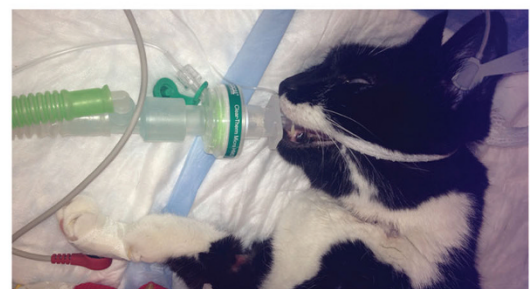
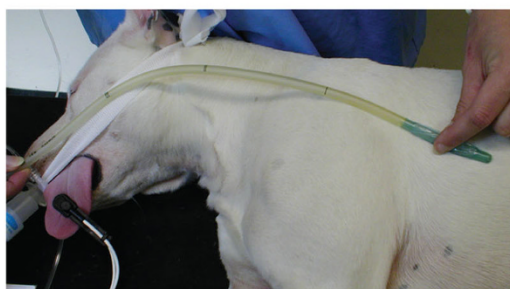
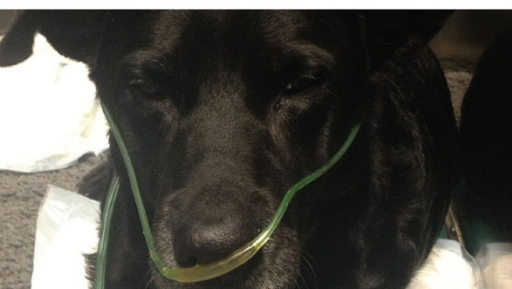


Second Edition

Veterinary Anaesthesia

Principles to Practice

Alexandra H. A. Dugdale | Georgina Beaumont | Carl Bradbrook | Matthew Gurney



WILEY Blackwell

Veterinary Anaesthesia

Veterinary Anaesthesia

Principles to Practice

Second Edition

Alexandra H. A. Dugdale MA, VetMB, PhD, PGCert(LTHE), DVA, Dip.ECVAA, FHEA, MRCVS
ChesterGates Veterinary Specialists CVS (UK), Ltd.
Cheshire, UK

Georgina Beaumont BVSc(Hons.), MANZCVS (Veterinary Anaesthesia,
Emergency and Critical Care), Dip.ECVAA, MRCVS
Christopher Beaumont Consulting Ltd.
Manchester Veterinary Specialists
Manchester, UK

Carl Bradbrook BVSc, CertVA, Dip.ECVAA, MRCVS
Anderson Moores Veterinary Specialists
Winchester, UK

Matthew Gurney BVSc, CertVA, PgCertVBM, Dip.ECVAA, MRCVS
Anderson Moores Veterinary Specialists
Winchester, UK

WILEY Blackwell

This edition first published 2020
© 2020 John Wiley & Sons Ltd

Edition History

John Wiley & Sons (1e, 2016)

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by law. Advice on how to obtain permission to reuse material from this title is available at <http://www.wiley.com/go/permissions>.

The right of Alexandra H. A. Dugdale, Georgina Beaumont, Carl Bradbrook, and Matthew Gurney to be identified as the authors of this work has been asserted in accordance with law.

Registered Offices

John Wiley & Sons, Inc., 111 River Street, Hoboken, NJ 07030, USA

John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial Office

9600 Garsington Road, Oxford, OX4 2DQ, UK

For details of our global editorial offices, customer services, and more information about Wiley products visit us at www.wiley.com.

Wiley also publishes its books in a variety of electronic formats and by print-on-demand. Some content that appears in standard print versions of this book may not be available in other formats.

Limit of Liability/Disclaimer of Warranty

The contents of this work are intended to further general scientific research, understanding, and discussion only and are not intended and should not be relied upon as recommending or promoting scientific method, diagnosis, or treatment by physicians for any particular patient. In view of ongoing research, equipment modifications, changes in governmental regulations, and the constant flow of information relating to the use of medicines, equipment, and devices, the reader is urged to review and evaluate the information provided in the package insert or instructions for each medicine, equipment, or device for, among other things, any changes in the instructions or indication of usage and for added warnings and precautions. While the publisher and authors have used their best efforts in preparing this work, they make no representations or warranties with respect to the accuracy or completeness of the contents of this work and specifically disclaim all warranties, including without limitation any implied warranties of merchantability or fitness for a particular purpose. No warranty may be created or extended by sales representatives, written sales materials or promotional statements for this work. The fact that an organization, website, or product is referred to in this work as a citation and/or potential source of further information does not mean that the publisher and authors endorse the information or services the organization, website, or product may provide or recommendations it may make. This work is sold with the understanding that the publisher is not engaged in rendering professional services. The advice and strategies contained herein may not be suitable for your situation. You should consult with a specialist where appropriate. Further, readers should be aware that websites listed in this work may have changed or disappeared between when this work was written and when it is read. Neither the publisher nor authors shall be liable for any loss of profit or any other commercial damages, including but not limited to special, incidental, consequential, or other damages.

Library of Congress Cataloging-in-Publication Data

Names: Dugdale, Alex, 1966– author. | Beaumont, Georgina, author. | Bradbrook, Carl, author. | Gurney, Matthew, author.

Title: Veterinary anaesthesia : principles to practice / Alexandra H. A. Dugdale, Georgina Beaumont, Carl Bradbrook, Matthew Gurney.

Description: Second edition. | Hoboken, NJ : Wiley-Blackwell, 2020. | Includes bibliographical references and index.

Identifiers: LCCN 2020013288 (print) | LCCN 2020013289 (ebook) | ISBN 9781119246770 (paperback) | ISBN 9781119246756 (adobe pdf) | ISBN 9781119246787 (epub)

Subjects: MESH: Anesthesia–veterinary | Handbook

Classification: LCC SF914 (print) | LCC SF914 (ebook) | NLM SF 914 | DDC 636.089/796–dc23

LC record available at <https://lcn.loc.gov/2020013288>

LC ebook record available at <https://lcn.loc.gov/2020013289>

Cover Design: Wiley

Cover Images: © Alexandra H. A. Dugdale

Set in 9.5/12.5pt STIXTwoText by SPi Global, Pondicherry, India

Contents

Preface *ix*

Acknowledgements *xi*

About the Companion Website *xiii*

- 1 Concepts and Mechanisms of General Anaesthesia** 1
- 2 Patient Safety** 7
- 3 Pain** 19
- 4 Sedation and Premedication: Small Animals** 55
- 5 Injectable Anaesthetic Agents** 77
- 6 Analgesic Infusions** 95
- 7 Intravascular Catheters/Cannulae: Some Considerations and Complications** 99
- 8 Inhalation Anaesthetic Agents** 117
- 9 Anaesthetic Breathing Systems and Airway Devices** 139
- 10 Anaesthetic Machines, Vaporisers, and Gas Cylinders** 167
- 11 Anaesthetic Machine Checks** 187
- 12 Local Anaesthetics** 191
- 13 Local Anaesthetic Techniques for the Head: Small Animals** 205
- 14 Local Anaesthetic Techniques for the Limbs: Small Animals** 215
- 15 Miscellaneous Local Anaesthetic Techniques: Small Animals** 237
- 16 Local Anaesthetic Techniques: Horses** 243
- 17 Muscle Relaxants** 259
- 18 Monitoring Animals during General Anaesthesia** 279

