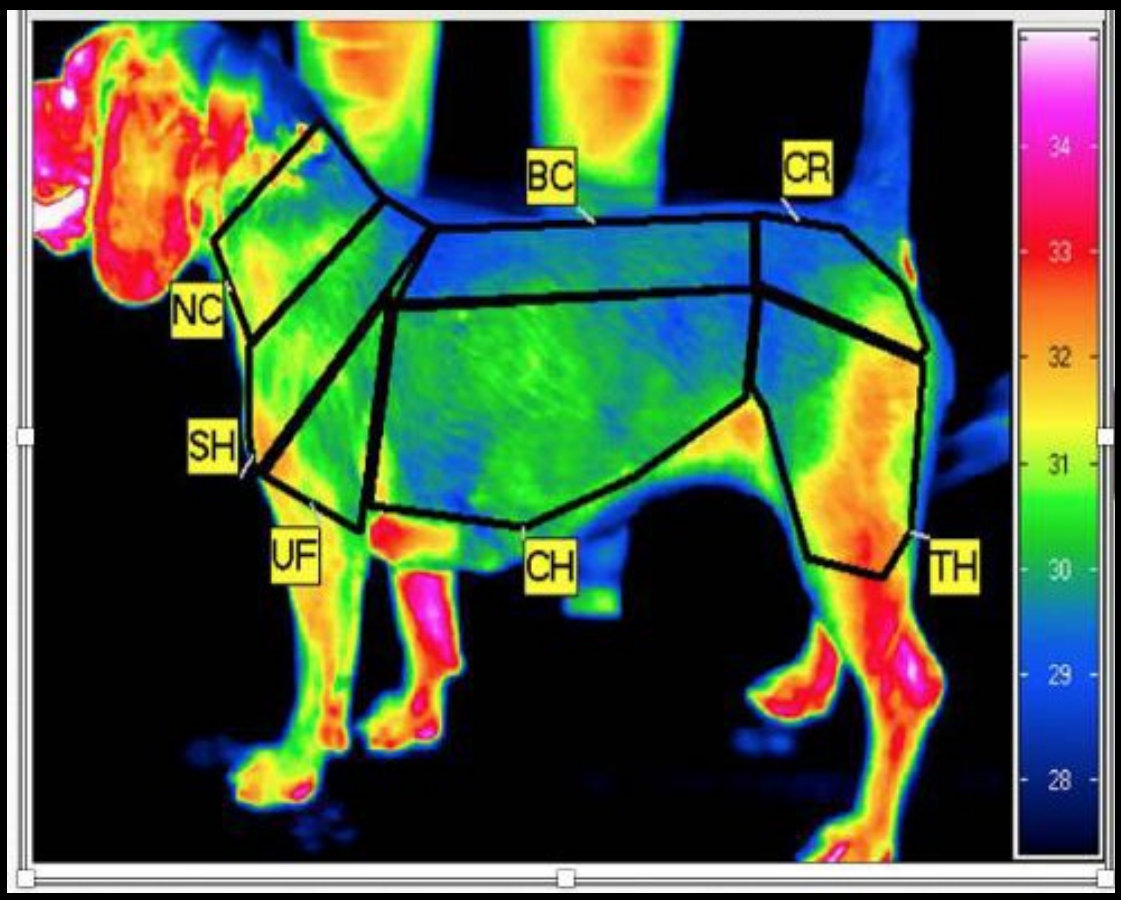
Australian
Veterinary Practitioner
Volume 54 (2) June 2024



In this edition:

AVA AUSTRALIAN VETERINARY ASSOCIATION
AUSTRALIAN SMALL ANIMAL VETERINARIANS

Galliprant™

The next generation in canine osteoarthiritis pain relief is here

Available now from your preferred wholesaler

Galliprant works differently

- First in class, non-COX inhibiting NSAID
- A unique mode of action
- Selectively blocks the EP4 receptor, a key mediator of OA pain

Recommend Galliprant as first-line treatment

INDICATION For the treatment of pain and inflammation associated with osteoarthitis in dogs. Please read product leaflet for full instructions.

Elanco™

www.elanco.com.au

Contents

ASAV Office

Elaine Robertson
Mob: +61 2 9431 5082
Email: elaine.robertson@ava.com.au

Editor
Professor Caroline Mansfield
Email: editor.avp@ava.com.au

AUSTRALIAN SMALL ANIMAL VETERINARIANS

The Australian Veterinary Practitioner is the official journal of the Australian Small Animal Veterinarians. The AVP is an independently refereed clinical journal, published four times per year to further the clinical and educational interests of veterinary practitioners and students throughout Australia. The AVP is abstracted by: Biological Abstracts; CAB International; Current Contents (Agriculture etc); First Move – Veterinary Librarian; Index Veterinarius; Science Citation Index; Small Animal Practice; Veterinary Bulletin; Veterinary Reference Service; VIN. Guidelines for authors can be found at https://www.ava.com.au/siteassets/library/journal/australian-veterinary-journal/australian-veterinary-practitioner---guide-to-authors-12.9.22.pdf

To submit an article to the AVP, email the manuscript to editor.avp@ava.com.au as an attachment. All scientific material is peer reviewed. All published material is subject to copyright. Every effort is made by the publishers, reviewers and editor to ensure that inaccurate or misleading data, opinion or statement is not published in this journal.

ISSN 0310 - 138X

MESSAGE FROM THE EDITOR

This edition of the Australian Veterinary Practitioner is the second for the year and includes three interesting papers as well as a review of open or free access journal articles that may be of relevance to those in clinical practice.

The hyperlinks for these papers are included in the review, and readers are encouraged to look further into the studies that may interest them.

Starting the original papers is a consensus statement developed by the anaesthesia and analgesia chapter of the Australian and New Zealand College of Veterinary Scientists. The review follows on from previous work this group has done trying to standardise anaesthesia protocols for small animal patients in Australia. This paper focuses on anaesthetic monitoring, including the minimum number of personnel, method of training and common parameters required to monitor safely. This is a useful article for veterinary clinics to refer to when upskilling and maintaining appropriate anaesthesia protocols.

This is followed by two papers, the first focusing on an unusual cardiac arrhythmia identified in a dog and the appropriate management of this. The third paper is focused on the greyhound, and focuses on thermoregulation. As well as being incredibly relevant for those practitioners who work with greyhounds, some of the principles discussed may be useful for managing heat stress and strike in other breeds.

Happy reading, and as always feedback and suggestions to improve this journal are welcome.

Best wishes,
Caroline Mansfield, Editor AVP

AUSTRALIAN SMALL ANIMAL VETERINARIANS

VETERINARY BUSINESS GROUP

BEATS, BREATHS & BUSINESS

49th ASAV Annual Conference 2024

Together with the Veterinary Business Group

Novotel Twin Waters, Sunshine Coast, Qld

12-15 August 2024

EARLY BIRD REGISTRATION **NOW OPEN**

Find out more

AVA

Journal review AVP Issue 2, 2024

All the papers summarized here are open access (so freely available) peer reviewed articles that have been published to date in 2024. The ones chosen hopefully have the most clinical impact and will be useful to those in practice.

JOURNAL OF VETERINARY INTERNAL MEDICINE

Clinical manifestations of chronic pancreatitis in English cocker spaniels - Coddou - Journal of Veterinary Internal Medicine - Wiley Online Library

This is a UK study of English cocker spaniels with chronic pancreatitis. To date a genetic deficiency has not been identified for pancreatitis in this breed, but the group from Cambridge have been focusing on immune mediated complexes, similar to a form of autoimmune pancreatitis seen in people. In this study, they showed that chronic pancreatitis in this breed was also associated with other immune-mediated organ disorders such as (potentially) glomerulonephritis and keratoconjunctivitis sicca (KCS). The authors recommend screening for these other conditions proactively in these dogs. It is unclear whether a similar finding in non-UK cocker spaniels would exist.

Thyroid function tests during nonthyroidal illness syndrome and recovery in acutely ill dogs - Bolton - 2024 - Journal of Veterinary Internal Medicine - Wiley Online Library

One area of endocrinology that is complex is interpreting thyroid function in dogs with other disease or medications that can influence the thyroid axis. This conundrum is increased with the inclusion of T4 in many 'sick' dog biochemistry panels both in house and through commercial pathology laboratories. In this prospective observational study serum total thyroxine (TT4) concentration was measured in 103 dogs within 4 hours of admission. If below the reference interval (RI), subsequent serum samples were obtained every 24 hours from admission until discharge (acute phase) and at 2 weeks and 4 weeks after discharge (recovery phase). Additionally, free thyroxine (fT4), total 3,5,3′-triiodothyronine (TT3), and thyroid-stimulating hormone (TSH) concentrations were determined.

ALL dogs had TT4, TT3 and free T4 below reference interval at the time of admission, whilst all dogs had normal TSH concentrations. By the time of discharge, 20% dogs had decreased TT4 with none having subnormal free T4. All dogs had all parameters within reference interval by 4 weeks post discharge.

Based on this, the authors recommend that if T4 is low during acute illness, there should be a period of 2-4 weeks before retesting free T4.

The impact of single-dose trazodone administration on plasma endogenous adrenocorticotropic hormone and serum cortisol concentrations in healthy dogs - Brown - 2024 - Journal of Veterinary Internal Medicine - Wiley Online Library

This study showed that a single dose of trazadone 1 hour before testing did not affect basal cortisol or the results of an ACTH stimulation test in 14 healthy dogs.

Further work is needed to know whether this holds true for sick dogs, or when dogs have received trazadone chronically.

Hypothalamic-pituitary-adrenal axis recovery after intermediate-acting glucocorticoid treatment in client-owned dogs - Del Baldo - 2024 - Journal of Veterinary Internal Medicine - Wiley Online Library

Another clinical conundrum is when to test the adrenal axis in dogs that have received glucocorticoids. There are many recommendations as to when to tests after corticosteroids have been discontinued, ranging from 4-6 weeks when there has been short term administration (< 4 weeks) up to 12 weeks if longer administration. Most studies have focused on healthy dogs.

In this prospective study, an ACTH stimulation test and endogenous ACTH concentration, were performed in 20 sick dogs at the time of glucocorticoid discontinuation, at T0 (2-6 days and then every 2 weeks (e.g., T1, T2, T3) until HPA axis recovery was documented.

The median time of HPA axis recovery was 3 days (range, 2-133 days). Eleven of 20 dogs showed recovery of the HPA axis at T0, 6/20 at T1, and 1 dog each at T2, T5, and T9. Most importantly, dose and duration of treatment were not correlated with timing of HPA axis recovery. Neither did tapering of prednisolone versus suddenly stopping impact this.

This suggests that most dogs experienced HPA axis recovery within a few days after glucocorticoid discontinuation. However, 2/20 dogs required >8 weeks. Therefore, screening the HPA axis can occur earlier than previously thought in most dogs, with caution being needed to diagnose hypoadrenocorticism as some dogs may take a long time to recover their normal HPA axis.

Concurrent hepatopathy in dogs with gallbladder mucocele: Prevalence, predictors, and impact on long-term outcome - Jablonski - 2024 - Journal of Veterinary Internal Medicine - Wiley Online Library

The incidence and diagnosis of gall bladder mucocele have increased recently, but there remains little to determine about the underlying prognosis. In this multi-centre study from the US, 98% of dogs (51/52) had at least one hepatic abnormalities on histology. Prognosis appeared to be inversely proportional to the presence of hepatic fibrosis but not inflammation. It is unclear whether the presence of fibrosis is a direct consequence of GBM however.

Comparison of timing of relapse in dogs with nonassociative immune-mediated hemolytic anemia, thrombocytopenia, or polyarthritis - Sparrow - 2024 - Journal of Veterinary Internal Medicine - Wiley Online Library

This retrospective review of one hundred sixty client-owned dogs (73 with IMHA, 55 with ITP, 32 with IMPA) was performed to determine the relapse rate, and if there were known trigger.

Relapse rates at 12 months was highest for IMPA (35%) compared to IMHA (11%) or ITP (11%). Relapse rate at 24 months was 41% for IMPA, 18% for IMHA, and 23% for ITP. Ninety percent of IMPA relapses occurred in the first 12 months after diagnosis, compared with 56% for IMHA and 50% for ITP.

Also importantly, vaccine administration after diagnosis was not associated with relapse.

ACVIM Consensus Statement on the management of status epilepticus and cluster seizures in dogs and cats - Charalambous - 2024 - Journal of Veterinary Internal Medicine - Wiley Online Library

An amazing review of the pathophysiology of status and cluster seizures in companion animals. This is a fantastic resource for practitioners to utilise, incorporating in and out of hospital recommendations.

The main recommendations from the panel for initial control of seziures include:

- Intravenous (IV; in-hospital settings) and intranasal (IN; out-of-hospital and in-hospital settings) routes currently are considered the most effective and safest methods of benzodiazepine (BZD) administration.
- Rectal diazepam is less supported by evidence.
- A BZD bolus should be considered effective if seizure cessation occurs <5 minutes after administration and seizures do not relapse in <10 minutes after cessation.
- In the case of recurrent SE or SE that does not cease after the first bolus, a second bolus of BZD should be administered after a minimum 2-minute interval.
- If seizures persist after 2 BZD boluses, then (i) in case of recurrent SE, administration of another BZD bolus followed immediately by a BZD IV CRI should be instituted, and (ii) if SE does not cease, a final BZD bolus should be administered followed by second-line interventions.
- Midazalom is the preferred BDZ for both cats and dogs to use as a CRI (diazepam CRI should be avoided in cats).

Second line treatment options include levetiracetam, phenobarbital and fosphenytoin.

- IV levetiracetam and phenobarbital typically are started as second-line medications when the first-line treatment has failed to terminate the seizures, however these medications also can be administered earlier, regardless of the response to first-line treatment, with the aim to maintain adequate seizure control in the short- and long-term (particularly in cases diagnosed with non-cryptogenic epilepsy).
- IM or rectal administration of levetiracetam is an option if there is no IV route.
- An IV bolus of fosphenytoin can be administered in dogs when there is no or inadequate response to levetiracetam or phenobarbital

Third line treatment consists of anaesthetic medications, and a four-step approach is clearly and easily described, with the use of ketamine being the main initial component.

Similarly, a clear approach to management of cluster seizures is described. In this aspect, the use of levetiracetam pulse treatment is recommended initially.

JOURNAL OF FELINE MEDICINE AND SURGERY

Use of orally administered dexmedetomidine to induce emesis in cats - Kathleen M Maxwell, Adesola Odunayo, Charlotte Wissel, 2024 (sagepub.com)

Inducing emesis in cats can be difficult with the current options available. This paper outlines successful treatment in 5 out of 6 cats with an oral dose of dexmedetomidine at 20 µg/kg. All cats were sedated following administration, but no other adverse effects were documented. This may therefore be a useful method for inducing emesis in cats.

Neuropathic pain in cats: Mechanisms and multimodal management - Clare Rusbridge, 2024 (sagepub.com)

This paper is a useful descriptive reference about pain, particularly neuropathic pain, in cats. The pathophysiology of acute and chronic pain is clearly detailed. Specifically, the phenomenon of central sensitisation is discussed; whereby a heightened sensitivity to pain can persist in an animal even after the initial injury or inflammatory event has resolved. This phenomenon, along with how chronic pain may manifest as behavioural changes, is clearly demonstrated using case examples. Discussion regarding multimodal management of pain, including pharmacological manipulation and methods such as acupuncture is extensive and clinically relevant.

Frequency of diabetic remission, predictors of remission and survival in cats using a low-cost, moderate-intensity, home-monitoring protocol and twice-daily glargine - Susan Gottlieb, Jacquie S Rand, Stephen T Anderson, 2024 (sagepub.com)

This paper outlines a method by which owners can manipulate insulin dosing of cats using minimal intervention thereby reducing costs of continuous monitoring or veterinary clinical visits. With this protocol, owners obtain a blood glucose reading prior to insulin administration and use this to adjust the insulin dosage. Interestingly, all blood glucose measurements were performed using human glucometers rather than specific feeling glucometers such as the Alpha Trak system. Approximately 50% of cats using this protocol did achieve remission, but 40% of those later relapsed. There is not enough evidence to support this as being the gold standard for managing diabetes in cats, but it is a very economical and practical option to consider.

Feline Aortic Thromboembolism: Recent advances and future prospects - Julien Guillaumin, 2024 (sagepub.com)

This is a useful review of a common condition that has historically been associated with a poor outcome. More recent studies would suggest that with appropriate care approximately 30 to 40% of cats will survive, with median survival times over one year of age being achieved. There is a good description of pharmacological management and recent studies on efficacy for these products.

JOURNAL OF SMALL ANIMAL PRACTISE

2024 guidelines for the vaccination of dogs and cats – compiled by the Vaccination Guidelines Group (VGG) of the World Small Animal Veterinary Association (WSAVA) - Squires - 2024 - Journal of Small Animal Practice - Wiley Online Library

This extensive document provides guidelines for vaccination in both dogs and cats, developed by the vaccination guidelines group. These guidelines have been updated to revise the definition of core vaccination, when to consider serological testing and also the difference between shelter versus individual vaccinations.

