then worked for two years in a small-animal and equine practice in rural NSW, before heading overseas to do a mix of locum and vet practice jobs for the next six years. She has been involved in tuberculosis research in Uganda at the domestic-wildlife interface, neutering clinics in the Peruvian Amazon, and vaccination clinics in the informal settlements of Nairobi. She completed a Masters in Control of Infectious Disease in Animals at the Royal Veterinary College in London. Liz did a joint wildlife/anatomic pathology residency at Cornell University in Upstate New York, relocating to the Bronx Zoo, NYC for the final year of her residency with the Wildlife Conservation Society, and later became a board qualified veterinary pathologist in 2010. After returning to Australia in 2011, Liz worked as a veterinary pathologist for major veterinary diagnostic companies.

liz.dobson@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Identify the more common disease conditions of the stomach/abomasum in domestic animals.
- Outline the pathogeneses of common diseases of the stomach/abomasum in domestic animals.

KEY WORDS

Gastric dilatation, gastric impaction, gastric displacement, abomasal displacement, gastritis, gastric ulceration, pyloric stenosis.

LECTURE OVERVIEW

In this lecture, we will overview various diseases of the stomach and abomasum.

The stomach and abomasum can become enlarged with feed (**impaction**) or water, food and gas (**dilation**). The stomach and abomasum can also become displaced (**GDV** in dogs and **abomasal displacement**). **Gastric ulceration** is a common finding in a variety of species and can lead to pain, poor performance

(horses) or production (livestock) and secondary infection. **Gastritis** is inflammation of the stomach wall and occurs due to a variety of reasons including *Clostridium septicum* infections in cattle, many chemicals, non-steroidal anti-inflammatory drugs and uraemia. Unlike in humans, there is no proven association between *Helicobacter spp* infection and ulceration in domestic animals.

Gastric/abomasal parasites are common in horses, cattle, sheep and goats and can cause death in severe cases. Adenocarcinomas, squamous cell carcinomas and lymphomas are the most important **gastric neoplasms** and will be discussed in this lecture.

FURTHER READING

Zachary, J. F., *Pathologic basis of veterinary disease* (2017), 6th edition, Chapter 7, p 324-411 (selected sections on the stomach)

Jubb, Kennedy and Palmer's, Pathology of domestic animals, 6th edition, Ed M Grant Maxie, volume 2, chapter 1, p 44-60

Veterinary Bioscience: Digestive System

LECTURE 20
DISEASES OF THE EXOCRINE PANCREAS

LECTURER

DR LIZ DOBSON
liz.dobson@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Explain the defence mechanisms that prevent pancreatic auto-digestion in health and the circumstances in which they can fail.
- Recognise the clinical signs that may be indicative of exocrine pancreatic disease.
- Explain the aetiopathogenesis and describe the associated lesions of the common disorders of the exocrine pancreas of domestic animals.

KEY WORDS

Protease inhibitor, pancreatic hypoplasia, pancreatic atrophy, juvenile pancreatic atrophy, exocrine pancreatic insufficiency, jaundice, steatorrhoea, amylorrhoea, creatorrhoea, pancreatic necrosis, pancreatitis, pancreatic calculi, exocrine pancreatic nodular hyperplasia, pancreatic adenoma, pancreatic adenocarcinoma.

LECTURE OVERVIEW

The exocrine pancreas plays a pivotal role in the digestion of food via the secretion of digestive enzymes/pro-enzymes and bicarbonate. Several defence mechanisms exist to prevent auto-digestion of this organ and of other host tissues. However, in certain circumstances, these defences can go awry, be circumvented or become exhausted, resulting in pancreatic necrosis (necrotising pancreatitis).

Although injury to the exocrine pancreas may provoke clinical signs during the acute phase, the large functional reserve of this organ allows many disease processes to smoulder subclinically for prolonged periods. When clinical signs do emerge, they may largely reflect maldigestion (for example, weight loss despite a normal to increased or depraved appetite, gross abnormalities in the volume, colour and/or odour of the faeces or in the frequency of defaecation), jaundice (due to obstruction of the distal biliary tree) or metabolic disturbances arising from concurrent destruction of the endocrine components of the pancreas (diabetes mellitus).

We will review the important disorders of the exocrine pancreas in domestic animals, including developmental anomalies, diffuse and focal atrophy, pancreatic necrosis, pancreatitis, pancreatic duct obstruction, nodular hyperplasia and neoplasia. The known causes and characteristic diagnostic features of the various exocrine pancreatic disorders will also be discussed.

FURTHER READING

Pancreas. In: *Jubb, Kennedy and Palmer's Pathology of Domestic Animals*. 6th edition, Volume 2. Ed. MG. Maxie. Elsevier Saunders, Philadelphia, USA (2016). pp. 353-375.

Liver, biliary system, and exocrine pancreas. In: *Pathologic Basis of Veterinary Disease*. 6th edition. Ed. M. D. McGavin and J. F. Zachary. Mosby Elsevier, St Louis, USA (2017). pp. 432-434 and pp. 464-470.

Veterinary Bioscience: Digestive System

LECTURE 21
DISEASES OF THE PERITONEAL CAVITY

LECTURER

ASSOCIATE PROFESSOR JENNY CHARLES
charlesj@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- discuss the routes by which peritoneal fluid drains in health and explain the mechanisms that can be responsible for accumulation of excess fluid within the cavity.
- describe the common disease processes that affect the peritoneal cavity and retroperitoneum of domestic animals and outline their causes and potential consequences.
- identify the characteristic gross and/or microscopic lesions that are used to reach a specific diagnosis.