19	Troubleshooting Some of the Problems Encountered in Anaesthetised Patients	307
20	Inadvertent Peri-operative Hypothermia	313
21	Blood Gas Analysis	321
22	Lactate	337
23	Fluid Therapy	347
24	Electrolytes	377
25	Drugs Affecting the Cardiovascular System	393
26	Shock, SIRS, MODS/MOF, Sepsis	401
27	Gastric Dilation/Volvulus (GDV)	423
28	Equine Sedation and Premedication	427
29	Equine Heart Murmurs	443
30	Equine Anaesthesia	445
31	Equine Intravenous Anaesthesia in the Field and Standing Chemical Restraint	477
32	Donkeys	481
33	Ruminants: Local and General Anaesthesia	485
34	Lamoids (South American Camelids)	519
35	Pigs: Sedation and Anaesthesia	529
36	Rabbit Anaesthesia	541
37	Neonates/Paediatrics	547
38	Senescent/Geriatric Patients	551
39	Pregnancy and Caesarean Sections	555
40	Obesity	561
41	Dental and Oral Considerations	567
42	Ocular Surgery Considerations	571
43	Orthopaedic and Neurosurgery Considerations	575
44	Renal Considerations	579

45	Hepatic Considerations	583
46	Endocrine Considerations	587
47	Background to Neuroanaesthesia for the Brain	595
48	Cardiac Considerations	603
49	Respiratory Considerations	607
50	Respiratory Emergencies	611
51	Cardiopulmonary Cerebral Resuscitation (CPCR)	627
	Appendix A Canine Emergency Drug Doses	637
	Appendix B Feline Emergency Drug Doses	639
	Appendix C Equine Emergency Drug Doses	641
	Answers to Self-test Questions	643
	Index	651

Preface

Welcome to the second edition of *Veterinary Anaesthesia: Principles to Practice*, which only really came to fruition thanks to all the encouraging messages received in response to the first edition.

The updating and expansion took a little while longer than originally intended, but I and my co-authors hope

that this edition remains a 'go-to' source of information for veterinary nurses, veterinary practitioners, veterinary students, and particularly, post-graduate students studying for professional veterinary anaesthesia qualifications.

We also hope to share our fascination, passion, and sheer enjoyment of the subject and wish you all 'Happy Reading!'

Acknowledgements

My very grateful thanks are extended to my co-authors: Georgina Beaumont, Carl Bradbrook, Fran Downing, Nicki Grint, and Matt Gurney, and also to Kate Brooks for her amazing artwork.

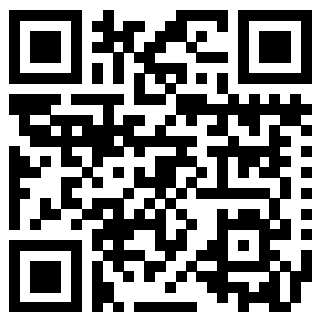
In addition, I would like to thank all my work colleagues and students, past and present, for their unending encouragement and support.

And finally, my heartfelt thanks to Jayadivya Saiprasad at Wiley-Blackwell for having more patience than anyone I know!

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/dugdale/veterinary-anaesthesia



Scan this QR code to visit the companion website

The website contains downloadable figures, appendices, self assessment questions and answers from the book.

1

Concepts and Mechanisms of General Anaesthesia

LEARNING OBJECTIVES

- To be able to define general anaesthesia.
- To be able to discuss general anaesthesia in terms of its component parts, i.e. the triad of general anaesthesia.
- To be able to define balanced anaesthesia.

1.1 Definitions

Anaesthesia literally means ‘lack of sensation/feeling’ (from *an* meaning ‘without’ and *aesthesia* pertaining to ‘feeling’). Therefore, general anaesthesia means global/total lack of sensations, whereas local anaesthesia relates to lack of sensation in a localised part of the body.

General anaesthesia can be defined as a state of unconsciousness produced by a process of controlled, reversible, intoxication of the central nervous system (CNS), whereby the patient neither perceives nor recalls noxious (or other) stimuli.

General anaesthesia is, however, often referred to as the state of the patient when the three criteria in the triad of general anaesthesia have been met.

1.1.1 The Triad of General Anaesthesia

- 1) **Unconsciousness**: no perception or memory (therefore including **amnesia**), of any sensory, or indeed motor, event.
- 2) **Analgesia** (or, more correctly in an unconscious patient, **antinociception**): can also be thought of as suppressed responses/reflexes to nociceptive sensory inputs.
- 3) **Suppressed reflexes**: autonomic (e.g. haemodynamic, respiratory and thermoregulatory) and somatic (e.g. proprioceptive reflexes such as the righting reflex).
 - Suppression of somatic reflexes can be useful, e.g. it can provide a degree of **muscular weakness/relaxation**.

- Suppression of autonomic reflexes can be a nuisance (see Chapter 18 on Monitoring), but **autonomic stability** can be a desirable component of anaesthesia and is often listed as a fourth component.

All these components could potentially be achieved in a patient following administration of a single ‘anaesthetic’ drug but, e.g. if that drug did not have very good analgesic properties, then large doses would be required to produce sufficiently ‘deep’ unconsciousness to reduce the response to noxious stimuli. Such deep anaesthesia is often associated with extreme depression of the CNS and homeostatic reflexes (Table 1.1).

An alternative approach, therefore, would be to produce each component (of the ‘triad’) separately by administering several drugs, each of which targets one component more specifically. This latter approach is theoretically advantageous because, by ‘titrating to specific effect’, relatively smaller doses of each individual drug tend to be sufficient, thereby minimising both each individual drug’s, and the overall, side effects. This ‘polypharmacy’ approach is often referred to as **balanced anaesthesia**.

1.1.2 Balanced Anaesthesia

The administration of a number of different drugs, each with different actions, given during the immediate peri-operative period, to produce an overall state of general anaesthesia, which fulfils the criteria of unconsciousness, analgesia, and muscle relaxation.