A retrospective observational cohort study on the postoperative respiratory complications and their risk factors in brachycephalic dogs undergoing BOAS surgery: 199 cases (2019-2021) - Filipas - 2024 - Journal of Small Animal Practice - Wiley Online Library

This paper is a nice retrospective review of complications following airway surgery in brachycephalic dogs. The type of postoperative cut complication were identified and analysed in a univariate an multivariate manner. There was no association between preoperative factors and the development of postoperative aspiration pneumonia. However, the risk of a tracheostomy being needed increased with pre or post operative aspiration pneumonia and increasing severity grade of the brachycephalic syndrome. The presence of aspiration pneumonia prior to surgery should be considered a negative prognostic indicator and carefully explained to clients.

Current evidence for non-pharmaceutical, non-surgical treatments of canine osteoarthritis - Pye - 2024 - Journal of Small Animal Practice - Wiley Online Library

The number of products available in the nutraceutical market for treatment of osteoarthritis in dogs is vast and clear evidence supporting use is sparse. Additionally, there are multiple nonpharmacological methods such as acupuncture, physiotherapy, weight management and emerging treatments such as electromagnetic field therapy that are being recommended . This review critically evaluates each of these methods and provides some guidance as to when and if they should be used in individual cases. In particular, the evaluation of nutraceuticals is highly useful and beneficial for veterinary practitioners.

JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

Measurement of feline-specific pancreatic lipase aids in the diagnosis of pancreatitis in cats in: Journal of the American Veterinary Medical Association Volume 262 Issue 1 (2024) (avma.org)

In this multicenter prospective study, that clinical records of cats with clinical signs consistent with pancreatitis were reviewed. Cats were then classified on a scale of one to six on the likelihood of having pancreatitis as determined by two internal medicine specialists blinded to the pancreatic lipase result. The classification was then compared to the serum pancreatic lipase concentration. This resulted in a positive predictive value of 69% and a negative predictive value of 87%. In other words, pancreatic lipase concentration greater than or equal to 5.4 µg/L had a sensitivity of approximately 80%, whilst a result less than that had a specificity also about 80%. This study suggests that the use of pancreatic lipase (when the feline specific assay is used) has a higher clinical utility than previously thought.

Pathways to sustainable antimicrobial use in cats in: Journal of the American Veterinary Medical Association Volume 261 Issue 12 (2023) (avma.org)

This interesting paper addresses some of the challenges that we have as a profession in addressing appropriate antimicrobial usage in clinical practice. It appears to be more difficult to implement appropriate antimicrobial usage in cats than in dogs. There are many reasons why this may occur, including owner and patient compliance, as well as a lack of veterinary commitment to antimicrobial stewardship. In this paper some of those roadblocks are identified and potential pathways to overcoming them discussed. Although not highly practical paper it is thought provoking and worthwhile reading to implement practice wide strategies.

Prognostic value of C-reactive protein in dogs with elevated serum pancreatic lipase immunoreactivity concentrations in: Journal of the American Veterinary Medical Association Volume 262 Issue 3 (2024) (avma.org)

Pancreatitis in dogs is a common and often very serious condition in veterinary practice. Methods of predicting severity and therefore prognostic location have been described, with many multi parameter clinical scoring indices developed. The use of pancreatic lipase immunoreactivity to aid in prognostication is controversial, and poorly supported in the literature. This paper analyses C-reactive protein (CRP) in dogs with pancreatic lipase greater than 600 µg/L. The study identified that CRP greater than 10 mg/L was associated with an increased risk of hospitalisation or death. Therefore, the concurrent measurement of this analyte along with CRP and pancreatic lipase may aid in the identification of the animals requiring hospitalisation and intervention.

Nutritional management of pancreatitis and concurrent disease in dogs and cats in: Journal of the American Veterinary Medical Association Volume 262 Issue 6 (2024) (avma.org)

This is an informative and easy to read review on the current recommendations for nutritional management of pancreatitis in both dogs and cats. There are some useful supplementary materials as well that can be applied in clinical practise.

Monitoring of anaesthetised dogs and cats: Australian and New Zealand College of Veterinary Scientists Veterinary Anaesthesia and Analgesia Chapter position statement

Musk GC[a*], Bass L[b], Best P[c], Bradbury L[d], Davis J[e], Lehmann H[f], Neck D[g], Santos LCP[h], Goodwin W[i]

[a] *School of Veterinary Medicine, Murdoch University, Murdoch 6150, Western Australia in addition to the School of Human Sciences at the University of Western Australia*

[b] *Perth Veterinary Specialists, Osborne Park 6017, Western Australia*

[c] *Tamworth 2340, New South Wales*

[d] *Veterinary Anaesthesia Specialists, Melbourne 3000, Victoria*

[e] *Animalius Vet, Bayswater 6053, Western Australia*

[f] *Charles Sturt University, Wagga Wagga 2650, New South Wales*

[g] *Cottesloe Veterinary Hospital, Cottesloe 6011, Western Australia*

[h] *University of Glasgow, Bearsden, Glasgow G611QH, United Kingdom*

[i] *The University of Queensland, Gatton Campus, Gatton 4343, Queensland*

[*] *corresponding author: Gabrielle Musk gabrielle.musk@uwa.edu.au*

POSITION STATEMENT:

During anaesthesia of healthy dogs and cats there must be a person dedicated to the role of monitoring and recording the physiological status of the animal and any events related to the safe conduct of anaesthesia. This person must be appropriately trained and experienced or must work under direct supervision of someone who is appropriately trained and experienced.

In addition to clinical observations of the animal (palpation of a peripheral pulse, measurement of pulse rate and respiratory rate, observation of mucous membrane colour/ capillary refill time and subjective assessment of depth of anaesthesia), monitoring of anaesthesia should be complimented by the use of equipment including a pulse oximeter, capnograph, blood pressure monitor, and thermometer. A record of anaesthesia must be created in every case. Animals must always be observed from the time that drugs are first administered to the time that adequate recovery from the procedure has been confirmed.

INTRODUCTION

The Veterinary Anaesthesia and Analgesia Chapter of the Australian and New Zealand College of Veterinary Scientists (ANZCVS) is the only official veterinary special interest group for anaesthesia and analgesia in Australasia. Members of the Chapter identified a gap in the resources available to veterinarians in Australia and New Zealand with regards to defining the minimum standards for the monitoring of anaesthesia in companion animal practice. This position statement provides clear guidance on the minimum requirements for adequate monitoring during anaesthesia to optimise animal safety and meet the expectations of registering bodies, professional colleagues, and pet owners.

Currently there are no Australian and New Zealand standards or guidelines pertaining specifically to the provision of monitoring during anaesthesia and the various state, territory and national Veterinary Surgeons' Boards (or equivalent) do not consistently articulate their expectations of appropriate

and safe monitoring of dogs and cats during anaesthesia. There are extensive resources on the process of anaesthesia and many comprehensive publications and textbooks on the subject, including a recent publication endorsed by the Australian Small Animal Veterinarians (ASAV)[1]. However, resources such as this example do not clarify expectations on a minimum standard of practice.

Furthermore, the ANZCVS Veterinary Anaesthesia and Analgesia Chapter is committed to "empower veterinarians and veterinary professionals to safely and skilfully work in this discipline with pride, confidence and acumen."[2] To this end, the creation of a position statement on monitoring during anaesthesia will provide clarity to veterinary practitioners on how to, as a minimum, monitor dogs and cats during anaesthesia.

In 1941 a system was proposed to define the "physical state of a human patient" prior to anaesthesia and surgery.[3] The system is referred to as the American Society of Anesthesiologists (ASA) physical status classification and it helps to predict or determine the extent of monitoring required for a patient. A higher physical status (ASA 3-5) is attributed to patients with severe systemic disease from any cause or causes.[3] As this position statement is intended to define minimum standards of monitoring, it focuses on animals that would be categorised as either ASA 1 or 2 (healthy or with mild systemic disease). More complex cases that require a more complex approach to all aspects of care are not within the scope of this position statement.

The aims of the working group were to review the current international recommendations for monitoring during veterinary anaesthesia and to review the literature, where possible and appropriate, to justify a position statement for minimum monitoring requirements.

APPROACH AND METHODS

The ANZCVS Veterinary Anaesthesia and Analgesia Chapter established a working group of voluntary members of the Chapter. Members of the Chapter, by examination or by recognition of equivalent specialist level qualifications (Associate Members) were included. Active and engaged members were nominated by the Chapter Executive in late 2018 to include members with extensive experience in general, as opposed to specialist, practice.

Within the working group tasks were delegated for subgroups to focus on, including: review of the existing local (individual veterinary boards' webpage review) and international guidelines [Association of Veterinary Anaesthetists (Assoc VA), the European College of Veterinary Anaesthesia and Analgesia (ECVAA), the American College of Veterinary Anaesthesia and Analgesia (ACVAA), the American Animal Hospital Association (AAHA), the World Small Animal Veterinary Association (WSAVA) and the Australian Small Animal Veterinarians (ASAV)] for monitoring during anaesthesia: allowances for veterinary nurse involvement in anaesthesia in the various states and territories of Australia and New Zealand (individual veterinary boards' webpage review); and a literature review of evidence based practices for monitoring in veterinary and where necessary, human anaesthesia practice.

Webpage review was performed using search terms including "anaesthesia, monitoring, veterinary, resources, nurses, guidelines and requirements" to navigate the website and find reference to expectations for monitoring during anaesthesia.

For the literature review, multiple databases were used including Google Scholar, PubMed, Web of Science, and Scopus, to ensure a wide-ranging collection of literature pertaining

to monitoring guidelines, and patient safety in veterinary and human anaesthesia. Initially, the search yielded a total of 40,500 papers. Papers that focused on monitoring guidelines and safety aspects of anaesthesia were then selected.

RESULTS AND FINDINGS

Working group meetings of the 9 members were conducted intermittently online, with a significant hiatus in progress during 2020 and 2021.

Australian and New Zealand Veterinary Boards webpage review

There are no specific references to monitoring during anaesthesia within the policies and guidelines of the boards of Western Australia,[4] South Australia,[5] Queensland,[6] Victoria,[7] Tasmania,[8] or the Australian Capital Territory.[9] However, the Veterinary Board of the Northern Territory states the clear expectation that anaesthesia will be monitored as a strategy to avert anaesthetic death and that for routine cases in normal business hours a second person would be available to assist with the task of monitoring and record keeping.[10] Furthermore, the advice of the New South Wales Veterinary Practitioners Board on how to avoid anaesthesia related complaints is to: "Ensure appropriate monitoring of anaesthesia including skills, knowledge and experience of staff and use of appropriate and required equipment".[11]

The Veterinary Council of New Zealand maintains a listed set of Competency Standards for performance indicators for veterinarians and the Veterinarians Act 2005 requires the veterinary council to prescribe minimum standards for practicing veterinarians. Standard 5 of these Competency Standards is "Implement safe and effective veterinary procedures and therapeutic strategies", which outlines the requirements "Safely induce(s), maintain(s) and monitor(s) analgesia and anaesthesia and takes( )steps to ensure safe and humane recovery".[12]

The Australasian Veterinary Boards Council does not provide detailed guidance on their expectations of veterinarians working in this region with the statement: "Safely perform sedation and general and regional anaesthesia; implement chemical methods of restraint".[13]

For veterinary nurses, Western Australia is the only jurisdiction which approves persons to perform specific duties prescribed for veterinary nurses and Queensland is the only jurisdiction that defines the term 'veterinary nurse' in legislation.[14] In Western Australia, registered veterinary nurses are authorised to "assist with and monitor the administration of anaesthetics" and "monitor the recovery of animals from anaesthesia" under personal supervision of a registered veterinarian.[15] In Queensland, there are no specific references to how nurses can contribute to the safe anaesthesia of veterinary patients.

The Veterinary Nurses Council of Australia refers to various specific skills of veterinary nurses including their role in veterinary anaesthesia and analgesia in a position statement related to promoting the utilisation of veterinary nurses and technicians in veterinary practice.[16] Herein, anaesthesia and analgesia are recognised as important components of a career development path for veterinary nurses.[16]

International guidelines review

The details of content within the resources produced by veterinary anaesthesia and analgesia organisations were varied (*Table 1*). The ECVAA did not have information on their website pertaining to guidelines or benchmarks for the provision of anaesthesia in small animals;[17] whilst the ACVAA publishes a set of guidelines pertaining specifically to monitoring anaesthetised small animals.[18] The ACVAA guidelines discuss monitoring of circulation, oxygenation, ventilation, temperature and neuromuscular blockade along with record keeping, expectations during the recovery period and the role of

personnel. Minimum expectations are not defined in the ACVAA guidelines.

The Assoc VA has a position statement on anaesthetic monitoring requiring "dedicated anaesthetist monitoring [for] each case and additional monitoring equipment of pulse oximetry, capnography and blood pressure".[19] The rationale for this approach is that "a suitable trained and focused person responsible for anaesthesia is the most vital instrument for safely monitoring anaesthesia and cannot be replaced by a machine. The additional information gained from pulse oximetry, capnography and blood pressure measurement cannot be objectively gained by a person alone and the interpretation of this information will regularly improve case management."[19] The Assoc VA position is based on best available evidence and/or endorsement by six veterinary anaesthesia professionals.[19]

The AAHA consensus statement for monitoring are within a comprehensive publication, where statements such as "dedicated anaesthetist", "monitoring of respiratory function (respiratory rate, EtCO2, SpO2)", "monitoring of cardiovascular function (heart rate, blood pressure, assessment of cardiac rhythm)", "monitoring and support of normal body temperature", "monitoring in recovery", and "documentation of patient parameters during anaesthesia and recovery" are clear requirements to mitigate risk during anaesthesia.[20]

The WSAVA does not provide guidance on monitoring during anaesthesia.[21] Currently, ASAV resources can only be accessed by members of the Australian Veterinary Association, however, the aforementioned anaesthesia guidelines produced by the ASAV are readily available and comprehensive.[1]

The common factors in all guidelines were ensuring adequate monitoring of the cardiovascular and respiratory systems (oxygenation and ventilation), thermoregulation and the dedication of appropriate trained and skilled personnel to the task of monitoring anaesthesia with continuous attention and frequent recording of monitored variables (at least every 5 minutes).[18-20]

Organisation	Guidelines or other relevant guidance for monitoring
European College of Veterinary Anaesthesia and Analgesia (ECVAA)[17]	None applicable. (Activities are limited to assessment and registration of specialists in veterinary anaesthesia and analgesia)
American College of Veterinary Anaesthesia and Analgesia (ACVAA)[18]	Minimum expectations not defined. Reference to importance of monitoring the circulation, oxygenation, ventilation, temperature and neuromuscular blockade. Record keeping, monitoring during recovery and role of personnel also described.
Association of Veterinary Anaesthetists (Ass Vet Anaes)[19]	Dedicated trained person monitoring each case with additional monitoring equipment of pulse oximetry, capnography and blood pressure.

American Animal Hospital Association (AAHA)[20]	Dedicated trained person, monitoring of respiratory function, cardiovascular function, body temperature and recovery, and documentation of patient parameters during anaesthesia and recovery.
World Small Animal Veterinary Association (WSAVA)[21]	Nil
Association Small Animal Veterinarians (ASAV)[1]	Minimum expectations not defined. Comprehensive description of how to monitor the cardiovascular system, respiratory system, thermoregulation and recovery.

Table 1: Summary of guidelines produced by Australian and International organisations representing the discipline of veterinary anaesthesia and analgesia.

Literature Review

Twenty-seven papers were found to be relevant to this literature review to inform the position statement. Ten of these papers were from the veterinary literature and 17 were medical.

One medical study analysed 2,000 anaesthetic incidents reported to the Australian Incident Monitoring Study (AIMS).22 In > 50% of incidents, at least one monitor detected the problem before clinical signs were apparent. The combination of pulse-oximetry and capnography detected more than half of the incidents. Furthermore, ECG and blood pressure monitors were responsible for 19% and 12% reduction on incidents, respectively.[22]

However, the presence of an appropriately trained and experienced anaesthetist is considered the main determinant of patient safety during anaesthesia and should not be replaced by monitoring equipment.[23,24] The patient and the information provided by monitoring devices must be regularly observed and recorded.[25] If monitoring equipment is available it should be utilised by skilled and experienced personnel dedicated to monitoring anaesthesia.[26] Regular observations of the patient´s mucosal colour, movement of the chest wall, palpebral reflex, pupil size, and response to painful stimuli are considered essential even when multi-parameter monitors are present.[27] The same standard applies when an anaesthetist is responsible for a 'twilight' procedure.[27] It is also essential that the standard of monitoring and care during transfer from the operating room to recovery areas are as high as in the operating room, and that skilled staff accompany the patient.

In one veterinary study, anaesthesia monitoring by dedicated personnel decreased the odds of anaesthetic death in both dogs and cats[28] whereas in another study, cats that were not adequately monitored had an increased odds of anaesthetic death by a factor of 5 – 35.[29]

Another veterinary study conducted in New Zealand concluded that the use of appropriate anaesthetic monitoring equipment by a skilled, attentive anaesthetist is likely to reduce morbidity and mortality.[30] Amongst respondents to this survey, most practices had access to a pulse-oximeter, oesophageal stethoscope, apnoea alarm and a thermometer. Apnoea alarms are easy to use, non-invasive and relatively inexpensive but the common availability of these alarms in private practices was of concern, because they only provide information about

respiratory rate and tend to be unreliable and inaccurate.[31] Capnographs were only available in approximately 10% of practices studied. Blood pressure monitors were only present in under half the practices. It was encouraging that most respondents reported that a dedicated anaesthetist usually monitored the patient during anaesthesia in their practice. Most frequently the dedicated anaesthetist utilised was a veterinary nurse.