KEY WORDS

Peritoneum, mesothelium, fibrin, fibrous adhesion, internal and external herniation, hernia, haemoperitoneum, ascites, transudate, modified transudate, uroperitoneum, chyloperitoneum, peritonitis, exudate, fat necrosis, lipoma, mesothelioma, retroperitoneum, retroperitonitis.

LECTURE OVERVIEW

What comprises the peritoneal cavity and retroperitoneum? What are the routes by which peritoneal fluid drains? How does the peritoneum respond to injury? What are the important disease processes that affect the peritoneal cavity and retroperitoneum of the domestic animals? What are the causes and potential consequences of these diseases?

In health, the peritoneal (or abdominal) cavity is lined by a monolayer of mesothelial cells and contains a small volume of transudative fluid that is constantly recycled via lymphatic drainage. Several conditions may result in distension of the cavity by excess non-inflammatory oedema fluid, because of obstruction of drainage pathways and/or over-production of fluid.

Injury to the lining mesothelium permits exudation of fibrin and leukocytes into the peritoneal cavity. If the inflammatory response is severe or persistent, it may promote the formation of permanent fibrous tissue adhesions between cavity structures. Adhesions can be advantageous in that they may help to wall off sites

of inflammation within the cavity to prevent systemic spread of infectious agents but they can also compromise the function of the various visceral organs that are suspended within the cavity.

In this lecture, we will review the important disease processes that involve the peritoneal cavity of domestic animals. These disorders include developmental and acquired lesions that permit internal or external herniation of cavity viscera, abnormal cavity contents (including foreign bodies, parasites, gastrointestinal contents, oedema fluid, blood, urine, chyle and bile), inflammation of the cavity (peritonitis) and its causes and consequences, and neoplasia.

The retroperitoneum lies immediately external to the peritoneal cavity and in health contains loose fibrofatty connective tissues and organs such as the kidneys and adrenal glands. The most significant processes that can affect the retroperitoneum of domestic animals and that will be reviewed in the lecture are retroperitoneal fat necrosis, inflammation (retroperitonitis), accumulation of haemorrhage or other fluids, and neoplasia..

FURTHER READING

C.A. Brown, D. C. Baker and I. K. Barker. Alimentary system. In: *Jubb, Kennedy and Palmer's Pathology of Domestic Animals*. 5th edition, Volume 2. Ed. M. G. Maxie. Elsevier Saunders, Philadelphia, USA (2007). pp. 279-296

Veterinary Bioscience: Digestive System

THEME 5: DIGESTION AND ABSORPTION

Week 9 and Week 10

In these two weeks we will answer the question: “How is food digested and absorbed?”. This involves understanding not just the processes involved in digestion of each of the major food stuffs, but also the structural adaptations of the different regions of the intestines that enable absorption to occur, in a range of species, including the role of microbial fermentation in these processes. We will consider the mechanisms of diarrhoea – that may occur as a consequence of perturbation of absorptive processes, or of intestinal secretory processes.

LECTURES

Mechanisms of Digestion and Absorption	• Structural adaptations of the intestines for their absorptive role • Intestinal secretion of chloride • Digestion and absorption of protein, carbohydrates and fats • Absorption in the large intestine
Intestines of Herbivores and Omnivores	• Comparative anatomy of the small and large intestines of the horse, cow, sheep, pig and bird • Comparative pancreatic anatomy
Role of Microbes in Digestive Function	• Why do herbivores rely on bacteria for digestion? • Foregut fermenters: ruminants, kangaroos • Hindgut fermenters: horses, rabbits and guinea pigs, birds • Disturbances of the intestinal bacterial flora
Diarrhoea	• Small intestinal versus large intestinal diarrhoea • Mechanisms of diarrhoea: hypersecretion; malabsorption; increased mucosal permeability • Large intestinal diarrhoea; osmotic overload in the large intestine
Development of the Mouth and Gastrointestinal Tract in the Embryo	• Early formation of the gut tube • Development of the mouth, formation of the stomach and intestines • The body cavities

PRACTICAL CLASSES AND CASE STUDIES

Comparative Intestines	Gross anatomy of the small and large intestines of various domestic animal species	WEBS Dissection Lab (B104)
Histology of the Intestines	Microscopic anatomy of the intestines	Online
Case study: Suki the Cat	Application of knowledge to clinical case	WEBS Collaborative Learning Centre (G04)

LEARNING OUTCOMES

	**SCIENTIFIC BASIS OF CLINICAL PRACTICE** • Apply an understanding of porto-systemic circulation to the principles of digestive function. • Relate the salient cellular and architectural features of each organ to their function in health and disease. • Describe the histological structure of the intestinal wall, and link this to intestinal function
	**CLINICAL SKILL ACQUISITION** • Identify the organs of the gastrointestinal system on gross and histological examination in different animal species. • Use clinical information to predict the consequences of maldigestion or malabsorption.
	**PERSONAL AND PROFESSIONAL DEVELOPMENT** • Engage in a collaborative approach to solving clinical problems.