Table 1.1 Summary of effects of general anaesthesia.**Central Nervous System Depression**

- Loss of consciousness
- Damping of reflexes
 - Cardiovascular → Hypotension
 - Respiratory → Hypoventilation
 - Thermoregulatory → Hypothermia
 - Postural → Reduced muscle tone
- Central modulation of nociception (hopefully providing analgesia/antinociception)

Cardiovascular System Depression (→ Hypotension)

- Reflex (e.g. baroreflex) suppression (centrally and peripherally)
- Changes in autonomic balance
- Changes in vasomotor tone (drug effects, centrally and peripherally)
- Myocardial depression
 - Direct (drugs)
 - Indirect (e.g. hypoxaemia, hypercapnia [acidosis])

Respiratory Depression (→ Hypoventilation; resulting in hypercapnia/hypoxaemia)

- Reflex suppression (↓ventilatory response to ↑PCO₂ [↓pH], and ↓PO₂)
- Reduced respiratory muscle activity (↓ sighing and yawning)
- Alveolar collapse/small airway closure (atelectasis)
- Reduced functional residual capacity
- Ventilation/perfusion mismatch

1.1.2.1 Components of the Peri-operative Period

- **Pre-operative assessment:** patient stabilisation; provision of (pre-emptive) analgesia.
- **Premedication:** anxiolysis/sedation and initiation/continuation of analgesia provision if not already provided.
- **Induction** of anaesthesia.
- **Maintenance** of anaesthesia; provision of muscle relaxation; continuation of analgesia/antinociception provision.
- **Recovery** from anaesthesia (sometimes referred to as ‘reanimation’): aftercare; continuation of (‘preventive’) analgesia provision.

1.2 The Depth of General Anaesthesia

Some texts refer to various stages and planes of anaesthesia that try to mark the progression of the continuum between consciousness and death. When ether was used as the sole anaesthetic agent, five ‘degrees’ of progression through ever ‘deeper’ stages of anaesthesia in people, from consciousness to deep coma, were described by John Snow; Overton did similar for chloroform. Guedel developed Snow’s ideas further and, in 1937, produced a chart outlining the patient’s responses at each of four successive stages of diethyl ether anaesthesia. This was developed still

further by Artusio in 1954, who divided Guedel’s stage 1 into three planes.

Table 1.2, included purely for historical interest, describes the features of diethyl ether anaesthesia in the dog, after Guedel. The features of these stages and planes, however, do not necessarily apply similarly to other inhalant agents, and apply even less to injectable agents, to say nothing of the combination of inhalational and injectable agents that can be administered when balanced anaesthesia is practised. Furthermore, the chart is not necessarily transferrable to other species.

So, when we do not want to use ether, when we need to consider species other than dogs, when we prefer to practise ‘polypharmacy’ to achieve the desired state/depth of general anaesthesia, and when we add surgical stimulation to the anaesthetised patient (because depth of anaesthesia is not only related to the ‘dose’ of drug/s administered, but is also dependent upon the degree of stimulation [usually surgery] at the time), we should still monitor the patient’s physiological responses to, and status during, anaesthesia, which are considered in more detail in Chapter 18.

Although Table 1.2 is included purely for interest, it is important to note that during induction of anaesthesia, stage II (involuntary excitement/movement) may be witnessed; and during recovery from anaesthesia, all the stages are traversed in the reverse order, such that emergence excitement/delirium (stage II) may be observed.

1.3 Mechanisms of Action of General Anaesthetic Drugs

Compounds that exert general anaesthetic effects exhibit a wide diversity of chemical structure and can be administered by injection (usually intravenously), or by inhalation. Although a unifying target for their action has been sought, the diversity in their structure makes a single target site unlikely.

Nevertheless, Meyer (1899) and Overton (1901), independently, reported that anaesthetic potency was strongly correlated with lipid solubility which sparked interest in lipid membranes as the site of action. It was variously hypothesised that anaesthetic agents may exert a non-selective physical perturbation of a lipid site within the membrane or possibly perturb the volume or fluidity of the membrane itself. That physical dissolution of lipid-soluble agents within plasma membranes caused their expansion, sparked the ‘critical volume’ and ‘membrane expansion’ hypotheses, with some demonstration of pressure-reversal. The **lipid theory**, however, had several problems, including the fact that some isomers with identical lipid solubilities had different anaesthetic potencies, not all anaesthetic

Table 1.2 Stages of ether anaesthesia in the dog, after Guedel.

Stage of anaesthesia	Depression of CNS	MM colour	Pupil size	Eyeball activity	Breathing		
Stage I: stage of voluntary movement/excitement	?Sensory cortex	N / flushed	Small	Voluntary	Rapid/irregular		
Stage II: stage of involuntary movement/excitement 'delirium'	Motor cortex Decerebrate rigidity	Flushed	Dilated	Increased	Irregular		
Stage III (light surgical): plane 1	Midbrain	Flushed / N	Smaller	Increased	Slow/regular		
Stage III (moderate surgical): plane 2	Spinal cord	N	Miotic	Fixed, ventral rotation	Slow/regular		
Stage III (deep surgical): plane 3	Spinal cord	N / pale	Miotic	Ventral rotation	Large abdominal component		
Stage III (excessive surgical): plane 4	Spinal cord	Pale	Bigger		Abdominal/shallow		
Stage IV: paralysis (death follows respiratory and subsequent cardiac arrest)	Medulla	Pale/cyanotic	Mydriatic	Central	None/agonal gasps		

Stage	Pulse rate & BP	Palpebral reflex	Corneal reflex	Swallowing	Cough	Pedal withdrawal	Comments
I	Rapid/high	+	+	+	+	+	Analgesia?
II	Rapid/high	+	+	+	+	+	Unconscious
III (plane 1)	N/N	Poss slight	+	-	+	+	Some lacrimation persists
III (plane 2)	N/N	-	Slight	-	-	-	
III (plane 3)	Rapid/low	-	-	-	-	-	
III (plane 4)	Rapid (or slow)/low	-	-	-	-	-	Anal reflex poor
IV	'Shocky'	-	-	-	-	-	Anal/bladder sphincters relax

N = normal.

Changes tabled above refer specifically to those observed during ether anaesthesia in the dog.

Surgical stimulation may alter haemodynamic and respiratory variables via autonomic reflexes which persist into stage III, planes 2-3.

effects were reversible with applied pressure, and small temperature changes could also change membrane volume but without anaesthetic effects.

Although microtubule and even bubble theories have also been proposed, the biggest step forwards came with the discovery, by Franks and Lieb (1984) that anaesthetic agent potency correlated with inhibition of firefly luciferase, a large globular protein. This **protein theory** was a turning point for research and focused attention on proteins as potential targets for anaesthetic agents, in particular membrane receptors and ion channels that control ionic permeabilities.

Accepting that anaesthesia results from reversible CNS depression, it is plausible that either enhancement of inhibitory neurotransmission and/or inhibition of excitatory neurotransmission could produce a state of unconsciousness. Anaesthetics have therefore been proposed to act by modulation of such neurotransmission.