PULSE-OXIMETRY

The aforementioned AIMS paper[22] showed that more than 26% of anaesthetic incidents were first detected by pulse-oximetry.[32] In 1993, Moller and colleagues published data on the use of pulse oximetry in anaesthesia, in a study population of 20,000 patients.[33] During anaesthesia and in the recovery area, substantially more patients in the oximetry group had at least one respiratory event than the control patients, due to an increase in the incidence of diagnosed hypoxaemia in the oximetry group. Despite these findings, no significant difference in clinical outcome could be identified.

However, in 1986, the American Society of Anesthesiologists (ASA) approved the first anaesthetic monitoring guidelines with an emphasis on pulse-oximetry.[24] Anaesthesia risk dramatically decreased during the first decade that the pulse oximeter was mandated in the ASA guidelines.[34] In fact, the World Health Organisation's Checklist for International Standards for Safe Practice of Anaesthesia clearly states that it is no longer acceptable for routine anaesthesia to be conducted without a pulse-oximeter.[35]

Capnography

Capnography is the instantaneous measurement of carbon dioxide (CO^2) concentration in the expired gases during a respiratory cycle. Although the first infra-red CO^2 measuring and recording apparatus was introduced in 1943 by Luft, capnography gained widespread popularity in healthcare only in the early 1980s.[36] Continuous waveform capnography, combined with clinical assessment is the most reliable method of confirming and monitoring correct placement of an endotracheal tube at induction of anaesthesia, and during the procedure in people.[37] Given that one veterinary study reported 16 incidences of temporarily unrecognised oesophageal intubation in a 12 month period,[38] capnography has the potential to reduce potentially fatal anaesthetic mishaps, especially during induction of anaesthesia, in veterinary practice. Capnography can also be used to ensure ventilation with supraglottic devices like the V-gel, and to confirm that a spontaneously ventilating patient is breathing.

Blood Pressure

Continuous observation of cardiovascular changes has been considered essential since the early days of general anaesthesia.[24] Blood pressure monitoring in anaesthesia may be useful in two respects: to titrate anaesthetic drugs and fluid management, and to provide a warning of unexpected incidents manifesting as hypo- or hypertension, which could affect patient safety. The recommendation of the Australian and New Zealand College of Anaesthetists is that "the circulation must be monitored at frequent and clinically appropriate intervals by ... measurement of the arterial blood pressure", and this is echoed by the "International Standards for a Safe Practice of Anaesthesia" which state "Arterial blood pressure should be determined ... at least every five minutes, and ... continual registration of arterial pressure is encouraged in appropriate cases".[35]

Of the incidents reported to AIMS,[22] 1,256 out of 2000 occurred in relation to general anaesthesia and 81 of these were first detected by blood pressure (BP) monitoring. A further 25 incidents not associated with general anaesthesia were first detected by blood pressure monitoring. In the detection of incidents in relation to general anaesthesia,

BP monitoring ranked fourth after oximetry, capnography and low-pressure alarms. On the other hand, 38 incidents in which the problem was primarily one of significant change in BP were first detected by means other than the BP monitor (20 clinically, 12 by pulse oximetry and 6 by ECG). In a theoretical analysis of the 1256 anaesthesia incidents, it was considered that on its own, BP monitoring would have detected 919 (73%) incidents, but in the vast majority, by the time this detection has occurred, potential organ damage could have occurred.[22]

Recovery period

Although many complications occur throughout anaesthesia, most anaesthetic associated deaths in animals occur during recovery, especially in the first 3 hours.[39] Forty-seven percent of anaesthesia mortalities in dogs and 60% of anaesthesia mortalities in cats have been reported to occur in the immediate postoperative period.[39]

DISCUSSION

The requirement for this position statement is multifactorial: anaesthesia is performed daily in most veterinary practices in Australia and New Zealand; anaesthesia is not without risk (mortality reported to be 0.1-0.3% in healthy dogs and cats [39-48]); monitoring practices vary greatly;[49] and registration bodies do not provide adequate detail on minimum expectations. The last point has created a situation where many practitioners find themselves in a mandate vacuum, not knowing what, how, when, and why to monitor their patients during anaesthesia.

The rationale for the content of this position statement is based upon a combination of evidence-based detail regarding anaesthesia related complications[28, 39, 41, 44-47,50] and empirical evidence that monitoring of the cardiovascular system (by palpation of a peripheral pulse, measurement of pulse rate, observation of mucous membrane colour and capillary refill time), the respiratory system (measurement of respiratory rate and observation of mucous membrane colour) and the central nervous system (subjective assessment of depth of anaesthesia) is informative and simple to achieve with a stethoscope, timer or watch and targeted observations. To compliment these variables more sophisticated monitoring equipment is suggested to enhance understanding of changes associated with the cardiovascular system (pulse oximetry, capnography and blood pressure), the respiratory system (capnography) and the central nervous system and thermoregulatory system (thermometer). Nevertheless, the most important component of the position statement is that someone is dedicated to the role of monitoring during anaesthesia and ensuring that their attention is focused solely on anaesthesia. This requirement is specified by the Veterinary Board of the Northern Territory,[10] the Assoc VA and the AAHA[19,20] and implied by the other aforementioned guidelines and policies. Interestingly, monitoring of depth of anaesthesia was not referred to in any of the guidelines that were reviewed by the working group. This important factor is included here as it is essential to ensure that animals under veterinary care do not experience any unnecessary pain or suffering during the procedure for which they are being anaesthetised.

The role veterinary nurses and technicians in monitoring anaesthesia cannot be understated. This workforce may often be best placed to fulfil the requirements proposed in this position statement as they are more available to dedicate time and attention to monitoring animals during anaesthesia. Ongoing professional development for these staff is encouraged and supported by the Veterinary Anaesthesia and Analgesia Chapter.

Monitored parameters

Pulse oximetry: despite inconsistent evidence to demonstrate better safety associated with pulse oximetry, medical clinicians agree that

pulse-oximetry should be used in every patient for two reasons: first, the consequences for the patient with arterial hypoxaemia are severe and can result in hypoxic tissue damage; second, hypoxaemia secondary to hypoventilation could be prevented with pulse-oximetry in most patients, especially those patients transitioning from 100% to 21% fraction of inspired oxygen, as seen in the recovery period of general anaesthesia.[33] These risks are also important in veterinary medicine as mortality associated with anaesthesia is most likely to occur in the post-operative period (6-24 h after extubation) for both dogs and cats.[28,51] Additionally, observation of mucous membrane colour is not a sensitive indicator of hypoxaemia.[52]

Capnography: various factors result in either increased, decreased or absent EtCO2. It is more advantageous to have a continuous recording of the capnogram (the waveform): than simply a numerical display of EtCO2 since an analysis of the capnogram gives more information and better insight into the clinical situation. This information permits early diagnosis and intervention when abnormalities develop. There is only one normal capnogram and all variations must be recognised and corrected where possible. As an example, airway pressure monitors used to detect breathing system leaks occasionally fail to detect some disconnections. Under these circumstances a capnograph will detect disconnection instantaneously. In addition, capnography gives an early warning of CO^2 retention by the patient due to a faulty anaesthetic machine, exhausted CO^2 absorber and malfunction of unidirectional valves in circle anaesthetic systems. Further, complete occlusion or accidental disconnection of the endotracheal tube results in an abrupt decrease in EtCO2, whereas a partially kinked or obstructed tube can result in either increased or decreased EtCO2. No studies specifically focus on the association of abnormal capnograms and problem identification and management, but this monitoring equipment provides valuable information about ventilation, pulmonary circulation and equipment.

In the event of cardiopulmonary arrest, cardiac output drops to zero, and thus no transport of CO^2 from the tissues to the lungs can occur. Once chest compressions are initiated, circulation of blood will again deliver CO^2 to the lungs, and the capnogram will rise and fall with each breath. EtCO2 levels of 20 mmHg or greater indicate adequate chest compressions during cardiopulmonary resuscitation (CPR), and failure to achieve a level of at least 10 mmHg after 20 minutes of CPR may help in making the decision to terminate resuscitative efforts.[37,53]

Blood pressure: in dogs and cats, arterial blood pressure can be measured invasively via a catheter placed in a peripheral artery connected to a transducer and noninvasively via a Doppler ultrasonic probe or oscillometric technology often incorporated in a multiparameter monitor.

Body temperature: hypothermia is the most common anaesthetic complication in veterinary anaesthesia, occurring in approximately 40% of anesthetized animals.[54] Hypothermia can be detrimental to overall outcomes because it causes or contributes to sympathetic activation, pharmacokinetic alterations, coagulation abnormalities, blood loss, cardiac morbidity, wound infection, and shivering.[55] Factors contributing to perioperative hypothermia are convection, conduction, radiation, evaporation, intravenous fluid administration, cold and dry inhaled gases, and patient surgical preparation using cold solutions.[55] Anaesthesia prevents heat-seeking behaviours and activities that cause heat production (movement and shivering), and some anaesthetic drugs cause vasodilation, which could contribute to heat loss, although this loss is minimal compared with losses due to blood redistribution.[56] Hypothermia reduces anaesthetic dose requirements, so that relative overdoses are possible if

patient cooling is not detected.[50] Furthermore, recovery from anaesthesia can be prolonged in hypothermic patients, resulting in increased morbidity.[57] Monitoring and management of body temperature is easy to achieve with consideration of ambient temperature, insulation and the use of active warming devices.

Depth of anaesthesia: in veterinary practice monitoring the depth of anaesthesia remains a subjective exercise where a composite approach to evaluate whether the animal is adequately anaesthetised must be performed throughout the process of anaesthesia. The factors that contribute to this composite assessment include autonomic nervous system alterations, muscle tone, eye position, response to stimulation, and respiratory character. As the use of direct monitoring of the central nervous system is not available for veterinarians in a clinical setting, the person responsible for monitoring anaesthesia must use all the information acquired during their continuous observations and integrate these details into the context of the procedure, personnel, the environment and the animal. The ultimate aim is to ensure that the depth of anaesthesia is sufficient to facilitate the performance of the procedure (surgery, dentistry, radiology etc) whilst minimising the adverse effects of anaesthesia and ensuring the patient does not perceive noxious stimuli.

The Veterinary Anaesthesia and Analgesia Chapter hopes this position statement will highlight the importance of monitoring during anaesthesia and provide guidance on how to improve patient safety. However, there are many steps to providing safe anaesthesia, of which monitoring is only one. Some or all the recommendations in this document may need to be exceeded depending on the results of the pre-anaesthetic patient evaluation or changing intra-operative demands. Anaesthetic agent monitoring, airway pressure measurement and direct arterial blood pressure monitoring are examples of more invasive and comprehensive monitoring strategies. Increasing patient comorbidities and sometimes complex procedural requirements also add to the complexity of care and the anaesthetic monitoring requirements. Additionally, it is hoped that this position statement will standardise the expectations of registration bodies to promote improvements in the monitoring of anaesthetised healthy dogs and cats.

REFERENCES

1. Warne, L., et al., *STANDARDS OF CARE Anaesthesia guidelines for dogs and cats*. Australian Veterinary Journal, 2018. 96(11): p. 413-427.
2. ANZCVS. *Veterinary Anaesthesia and Analgesia Chapter*. 2023 [cited 2023 8/11/23]; Available from: https://www.anzcvs.org.au/chapters/veterinary+anaesthesia+and+analgesia+chapter.
3. Saklad, M., *GRADING OF PATIENTS FOR SURGICAL PROCEDURES*. Anesthesiology, 1941. 2(3): p. 281-284.
4. Veterinary Practice Board of Western Australia, *Veterinary Practice Board Western Australia Codes of Practice and Guidelines*. 2023 [cited 2023 6/11/23]; Available from: https://www.vsbwa.org.au/Public/_VSBWA/Vets/Code%20of%20Practice%20and%20Guidelines.aspx?hkey=193b44b4-320d-4685-a61f-7e0de614b1b0.
5. Veterinary Surgeons Board of South Australia, *Codes of conduct, professional standards and guidelines*. 2023 [cited 2023 8/11/23]; Available from: https://vsb.sa.gov.au/information-for-veterinary-surgeons/codes-of-conduct-professional-standards-and-guidelines/.
6. Veterinary Surgeons Board of Queensland, *Guidelines and policies*. 2023 [cited 2023 8/11/23]; Available from: https://www.vsb.qld.gov.au/for-vets/guidelines-and-policies.
7. Veterinary Practitioners Registration Board of Victoria, *Guidelines of the Veterinary Practitioners Registration Board of Victoria*. 2023 [cited 2023 6/11/23]; Available from: https://www.vetboard.vic.gov.au/VPRBV/VPRBV/VPRBV_Guidelines/Guidelines_TOC.aspx?hkey=76c45aa3-8635-42ec-b5be-bb3a03d2a9bd.
8. Veterinary Board of Tasmania, *Veterinary Board of Tasmania*. 2023 [cited 2023 9/11/23]; Available from: https://nre.tas.gov.au/biosecurity-tasmania/animal-biosecurity/veterinary-board-of-tasmania.
9. Veterinary Practitioners Board ACT Government, *Guidelines*. 2023 [cited 2023 10/11/23]; Available from: https://www.cityservices.act.gov.au/pets-and-wildlife/veterinary-practitioners-board/resources/guidelines.
10. Veterinary Board of the Northern Territory, *Guidelines*. 2023 [cited 2023 13/11/23]; Available from: https://industry.nt.gov.au/boards-and-committees/vetboardnt/guidelines.
11. New South Wales Veterinary Board, *2018 June - Avoiding anaesthesia related complaints*. 2023 [cited 2023 3/11/23]; Available from: https://www.vpb.nsw.gov.au/2018-june-avoiding-anaesthesia-related-complaints.
12. New Zealand Veterinary Council, *Standards and Guidance - Competency Standards and Performance Indicators for Veterinarians*. 2023 [cited 2023 3/11/23]; Available from: https://hub.vetcouncil.org.nz/competency-standards-and-performance-indicators-for-veterinarians.
13. Australian Veterinary Board Council, *Day One Competencies*. 2023 [cited 2023 13/11/23]; Available from: https://avbc.asn.au/veterinary-education/day-one-competencies/.