Veterinary Bioscience: Digestive System

LECTURE 22
DIGESTION AND ABSORPTION

LECTURER

PROFESSOR ELIZABETH TUDOR

etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the histology of the small and large intestine, and Identify different anatomical regions of the intestine in tissue specimens prepared for light microscopy.
- Describe the structure of the intestinal villus and of intestinal mucosal cells and explain the functional significance of their structure.
- Explain how proteins, carbohydrates and fats are digested and absorbed in the small intestine, and describe how brush border enzymes and contribute to the digestive process.
- Explain how absorption of sodium and secretion of chloride contribute to water balance in the intestines.
- Describe the role of secretory and absorptive cells in the crypt-villus unit and explain how disease processes can perturb the balance of secretion and absorption, resulting in diarrhoea.

KEY WORDS

Villus, crypt-villus unit, microvillus, brush border, goblet cell, crypt cell, duodenum, jejunum, ileum, colon, dipeptidase, disaccharidase, aminopeptidase, sucrase, lactase, micelle, chylomicron, oral rehydration therapy (ORT), lacteal.

LECTURE OVERVIEW

The small intestine is the site where digestion of foodstuffs is completed, and absorption occurs. The greatest proportion of absorption takes place in the duodenum- but ileal absorption is important particularly for absorption of bile salts, fat soluble vitamins and vitamin B12. There are specific absorption mechanisms for each of the major nutrients as well as for sodium. Clinically significant secretion of electrolytes also occurs in the small intestine.

The small and large intestines are differentiated structurally for their roles in digestion and absorption. Surface area is increased by the presence of folding (plicae and rugae), by the villus structure of the small intestinal mucosa and by the presence of microvilli on mucosal absorptive cells. The microvillus brush

border contains enzymes that catalyse the final breakdown of carbohydrates and peptides to absorbable units, and also the transport pumps required for absorption.

Intestinal secretion of chloride

Chloride is secreted by intestinal crypt cells, by means of an active transport process. The movement of chloride into the lumen is accompanied by sodium and water. The secretion of chloride is mediated by second messenger systems involving cAMP and cGMP. Changes in these second messenger levels induced by microbial pathogens can result in secretory diarrhoea.

Digestion and absorption of protein

The breakdown products of pancreatic proteases are amino acids and small peptides. These are split by dipeptidases bound to the brush border membrane, and amino acids are absorbed by a carrier mediated Na dependent secondary active transport process in the apical cell membrane.; the carrier is specific for each amino acid. Absorbed amino acids then move by diffusion down a concentration gradient into the blood stream.

Digestion and absorption of carbohydrate

Dietary carbohydrate is presented for absorption as the disaccharides: maltose, sucrose and lactose. Disaccharidases located in brush border facilitate their breakdown to monosaccharides. Glucose and galactose both move through the apical membrane via secondary active transport: a carrier on the luminal surface simultaneously transfers glucose against a concentration gradient, and Na+ down a concentration gradient from the lumen to the cell. The Na concentration gradient is established by basolateral Na+/K+ pump; no energy is directly used to move glucose up a concentration gradient. Cotransport is driven by Na+ gradient established by the Na/K pump. Glucose and galactose diffuse down a concentration gradient through the basolateral surface of cell; fructose is absorbed into the blood purely by facilitated diffusion.

Digestion and absorption of fat

Because of the insolubility of fat in water, fat must undergo physical and chemical transformation to facilitate absorption. Dietary fat in the form of triglycerides is emulsified by the detergent action of bile salts to form micelles, with increased surface area for pancreatic lipase activity. Lipase hydrolyses triglycerides to monoglycerides and free fatty acids, and the water insoluble products are carried in the interior of water-soluble micelles to the brush border. Monoglycerides and free fatty acids leave the micelle and passively diffuse through the luminal membrane. Within the cell, triglycerides are resynthesised, aggregate and are coated with lipoprotein to form water soluble chylomicrons that are extruded from the basal cell surface by exocytosis. Chylomicrons unable to enter blood capillaries, so enter lymphatic lacteals. Following feeding, lacteals are visible due to the “milky” character of the lymph within them.

Absorption of sodium

Absorption of sodium, that drives also the absorption of water from the gut, occurs in a number of ways. When the electrochemical gradient is favourable, sodium will diffuse down a concentration gradient into the cell. At other times, absorption of sodium is driven by a Na/K active transport pump on the basolateral cell membrane, that creates a favourable diffusion gradient across the apical cell membrane into the cell. In addition, Na entry to the cell occurs in a cotransport mechanism linked to both glucose and amino acid absorption, by means of the sodium linked glucose transport (SLGT) protein. This latter mechanism forms the basis of oral rehydration therapy with isotonic glucose solutions, as this cotransport may continue to function in intestinal disease states that cause diarrhoea.

Absorption in the large intestine

With the exception of the hind-gut fermenters, that are the topic of a later lecture, the largest proportion of water, and almost all nutrients are absorbed from the small bowel. Approximately 12% of water absorption occurs in the colon, but the efficiency of absorption is much higher here; (90% compared to 50% in the small intestine). In addition, water absorption from the large bowel is regulated by the presence of aldosterone responsive Na channels in the basal membrane of colonic mucosal cells.