The main inhibitory neurotransmitter in the brain is gamma-aminobutyric acid (GABA), and the main excitatory neurotransmitter is **glutamate**. Between them, these neurotransmitters act at several key synaptic, ligand-gated ion channels: GABA at GABA_A receptors; and glutamate at N-methyl-D-aspartate (NMDA), alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and kainate receptors. (Although primarily ligand-gated, the NMDA receptor also displays a voltage-dependent magnesium block.) The majority of anaesthetic agents have been shown to interact with at least one of these targets.

Barbiturates, benzodiazepines, neuroactive steroids, propofol and, to a lesser extent, the volatile agents, have been shown to be **positive allosteric modulators of the GABA_A receptor**; that is, they produce little direct effect alone (except barbiturates and alfaxalone at higher doses), but enhance GABA-mediated chloride currents in post-synaptic membranes. This produces membrane hyperpolarisation with consequent reduction in neuronal activity, resulting in depressant effects. The different anaesthetic agents, however, appear to have different preferred sites of allosteric modulation within the receptor complex. Furthermore, variations in both receptor structure (many different isoforms exist), and distribution (not only

throughout the CNS but also between pre-, extra-, and post-synaptic sites), increases the possibilities for differential effects.

Ketamine, nitrous oxide and xenon are **NMDA receptor antagonists**, again having different sites of action within the receptor complex. These anaesthetic agents reduce the activation/permeability of NMDA receptors to calcium and sodium, thus reducing excitation of the neurone, resulting in overall depression. NMDA receptors are also involved in the development of nociceptive- and memory-processing, hence NMDA receptor antagonists produce analgesic, anti-hyperalgesic, anti-allodynic, and other effects.

More recently, the volatile anaesthetic agents have also been shown to interact with a family of so-called tandem-pore-domain or **two-pore-domain potassium (K_{2P}) channels**, which are widely expressed in the brain and have roles in regulation of sleep and membrane excitability generally. Local anaesthetic agents also have actions at these channels.

Finally, many anaesthetics have also been shown to affect other, unrelated, receptors, resulting in a multitude of possible side effects: these are often undesirable but may, on occasion, be beneficial. Some of these **other sites of action** include: glycine receptors, other glutamate receptors, cholinergic receptors, potassium channels (e.g. voltage-gated, ATP-sensitive, etc.), voltage-gated calcium channels, voltage-gated sodium channels, and others (e.g. hyperpolarisation-activated cyclic nucleotide-gated non-selective cation channels such as neuronal HCN1).

From this multiplicity of sites of action, we can begin to see how 'balanced' anaesthesia developed; that is, the administration of several different anaesthetic agents, each with a slightly different site/mode of action (or spectrum of activities), to produce an overall state of what we refer to as 'general anaesthesia', with, hopefully, fewer overall side effects because usually a lower dose of each agent suffices.

No matter which drug/s we use to produce general anaesthesia, our main objective is to maintain tissue perfusion, with delivery of oxygen and removal of waste products. If this fails, we can expect increased patient morbidity and mortality. There are no safe anaesthetics; there are only safe anaesthetists.

References

- Franks, N.P. and Lieb, W.R. (1984). Do general anaesthetics act by competitive binding to specific receptors? *Nature* 310: 599–601.
- Meyer, H.H. (1899). Zur Theorie der Alkoholnarkose. *Archiv für Experimentelle Pathologie und*

Pharmakologie 42 (2–4): 109–118. <https://doi.org/10.1007/BF01834479>.

- Overton, C.E. (1901). Studien über die Narkose zugleich ein Beitrag zur allgemeinen Pharmakologie. Jena, Switzerland: Gustav Fischer.

Further Reading

- Campagna, J.A., Miller, K.W., and Forman, S.A. (2003). Mechanisms of actions of inhaled anesthetics. *The New England Journal of Medicine* 348: 2110–2124.
- Foster, P. (2003). How deep is the sleep? Looking into anaesthesia depths. *Southern African Journal of Anaesthesia and Analgesia* 91: 6–8.
- Jones, R.S. (2002). A history of veterinary anaesthesia. *Annales de Veterinaria de Murcia* 18: 7–15.
- Kaul, H.L. and Bharti, M. (2002). Monitoring depth of anaesthesia. *Indian Journal of Anaesthesia* 46: 323–332.
- Lambert, D.G. (2010). Mechanisms of action of general anaesthetic drugs. *Anaesthesia and Intensive Care Medicine* 12: 1410143.
- Laws, D., Verdon, B., Coyne, L., and Lees, G. (2001). Fatty acid amides are putative endogenous ligands for anaesthetic recognition sites in mammalian CNS. *British Journal of Anaesthesia* 87: 380–384.
- Lugli, K.A., Yost, C.S., and Kindler, C.H. (2009). Anaesthetic mechanisms: Update on the challenge of unravelling the mystery of anaesthesia. *European Journal of Anaesthesiology* 26: 807–820.
- Mashour, G.A., Forman, S.A., and Campagna, J.A. (2005). Mechanisms of general anesthesia: From molecules to mind. *Best Practice and Research Clinical Anaesthesiology* 19: 349–364.
- Matta, J.A., Cornett, P.M., Miyares, R.L. et al. (2008). General anesthetics activate a nociceptive ion channel to enhance pain and inflammation. *Proceedings of the National Academy of Science, USA* 105: 8784–8789.
- Pascoe, P.J. and Steffey, E.P. (2013). Introduction to drugs acting on the central nervous system and principles of anesthesiology. In: *Veterinary Pharmacology and Therapeutics*, 9e (eds. J.E. Riviere and M.G. Papich), 183–210. Ames, Iowa, USA: Wiley-Blackwell.
- Schupp, M. and Hanning, C. (2003). Physiology of sleep. *British Journal of Anaesthesia: CEPD Reviews* 3: 69–74. **(Distinguishes sleep from general anaesthesia; useful information on effects of sleep deprivation for the anaesthetist).**
- Tranquilli, W.J. and Grimm, K.A. (2015). Introduction: use, definitions, history, concepts, classification and considerations for general anesthesia. In: *Veterinary Anesthesia and Analgesia: The Fifth Edition of Lumb and Jones* (eds. K.A. Grimm, L.A. Lamont, W.J. Tranquilli, et al.), 3–10. Ames, Iowa, USA: Wiley Blackwell.
- Urban, B.W. and Bleckwenn, M. (2002). Concepts and correlations relevant to general anaesthesia. *British Journal of Anaesthesia* 89: 3–16.
- Weir, C. (2006). The molecular mechanisms of general anaesthesia: dissecting the GABA_A receptor. *Continuing Education in Anaesthesia, Critical Care and Pain*. 6: 49–53.
- Whelan, G. and Flecknell, P.A. (1992). The assessment of depth of anaesthesia in animals and man. *Laboratory Animals* 26: 153–162.