14. Australian Veterinary Board Council, *AVBC Sustainable Practice Committee - Options for Registration of Veterinary Nurses*. 2022: Melbourne, Victoria.
15. Veterinary Practice Board of Western Australia. *Veterinary Nurses Prescribed Duties*. 2023 [cited 2023 13/11/23]; Available from: https://www.vsbwa.org.au/Public/_VSBWA/Nurses/VNaTVN-duties.aspx?hkey=ad8c9ac6-131b-413b-9e3e-40570c90ae17.
16. Veterinary Nurses Council of Australia, *VNCA Position Statement: Utilisation of Veterinary Nurses and Veterinary Technicians in veterinary Practice, December 2021*. 2021 [cited 2023 13/11/23]; Available from: https://www.vnca.asn.au/resources/vnca-position-statements/.
17. ECVAA. *European College of Veterinary Anaesthesia and Analgesia. 2023* [cited 2023 6/11/23]; Available from: https://www.ecvaa.org/.
18. ACVAA. *American College of Veterinary Anaesthesia and Analgesia.* 2023 [cited 2023 7/11/23]; Available from: https://acvaa.org/.
19. Association of Veterinary Anaesthetists. *Guidelines for Safer Anaesthesia*. 2023 [cited 2023 6/11/23]; Available from: https://ava.eu.com/resources/anaesthesia-guidelines/.
20. Grubb, T., et al., *2020 AAHA Anesthesia and Monitoring Guidelines for Dogs and Cats*. J Am Anim Hosp Assoc, 2020. 56(2): p. 59-82.
21. WSAVA. *World Small Animal Veterinary Association Guidelines*. 2023 [cited 2023 6/11/23]; Available from: https://wsava.org/global-guidelines/animal-welfare-guidelines/.
22. Webb, R.K., et al., *The Australian Incident Monitoring Study: an analysis of 2000 incident reports*. Anaesth Intensive Care, 1993. 21(5): p. 520-8.
23. Eichhorn, J.H., *Prevention of intraoperative anesthesia accidents and related severe injury through safety monitoring.* Anesthesiology, 1989. 70(4): p. 572-7.
24. Eichhorn, J.H., et al., *Standards for patient monitoring during anesthesia at Harvard Medical School.* JAMA, 1986. 256 8: p. 1017-20.
25. Gaba, D.M., M. Maxwell, and A. DeAnda, *Anesthetic mishaps: breaking the chain of accident evolution*. Anesthesiology, 1987. 66(5): p. 670-6.
26. Cheney, F.W., *The American Society of Anesthesiologists Closed Claims Project: what have we learned, how has it affected practice, and how will it affect practice in the future?* Anesthesiology, 1999. 91(2): p. 552-6.
27. Gravenstein, J.S., *Let No Patient be Harmed by Anesthesia.* Journal of Clinical Monitoring and Computing, 2000. 16(3): p. 233-235.
28. Brodbelt, D., *Perioperative mortality in small animal anaesthesia*. Vet J, 2009. 182(2): p. 152-61.
29. Matthews, N.S., et al., *Factors associated with anesthetic-related death in dogs and cats in primary care veterinary hospitals*. J Am Vet Med Assoc, 2017. 250(6): p. 655-665.
30. Sano, H., et al., *A survey of dog and cat anaesthesia in a sample of veterinary practices in New Zealand*. N Z Vet J, 2018. 66(2): p. 85-92.
31. Southall, D.P., et al., *An explanation for failure of impedance apnoea alarm systems*. Arch Dis Child, 1980. 55(1): p. 63-5.
32. Runciman, W.B., et al., *The Australian Incident Monitoring Study. The pulse oximeter: applications and limitations--an analysis of 2000 incident reports. Anaesthesia and intensive care*, 1993. 21(5): p. 543-550.
33. Moller, J.T., et al., *Randomized evaluation of pulse oximetry in 20,802 patients: II. Perioperative events and postoperative complications.* Anesthesiology, 1993. 78(3): p. 445-53.
34. Shah, A. and K.H. Shelley, *Is pulse oximetry an essential tool or just another distraction? The role of the pulse oximeter in modern anesthesia care*. J Clin Monit Comput, 2013. 27(3): p. 235-42.
35. Merry, A.F., et al., *International Standards for a Safe Practice of Anesthesia 2010*. Can J Anaesth, 2010. 57(11): p. 1027-34.
36. Smalhout, B., *The first years of clinical capnography*. 2011: p. 430-456.
37. Link, M.S., et al., *Part 7: Adult Advanced Cardiovascular Life Support.* Circulation, 2015. 132(18_suppl_2): p. S444-S464.
38. Hofmeister, E.H., et al., *Development, implementation and impact of simple patient safety interventions in a university teaching hospital*. Vet Anaesth Analg, 2014. 41(3): p. 243-8.
39. Brodbelt, D.C., et al., *The risk of death: the confidential enquiry into perioperative small animal fatalities*. Vet Anaesth Analg, 2008. 35(5): p. 365-73.
40. Clarke, K.W. and L.W. Hall, *A survey of anaesthesia in small animal practice: AVA/BSAVA report*. Journal of the Association of Veterinary Anaesthetists of Great Britain and Ireland, 1990. 17(1): p. 4-10.
41. Dyson, D., M. Maxie, and D. Schnurr, *Morbidity and mortality associated with anesthetic management in small animal veterinary practice in Ontario*. Journal of the American Animal Hospital Association, 1998. 34(4): p. 325-335.
42. Hosgood, G. and D.T. Scholl, *Evalution of Age as a Risk Factor For Perianesthetic Morbidity and Mortality in the Dog*. Journal of Veterinary Emergency and Critical Care, 1998. 8(3): p. 222-236.
43. Hosgood, G. and D.T. Scholl, *Evaluation of age and American Society of Anesthesiologists (ASA) physical status as risk factors for perianesthetic morbidity and mortality in the cat.* Journal of Veterinary Emergency and Critical Care, 2002. 12(1): p. 9-15.
44. Brodbelt, D.C., et al., *Risk factors for anaesthetic-related death in referred dogs*. Veterinary Record, 2006. 158(16): p. 563-564.
45. Brodbelt, D.C., et al., *Risk factors for anaesthetic-related death in cats: results from the confidential enquiry into perioperative small animal fatalities (CEPSAF)[†]*. British Journal of Anaesthesia, 2007. 99(5): p. 617-623.
46. Brodbelt, D.C., et al., *Results of the Confidential Enquiry into Perioperative Small Animal Fatalities regarding risk factors for anesthetic-related death in dogs*. Journal of the American Veterinary Medical Association, 2008. 233(7): p. 1096-1104.
47. Bille, C., et al., *Risk of anaesthetic mortality in dogs and cats: an observational cohort study of 3546 cases*. Veterinary Anaesthesia and Analgesia, 2012. 39(1): p. 59-68.
48. Gil, L. and J.I. Redondo, *Canine anaesthetic death in Spain: a multicentre prospective cohort study of 2012 cases*. Veterinary Anaesthesia and Analgesia, 2013. 40(6): p. e57-e67.
49. Truchetti, G., et al., *Management of veterinary anaesthesia in small animals: A survey of current practice in Quebec.* PLoS One, 2020. 15(1): p. e0227204.
50. Brock, N., *Anesthesia safety through monitoring.* Can Vet J, 1994. 35(10): p. 655-6.
51. Redondo, J.I., et al., *Anaesthetic mortality in dogs: A worldwide analysis and risk assessment*. Vet Rec, 2023: p. e3604.
52. Kelman, G.R. and J.F. Nunn, *Clinical recognition of hypoxaemia under fluorescent lamps*. Lancet, 1966. 1(7452): p. 1400-3.
53. Paiva, E.F., J.H. Paxton, and B.J. O'Neil, *The use of end-tidal carbon dioxide (ETCO(2)) measurement to guide management of cardiac arrest: A systematic review*. Resuscitation, 2018. 123: p. 1-7.
54. Kennedy, K.C., K.R. Tamburello, and R.J. Hardie, *Peri-operative morbidity associated with ovariohysterectomy performed as part of a third-year veterinary surgical-training program*. J Vet Med Educ, 2011. 38(4): p. 408-13.
55. Taguchi, A. and A. Kurz, *Thermal management of the patient: where does the patient lose and/or gain temperature?* Curr Opin Anaesthesiol, 2005. 18(6): p. 632-9.
56. Matsukawa, T., et al., *Heat flow and distribution during induction of general anesthesia*. Anesthesiology, 1995. 82(3): p. 662-73.
57. Pottie, R.G., et al., *Effect of hypothermia on recovery from general anaesthesia in the dog*. Aust Vet J, 2007. 85(4): p. 158-62.

Isorhythmic atrioventricular dissociation in a Shih-tzu dog

Beluque T,[a*] Endrighi M,[b] Vila BCP,[b] Costa BN,[b] Sousa MG[b]

[a]*Department of Veterinary Clinic and Surgery, Sao Paulo State University, Jaboticabal, SP, Brazil, 14884-900*

[b]*Department of Veterinary Medicine, Federal University of Paraná, Curitiba, PR, Brazil, 80035-050.*

Corresponding author: Tamyris Beluque tamyrisbeluque@gmail.com

ABSTRACT

Isorhythmic atrioventricular dissociation (IAVD) is a conduction disorder between the atria and ventricles in which both a sinus rhythm and a separate rhythm originating in the atrioventricular junction or ventricular myocardium coexist. The P waves and QRS complexes are dissociated and can be classified as type I and type II synchronisation patterns. This study describes electrocardiographic findings from a Shih-tzu dog with IAVD with type I synchronisation, characterised by the fluctuation of P waves in relation to the QRS complexes. This condition was associated with heart disease, namely mitral valve disease and pulmonary hypertension. The animal did not require treatment with antiarrhythmic drugs. Structural heart disease and its implications may have contributed to the development of this arrhythmia.

Keywords: arrhythmia, myxomatous mitral valve disease, electrocardiography, pulmonary hypertension

Abbreviation List

A wave: mitral end-diastolic inflow peak velocity

ALP: alkaline phosphatase

ALT: alanine aminotransferase

Ao: aorta

BUN: blood urea nitrogen

ECG: electrocardiography

E wave: mitral early-diastolic inflow peak velocity

IAVD: isorhythmic atrioventricular dissociation

Hb: hemoglobin

Ht: hematocrit

IV: intravenous

LA: left atrium

LA/Ao: left atrium/aorta ratio

ms: millisecond

mV: millivolt

MVD: mitral valve disease

PH: pulmonary hypertension

RBC: red blood cells

VHS: vertebral heart size

VLAS: vertebral left atrial size

UPC: urinary protein to creatinine ratio

INTRODUCTION

Isorhythmic atrioventricular dissociation (IAVD) is a conduction disorder between the atria and ventricles characterised by two concomitant rhythms, in which the atria and ventricles are stimulated by independent pacemakers with similar trigger rates. There is a sinus rhythm as well as a second separate rhythm that originates at either the atrioventricular junction or the ventricular myocardium.[1]

Synchronisation is related to the association between P waves and QRS complexes and can be classified into two types; in IAVD with type I synchronisation, P waves of sinus origin are observed moving towards the QRS complexes and then returning, without a clear association between them.[1,2] This disorder can result from cyclical variations in blood pressure, affecting atrioventricular mechanical coupling and contributing to changes in heart rate and arterial and atrial pressures.[1-5] Isorhythmic atrioventricular dissociation with type I synchronisation is not a routine diagnosis in veterinary medicine, with few descriptions in the literature. [2,6-10]

This study describes the electrocardiographic findings from a Shih-tzu dog with type I IAVD and the conditions that contributed to the emergence of this arrhythmia.

CLINICAL FEATURES

A 14-year-old, 5.4-kg, neutered male Shih-tzu was referred to a veterinary teaching hospital due to episodes of syncope that intensified over the two previous days. The owner reported that four years earlier, the patient presented with occasional seizures that intensified in frequency and intensity and occurred daily during the few weeks preceding the referral. For the two days prior to the consult, the patient experienced approximately seven seizures throughout the day; the owner reported urination and defecation in some cases. After the seizures the dog wheezed, but at other times the respiratory pattern was unremarkable. The history also included hypodipsia and inappetence for four days.

The animal had already been diagnosed with stage C myxomatous mitral valve disease,[11] pulmonary hypertension (PH), and neurological dysfunction, and was being treated with furosemide, enalapril, and pimobendan. Sildenafil was also prescribed, but the owner did not start the medication. Physical examination found a regular heart rhythm with a rate of 150 beats/min, systolic blood pressure of 90 mmHg, pink mucous membranes, capillary refill time of two to three seconds, and rectal temperature of 36.7 °C (98.1° F). Cardiac auscultation revealed a grade 4/6 holosystolic murmur over the left cardiac apex and a grade 3/6 holosystolic murmur over the right cardiac apex. Cranial bilateral inspiratory pulmonary crackles and increased broncho-bronchiolar sounds in the caudal lung fields were also documented upon pulmonary auscultation. Abdominal palpation detected a painless, rounded structure with increased stiffness and irregular edges adjacent to the liver and spleen. The right prescapular lymph node was slightly enlarged, and the animal was moderately dehydrated (7-8 %). The dog was hospitalised and administered lactated Ringer solution 360 mL/24 hours IV and oxygen therapy. The medications that had been previously prescribed were maintained at the beginning of hospitalization, however, after a new evaluation and after chest x-rays and echocardiography, diuretics and ACEi were suspended. The medications were furosemide (2.1 mg/kg [0.96 mg/lb] orally, q 12 h), pimobendan (0.27 mg/kg [0.13 mg/lb], orally, q 12 h), enalapril (0.27 mg/kg [0.13 mg/lb] orally, q 24 h), and sildenafil (2 mg/kg [0.9 mg/lb] orally q 8 h).

Blood samples were collected for laboratory analysis, and mild normochromic normocytic anaemia was observed (RBC: 4,600,000/mm^3; Ht: 33 %; Hb: 11 g/dL). Other abnormalities were also identified in ALT

(3,548 UI/L; reference interval [RI] 21–102 UI/L), ALP (3,875 UI/L; RI 20–156 UI/L), cholesterol (228.4 mg/dL; RI135–210 mg/d), BUN 80.64 mg/dL; (RI 9.8–28.0 mg/dL), urea (172.8 mg/dL; RI 21–60 mg/dL), phosphate (6.8 mg/dL; RI 2.6–6.2 mg/dL), and blood glucose (185.5 mg/dL; RI 60–110 mg/dL). Urinalysis found granular casts, leukocytes, desquamative and transitional cells, and mild proteinuria (UPC: 1.39). Three-view thoracic radiography revealed cardiomegaly as assessed subjectively (VHS: 11, VLAS: 2.05) and a diffuse bronchointerstitial pulmonary pattern, combined with reduced lung volume and loss of lung border definition in lateral projections. The trachea presented a dorsal deviation in lateral projection and a preserved lumen, and mild pleural effusion and a markedly increased hepatic silhouette with rounded edges were also identified. Ultrasound confirmed hepatomegaly, and signs of concomitant hepatic and caudal vena cava congestion were also identified.

Two-dimensional echocardiography revealed significant thickening and irregularity in the anterior cusp of the mitral valve compared to the posterior cusp (differential diagnoses were degenerative valve disease, endocarditis, and myxoma), aortic and pulmonary cusps were irregular and hyperechoic (suggesting a valvular sclerosis), and the left atrium was slightly increased (LA/Ao 1.62; reference upper limit 1.59). The right atrium was enlarged when assessed subjectively, the left ventricular eccentricity index was elevated (1.7; reference limit ≤1.0), and the right pulmonary artery distensibility index was decreased (28.3%; reference limit ≥30%),12 but the normalised left ventricular internal diameter in diastole (LVIDDn) was normal (1.03; reference limit 1.69). Doppler ultrasonographic findings confirmed mitral, tricuspid, and pulmonic valve insufficiency, decreased ratio of early-to-late transmitral peak velocities (E:A) (0.85; reference range 1.18–1.89), and normal E-to-isovolumic relaxation time ratio (1.62; values associated with myxomatous mitral valve disease are typically <2.5). Estimated systolic pulmonary artery pressure was 108 mmHg and diastolic pulmonary artery pressure was 48 mmHg. In the absence of pulmonic stenosis, these findings are consistent with severe pulmonary hypertension.[13]

Ten-lead electrocardiography was recorded for 5 minutes and revealed an underlying regular rhythm with a mean heart rate of 136 beats/min, and an electrical axis of +60° (reference range +40° to +100°) (Figure 1). Wandering P waves were noticed moving back and forth into QRS complexes, characterizing IAVD with type I synchronisation. Additionally, P wave duration was increased (47 ms; reference range <40 ms), which might indicate either left atrial enlargement or atrial conduction delay. The amplitude of the P waves was normal (0.34 mV; reference range <0.4 mV).

The P-wave morphology remained unaltered throughout the ECG tracing, yet the QRS complex morphology changed, and the P-wave axis varied from +55 to +60º (reference range for sinus axis -20 to +90º). The R-wave became taller (1.0–1.28 millivolt; reference range < 3.0 millivolt) and notched when superimposed over the P waves. All QRS complexes not superimposed by the P wave were narrow (57 milliseconds; reference range <70 milliseconds), with amplitude within the reference interval.

Unfortunately, the dog remained in critical condition during hospitalization due to neurological and respiratory disease and died 5 days after electrocardiography.

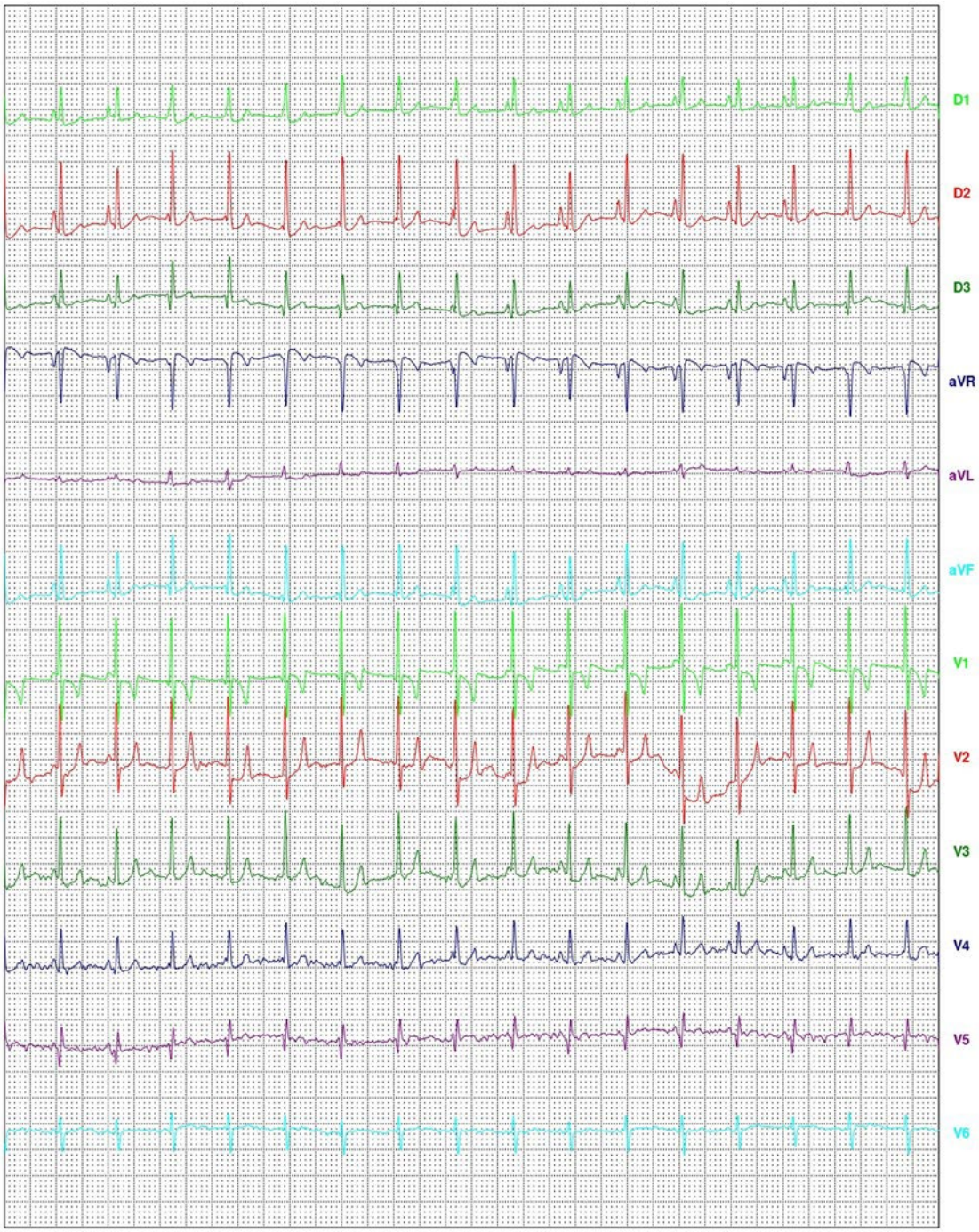


Figure 1. Section of 10-lead ECG trace recorded from a Shih-tzu that presented recurring episodes of syncope over the previous two days. This ECG trace shows isorhythmic atrioventricular dissociation with type I synchronisation and a mean electrical axis of +60°. Can you please also provide a longer rhythm strip (lead II). Paper speed = 25 mm/s; 1 cm = 1 mV.