FURTHER READING

Sherwood, L. *Human Physiology from Cells to Systems* 8th Edition 2013 Ch 16

Cunningham's *Textbook of Veterinary Physiology* 6th Ed Elsevier 2020 Ch 27 & 28

Maddison J, Page S and Church D *Small Animal Clinical Pharmacology* 2nd Ed 2008

Riviere JE, & Papich MG *Veterinary Pharmacology & Therapeutics* 10th Edn 2018 Wiley Blackwell (ebook)

section 46

Rang and Dale's *Pharmacology* 9th edn Ch 31 (ebook)

Young B *Wheater's Functional Histology : A text and colour atlas* 4th Edn 2014 Churchill Livingstone (ebook)

Bacha WJ & Bacha LM *Color Atlas of Veterinary Histology* 2nd Edn 2012 Wiley-Blackwell

Sjaastad OV, Sand O & Hove K *Physiology of Domestic Animals* Scandinavian Veterinary Press 2010 Ch 15

Veterinary Bioscience: Digestive System

LECTURE 23
COMPARATIVE INTESTINES

LECTURER

DR NICHOLAS BAMFORD

n.bamford@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the comparative gross anatomy of the small and large intestines of the horse, cow, sheep, pig and bird, including their course and position within the abdominal cavity and relationships to other organs.
- Describe the comparative gross anatomy of the pancreas and duodenal papillae in the horse, cow, sheep, pig and bird.
- Apply an understanding of intestinal anatomy of the horse, cow, sheep, pig and bird to explain how these species utilise different diets.

KEY WORDS

Digestion; duodenum; pancreatic ducts; bile duct; jejunum; ileum; ileocaecal orifice; caecum; ascending colon; taeniae; haustra; pelvic flexure; transverse colon; descending colon; rectum.

LECTURE OVERVIEW

This lecture will detail the comparative gross anatomy of the intestinal tracts of selected herbivore and omnivore species: horses, ruminants, pigs and birds. The intestinal tracts of these species are highly specialised, reflecting the different ways in which different species utilise different diets. Of particular interest is the way in which the hindgut of these species is adapted to facilitating microbial fermentation otherwise indigestible carbohydrates. The course and position of the different segments of the small and large intestines will be described, as well as comparative anatomy of the pancreas in these species and configuration of the duodenal papillae.

The small intestine

As in the dog and cat, the small intestines in the other domestic species consist of the duodenum, jejunum and ileum. Apart from the great length of the small intestine in the horse, ruminants and pig, there are some

differences in the ducts opening on the major and minor duodenal papillae (either one or two pancreatic ducts).

The large Intestine

While in the dog, the large intestine is short and unspecialised (a simple tube only slightly larger in diameter than the small intestine), in the ruminants, horse and pig it is greatly enlarged and coiled. This reflects the contribution of bacterial digestion in the hindgut and the absorption of nutrients in the form of volatile fatty acids.

FURTHER READING

Singh: *Dyce, Sack & Wensing's Textbook of Veterinary Anatomy*, 5th Ed. Elsevier, 2018.

Getty: *Sisson and Grossman's The Anatomy of the Domestic Animals*, Volumes I and II, 5th edition, 1975.

Smallwood: *A Guided Tour of Veterinary Anatomy*, Saunders, 1992.

Ashdown and Done: *Color Atlas of Veterinary Anatomy* The Ruminants, 1984.

Ashdown and Done: *Color Atlas of Veterinary Anatomy* The Horse, 1984.

Fails: *Anatomy and physiology of farm animals*, Wiley, 2018.

Veterinary Bioscience: Digestive System

LECTURE 24
ROLE OF MICROBES IN DIGESTION

LECTURER

DR NICHOLAS BAMFORD
n.bamford@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Compare the roles of microbes in the digestive function of different domestic animal species, and explain how disruption of the microbiome can result in disease.
- Explain the role of microbes in the digestive processes of hindgut and foregut fermenting animals.
- Discuss the relative advantages and disadvantages of microbial digestion in the foregut and hindgut.

KEY WORDS

Foregut fermenter; ruminant; hindgut fermenter; bacteria; nutrition; volatile fatty acids; carbohydrates; rabbits; guinea pigs; kangaroos; herbivores.

LECTURE OVERVIEW

This lecture considers the role of microbes in digestive function for various animal species. Herbivores have developed different means of accommodating microbes in their digestive tract, in order to digest plant structural carbohydrates. The relative advantages and disadvantages of having bacterial digestion in the forestomach or the hindgut can be compared. While intestinal microbes bring great benefits to the animal, disruption of the microbiome can result in severe disease, and several clinical examples will be illustrated.

FURTHER READING

McDonald, Edwards, Greenhalgh, Morgan. *Animal Nutrition.* (1995) 5th Edition. Prentice Hall Publishers.

The ruminant animal digestive physiology and nutrition. Church DC Ed. (1988). Eaglewood Cliffs, Prentice Hall Publishers.

Cunningham and Klein. *Textbook of Veterinary Physiology*. (2007) 4th edition. Saunders.

Veterinary Bioscience: Digestive System

LECTURE 25
DIARRHOEA

LECTURER

ASSOCIATE PROFESSOR JENNY CHARLES
charlesj@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- describe some of the most common disease conditions of the intestines in domestic animals
- explain the pathogeneses of diarrhoea.

KEY WORDS

Diarrhoea, enteritis, colitis, typhlitis, hypersecretion, malabsorption, maldigestion, exudation, steatorrhoea, ileus, lymphangectasia.