Self-test Section

- Induction excitement occurs during which classical 'stage' of anaesthesia?
 - Stage I
 - Stage II
 - Stage III
 - Stage IV
- The main inhibitory and excitatory neurotransmitters in the brain are, respectively:
 - GABA and glycine
 - Glutamate and GABA
 - Glycine and glutamate
 - GABA and glutamate

2

Patient Safety

LEARNING OBJECTIVES

- Provide an overview of risk in veterinary anaesthesia.
- Provide an overview of medical error and how risk can be reduced.

2.1 Introduction

In 1999, the U.S. Institute of Medicine released its report 'To Err Is Human', highlighting the enormity of the problem of medical error and harm, and catapulting human patient safety firmly into both the public and health sector psyches worldwide. Human patient safety research subsequently rose by almost twofold in the first 5 years alone and the focus shifted from malpractice (fault/blame of the individual/team) to organisational culture (fault/blame of the organisation/procedures). As a result of the research in human patient safety, six main target areas have been identified:

- Hospital acquired infection
- Surgical complications
- Medication errors
- Device complications
- Identification errors
- Death

The World Health Organisation (WHO) has already targeted the first 3 of these with its 2005 'Clean Care is Safer Care', 2008 'Safe Surgery Saves Lives' and 2017 'Medication Without Harm' Global Patient Safety Challenges. The implementation of hand-washing protocols (Clean Care) and peri-surgery checklists (Safe Surgery) has had a significant, positive impact on patient safety with improved outcomes demonstrated in many countries (high, low, and middle income) and it is hoped that the Medication

Without Harm campaign will yield similar results by reducing medication errors.

Over the past 30 years, advancements in veterinary anaesthesia have led to improved anaesthetic risk for cats, dogs, and horses, particularly in ill patients (Table 2.1). However, veterinary patient safety has only recently emerged as a subject in its own right, over the last 10 years, with increases in patient safety publications and a shift in focus from primarily mortality risk reporting, to reporting on broader aspects of safety culture.

2.2 Anaesthetic Risk

Anaesthesia, and veterinary medicine in general, carries the risk of inadvertent medical error and/or harm. An understanding of the patient, human and system factors that contribute to anaesthetic error and risk, and those that may mitigate it, is of key importance to anaesthetists. Of all Veterinary Defence Society claims made against veterinary practices in the UK, only 2% related to anaesthesia (2013–2014). However, as in human medicine, focus group work indicates that the rate of actual significant events occurring is likely to be higher. The worst outcome of error and harm is death (usually of a patient, rarely the death of a member of staff or the public) and in the UK National Health Service there are approximately 7000 significant events or near misses for every catastrophic significant event. Similar data for veterinary medicine is currently unavailable.

Table 2.1 Peri-anaesthetic risk of mortality (%) reported for various species. Human mortality figures are shown as rate and percentage. Rates vary according to study year, country, and methodology. Healthy = ASA 1–2; ill = ASA ≥3.

Species	Overall risk (%)	Risk healthy (%)	Risk ill (%)	Country	Year
Cats	0.29	0.18	3.33	UK	1990
	0.24	0.11	1.4	UK	2008
Cats and dogs (pooled)	1.35	0.12	4.77	France	2012
Dogs	0.23	0.11	3.12	UK	1990
	0.17	0.05	1.33	UK	2008
	1.29	0.33	4.06	Spain	2013
	0.65	0.28	1.74	Japan	2017
Equine	1.9	0.9	7.8	International	2002
	1.6	0.9	—	International	2004
	1.1	0.9	1.6	UK	2016
Great apes	0.88	—	—	USA	1986
	1.35	—	—	UK	2007
Rabbits	1.39	0.73	7.37	UK	2008
	4.8	—	—	UK	2018
Guinea pigs	3.8	—	—	UK	2008
Ferrets	0.33	—	—	UK	2008
Hamsters	3.66	—	—	UK	2008
Chinchillas	3.29	—	—	UK	2008
Rats	2.01	—	—	UK	2008
Other small mammals	1.72	—	—	UK	2008
Budgerigars	16.33	—	—	UK	2008
Parrots	3.94	—	—	UK	2008
Other birds	1.76	—	—	UK	2008
Reptiles	1.49	—	—	UK	2008
Human					
Adult	8.8:100 000 (0.0088%)	—	—	Netherlands	2001
	4.7:100 000 (0.0047%)	0.4:10 000 (0.0004%)	—	France	2006
	1:57 023 (0.0018%)	7% of deaths	93% of deaths	Australia and New Zealand	2017
Child	1–7:10 000 (0.01–0.07%)	—	—	International	2011
	Neonate	19–24:10 000 (0.19–0.24%)	—	—	International

- **Significant events** are deviations from the usual medical care that are considered undesirable or that pose a risk of unnecessary harm to the patient, whether or not actual injury occurs as a result (also called safety incidents, critical incidents, or critical events)

- **Catastrophic significant events** are those that result in major harm or death of the patient
 - Major harm: sickness or injury leading to long-term incapacity/disability and/or increased duration of hospitalisation by more than 15 days and/or subject to statutory reporting

- Catastrophic harm: irreversible health effects and/or multiple permanent injuries or death
- A **near miss** is a significant event that specifically did not result in harm (on this occasion)

For the risk of mortality for selected species, see Table 2.1. The majority of veterinary peri-anaesthetic mortality occurs during maintenance of, or recovery from, anaesthesia. Systemically healthy animals (American Society of Anesthesiologists [ASA] physical status classification 1 and 2) are, unsurprisingly, at a lower risk of death than unhealthy (\geq ASA 3) animals. See below for more information on ASA classification. Veterinary peri-anaesthetic mortality is generally reported as a percentage, whilst human mortality is reported as a rate.

2.2.1 Factors Affecting Anaesthetic Risk

The factors affecting anaesthetic risk are listed below; not all are patient-related. The duration of anaesthesia and surgery are particularly related to risk.

- Patient's health
- Urgency: elective or emergency procedure
- Surgery: surgeon's experience, duration of surgery, type of surgery, gravity of surgery, surgery that involves the airway/lungs and interferes with the anaesthetist's 'space'
- Facilities available (surgical and anaesthetic): equipment, drugs, referral hospital, general practice or field
- Help available and experience of available personnel
- Anaesthetist: experience, duration of surgery (tiredness/vigilance/boredom), type of surgery
- Duration of anaesthesia and surgery

2.2.1.1 Patient Factors

Poor oxygen delivery to the tissues means trouble. Tissues susceptible to hypoperfusion/hypoxia are:

- Central nervous system (visual cortex)
- Myocardium
- Kidneys
- Liver

To reduce peri-operative morbidity and mortality, we must consider the effects of anaesthesia on any disease processes already present and the problems that those disease processes pose for anaesthesia.

We can improve the overall safety of anaesthesia with adequate pre-operative assessment, medical treatment and stabilisation of the patient where possible, and anticipation of the possible complications.