DISCUSSION

In IAVD, the atria and ventricles are stimulated by independent pacemakers in two concomitant but separate rhythms.[1] In the electrocardiographic tracing, P waves of sinus origin (axis: +55 to +60 º) are seen to move towards the QRS complexes and then back in a cycle. There is clearly no association between P waves and QRS complexes; this P-wave fluctuation occurs because of the variation in the rate of one of the sites. Although it can overlap the QRS complexes at times, the P wave is never superimposed on the ST segment, since the two rhythms have very similar rates.[1,2]

Different mechanisms have been proposed to explain this movement of the P wave, which characterizes type I synchronisation. Experimental animal models suggest it can result from cyclic variations in blood pressure. Whenever the P wave is very close to the QRS complex, the atrioventricular mechanical coupling is impaired, in turn reducing systolic volume and contributing to a slight reduction in blood pressure. This stimulates baroreceptors and the sympathetic autonomic nervous system is activated, increasing the heart rate and decreasing the P-P interval. Blood pressure increases after P waves are an adequate distance from the QRS complex due to the resumption of atrioventricular mechanical coupling. But the cycle begins once more when the heart rate drops, causing the P wave to move towards the QRS complex once again.[1-4]

Another theory for IAVD with type I synchronisation involves a mismatch in atrial and arterial pressures. When the P wave is close to the QRS complex, the simultaneous contraction of the atria and ventricles results in the right atrium contracting against a closed tricuspid valve. This increases right atrial pressure with subsequent stretching of the right atrium myocardium. This may cause distension of the sinus node fibres, which increases the trigger rate.[2,5] On the other hand, IAVD with type II synchronisation presents a constant relationship between the P wave and the QRS complex, and generally the P wave is positioned in the ST segment or superimposed on the QRS complex. This causes atrial and ventricular contraction to occur at the same time or very close together.[3]

Notably, P-P and R-R intervals are usually similar and variations in the PQ interval are mild,[1] although the PQ interval found in this dog's electrocardiogram ranged from 23–70 ms (reference interval 60–130 ms).

The dog in this present report was not administered antiarrhythmic drugs because IAVD is unlikely to cause significant hemodynamic impairment and consequently does not require treatment.[14] The patient received fluid therapy to correct dehydration, however, and was also treated with oxigeniotherapy, pimobendan andthe pulmonary artery vasodilator sildenafil in an attempt to control PH. In another study describing IAVD in a spayed female Labrador retriever, the dog did not receive antiarrhythmic therapy and did not develop complications as a result of the arrhythmia.[10]

In a set of cases that included 10 Labrador dogs with IAVD and type I synchronisation, 5 had congestive heart failure, and the authors suggested that the elevated sympathetic tone may have increased the automaticity of the atrioventricular junction and contributed to worsening hemodynamics.[2] Although no studies have evaluated the occurrence of IAVD and dogs with PH, we speculate that right atrial wall distension ascribed to hypertension might have affected the sinus node conduction fibres resulting in an accelerated rate with a similar discharge rate to the ectopic focus. In human patients with PH, the occurrence of supraventricular arrhythmias has already been described, as well as a significant increase in mortality in these patients compared to other patients with PH who did not have supraventricular arrhythmias.[15] In another study evaluating human patients with PH, right atrial enlargement was associated with a greater occurrence of supraventricular arrhythmias;[16]

over 46% of patients were found to have supraventricular arrhythmias, as well as larger right atrial area and higher right atrial pressure compared to patients without arrhythmias. Furthermore, a study on Beagle dogs with induced PH showed that right atrial remodelling incited supraventricular arrhythmias.[17]

The dog in this present report had a diagnosis of mitral valve disease (MVD), which might have predisposed the onset of arrhythmia, even though the left-side cardiac remodelling was only mild. Animals at different stages of MVD can develop atrial and ventricular arrhythmias,[18-20] and those with left atrial enlargement may have more supraventricular arrhythmias.[18] Nonetheless, no research has evaluated the prevalence of IAVD in dogs with MVD.

Some other suspected mechanisms for triggering IAVD with type I synchronisation were present in our patient; endocarditis was ruled out by negative blood cultures, for example. Unfortunately, further study to investigate suspected valve neoplasia was not possible because the owner declined necropsy.

Although IAVD with type I synchronisation is uncommon in veterinary medicine, this case highlights the importance of electrocardiographic evaluation in dogs with structural heart disease, even without prior auscultation of arrhythmia, since the autonomic nervous system and cardiac remodelling play essential roles in the occurrence of this arrhythmia.

ACKNOWLEDGMENTS

This research was supported by Coordination of Superior Level Staff Improvement (CAPES), Brazil.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest for the work presented herein.

REFERENCES

1. Santilli R, Moïse NS, Pariaut R, et al. Supraventricular tachycardias. In: Santilli R, Moïse NS, Pariaut R, et al., eds. *Electrocardiography of the dog and cat: diagnosis of arrhythmias.* 2nd ed. Trento: Edra S.p.A., 2019;145–189.
2. Perego M, Ramera L, Santilli RA. Isorhythmic atrioventricular dissociation in Labrador Retrievers. *J Vet Intern Med* 2012;26:320–325.
3. Levy MN, Edflstein J. The mechanism of synchronization in isorhythmic AV dissociation. II. Clinical studies. *Circulation* 1970;42:689–699.
4. Levy MN, Zieske H. Mechanism of synchronization in isorhythmic A–V dissociation. 3. Computer model. *Circ Res* 1971;109:23–33.
5. Paulay KL, Damato AN, Bobb GA. Atrioventricular interaction in isorhythmic dissociation. *Am Heart J* 1971;82:647–653.
6. Ettinger S, Buergelt CD. Atrioventricular dissociation (incomplete) with accrochage in a dog with ruptured chordae tendineae. *Am J Vet Res* 1968;29:1499-1503.
7. Bright JM, Lombard CW. ECG of the month: atrioventricular junctional tachycardia producing atrioventricular dissociation. *J Am Vet Med Ass* 1983;182:580-581.
8. Pereira NJ, Glaus T, Matos JN. ECG of the Month. *J Am Vet Med Ass* 2014;244:1384-1386.
9. Santarelli G, Toaldo MB. ECG of the Month. *J Am Vet Med Ass* 2015;247:1019-1021.
10. Wiggen KE, Saelinger C. ECG of the Month. *J Am Vet Med Ass* 2019;254:350-352.
11. Keene BW, Atkins CE, Bonagura JD, Fox PR, Häggström J, Fuentes VL, Oyama MA, Rush JE, Stepien R, Uechi M. ACVIM consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease in dogs. *J Vet Intern Med.* 2019;33(3):1127-1140.
12. Visser LC, Im MK, Johnson LR, Stern JA. Diagnostic Value of Right Pulmonary Artery Distensibility Index in Dogs with Pulmonary Hypertension: Comparison with Doppler Echocardiographic Estimates of Pulmonary Arterial Pressure. *J Vet Intern Med* 2016;30:543-52.
13. Reinero C, Visser LC, Kellihan HB et al. ACVIM consensus statement guidelines for the diagnosis, classification, treatment, and monitoring of pulmonary hypertension in dogs. *J Vet Intern Med* 2020;34:549-573.
14. Kittleson MD. Diagnosis and treatment of arrhythmias (dysrhythmias). In: Kittleson MD, Kienle RD, eds. *Small animal cardiovascular medicine*. St Louis: Mosby, 1998;449–494.
15. Cannillo M, Marra WG, Gili S, D'Ascenzo F, Morello M, Mercante L, Mistretta E, Salera D, Zema D, Bissolino A, Fusaro E, Marra S, Libertucci D, Gaita F. Supraventricular Arrhythmias in Patients With Pulmonary Arterial Hypertension. *Am J Cardiol* 2015;116:1883-1889.
16. Waligóra M, Tyrka A, Miszalski-Jamka T, Urbańczyk-Zawadzka M, Podolec P, Kopeć G. Right atrium enlargement predicts clinically significant supraventricular arrhythmia in patients with pulmonary arterial hypertension. *Heart Lung* 2018;47:237-242.
17. Zhao Q, Deng H, Jiang X, Dai Z, Wang X, Wang X, Guo Z, Hu W, Yu S, Yang B, Tang Y, Huang C. Effects of Intrinsic and Extrinsic Cardiac Nerves on Atrial Arrhythmia in Experimental Pulmonary Artery Hypertension. *Hypertension* 2015;66:1042-1049.
18. Crosara S, Borgarelli M, Perego M, Häggström J, La Rosa G, Tarducci A, Santilli RA. Holter monitoring in 36 dogs with myxomatous mitral valve disease. *Aust Vet Journal* 2010;88;386-392.
19. Oliveira MS, Muzzi RAL, Araújo RB, Muzzi LAL, Ferreira DF, Silva EF. Heart rate variability and arrhythmias evaluated with Holter in dogs with degenerative mitral valve disease. *Arq Bras Med Vet Zootec* 2014;66:425-432.
20. Beluque T, Camacho AA, Ampuero RN, Braz JB, Kirnew MD, Canola RAM, Carvalho E, Sousa MG. Heart rate variability and quality of life in dogs with mitral valve disease treated with metoprolol. *Braz J Vet Med* 2021;43:1-10.

Clinical Perspective Article:

Hot Dogs: Thermoregulatory function and dysfunction in the racing Greyhound (*Canis familiaris)* and the rationale for cooling strategies

Brownlow M[1], Streckfuss E[2]

Greyhound Welfare and Integrity Commission, Bathurst, New South Wales, Australia.

Corresponding Author: mbro8605@uni.sydney.edu.au

ABSTRACT

The greyhound (GH) is a unique canine athlete that has been selectively bred over millennia for coursing and racing. The GH can utilise oxygen at a prodigious rate, with extremely rapid energy kinetics, enabling it to accelerate from a standing start to maximum speed in a few seconds. The multiple demands imposed by such strenuous exercise require complex coordination between the respiratory, cardiovascular, and neuromuscular systems, and the resulting high rate of metabolic heat production necessitates a finely tuned thermoregulatory system. This is provided by a powerful 'panting' mechanism, where variations in breathing pattern determine its efficiency by controlling the rate and direction of air flow over respiratory and buccal evaporative surfaces.

Disorders of thermoregulation referred to in athletic species as Exertional Heat Illness (EHI) occur from very minor to severe and can be potentially fatal. Veterinarians working at racetracks should be aware of the earliest clinical signs and trainers and handlers should be educated in their recognition. Treatment may require proactive management and specific cooling interventions. The concept of 'thermal windows', which are specific parts of the body surface with specialised vascular structures for heat dissipation, are discussed and rationale for cooling interventions suggested. These principles may be applied to other situations of heat stress in non-GH dogs.

Keywords: Thermoregulation Greyhound 1: Respiratory evaporative heat loss mechanism (REHL) 2: respiratory (panting) 3: exertional heat illness 4: thermal windows 5: cooling interventions GHs 6.

INTRODUCTION

The racing greyhound (GH) has been selectively bred over several thousand years purely for their speed in coursing and racing. The result has been not only structural specialisation but also the acquisition of unique physiological traits crucial to the uptake, transport, and utilization of oxygen at very high rates.[1] To achieve this, the key systems, cardiovascular-respiratory-neuromuscular and thermoregulatory, associated with the intense exercise of racing have been intricately linked, so that oxygen uptake response kinetics show minimal inertia as the GH transitions from a standing start to full speed in just a few seconds, and the substantial quantity of metabolic heat produced is effectively dissipated.[2]

Researchers studying the canine genome have found that the modern GH lacks the main haplotype found in most dogs,[3] supporting the claim for an ancient lineage which has been protected by deliberate artificial selection and reproductive isolation.[4]

Li has suggested that the genes under positive selection have contributed to the unique GH phenotype, specifically its musculature and appearance, and the specialized physiological and biochemical traits required for its athleticism.[3]

It is important to note that there are differences between the racing GH and other domestic dogs.[1,2,3] Of particular importance to thermoregulation is the fact that the GH muscle mass accounts for approximately 60% of its total body weight, so it is expected that high-intensity sprint exercise may generate heat rapidly.[5] This has been confirmed by Nold, who demonstrated in four GHs that blood temperature in the pulmonary artery rose at a rate of 1.8ºC min^{-1} during a 700m sprint and was associated with an estimated increase in metabolic rate to approximately 35 times the basal metabolic rate.[6] Similarly, there are thermoregulatory advantages to the GH body shape in terms of its surface area to body weight ratio, the lack of body fat, and the very thin hair coat, usually absent on the ventral surface of the abdomen and the upper medial surface of the hind legs, which decreases insulation and enhances heat dissipation.[7]

As articulated by Jessen,[8] the rates of heat production and heat loss must be brought into balance if thermoregulatory homeostasis is to be maintained. If heat loss in athletic individuals is impaired for any reason, they may be predisposed to a condition referred to as exertional heat illness (EHI) and if this condition is either not recognised or left untreated it may progress to the more serious state of heat stroke (HS), where death may occur. This article deals with normal and abnormal thermoregulatory function in the racing GH, defining thermoregulatory dysfunction and providing recommendations for care and cooling strategies.

Part 1: Thermoregulatory Function in the racing greyhound

In athletic species such as the GH, muscular contraction produces substantial metabolic heat. This is essentially a normal process, acting to increase enzyme function and allowing proteins to function at lower pH levels. During exercise, body temperature rises, and this activates the dominant heat-loss (HL) mechanisms such as panting.

As exercise continues, HL increases to match heat production (HP). The estimated maximum metabolic rate (MMR) during exercise for the GH is 35 times resting MR, and the efficiency of transforming chemical energy into mechanical work under optimal conditions is only about 20%.[1,2,8] This means that approximately 80% of MR during exercise is simply HP. In view of this, it is obvious that dissipating the waste heat can pose serious problems.

The maximum rate of HL will be determined by three factors: firstly, the capacity of the HL mechanism; secondly, the cooling power of the environment; and lastly, redistribution of blood flow to transfer heat from the exercising muscles to the relevant parts of the body surface. Jessen[8] maintains that if the capacity of these three factors exceeds HP, Tcore will stabilize at an elevated level, but if the rate of HP is beyond the HL limit set by the three factors, Tcore will continue to rise. Core temperature measurements greater than 41.0ºC have been commonly recorded in the racing GH post exercise.[5,6,8,9]

1.0: 'Dry' or sensible heat loss

Heat is transferred by the circulating blood from an area of high temperature (heated core) to one of low temperature (skin surface), to be dissipated to the environment by the process of 'dry' or sensible heat loss, which is largely dependent upon the temperature difference between the skin surface and the environment. *Figure 1* demonstrates a general principle shared by various mammals in which evaporative cooling plays a part in

heat dissipation. The same graph can be a template for humans, horses and dogs, illustrating the relationship between total heat loss and the contributions of 'dry' or sensible heat loss as environmental temperatures change. For instance, at ambient temperatures around 12.0°C, more than 70% of total heat loss is through the 'dry' insensible route, but as ambient temperature increases, evaporative heat loss becomes more important.[10]

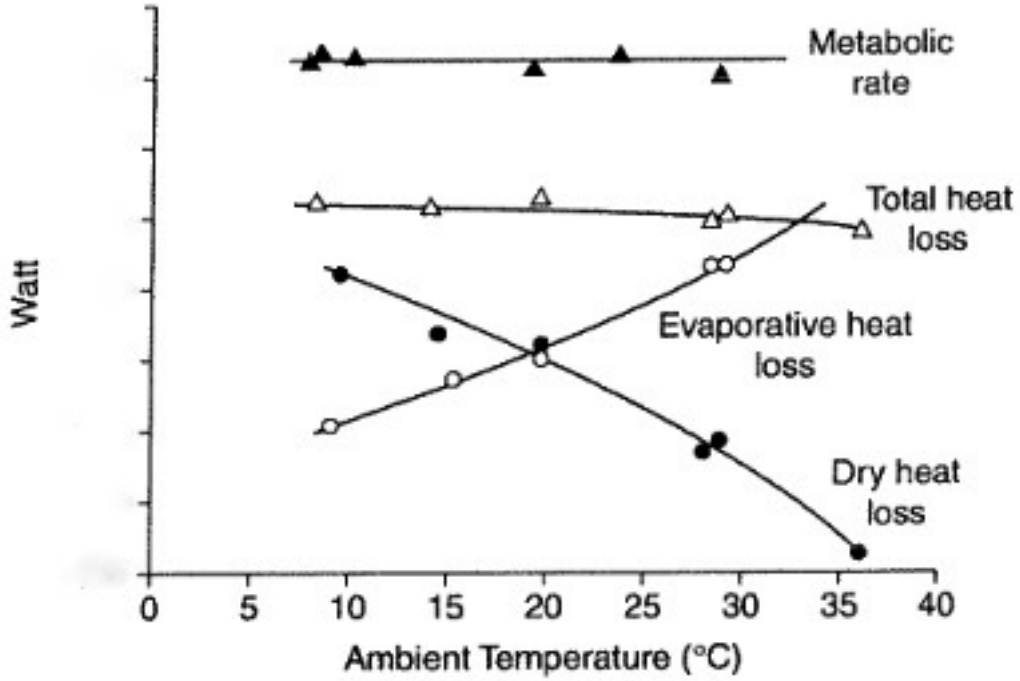


Figure 1: This diagram shows that the contribution of 'dry or insensible heat loss', involving the physical processes of radiation and convection, is largely dependent on environmental conditions. When GHs race in cool conditions (15 to 20.0°C), their skin temperatures will be greater than air temperature, and convective and radiative heat exchanges alone will dissipate their heat load. However, at higher temperatures (up to 35.0°C) the skin-to-air temperature difference becomes progressively smaller and heat loss becomes increasingly dependent on the evaporative heat loss associated with panting. At air temperatures around 36.0°C the ability of the animal to cool by 'dry' heat loss is negligible and beyond this level radiative and convective heat exchange reverses and heat is added to the body instead of being lost (*Source: Sawka et al.,2006*).