LECTURE OVERVIEW

In this lecture we will overview various diseases of the small and large intestine focusing on various causes of diarrhoea. These will include developmental disorders, infectious diseases, autoimmune/immune mediated diseases, idiopathic diseases, and some of the more common neoplastic conditions of the small intestine.

FURTHER READING

McGavin General Veterinary Pathology, 2nd edition, Chapter 7, p 301-392. *Jubb, Kennedy and Palmer, Pathology of domestic animals*, 5th edition, Ed M Grant Maxie, volume 2, chapter 1, p 1-296.

Veterinary Bioscience: Digestive System

LECTURE 26
DEVELOPMENT OF THE MOUTH AND GASTROINTESTINAL TRACT IN THE EMBRYO

LECTURER

DR CHRISTINA MARTH
christina.marth@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Explain the developmental anatomy in the embryo of the mouth and associated structures, including the tongue in order to elaborate on the origin of anatomical structures in adult animals and common malformations.
- Describe the derivatives of the primitive gut tube (fore-, mid-, hindgut) in order to explain the relations between organs in adult animals.
- Discuss the developmental anatomy of the oesophagus, stomach and intestines in order to elaborate on their orientation in adult animals.

KEY WORDS

Embryology, coelom, gut tube, foregut, midgut, hindgut, mouth, palate, tongue, cloaca, gastrointestinal development.

LECTURE OVERVIEW

Early formation of the gut tube

Formation of the primitive gut is initiated by the establishment of the endodermal layer of the blastocyst. With folding of the body wall into a cylindrical shape, the primitive gut within the embryo is formed into a tube with three parts: foregut, midgut, hindgut and the part of the primitive gut that remains outside the embryonic body wall, the yolk sac.

Derivatives of the fore-gut include part of the mouth, the pharynx; the oesophagus, the stomach, the liver and the pancreas, the thyroid, the parathyroid and the thymus glands, the trachea and the lungs.

Derivatives of the mid-gut include the major part of the small intestine.

Derivatives of the hind-gut include the large intestine and the cloaca.

Development of the mouth

The Stomodaeum is a midline depression on the ventral surface of the head that is created by the cranial and lateral body folding. It subsequently enlarges into a definitive oro-nasal cavity. The oral plate (or pharyngeal membrane) is formed by the fusion of the ectoderm of the stomodaeum and the endoderm of the fore-gut. The oral plate subsequently breaks down and the cranial opening of the digestive tube is established.

After head folding, fleshy mesenchymal swellings, the branchial arches, develop on either side of the developing oro-nasal cavity. The derivatives of the branchial arches contribute to formation of the head and neck. For example, the first branchial arch divides into the left and right maxillary processes and mandibular processes which elongate to form the jaws and mouth.

Development of the palate

At an early stage of development the oral and nasal cavities are confluent, but palate development subsequently separates the oral and nasal cavities. The palate derives from three parts:

1. Left palatine process
2. Right palatine process
3. Medial palatine process (= inter-maxillary process)

These three processes fuse together to separate the oral and nasal cavities. This hard palate extends caudally as the soft palate that separates the rostral pharynx into the nasopharynx (dorsal) and oropharynx (ventral).

Early in development, the most cranial part of the fore-gut becomes very broad and flattens dorso-ventrally. This expanded fore-gut gives rise to a series of pouches - pharyngeal pouches - which extend laterally. These pharyngeal pouches provide numerous contributions to various organs and tissues, many of which are not associated with the digestive system. These include the eustachian tubes (auditory tube, the fossae of the tonsils and covering epithelium and cells of the parathyroid, thymus and thyroid glands.

Development of the tongue

The tongue arises as a protrusion from the floor of the pharynx into the primitive mouth. Tongue formation begins as 4 distinct mesenchymal swellings:

- A median tongue swelling;
- Two distal tongue swellings;
- A proximal tongue swelling.

The median and distal tongue swellings will form the body of the tongue, while the proximal tongue swelling will form the root of the tongue. The epithelium covering the surface of the tongue is completely renewed every 7-10 days.

Formation of the oesophagus

The fore-gut narrows to form the oesophagus and elongation of the oesophagus occurs during growth of the cervical and thoracic regions of the body. The epithelial lining of the oesophagus and any associated mucosal glands develop from the endoderm of the primitive fore-gut. The connective tissue and muscle layer of the oesophagus are derived from accumulating mesenchymal cells partially derived from the splanchnic mesoderm of the primitive gut.

Formation of the stomach

The stomach arises as a spindle-shaped swelling of the fore-gut located in midline. A dorsal mesentery (the dorsal mesogastrium) develops on the dorsal surface and the ventral mesentery (the ventral mesogastrium) develops on the ventral surface. It has a cranial opening - the cardia and a caudal opening - the pylorus. The developing stomach moves caudally and shifts away from the midline. Thus, the stomach rotates towards the left pulling with it the dorsal mesentery and leading to the formation of the greater omentum. There is species-specific differential enlargement and reshaping of the stomach, e.g. the ruminant stomach.

Formation of the small and large intestines

Initially, the primitive gut is straight with dorsal and ventral mesenteries. Later the ventral mesentery breaks down. The gut grows faster than the body so a hairpin-shaped loop is formed. The remnant of the yolk sac (the yolk-stalk) is at the tip of the loop. Rapid development of the liver forces the loop of gut into the umbilical stalk - this is physiological herniation. Later the intestines return to the abdomen and move into their final position. Further changes involve rotation of the loop and extensive coiling of the cranial arm of the loop to form the duodenum, jejunum and most of the ileum.