Familiarity with an anaesthetic technique is often a more important safety factor than the theoretical pharmacological advantage of an unfamiliar drug/technique.

A **careful history and thorough clinical examination** will reveal any problem areas. If there is time, further work-up may be warranted, such as laboratory tests, imaging or electrodiagnostics. The whole peri-anaesthetic period (including the pre-operative and post-operative periods) can then be tailored to suit each individual animal (see Chapter 18 on monitoring).

2.2.1.1.1 Body Mass

Is the animal overweight or too skinny, even debilitated? Is there a recent history of weight gain or loss? For obese animals, try to assess what their lean mass ought to be (See Chapter 40 on Obesity).

2.2.1.1.2 Age

Very young (neonatal) and very old (geriatric) animals may require dose adjustments (see Chapter 37 on neonates and Chapter 38 on geriatrics). Some chronologically old animals act as if they are still very young and some very young animals act as if they are very old, so be aware that the animal's chronological (true) age may not match its physiological/behavioural age. An animal's response to anaesthesia often matches its physiological age more than its chronological age.

2.2.1.1.3 Pre-existing Conditions

Hypovolaemia, cardiac disease, or respiratory disease may compromise the patient's ability to maintain adequate tissue perfusion/oxygen delivery, even before the physiological insult of anaesthesia.

Exercise tolerance is the **best indication of how compromised an animal is by its cardiac and/or respiratory disease**. Resting heart and breathing rates are also useful, especially in dogs.

Renal, hepatic, endocrine, neurologic, allergic, neoplastic, and musculoskeletal pathology/disease/dysfunction can all influence anaesthetic risk through derangements, or alterations, in:

- Homeostasis (glucose and electrolytes, acid-base, coagulation, thermoregulation, paraneoplastic syndromes, etc.)
- Drug pharmacokinetics and pharmacodynamics
- Drug interactions by pre-existing medicines or nutraceuticals (that may or may not be given under veterinary direction)
- Pain

2.2.1.1.4 ASA Physical Status Classification

Having completed the history and clinical examination, the animal is assigned to one of the ASA physical status classes (basic class descriptors are given below but, in line with the original medical classification system, a recent veterinary version has been devised with exemplars for

each class, see further reading), as this can help to decide whether anaesthesia can proceed, or whether further investigations or patient stabilisation are warranted first.

- 1) Normal healthy animal; no detectable underlying disease (cannot be an emergency)
 - 2) Mild systemic disease, but causing no obvious clinical signs or incapacity (animal compensating well)
 - 3) Severe systemic disease, causing clinical signs (animal not compensating fully, substantial functional limitations)
 - 4) Severe systemic disease that is a constant threat to life
 - 5) Moribund and not expected to survive without the procedure
- E) Add 'E' to any class to denote an emergency (where a delay in treatment significantly increases the threat to life or limb)

2.2.1.1.5 Stabilisation

Pre-operative support/stabilisation should be considered, which could involve:

- Anxiolysis/sedation
- Analgesia
- Pre-oxygenation/oxygen supplementation
- Fluid therapy/diuresis
- Attention to thermoregulatory requirements
- Medical support (e.g. for diabetes or cardiac arrhythmias)
- Surgical procedures (e.g. tracheostomy, chest, or pericardial drainage)

Appropriate monitoring should be considered and may be instigated in the pre-operative phase. Take extra care with very young, old or thin animals, and those with endocrinopathies or liver disease. Hypothermia will delay recovery. Remember that hypoglycaemia may be a confounding factor in very young animals, those with insulinomas, or poorly controlled diabetes mellitus.

2.2.1.1.6 Pre-anaesthetic Fasting

Feeding has traditionally been suspended for varying durations (averaging 6–12 hours), before premedication/anaesthesia because of the risk of vomiting or regurgitation, and subsequent aspiration. Whilst some agents can stimulate vomiting (e.g. morphine, α_2 agonists), this usually occurs during the 'onset' of sedation or premedication. Occult (undetected) gastro-oesophageal reflux (GOR), however, appears to be more common than regurgitation (where material refluxing from the stomach through the cardia becomes visible in the pharynx); but both can result in oesophagitis and even oesophageal stricture, and both pose the additional risk of aspiration

pneumonitis. GOR has been reported to occur in around 25+% of dogs and around 12% of cats; regurgitation in around 1% of patients, and aspiration/chemical pneumonitis (Mendelson's syndrome) in <1%. Material of adverse pH reaching the nasopharynx undetected can also result in choanal stricture.

The barrier pressure across the cardia (i.e. the difference between lower oesophageal sphincter pressure [LOSP] and intragastric pressure [IGP]) is important in determining whether GOR/regurgitation may occur. (Species differences exist in the muscular composition of the lower oesophagus: striated muscle is present in dogs and ruminants, whereas only smooth muscle is present in horses and cats.)

Lowered barrier pressure (increased risk of GOR/regurgitation), occurs with either decreased LOSP and/or increased IGP. Opioids, sedatives, anaesthetic, and ancillary drugs can reduce LOSP (and many of these also delay gastric emptying); and a recent large meal or drink can increase IGP, but so can factors increasing intra-abdominal pressure in general. Increased gastric acidity can also reduce LOSP, whereas reducing the acidity of gastric contents (with food or antacids) can help to increase LOSP.

Suggested **predisposing factors for GOR/regurgitation appear to include: opioid administration** (butorphanol > morphine >> pethidine), **age** (very young and very old), **increased intra-abdominal pressure** (e.g. obesity, pregnancy, abdominal surgery), **history of gastrointestinal disease** (including brachycephalics), **deep-chested conformation**, and **multiple changes in patient position** (common in orthopaedic patients undergoing pre- and post-operative imaging). Use of laryngeal mask airways has been reported to increase the risk of occult GOR in kittens.

As for aspiration pneumonitis, this most commonly appears to follow occult GOR. Whilst predisposing factors facilitating aspiration might include use of supra-glottic airway devices compared with suitably-inflated cuffed endotracheal tubes, the severity of the pneumonitis depends not only upon the volume, physical composition and pH of aspirated material but also on other factors, including the patient's ASA physical status.

A single optimum duration for denying access to food before anaesthesia/surgery is unlikely to exist because it will depend upon: the species (dogs appear more prone than cats), the individual patient, its health status (including pre-existing pathologies which may predispose to GOR, regurgitation or delayed gastric emptying), what meal size/s and intervals are usually adhered to, and what type of food is usually fed (wet or dry; caloric content and composition in terms of fat/carbohydrate [including fibre types]/protein).