1.1: Evaporative heat loss by the upper respiratory heat loss mechanism

Dogs as a species only have sweat glands on the glabrous or non-haired portions of the skin, which are their paw pads and nose, meaning that the evaporation of fluid from these areas has a very minimal role in heat dissipation.[11]

Evaporative heat loss is achieved in dogs by their powerful panting mechanism.- Evaporation is a two-step process involving the phase transition of heated mucosal and buccal secretions from a liquid to a vapour, followed by its diffusion into the surrounding air. For example, in a study of two small dogs running for 15 min at 15 kmh^{-1}, metabolic rate was ten times the resting level and about one half was dissipated through the respiratory tract and tongue, amounting to 15 Wkg^{-1}.[12]

It is important to note that while 'dry' heat loss is determined by ambient temperature, the rate of evaporative heat loss depends on the prevailing water vapour pressure, or environmental humidity, and convective air flow or wind speed. Thus, the maximum heat loss achieved by panting depends, firstly, on the evaporative capacity of the environment, and secondly, on the physiologically sustainable rate of ventilation by the animal.[8,13] This has

important implications for any heat policy because the worst conditions in terms of heat-related illness are those involving low environmental evaporative capacity (high absolute humidity), which has the potential to seriously impair any evaporative mechanism and can predispose the racing GH to thermoregulatory dysfunction.

1.2: Water for evaporative heat loss by panting is secreted from the lateral nasal gland in the dog

Just as sweat is produced by glands in the skin for evaporative cooling in horses and humans, a source of water is required for the evaporative process associated with panting in dogs. This is provided by the paired lateral nasal glands, positioned on the premaxilla, and draining into single ducts which open two centimetres inside each nostril. This location is considered important because it prevents drying of the nasal mucosa if panting is strident. The rate of fluid secretion varies with the respiratory rate but is estimated to account for between 19% and 36% of the total fluid required for heavy panting, the remainder being provided by the salivary glands. Examination of the lateral nasal gland fluid reveals it to be hypo-osmotic, in contradistinction to horses, where sweat fluid is hyperosmotic.[37] This tonicity has important ramifications because if the dog does not 'drool' excessively when panting there may be minimal electrolyte loss. One study[38] has shown that secretions from the dog's lateral nasal gland also contains large amounts of proteinaceous sialic acid and immunoglobulin A, which is suggestive of an immune function for the upper respiratory tract.

1.3: Anatomical specialisation of the upper respiratory tract

The dog has a complex system of folded mucosa-covered nasal turbinate bones with a surface area larger than that of its body *(see Figure 2).* The vascular structure is well developed and characterized by high rates of blood flow when thermoregulatory functions are at their peak.[16]

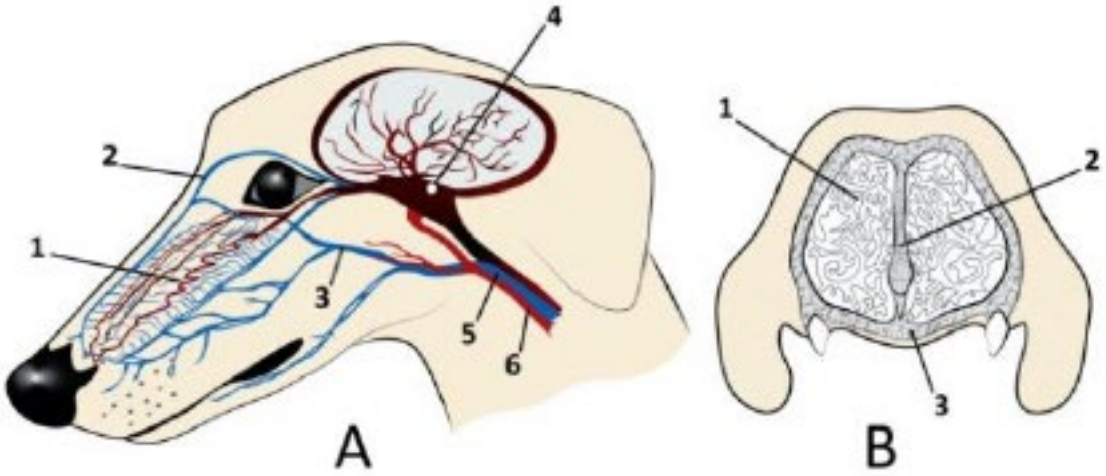


Figure 2: Part A is a simplified diagrammatic representation of the nasal turbinate structure (1) of the dog's head, showing its relationship to the brain and major vessels. Whilst the dog only has a rudimentary carotid rete, venous transporters from the scalp and face (2) take cooled blood to the cavernous sinus (4) and could be the source of unregulated direct cooling effect on the rostral brain stem (8,16). At the same time, venous blood returns from the upper respiratory tract (3) via the jugular vein (5) to the heart, where it mixes with venous blood from the rest of the body and provides further cooling for the general circulation and the brain via the carotid artery (6) (8,16). Part B shows a cross-sectional area of the dog's nose, showing the intricate folding of the turbinate bones (1). The respiratory system is divided into right and left sides by the medium septum (2) and the lower boundary of the hard palate (3) *(Source: Adapted from Baker [16]. Created by Meg Brownlow; Artwork S. Feeney).*

1.4: Studies of the canine nose show a remarkable vascularity and a complex venous drainage system

Two main arteries, the sphenopalatine and the ethmoidal group of vessels, supply the nasal cavity, the former providing blood to the respiratory portion of the nasal mucosa and the latter supplying the olfactory region. The most striking feature of the nasal cavity is the complexity of the venous system *(see Figure 3*). The veins are numerous and large with very thick muscular walls. Dawes and Prichard[17] suggested that such an arrangement could act as a sphincter and shut down parts of the venous plexuses when not required or obstruct the flow of blood out of the area, allowing the distal part of the venous system to be filled to capacity. More recently, Lung and Wang[18] postulated that the upper respiratory tract of dogs had two functionally separate venous passageways: firstly, a system of vessels characterized by high pressure and high flow drained the anterior nasal cavity via the left and right dorsal nasal veins, effected by the presence of arteriovenous anastomoses located only in the anterior nasal cavity. Secondly, there was a system of low pressure and low flow draining the more caudal parts of the nasal cavity via the sphenopalatine vein. Physiological experiments[18] proved that these two venous systems were functionally separated by the presence of valves that prevented retrograde flow and the mixing of blood in the different venous channels, enabling them to function independently in response to changes in environmental conditions and levels of ventilation.

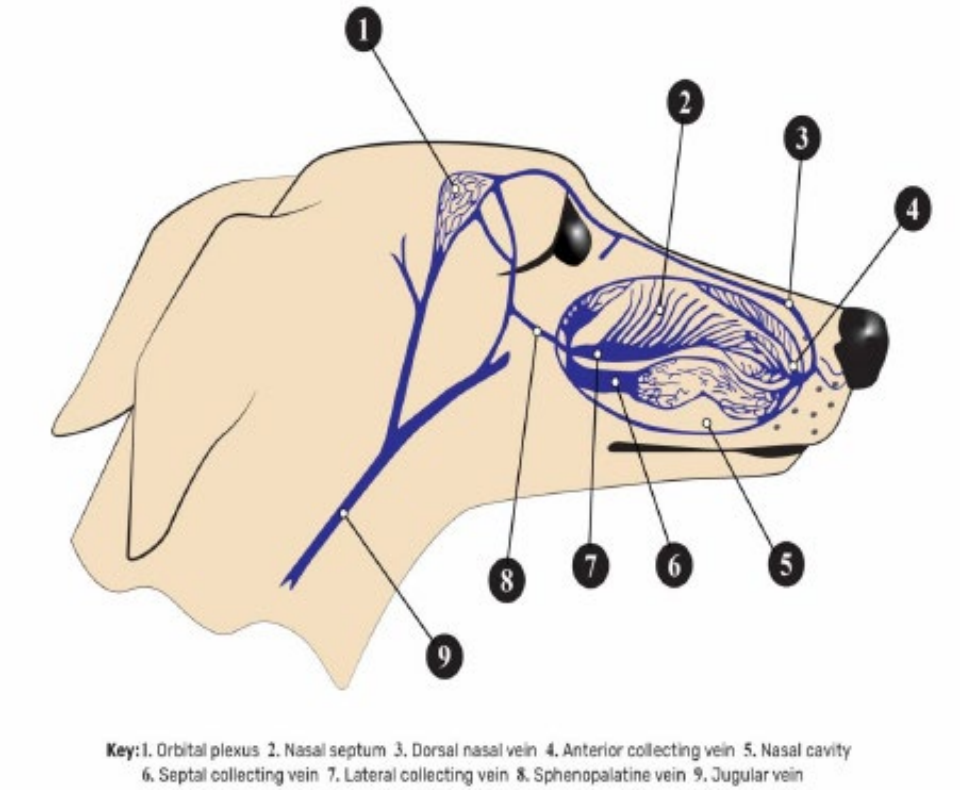


Figure 3: The intricate nature of the venous drainage system associated with the upper respiratory system. It has two functional and independent systems that allow for optimal nasal mucosal blood flow as required by exercise and environmental conditions (*Source: adapted Lung and Wang [18], created M Brownlow; Artwork Sonia Feeney*).

1.5: Specialised vascular structures which facilitate heat dissipation – the arteriovenous anastomoses (AVAs)

Arteriovenous anastomoses (AVAs) are direct connections between small arteries and veins. Anatomically, they are short vessel segments with thick muscular walls, a large internal diameter, and are densely innervated by the adrenergic nervous system, which controls blood flow by vasodilation and vasoconstriction (*see Figure 4*). While the diameter of the vessels varies, 10μm at rest and up to 150μm when open, they provide a low resistance connection between arteries and veins, shunting blood directly into the larger collecting veins, which have direct access either to specialised parts of the skin surface or the right heart, potentiating general body cooling.

In the panting dog, it has been estimated that 70% of the total tongue blood flow passes through AVAs.[19] They are present in many tissues but are most numerous in specific parts of the skin, particularly acral regions such as the ears, nose, toes, and paw pads.[8]

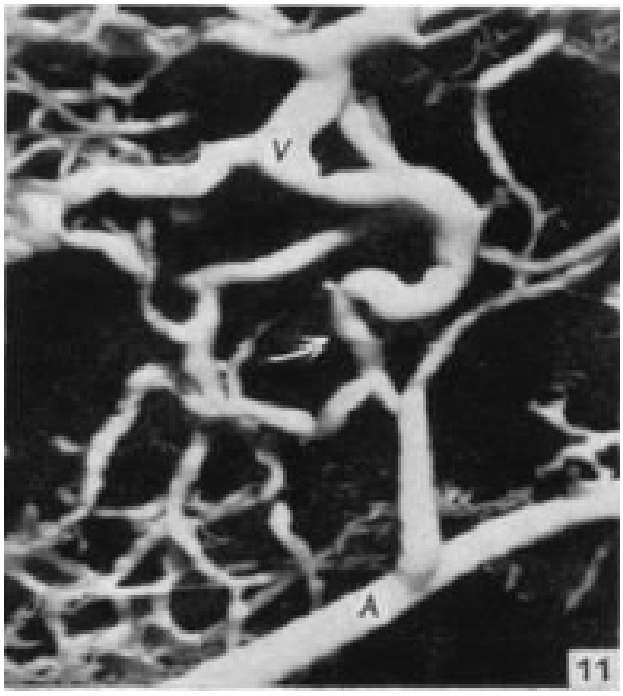


Figure 4: This photo shows the anatomical structure of the AVA. They are frequently tortuous, and their lumen is small. The arrow shows the area where the artery can change into the vein, giving cooled blood from evaporative respiratory surfaces access to the venous side of the circulation. AVAs can be seen singly or in groups of two to four vessels, each lying close to another. Occasionally, the coils of adjacent AVAs are intertwined to form a knot [19]. (*Source: Google Images).*

1.6: How the panting mechanism works – the various patterns of air flow through the nose and mouth enable the maximising or minimising of heat loss, depending upon the thermoregulatory requirements of exercise intensity and environmental heat loads

Inevitably, respiration involves the loss of heat and water because the water content of exhaled air is higher than that of inhaled air, but this passive loss of water and heat can be minimized. **A resting animal in a thermoneutral environment** inhales and exhales through the nose. During inhalation, air passes over the wet respiratory mucosal surfaces and is humidified and warmed. In so doing, the mucosa is cooled, as is the blood flowing through it. The inspired air continues into the lungs, where again it is warmed to

body temperature and fully saturated. During exhalation, as it passes back over the cooler surfaces of the nose, a substantial fraction of its water content recondenses on the mucosal surfaces.[8,20] This counter-current heat exchanger enables the dog to conserve much of the heat and water that would be lost if the air was simply exhaled at body temperature.

In a warm environment, increasing the amounts of respiratory evaporative heat loss becomes a necessary means of compensating for the decline in dry heat loss in a panting animal. To achieve this, in the first instance, the animal needs to secrete more water from the lateral nasal glands and to increase nasal mucosal blood flow, allowing the adjustment of mucosal surface temperature to the needs of heat dissipation. Higher blood flow effectively warms the mucosa quickly after inhalation and consequently recuperates less heat and water on exhalation. This means that more heat is dissipated, even though inhalation and exhalation is still occurring entirely through the nose.[8,13,21]

In a hot environment, even the resting animal must dissipate more heat, and this is achieved by increasing the rate of ventilation. At this time the nasal heat exchanger can be circumvented and although inhalation is still entirely through the nose, exhalation is now occurring increasingly through the open mouth in preference to the nose.[13,21]

During exercise and as the dog begins to pant more stridently, less air travels through the nose. This is because the resistance created by the turbinates is too great to sustain the high rates of airflow required and there is a switch to mouth breathing. Humidification and warming of the air inhaled through the mouth are achieved by a large saliva production and concomitant increases to blood flow of the tongue and buccal surfaces.[8,13,21] The tongue assumes a greater relevance as the major heat loss effector organ (*see Figures 5,6 and 7*), not only dissipating heat directly to the environment but also humidifying and warming the inhaled air. It is heavily vascularized with specialized heat transfer structures (AVAs and retia venosa), enabling a large volume of blood to flow in close apposition to the exposed tongue surfaces. In an amazingly efficient system, the cooled blood exiting the tongue then returns directly to the right heart, where it mixes with and cools the general circulation.

Figure 5: Changes to the tongue in the immediate recovery period after high intensity exercise. The corners of the mouth are pulled backwards into a smile, the tongue is wide, tie-like, and often curled at the end. In the right environmental conditions vapor can be seen to evaporate off the tongue's exposed surface. (*Photos courtesy of Lachlan Naidy – Greyhound Racing New South Wales Social Media Manager*).

Figure 6: A dog exhibiting the rapid, shallow type of panting immediately after racing. The mouth is not widely open, the corners of the mouth are pulled back into a smile, the tongue protrudes and is large and like a necktie in appearance. Drops of secretion can be seen on the tongue's tip (*Source: Meg Brownlow*).