The gut loop from the yolk-stalk to the proctodaeum contributes to the terminal part of the ileum, caecum, colon, rectum and anal canal. The caecum becomes established as a definite pouch-like diverticulum of the digestive tube caudal to the yolk stalk. Species-specific changes occur in the developing caecum and colon to account for the specific layouts discussed in anatomy.

Cloaca and proctadeum

The caudal portion of the primitive gut expands to form the blind cavity of the cloaca. The invagination of ectoderm beneath the tail forms the proctadeum. This boundary between the endoderm and the ectoderm forms the cloacal membrane, which degenerates resulting in the anal opening.

FURTHER READING

McGeady TA, Quinn PJ, Fitzpatrick ES and Ryan MT. (2008). *Veterinary Embryology.*

Noden, D.M. and De Lahunta, A. (1985) *The Embryology of Domestic Animals.*

Moore, K.L. and Persaud, T.V.N. (1993) *Before We are Born*, 4th edition.

Mitchell, B. and Sharma, R. (2005) *Embryology.*

Sadler T.W. (2006) *Langman's Medical Embryology*, 10th edition.

Veterinary Bioscience: Digestive System

THEME 6: GASTROINTESTINAL BARRIER FUNCTION AND FLUID MOVEMENT

Week 11 and Week 12

In the final two weeks, we will consider the gut as a dynamic barrier to the external environment: How is its integrity maintained in the face of caustic or infectious luminal content? What happens to the balance of absorption and secretion when passage of gut content along the length of the gut is obstructed? How might gastrointestinal health be evaluated using clinical pathology? There will also be ample opportunities to acquire practical skills in pathology of the upper and lower gastrointestinal tract.

LECTURES

The Gastrointestinal Barrier	• Structural aspects of the mucosal barrier • Secretions and the mucosal barrier • Cell turnover and the mucosal barrier • Mucosal blood flow • How do drugs cross the mucosal barrier?
When the Gut Stops Moving	• Functional obstruction - ileus • Impaction, obturation and space occupying lesions • Stenosis; extrinsic obstruction • Torsion, volvulus, strangulation & intussusception
Consequences of Gut Stasis for Fluid Balance	• The enterosystemic fluid cycle • Dehydration; electrolyte balance • Obstruction of the digestive tract
Clinical Pathology of the GI Tract	• Clinical pathology of the abdominal tract • Abdominocentesis, cytology and biochemistry

PRACTICAL CLASSES AND CASE STUDIES

Pathology of the Upper GI Tract	Gross pathology wet specimens	Werribee Dissection and Pathology Laboratory (Room 410)
Pathology of the Exocrine Pancreas and Peritoneal Cavity	Gross pathology wet specimens	Werribee Dissection and Pathology Laboratory (Room 410)
Pathology of the Lower GI Tract	Gross pathology wet specimens	Werribee Dissection and Pathology Laboratory (Room 410)
Case Study: A Pain in the Gut	Application of knowledge to clinical case	WEBS Collaborative Learning Centre (G04)

LEARNING OUTCOMES

	**SCIENTIFIC BASIS OF CLINICAL PRACTICE** • Explain how the barrier between gastrointestinal contents and the intestinal tissues is maintained. • Explain how the gut responds to injury and heals, and the role of the peritoneum in these processes. • Explain the implications of interruptions to the entero-systemic fluid cycle. • Relate the salient cellular and architectural features of each organ to their function in health and disease.
	**CLINICAL SKILL ACQUISITION** • Identify common pathological states of the gastrointestinal tract on examination of necropsy specimens. • Interpret clinical pathology data to assess gastrointestinal diseases. • Use clinical information to predict the consequences of gut stasis.
	**PERSONAL AND PROFESSIONAL DEVELOPMENT** • Engage in a collaborative approach to solving clinical problems.

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LECTURE 27
THE GASTROINTESTINAL BARRIER

LECTURER

PROFESSOR LIZ TUDOR
etudor@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe the histological structures of the gastrointestinal tract that contribute to its functional integrity and explain the structures and mechanisms that provide protection from damage caused by gut contents including parasites and microbes.
- Explain the functional significance of the crypt-villus unit in the replenishment of intestinal epithelial cells, and the kinetics of gastrointestinal turnover.
- Explain the importance of mucosal blood flow in maintaining the integrity of the mucosal barrier and describe the important factors that regulate mucosal blood flow.
- Describe the processes that govern drug absorption from the gastrointestinal tract, and the physiological and pathological factors that may limit drug absorption.

KEY WORDS

Villus, crypt-villus unit, microvillus, brush border, goblet cell, tight junction, prostaglandin, stem cell, mucosal barrier, mucus, bicarbonate, gastric ulcer, villus atrophy, drug absorption.

LECTURE OVERVIEW

How is it that the gastric mucosa can secrete acid at a pH of less than 2, and proteolytic enzymes that digest ingested meat and other proteins, yet the single celled mucosal lining of the stomach is not digested by its own secretions? What determines why a nutrient is absorbed, yet a potentially harmful molecule is not? How can drugs be designed to enhance or to limit their absorption from the gastrointestinal tract? How are bacteria and other microorganisms prevented from entering the mucosa? Understanding the answers to these questions involves understanding the biological principles that underpin the concept of the gastrointestinal barrier.