At the spring 2019 meeting of the Association of Veterinary Anaesthetists (AVA), lively debate was held

regarding opinions for food and water withhold before anaesthesia. This was sparked by recent changes to human guidelines following recognition that prolonged fasting is associated with dehydration, thirst, hunger, irritability (patients are often referred to as being ‘hangry’ = hungry and angry), and adverse metabolic consequences including the promotion of insulin resistance. Although no veterinary consensus was reached due to the many factors involved, one suggestion was:

- For dogs and cats, a light meal (supplying up to around half of the daily energy requirements), of ‘wet’ (canned) food, of low fat and low fibre composition (so as not to increase gastric emptying time), may be offered (4-)6 hours before premedication. (If dry, fatty, or high protein content food is given, then 10+ hours should be allowed before premedication.) (For puppies and kittens, see Chapter 37.)
- Water should be freely available: up to the time of premedication (or, if a restriction time is felt necessary, then access to water should be allowed up to one to two hours before ‘anaesthesia’ [which could be interpreted as premedication or induction]).

Should regurgitation be observed, then suction, with or without oesophageal warm-water lavage (this may be preferred for acidic material), may be indicated (taking care to protect the airway). This author also flushes the nasal passages with warm water in a retrograde fashion, ensuring the nasopharynx is cleared of material, to reduce the risk of choanal mucosal damage and subsequent stricture.

Delayed gastric emptying (due to stress, anaesthesia, and surgery), might also increase nausea and possibly delay resumption of feeding post-operatively. The subject of when to re-introduce feeding post-operatively, and with food of what quantity, frequency, and composition, remains to be discussed.

See Chapters 30, 33, and 35 for horses and farm species.

2.3 Error

Medical error is the failure to correctly complete an intended action, or the implementation of a wrong plan to achieve the goal. Errors may be **latent**, due to the system (e.g. poorly designed systems/procedures/buildings), or **active**, due to the people (e.g. dose/technical error).

High-reliability organisations (e.g. nuclear power, aviation, rail networks) recognise that people will always be fallible, particularly in high risk/stressful/emotional situations, and so they focus on constructing systems with as few latent risk factors as possible so that people are less

likely to find themselves in a situation where they can err. Safe industries also assume that errors will, at some point, occur and so they build in vigilance and damage-control to these systems. This protects employees, clients, and the public from error by:

- Minimising the risk of an error occurring in the first place
- Vigilantly looking for errors
 - Errors are detected quickly and reliably, allowing rapid correction/management
 - Errors are reported and reviewed to enable system improvements, staff training (or re-training) and further research/learning
- Actively managing errors to reduce their impact

It is interesting to note that medical professionals perceive themselves as less fallible than aviation pilots do. This denial of normal, human fallibility reflects a problematic discrepancy in safety culture and attitude towards risk between medicine and high-reliability organisations. It also sets medical professionals up for failure and the emotional and psychological distress that accompanies it. Veterinary professionals have similar levels of perfectionism, workplace complexity, moral conflict and expectations of professional infallibility (for themselves and each other) as our medical counterparts, and these unrealistic professional expectations contribute to poor mental health and career dissatisfaction. Recent safety culture studies have revealed significant overlap and commonality between veterinary and medical errors.

2.3.1 Latent (System) Error

Latent errors are often foreseeable and include:

- Communication
 - Within and between teams; written, oral and behavioural
 - Clinical handovers and transitions are particularly susceptible to communication error, especially when performed informally/without structure
 - Unwillingness or failure to ask for help
- Non-technical skills
 - Non-technical skills are cognitive, social, and personal resource skills that complement technical skills
 - The importance of non-technical skills in patient safety culture and efficient, edifying working relationships is well documented in high reliability organisations and human medicine
- Leadership (individuals or organisations)
 - Failure to take charge of situations or to clearly allocate/identify roles

- Failure to acknowledge strengths/contributions of team members
- Failure to acknowledge weaknesses (of team members and self) and provide appropriate support/supervision
- Weak industry regulation
- Product or equipment design flaws
 - Standards for veterinary products are not as robust or well developed as for human products, e.g. there is no requirement for veterinary anaesthetic machines to be fitted with hypoxic guards
 - Many drugs are similarly packed and look identical in an unlabelled syringe
 - If possible, similarly packaged or named products should be separated
- Productivity
 - Time and financial pressures
 - Inefficient and under-staffing
 - Over-time and shift structure
- Organisational failure
 - Lack of safety systems and protocols
 - Institution of policies or procedures that are not fit for purpose
- Owners
 - Poor compliance or refusal of recommended treatment
 - Loss to follow-up or further investigations
 - Conflicting owner: animal needs
 - Legally, animals are personal property; laws pertaining to property rights may conflict with laws safeguarding animal welfare, resulting in marked moral stress
- Veterinary specific
 - Animal behaviour, e.g. aggression or ability to out-run the veterinarian
 - Inability to control the surroundings, e.g. procedures performed in the field
 - Knowledge-based: the individual has never known the correct solution and has come to the wrong conclusion whilst attempting to work the problem out
- Conformational bias: the individual sees what they expect to see instead of what is actually there
- Memory failure
- Individual factors
 - Fatigue: 17 hours without sleep results in a reduction of psychomotor performance equivalent to a blood alcohol concentration of 50 mg/dl; and 24 hours of sleep deprivation reduces performance equivalent to a blood alcohol concentration of 100 mg/dl (the current UK legal driving limit is 80 mg/dl)
 - Illness, emotional distress, and stress all significantly impair decision-making
- Lack of technical ability
 - Failure of supervision and support, particularly of junior staff
 - Attempting a task beyond the training, capability or experience of the individual; this can be accompanied by emotional stress if the individual is aware of the deficiency but feels pressured into continuing with the course of action regardless
- Deliberate negligence/harm
 - Perpetrators of deliberate negligence/harm should be formally disciplined and criminal charges may be appropriate; thankfully this is an uncommon cause of error and harm

Most significant events are caused by multiple errors (the Swiss-cheese model), often arising from different levels within the organisation. Active (human) error, communication, and leadership are consistently the top three contributors to medical errors. Initial research indicates that this may also hold true for veterinary errors. An understanding of the types and patterns of error in veterinary medicine provides the opportunity to develop interventions to minimise risk and may highlight individuals that require additional (re)training, or systems that require review and overhaul.

2.3.2 Active Error

Active errors are often difficult to foresee and include:

- Cognitive limitations
 - Slips: the action is not carried out as planned, often due to distraction
 - Lapses: the action is missed out entirely, may be due to distraction but includes deliberate omissions where the individual believes it will not cause harm
 - Mistakes: the wrong action is carried out but the individual believes it to be the correct action
 - Rule-based: the incorrect application of previous learning

2.3.3 Cost of Error

Error has medical, psychosocial, and financial costs. The medical cost of error is borne through patient morbidity and/or mortality, i.e. the harm. The psychosocial cost is the emotional and psychological impact of error on the patient, owner, and staff member(s). It is important to remember that staff may become the hidden (second) victim(s) of an error and can experience significant emotional and psychological distress requiring appropriate support and

counselling. The financial cost lies in rectifying the error, which often requires additional treatment/hospitalisation, and the cost of litigation against the practice that erred.