Figure 7: A dog immediately after racing with deep alveolar or second phase panting. Note that the mouth is wide open, which decreases resistance to air flow. Inhalation and exhalation are occurring entirely through the open mouth. Note the curled end of the tongue (*Source: Meg Brownlow).*

Hales and Dampney have described two types of respiratory patterns in dogs:[22]
a rapid shallow panting, where high ventilation rates are achieved by a greatly increased respiratory frequency combined with a reduced tidal volume, so that alveolar ventilation and acid-base balance of the blood remain essentially unchanged, and in very hot environments and at a high Tcore, a deeper alveolar type of respiration is observed with a larger alveolar ventilation which is described as **second-phase panting.** In this mode there is a lower respiratory rate, and inhalation and exhalation take place mainly through an open mouth with the tongue protruding and becoming very large *(see Figure 7).* A potentially harmful side effect of second phase panting is respiratory alkalosis, with arterial pH levels rising from 7.4 to 7.6 or more. Carbon dioxide can also fall to very low levels, for example, from 40mmHg to 10mmHg, but it is metabolic acidosis due to the very high levels of lactate that drives the respiratory centre in the immediate post-race period.[8,20,21]

In the racing GH there are competing demands on the panting mechanism

As articulated by Jessen,[8] high intensity exercise during warm/hot conditions presents a complicated situation because there are competing demands. It is suggested that the rate of REHL under such conditions is governed by two factors: firstly, by the metabolic and exercise related requirements for blood gas control through deep alveolar ventilation, and secondly, by the animal's thermoregulatory status and its drive for heat dissipation through dead space ventilation. From the seminal work of Hales and Dampney,[21] it is suggested that to achieve physiological normality the respiratory pattern tends to oscillate between the rapid shallow and deep alveolar types of panting. Environmental conditions will also affect panting efficiency. When humidity levels are high, the evaporation of heat-laden secretions from the tongue surface may be compromised and track veterinarians may need to assist cooling. Overall, however, the Greyhound's REHL is an adequate and finely tuned mechanism.

PART 2: THERMOREGULATORY DYSFUNCTION IN THE RACING GREYHOUND

2.0: Disorders of thermoregulation: passive heat stress due to environmental conditions versus exertional heat illness

Thermoregulatory disorders due to extremes of environmental heat, such as heat waves and vehicular confinement, are common in all species, particularly affecting the aged, the very young, and the sick in human subjects. This is generally referred to as passive heat stress (PHS), and heat stroke (HS) is a common outcome.[22] Exercise-related thermoregulation disorders, referred to as EHI, presents in various ways and can also be followed by HS and death. These have been reported across species and are the third highest cause of mortality in human athletes during physical activity.[22] They have been reported in the racing Greyhound,[5,23,24] as well as sporting,[25] working, and military dogs.[26,27] In general, when comparing PHS to EHI it is considered that the latter causes more severe multiple organ injury at lower core temperatures than PHS,[28] because the redistribution of cardiac output occurring post exercise leads to hypoperfusion of splanchnic vascular beds. This can result in endotoxin release from the gut, which acts as a trigger for the systemic inflammatory response which has been identified as a major driver for EHI.[28,29] There may also be a greater degree of cerebral hypo-perfusion, which facilitates the onset of more severe neurological signs than those manifested by PHS.[22]

2.1: Misdiagnosis of EHI is common in all elite athletic species and may contribute to the increased levels of morbidity and even mortality in affected individuals because of the time taken to initiate treatment

The clinical manifestations of EHI in TB racehorses, GHs, and humans are those of a neurological disorder, which early in their onset can be vague and even bizarre, presenting a challenge to diagnosis.[23,30] Because the effects of EHI are determined not only by the absolute temperature elevation but also its duration, the importance of early recognition of EHI and prompt treatment cannot be overstated.[31] Time spent making a diagnosis based on the procurement of rectal temperature measurements may be wasted: most recently, a consensus of expert opinion has recommended that the presence of central nervous system (CNS) dysfunction is sufficient for a diagnosis of EHI/HS and is likely to define the condition with greater sensitivity and specificity.[32,33,34] In a review of why human athletes died after an EHI event, it was considered that misdiagnosis of the condition was most important, followed by delayed care, and inefficiency of the cooling modality used for treatment.[32]

2.2: An experimental study involving environmental heat load together with forced exertion in dogs provides descriptions of clinical manifestations, clinical pathological, and gross autopsy findings

In a study by Shapiro et al[33] that was considered inhumane, ethically unjustifiable, and poorly designed, mongrel dogs were forced to exercise on a treadmill with a superimposed high environmental heat load for prolonged periods of time until certain target core body temperatures were reached. Detailed observations concerning the correlation between heat illness severity, clinical manifestations, clinicopathological and autopsy findings were documented. In summary, ataxia was a pathognomonic finding in most cases, seizure activity was only seen in some dogs, along with the presence of gastrointestinal symptoms, vomiting, diarrhoea and hypersalivation. Despite substantial innate problems with this study, there were three important findings. Firstly, that dogs whose temperature did not reach 43.0°C showed no clinical signs of a heat-related illness. Secondly, that the duration of the dog's body temperature above 43.0°C was the most accurate predictor of death, and finally, that the severity of heat illness manifestations was directly and closely correlated to the duration of the rising phase of the temperature elevation. The authors have included this study not to support a benchmark temperature level for a diagnosis of heat illness, but rather to support the concept of a critical time frame within which to initiate cooling before thermal injury occurs.

2.3: Clinical reports of EHI in GHs in Australia

In Australia, a post-exercise distress syndrome (PEDS) and exercise-induced ataxia (EIA) have been described. In a study[23] involving 962 starters (n = 768 dogs) over a 6-month period, 15 dogs (0.4%) were found to be ataxic after racing; this included animals of both sexes. Ambient conditions were not described. Affected dogs exhibited a marked forelimb hypermetria within two minutes of race completion with a complete resolution of clinical signs within three minutes. No cases of collapse, seizure, or sudden death were recorded. Some dogs in this study were repeat offenders, and the authors suggested the possibility of a genetic link in those individuals.[23]

There are more severe cases reported anecdotally[23] of dogs with sudden bursts of motor activity, or marked overflexion with protraction of the limbs, which may suggest that the CNS dysfunction was of a cerebellar origin. In human subjects, cerebellar dysfunction is the most common neurological finding in EHI/HS victims and is characterized by incoordination, gait ataxia, nystagmus and tremor. It has been documented in human

subjects that the cerebellum is particularly vulnerable to the effects of hyperthermia, manifested by damage to the Purkinje cell layer, with cerebellar atrophy demonstrated on magnetic resonance imaging, sometimes many months after the initial insult.[64]

2.4: Clinical reports of EHI in dog breeds in the UK

In a seminal study, Hall et al.[36] reviewed the veterinary records of over 9 million dogs presented to primary care veterinary practices in the UK with heat-related illnesses and found that exercise was the most common trigger (74.2% of cases), followed by purely environmental causation (12.9%), and vehicular confinement (5.2%). It was concluded that in the UK, exertional heat illness affected more dogs and killed more dogs than confinement in a hot vehicle and occurred all year round, with a fatality rate of 7.6%. Reports from Israel also revealed that EHI was a more common cause of heatstroke in that country.[39] Identified risk factors included advanced age, respiratory disease, brachycephaly, obesity, long haircoat, lack of fitness, and limited opportunity for acclimatisation.

Hall further identified[38] nine breeds of dogs at extreme risk of a heat-related illness compared to the Labrador Retriever, which was chosen as the comparator breed due to their popularity and typical canine type of body conformation. At the head of the group was the Chow Chow (x 17 risk), English Bulldog (x 14 risk), French Bulldog (x 6 risk), Dogue de Bordeaux (x 5 risk) and the Greyhound (x 4 risk). Interestingly, the GH did not appear in the risk analysis for environmental heat-related illness but was second in line for vehicular confinement-related heat illness, behind the Chow Chow.[67] This is important information for Greyhound welfare in the UK, suggesting that some dogs required primary veterinary attention after racing or training, and often experienced a vehicular confinement-related heat illness. The fact is, that prevention of both exertional and vehicular heat illnesses relies on the dog's trainer or handler, firstly for recognition and secondly for the ability to efficiently cool the affected animal.

2.5: Proposed categorisation of CNS dysfunction for EHI in racing greyhounds

From the human literature it is apparent that the measurement of rectal temperature as a means for the diagnosis of EHI in human subjects is unreliable and has lost favour. In a consensus statement from the American College of Sports Medicine[22,31] it was stated that "there was no strong evidence for a temperature threshold to discriminate EHI/HS and that relying on a specific core temperature should be avoided…. instead signs of CNS dysfunction should be the main diagnostic criteria being the most reliable clinical indicators of EHI/HS". The authors agree with this view and previously have developed a case definition for EHI in TB racehorses based on signs of CNS dysfunction, enabling track veterinarians to recognise the earliest clinical signs more easily, make a rapid assessment, and institute treatment accordingly.[39,41]

Despite the sparse documentation in the veterinary literature describing the clinical manifestations of EHI in racing GHs, anecdotal information from professional GH trainers and racetrack veterinarians has enabled the authors to collate a 'case definition' or 'clinical grading tool', which describes neurological manifestations that are extremely diverse, although divisions between grades are not necessarily clear. In general, for the racing GH, CNS dysfunction is on a spectrum from minor to severe (*see Figure 8*). A similar clinical grading tool based on CNS dysfunction has been developed for other dogs by Hall et al.,[42]

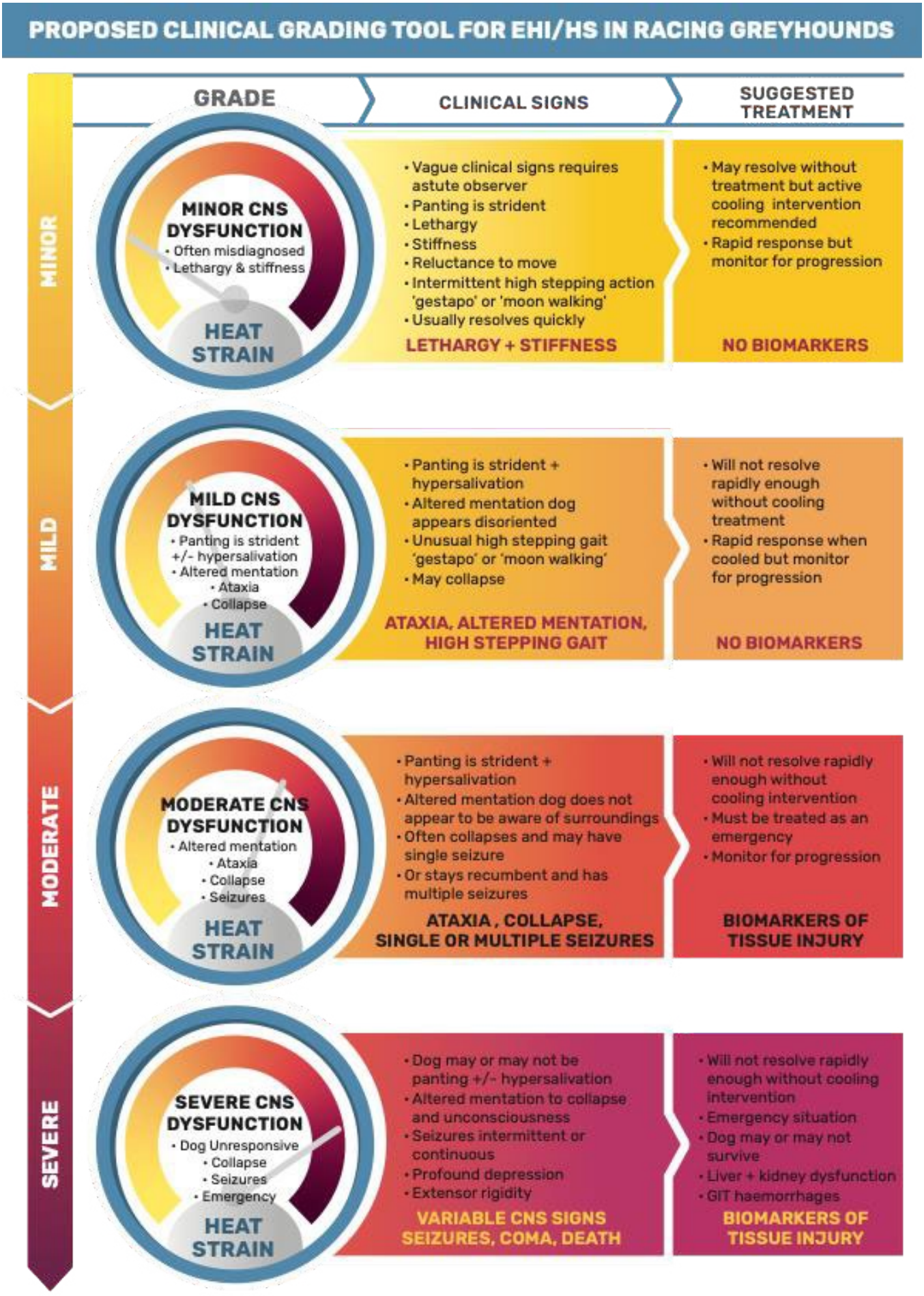


Figure 8: Proposed clinical grading tool for EHI/HS in the racing Greyhound, based on a consensus from professional dog trainers and experienced track veterinarians. Note that levels are arbitrary and may not be distinct, with progression occurring according to the efficiency of cooling treatments (*Source: Created Meg Brownlow; Artwork S.Feeney*).

Level 1: MINOR CNS DYSFUNCTION

The very first clinical signs of EHI may be vague and only seen by an astute observer. After the GH finishes the race, there is an elevated rate of panting and fluid may drip from the tongue as expected. There are very minor changes to mentation, with the dog appearing to be lethargic, stiff, and reluctant to move, with the handler having to encourage forward movement. This has often been viewed as exhaustion or cramping, which may become a confounder for early cases of EHI. When the dog walks forward there may be a few unusually high steps, which have been described anecdotally as 'Gestapo or moon walking', or it may be walking on 'clenched toes' but these may disappear quickly as the dog passively cools by panting on its way back to the kennelling area. It is important, however, to recognize these signs early and initiate active cooling, thus ensuring that the condition will be rapidly terminated without progression to a higher level. There should be no biomarkers of tissue injury at this minor level.

Level 2: MILD CNS DYSFUNCTION

Dogs finish their race and there may be a few minutes before the onset of clinical signs. Panting is strident and there may be hypersalivation, which is often mistaken for 'frothing at the mouth'. They have an altered mentation and appear disoriented, or may collapse momentarily, which can be mistaken for the dog 'having a fit'. They then follow their handler but display a more consistent, ataxic, high-stepping gait. These clinical signs may last for 3 to 4 minutes without any cooling intervention or become more severe. If actively cooled, they respond rapidly. There should be no biomarkers of tissue injury at this mild level.

Level 3: MODERATE CNS DYSFUNCTION

These dogs show signs of CNS dysfunction almost immediately after racing, either in the catching pen or during the walk back to the kennels. The have an altered mentation and appear to be unaware of their surroundings. They often collapse, remain recumbent, and may demonstrate a single seizure. Trainers describe some dogs falling to one side and adopting a galloping action. The longer the delay in returning these animals to normality by cooling, the greater the probability of progression of the illness and the presence of biomarkers of tissue injury.

Level 4: SEVERE CNS DYSFUNCTION – HEAT STROKE

These dogs display profound CNS dysfunction almost immediately after racing, often with loss of consciousness. They may be unresponsive on the ground and may demonstrate seizure activity. Others display generalized muscular spasticity (hypertonia), which is easily misdiagnosed as generalised cramping. Dogs can then become comatose, and death may quickly follow. This is an emergency situation and advanced veterinary management may or may not be effective in preventing death; nevertheless, cooling is mandatory as a first line of treatment and whole-body immersion is recommended.

It is important to note that seizures have a pathophysiological mechanism of both neuronal excitation and reduced inhibition. Animal studies have shown that prolonged or persistent hyperthermia-induced seizure activity can cause long-lasting changes in neuronal excitability of the limbic system, possibly establishing a permanent predisposition to seizure occurrences.[43]

The use of biomarkers as an assessment of multi-organ injury associated with a single episode of EHI/HS has been advocated as an objective tool in the determination of 'return to activity' policy for human subjects.[44] This involves the collection of blood and the determination of serum levels of alanine amino transferase (ALT), aspartate amino transferase (AST), creatinine phosphokinase (CPK), lactate dehydrogenase (LDH) and alkaline phosphatase (ALP). Overall disturbances in clinical laboratory analytes

peaked during the 4 days following injury and persisted outside their respective reference ranges for up to 16 days. The study by Ward and colleagues,[45] which included 2,529 EHS episodes from US military personnel, concluded that 16 days was the time frame for laboratory normalization and maintained that this represented a foundation for return-to-activity decisions following heat injury.

Sudden death on the racetrack:

Sudden death is defined as acute collapse followed by death and occurs in all athletic species. The individual can be affected in the running or after completion of the race, or there is collapse and death a short time later. Most cases are diagnosed as heart attack or heart failure. In all species it is difficult to determine the exact cause of sudden death, even with a full post-mortem examination, because lesions found are often not specific or the animal has died too quickly for them to become pathologically established.[46] In a study into the risk factors of sudden death in TB racehorses in the US and Canada, of 4,198,073 race starts made by 284,387 starters at 144 tracks, 15 risk factors were identified, of which racing in the summer months was one. The authors concluded that race starts in summer were likely to have been in warmer conditions, so it was reasonable to speculate that heat stress could contribute to metabolic processes leading to sudden death, most probably from fatal arrhythmias. A study in the UK[47] into sudden death in racehorses made a similar finding. Heat stroke can be a cause of sudden death due to acute, progressive cerebral oedema with catastrophic effects upon vital centres of the brain. To the author's knowledge there is no published information concerning the incidence or cause of sudden death in racing Greyhounds in Australasia.