Disruption of the gastrointestinal barrier is a significant contributor to disease processes. It allows intestinal microorganisms to cause systemic disease, and in other situations, systemic disease can result in disruption of the mucosal barrier, that further compromises the patient.

Structural aspects of the mucosal barrier

The intrinsic mucosal barrier is composed of the epithelial cells and the tight junctions that hold them together. Once toxins or microorganisms cross this epithelial surface, they have relatively unimpeded access to the circulation. The integrity of tight junctions is therefore critical and understanding factors that modulate their permeability is important.

Epithelial cells themselves differ in their permeability to ions and hence their resistance to damage from luminal content. Parietal cells in the stomach for example, are less permeable to protons than intestinal epithelial cells and hence less prone to acid damage than intestinal cells.

Secretions and the mucosal barrier

The surface of the gastrointestinal tract is coated with mucus that contributes to the barrier in a number of ways. Mucus reduces shearing forces on the epithelial surface, helps bind and aggregate bacteria, preventing their colonisation, and impairs absorption of some water-soluble molecules. A range of mucins are produced in different parts of the digestive tract. Some are membrane bound and recognise and repel bacteria, others exert their protective effects by creating a physical barrier. Some are involved in cell signalling, leading to secretion of bactericidal compounds by surface epithelial cells.

Epithelial cells in both the stomach and intestines also secrete bicarbonate ions, that help to create a neutral pH at the apical cell surface, despite widely fluctuating pH in the gastrointestinal lumen.

Cell turnover and the mucosal barrier

An important functional component of the intrinsic mucosal barrier is provided by the constant turnover and replenishment of epithelial cells in the stomach and intestines. Integrity of the mucosal barrier relies on a balance between stem cell proliferation, differentiation and epithelial cell death.

In the intestine, this dynamic cell population is described as the crypt-villus unit. Epithelial cells migrate from the base of a crypt to the tip of a villus where they are extruded, over a period of three to five days. As they do so, they differentiate from a stem cell to a secretory cell and finally to an absorptive epithelial cell. A number of disease states are characterised by changes in the balance of cell types in the crypt-villus unit as a result of changes to proliferation, differentiation or the rate of cell death. Villus atrophy is one outcome of such a perturbation.

In the stomach, stem cells that give rise to the various cell types are located in the mid gland region and migrate as they differentiate in both directions in the gland.

Mucosal blood flow

Another important component of the mucosal barrier is provided by the mucosal blood supply. The submucosa is richly supplied with blood vessels, that in addition to providing nutrients and oxygen to mucosal cells, allow for rapid removal of hydrogen ions that diffuse from the lumen. Any disease state that causes significant reduction in mucosal blood flow will result in damage to the mucosal barrier. Gastric ulceration associated with haemorhagic shock occurs by this mechanism.

Control mechanisms and the mucosal barrier

Each of the functional aspects of the mucosal barrier that have been described above is subject to regulation- some that are better understood than others. Important and well described regulators of a number of these functions are the prostaglandins (PGs)- particularly PGE and prostacyclin, that are synthesized and released locally in the gastrointestinal mucosa. PGs increase mucus and bicarbonate secretion, stimulate cell proliferation and increase mucosal blood flow. Nonsteroidal anti-inflammatory

Drugs (NSAID's) such as aspirin increase the risk of gastric ulceration because they impair prostaglandin synthesis.

Other chemicals known to be involved in maintenance of the mucosal barrier are epidermal growth factor (EGF) secreted in saliva and by duodenal glands and transforming growth factor alpha (TGF- alpha), that is secreted by gastric epithelial cells. Both bind to a common receptor to increase epithelial cell proliferation, and also increase gastric mucus secretion and decrease acid secretion.

How do drugs cross the mucosal barrier?

There are a number of potential routes for drugs to cross the mucosal barrier. How they cross and the rate of absorption depends on characteristics of the drug and also the functional state of the mucosa. A reduction in mucosal surface area (for example as a result of villus atrophy,) will reduce absorption of drugs as well as nutrients, and this needs to be taken into account if drugs are administered orally. Equally, disease states that increase permeability of the mucosal barrier can result in absorption of drugs not normally absorbed, or in increased rate of absorption of a drug, beyond what is expected.

Many drugs are absorbed in a concentration dependent manner; (the greater the concentration the greater the absorption of drug). For these drugs diluting the drug in food may result in reduced absorption of drug. The concentration gradient of the drug across the mucosal surface also depends on mucosal blood flow. Local variations in blood flow as a result of exercise or feeding can change the concentration gradient for absorption of drugs- so can be important when determining timing of drug administration.

Solubility of the drug is also important. For amphoteric drugs, that are weak acids or bases, their solubility varies depending on the pH of the luminal content- which of course may vary along the gut length and also with feeding status.

To be absorbed drugs must cross both the apical and basal epithelial cell surface. Lipid soluble drugs cross these membranes readily and so can establish a large concentration gradient between the lipid membrane and the cytosol. Some water-soluble drugs move readily across the membrane by bulk flow, with water absorbed in active co-transport mechanisms. Some very small drugs may also move by diffusion through tight junctions.