Of the estimated 237 million medication errors occurring in the UK each year, most (72%) have little or no potential to cause harm and many are detected before reaching the patient (near misses). Despite this, it is estimated that medication errors alone cost the UK National Health Service £98.5 million per year, requiring an additional 181,626 hospital bed-days, directly causing the death of 712 people and contributing to the death of an additional 1,708 people.

As there is no mandatory reporting in veterinary medicine, accurate information on the true magnitude and cost of veterinary error is, at this time, lacking.

2.3.4 Responding to Error

When responding to an error, the following questions should be considered:

- Was the harm deliberate?
 - If so, disciplinary action is required
- Were staff physically or mentally impaired (e.g. fatigue, distress, substance abuse, injury)?
 - If so, the staff involved require support
 - Disciplinary action may or may not be required (and may be harmful in some instances)
- Were established policies, protocols, and procedures followed correctly?
 - If not, the staff involved require additional (re)training
 - Disciplinary action may or may not be required (and may be harmful in some instances)
- Would the same error have occurred if different, similarly skilled staff members had been exposed to the same circumstances?
 - If so, the system causes/allows error and accountability for the error is shared with the organisation's leadership
 - The system must be changed so that harm is avoided rather than encouraged
- Has the same, or a similar, harm occurred previously?
 - If so, the repetitive safety failing must be addressed whether by systems overhaul if the system is at fault or, if a particular staff member has repeatedly caused the harms, by performance review

2.4 Safety Culture

The safety culture of an organisation is the product of individual and group values, attitudes, perceptions, competencies, and patterns of behaviour that determine the

commitment to, and the style and proficiency of, an organisation's health and safety management. Organisations with a positive safety culture are characterised by communications founded on mutual trust, by shared perceptions of the importance of safety and by confidence in the efficacy of preventive measures.

ACSNI Human Factors Study Group (1993)

Committed and engaged management, active employee participation and honest and blame-free communication are crucial for a healthy safety culture. Organisations with healthy safety cultures: monitor and review significant errors, proactively implement changes and training to reduce future risk, and cultivate a no blame environment. Checklists, significant event reporting, drills/training, and established, written procedures improve safety whilst also aiding in the development of a healthy safety culture. For example:

- Purposeful, regular team training (e.g. practical resuscitation training, handover training) improves teamwork, communication, and non-technical skills
- Checklists help to prevent errors due to cognitive limitations whilst also developing teamwork and non-technical skills

2.4.1 Checklists

Checklists help to prevent significant events by avoiding reliance on memory and vigilance, and by standardising common procedures. They promote teamwork, communication, and flat safety hierarchies. Checklists have been shown to improve outcomes in a variety of veterinary applications including:

- Small animal anaesthesia and surgery
- Equine anaesthesia
- Cardiology
- Clinical pathology laboratories
- Patient discharges to the owner

Checklists are used to ensure that the basic, common tasks that should always be completed are always completed (e.g. open the adjustable pressure limit valve before the animal is connected to the breathing system). A good checklist should be:

- Concise and focused on critical interventions/events, with no more than nine steps
- Brief, taking no more than 60 seconds to complete
- Actionable: every step is linked to a clear action
- A verbal exercise undertaken between multiple team members

- Modified only following collaboration with representatives of all of the team members who will be using it
- Tested before it is formally launched; feedback from testing feeds into additional improvements and facilitates collaboration
- Integrated into the existing framework; using the checklist should be the norm

Peri-operative checklists pertinent to anaesthesia have been developed by the WHO and the AVA, in conjunction with Jurox UK, and are freely accessible online. These documents comprise three separate checklists: one for before induction ('sign in'), one for before first incision ('time out') and one for surgery end, before everyone leaves theatre ('sign out'). The AVA has also produced a comprehensive anaesthetic workstation checklist for use daily and an abridged checklist for use between patients.

2.4.2 Significant Event Reporting

Significant event reporting is critical to the advancement of safety culture and development of strategies and tools that promote safety in veterinary practice. Significant event reporting acts as a sentinel, providing an early warning system for potential problems, before significant harm occurs. In human medicine, up to 20% of acute care patients experience at least one significant event during their hospitalisation. Of these significant events, ~65% result in minimal or no harm, providing an opportunity for corrective measures to be made before a patient is permanently disabled, or killed.

Many, larger veterinary practices and corporates have begun to implement significant event reporting systems. In the UK, the VetSafe voluntary reporting system was launched in 2019 to capture, report, and develop solutions for veterinary errors and harm. The VetSafe reporting system is operated by the Veterinary Defence Society Ltd (UK) and is available to their members. In addition to the anonymised collation and analysis of practice incidents, reports made via VetSafe form the first notification to the insurer (i.e. the Veterinary Defence Society) of an event that might result in a claim against the practice.

2.4.3 Drills/Training

Scenario training enhances teamwork, communication, situational awareness, and practical performance during high stress events, such as cardiopulmonary-cerebro resuscitation

(CPCR). Standardised drills/training also aim to improve adherence to protocols and guidelines

2.4.4 Established Protocols and Procedures

Organised, well-documented, written protocols and procedures aim to reduce the likelihood of cognitive limitation contributing to error by providing staff with evidence-based, safe and appropriate frameworks to work within. Familiarisation with the protocols and procedures and clear cognitive aids (algorithms, checklists, dosage charts, etc.) enable staff to provide a consistent standard of care across the team. Examples of written protocols and procedures include (non-exhaustive):

- RECOVER (REassessment Campaign On VEterinary Resuscitation) guidelines for CPCR (2012)
- Handover mnemonics, e.g. I-PASS (Illness severity, Patient summary, Action list, Situation awareness and contingency planning, Synthesis by receiver) or SBAR (Situation, Background, Assessment, Recommendation)
- Anaesthetic workstation checks
- Medication checks, e.g. double-checking doses and dispensed medication
- Controlled drug management (including disposal)
- Treatment pathways, e.g. management of hypotension, patient temperature, local anaesthetic toxicity, etc.
- Standard procedures for managing clinical scenarios, e.g. aggressive patients, intravenous catheter care, fire action plans, etc.

2.5 Where to Get Help

Humans make errors. Veterinarians make errors. When we do err, we may suffer emotional and psychological distress. This is called **second victim syndrome**. This includes feelings of isolation, shame, denial, and a sense of being unsupported. It is important that vets affected by these issues are able to talk about them in a safe way and receive the support they need.

In many countries, independent, confidential support is available 24 hours a day, 365 days a year, from organisations such as the Samaritans (UK, Republic of Ireland, Australia, New Zealand, Thailand, Singapore, Hong Kong) and Lifeline (Australia).

Reference

Health and Safety Commission (1993). ACSNI Study Group on Human Factors. 3rd Report: Organising for safety. Health and Safety Commission. London: HMSO.