FURTHER READING

Blikslager AT, Roberts MC: *Mechanisms of intestinal mucosal repair*. J Am Vet Med Assoc 211:1437-1441, 1997.

Cunningham's *Textbook of Veterinary Physiology* 6th Ed Elsevier 2020 Ch 27 & 28

Veterinary Bioscience: Digestive System

LECTURE 28
WHEN THE GUT STOPS MOVING

LECTURER

ASSOCIATE PROFESSOR JENNY CHARLES
charlesj@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Describe what happens to the body when the normal flow within the gastrointestinal tract is impeded.
- Explain the mechanisms/pathogeneses of major mechanical and physical obstructions to the gastrointestinal tract.
- Identify important causes of abnormal flow of gastrointestinal contents.

KEY WORDS

Volvulus, torsion, dilatation, infarction, hernia, incarceration, intussusception.

LECTURE OVERVIEW

In this lecture we will discuss abnormal gut movement, discussing what happens when there is a physical or mechanical obstruction to fluid flow within the gastrointestinal tract. Vascular accidents such as infarction of the tract will be discussed, along with other potentially catastrophic conditions such as volvulus, incarceration associated with hernias, dilatation and torsion.

FURTHER READING

McGavin *General Veterinary Pathology*, 2nd edition, Chapter 7, p 301-392.

Jubb, Kennedy and Palmer, Pathology of domestic animals, 5th edition, Ed M Grant Maxie, volume 2, chapter 1, p 1-296.

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LECTURE 29
CONSEQUENCES OF GUT STASIS FOR FLUID MOVEMENT

LECTURER

DR NICHOLAS BAMFORD
n.bamford@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Identify the fluids that are secreted into the gastrointestinal tract daily with a concept of their volume and composition in the different species.
- Apply an understanding of the enterosystemic fluid cycle in different animal species to explain the consequences of obstruction in different regions of the digestive tract for fluid and electrolyte balance.
- Describe how the gastrointestinal tract can be clinically assessed in large animal species.

KEY WORDS

Fluid movement; enterosystemic fluid cycle; plasma volume; electrolyte balance; gastrointestinal tract; mucosal barrier; clinical examination.

LECTURE OVERVIEW

Saliva, gastric fluid, bile, pancreatic and intestinal fluids are secreted into the digestive tract. The relative volume of these fluids as well as their composition varies considerably between the different species according to diet and type of digestive tract. Much of these fluid secretions are reabsorbed at a more distal part of the digestive tract, exactly where depends on gastrointestinal tract anatomy and physiology.

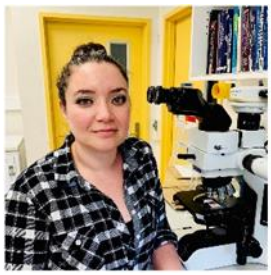
Obstruction to fluid movement along the digestive tract, either physical or functional, can have significant consequences for the fluid balance and circulation of the animal. This lecture looks at common clinical situations in different species where normal fluid movement in the gastrointestinal tract is disturbed. We will examine the clinical consequences for the animal when this occurs, including fluid, electrolyte and acid-base disturbances. We will also look at the different ways of assessing the gastrointestinal tract, particularly in the horse.

Veterinary Bioscience: Digestive System

LECTURE 30
CLINICAL PATHOLOGY OF THE GASTROINTESTINAL TRACT

LECTURER

DR ASTRID OSCOS SNOWBALL



Astrid is a graduate of the University of Guadalajara, Mexico (2007) and serves as a Lecturer in Clinical Pathology here at the University of Melbourne. After veterinary school, she completed a Master's in Veterinary Clinical Pathology in 2011, followed by a Doctor of Veterinary Science degree (research + residency) in Clinical Pathology at the Ontario Veterinary College, University of Guelph, Canada in 2017. She joined the faculty at Melbourne in October of 2018 and is currently pursuing Board Certification in Clinical Pathology.

marja.oscossnowball@unimelb.edu.au

INTENDED LEARNING OUTCOMES

At the end of this lecture, you should be able to:

- Explain how clinical pathology can help in defining and diagnosing gastrointestinal disease.
- Describe in basic terms how blood values may change in response to some common GI diseases.
- Explain the diagnostic tests for exocrine pancreatic insufficiency.
- Describe what disease mechanisms may result in changes to the peritoneal fluid.

KEY WORDS

Clinical pathology; vomiting; diarrhoea; exocrine pancreatic insufficiency; abdominocentesis; peritoneal fluid; faecal samples.

LECTURE OVERVIEW

The following topics will be covered in this session:

- Introduction to clinical pathology –The basic principles of blood biochemistry and haematology.
- The concept of normal reference ranges and what they mean.
- Changes in plasma electrolytes with vomiting, diarrhoea and enteropathy; effects of dehydration on blood biochemistry and haematology.

- Abdominocentesis and peritoneal fluid analysis – how this may be helpful as a diagnostic aid.
- Diagnosis of exocrine pancreatic insufficiency.
- Tests for malabsorption and maldigestion.
- Analysis of faecal samples.

FURTHER READING

Latimer KS. *Duncan & Prasse's Veterinary Laboratory Medicine Clinical Pathology*, 5th ed. Wiley, 2011.

- Ch. 5 Water, Electrolytes, and Acid-Base pgs. 156-167
- Ch. 8 Digestive System pgs. 231-246