2.6: Pathophysiology of CNS dysfunction associated with EHI

Rodent and dog heat stroke models[41] have clarified the CNS dysfunction related to experimentally induced hyperthermia *(see Figure 9)*. Firstly, there is a reduction in cerebral blood flow, which results in cerebral hypo-perfusion progressing to ischemia and is probably responsible for the earliest clinical signs of the disorder. These might be vague and include confusion, dizziness, and headache. If the hyperthermia continues unabated, there is a serotonin-mediated increase in permeability and breakdown of the blood-brain barrier (BBB) with leakage of plasma proteins from cerebral capillaries, leading to cerebral oedema.[48] This causes escalating levels of CNS dysfunction and if treatment is delayed, cerebral oedema may progress, causing neuronal injury. In the dog and rat heat stroke model, as in human victims, the Purkinje cell layer of the cerebellum shows a marked and irreversible degeneration.[41] Clinical effects at this stage are leading towards 'heat stroke' and involve stupor and/or delirium, with the affected individual unaware of their surroundings. End-stage heat stroke is characterized by a swollen, oedematous brain in a closed cranial compartment, causing compression and cellular damage to vital centres, with eventual collapse, loss of consciousness, coma, and ultimately death.[49] It has been documented by numerous researchers that elevated levels of CNS serotonin are a key driver in the heat stress-associated changes to the permeability of the BBB, leading directly to progressive cerebral oedema,[41] which if not reversed can result in heat stroke and death.[48]

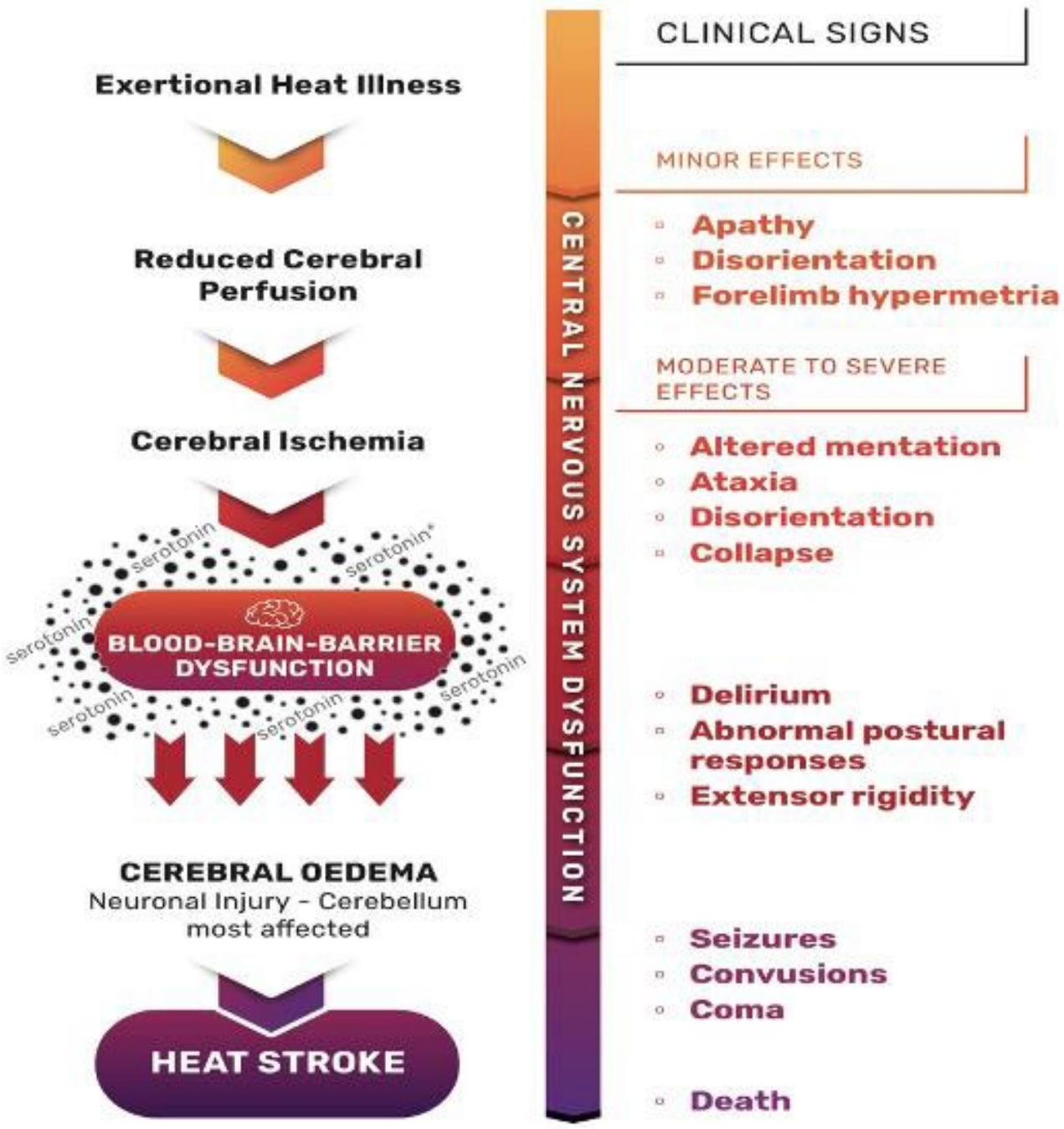


Figure 9: Postulated pathophysiological mechanism of central nervous system dysfunction and associated clinical manifestations according to cerebral pathology. Note that serotonin has been documented as the most potent neurochemical mediator involved in breakdown of the blood brain barrier, leading to cerebral oedema formation [41].(*Source: created M. Brownlow; Artwork S.Feeney.*)

2.7: Epidemiological considerations of EHI - the role of extrinsic and intrinsic risk factors and the 'triple hit' hypothesis

The simplest model of disease causation is illustrated by the epidemiological triad *(see Figure 10).* The agent of causation, which must be present in all cases for the disease to occur, is strenuous exercise, typified by the intense activity of racing. The other arms of the triad are represented by extrinsic (environmental or external-to-the animal) risk factors and intrinsic (host or internal-to-the animal) risk factors. A critical premise of epidemiological investigations is that disease conditions may not occur randomly in a population but may be more likely to occur in some individuals due to the presence of certain risk factors that predispose them to the

condition. New concepts of disease causation in the human heat illness literature have concluded that a more complex interaction between extrinsic and intrinsic factors may combine to create a 'triple hit' hypothesis which may predispose to disease occurrence.[50]

Extrinsic risk factors are represented mainly by environmental conditions, involving the four thermal essentials: ambient temperature, radiant heat, absolute humidity, and wind speed.

For some time EHI/HS events were thought to be purely related to environmental heat, thus the 'heatstroke' terminology describing the condition. In Australia, 'hot' days are characterised by, in the first instance, the combination of high levels of ambient and radiant heat. The impact of this on the animal body is that it increases skin temperature, which changes the dynamics of heat transfer, potentially resulting in additional environmental heat gain by the body. In Australia, GH racing is cancelled at an ambient temperature of 38.0ºC, but if racing is conducted just below that level on a cloudless day, radiant heat as measured by the black globe thermometer may be 10 to 15ºC higher than ambient temperature, causing considerable physiological strain due to environmental heat gain. Exposure to radiant heat can be reduced by racing in the later afternoon and into the night.[51] In Australia, a four-hour race meeting might commence at any time from about 8:30 a.m. to 7:00 p.m., so that GHs are regularly exposed to high levels of radiant heat.

In the second instance, warm to hot conditions in combination with high levels of humidity cause a different type of physiological heat strain. The GH thermoregulates primarily by panting, so when racing in warm to hot conditions, a high level of humidity will hinder evaporation and render the GH less able to dissipate heat by that route. This will result in a greater level of thermal strain than the direct effects of ambient and radiant heat alone.

A study into environmental conditions which predisposed TB racehorses in eastern Australia to EHI was performed by the senior author,[52] where it was found that an absolute humidity level between 2.0 and 2.5 kPa in the absence of significant air movement increased the risk of EHI. Wind speed has a marked effect on evaporative cooling because it modifies the immediate thermal environment at the boundary layer adjacent to the skin surface. By dispersing the hot air close to the skin, it accelerates the rate of heat transfer from those parts of the body specialised for heat transfer. Low wind speeds, less than 1.0 m/sec, will compromise evaporative cooling, whereas wind speeds above 2.5 m/sec create favourable conditions, despite high levels of absolute humidity.[52]

The importance of intrinsic risk or host factors to EHI

One of the most surprising findings from the Brownlow study in TB racehorses[52] was that the weather elements only accounted for approximately 50% of post-race EHI cases. According to the senior researcher in that study (JRB), these apparently low values agreed with the human experience in athletic, military, and other strenuous occupational fields.[53]

It is apparent that causes of EHI are not fully understood, and most recently, the human EHI literature has turned to the role of intrinsic risk factors as an explanation for its continued incidence, despite the establishment of significant regulatory and preventative strategies.[31,33,44] Those studies identified the lack of acclimatisation to exercise in the heat, lack of fitness, and poor hydration status as being most relevant, conclusions that are easily transferred to both the TB racehorse and the racing GH. Awareness of the possible presence of host factors may enable veterinarians to scrutinise individuals more carefully post-EHI and perhaps develop strategies to modify such factors, improving exercise-associated heat intolerance and mitigating EHI risk overall.

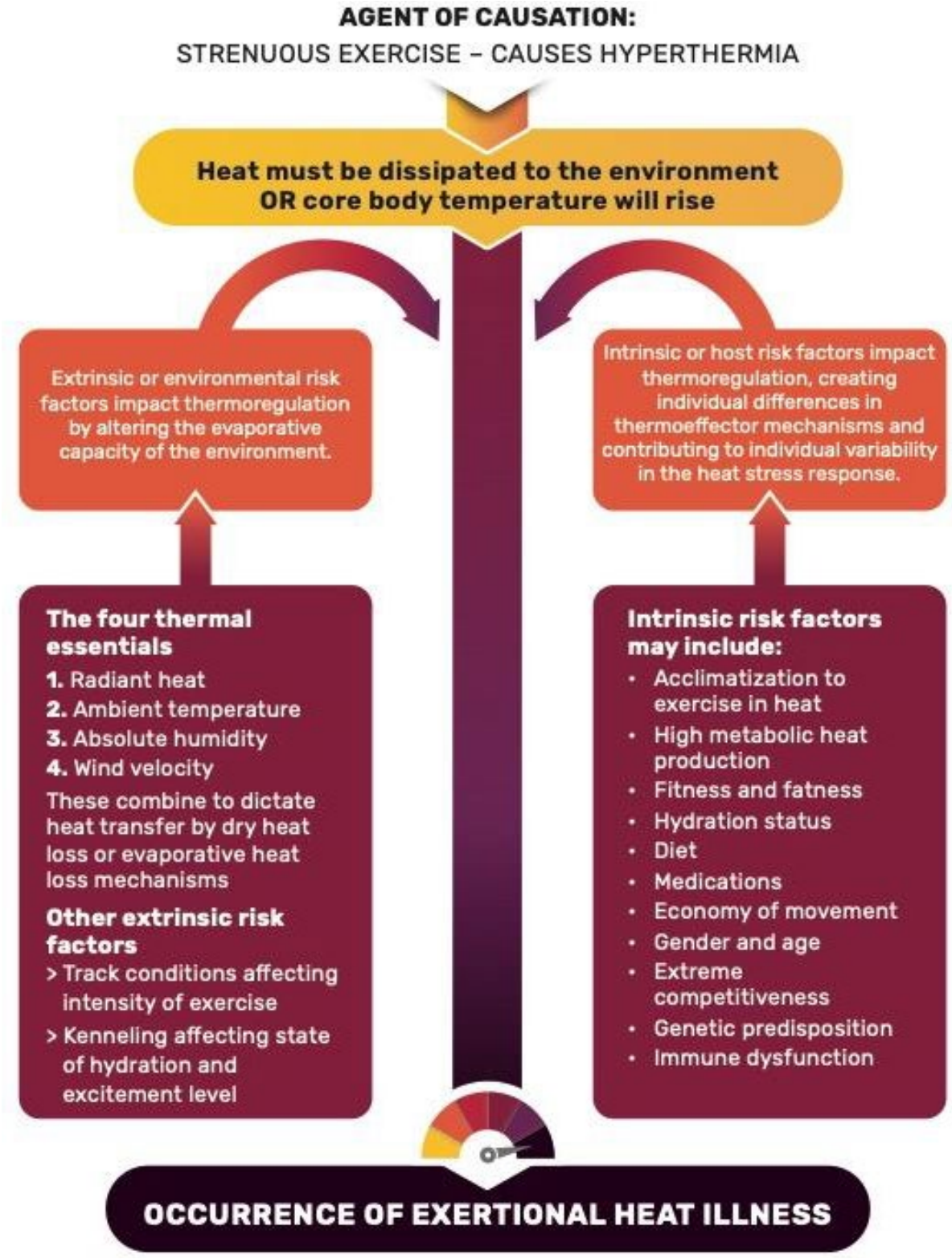


Figure 10: The proposed epidemiological triad for EHI in racing Greyhounds. The agent of causation is strenuous exercise, which produces substantial metabolic heat that must be dissipated to the environment to limit the elevation of core body temperature. Environmental factors are an is a common misconception that these are the direct important risk factor, and it cause of EHI, but it can also occur on cool days. Environmental factors act as catalyst, predisposing to EHI by diminishing the evaporative capacity of the environment or by increasing the radiant heat load. The final component of the triad is intrinsic or host factors, which have assumed greater relevance in recent studies and suggest a more complex interaction between multiple risk factors, the so called 'triple-hit' hypothesis, in the causation of EHI (*Source: created Meg Brownlow; Artwork S Feeney*).

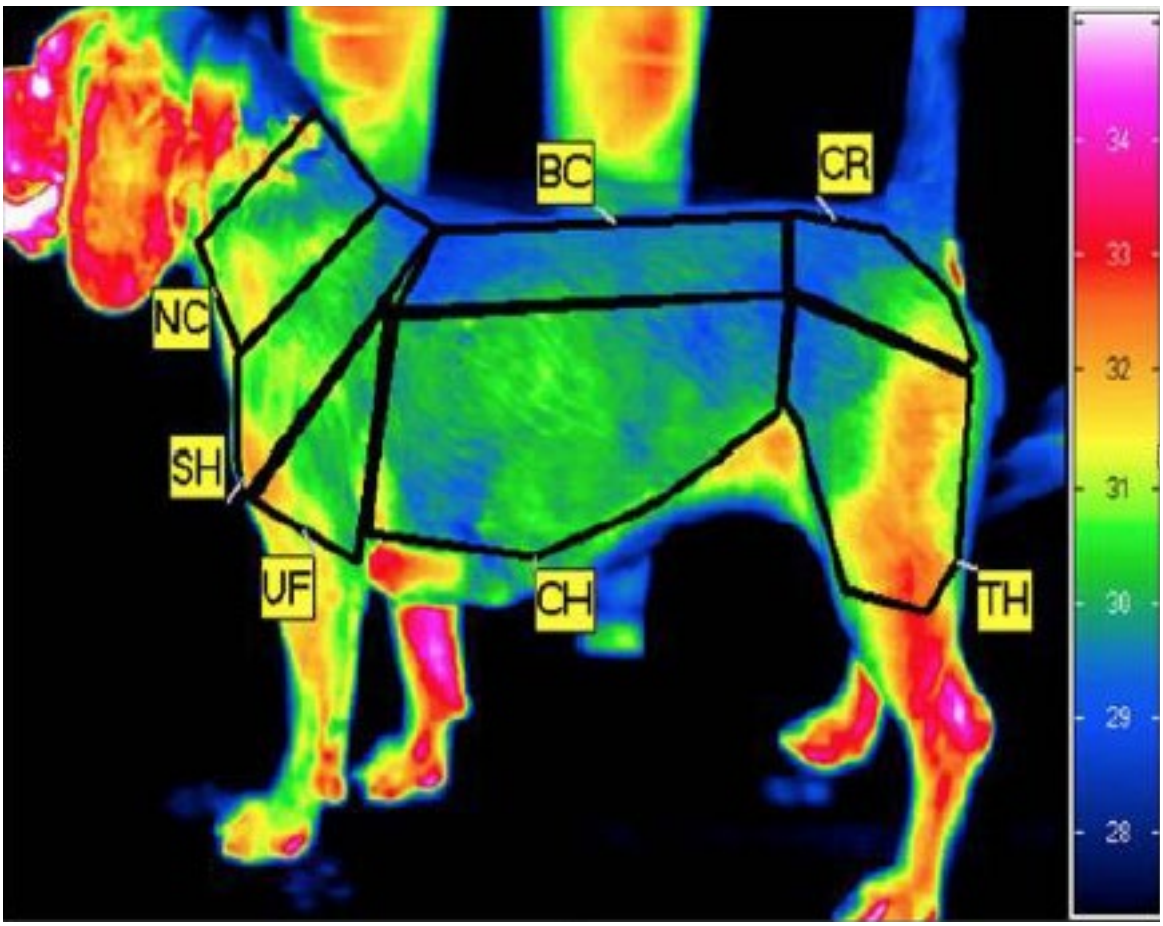
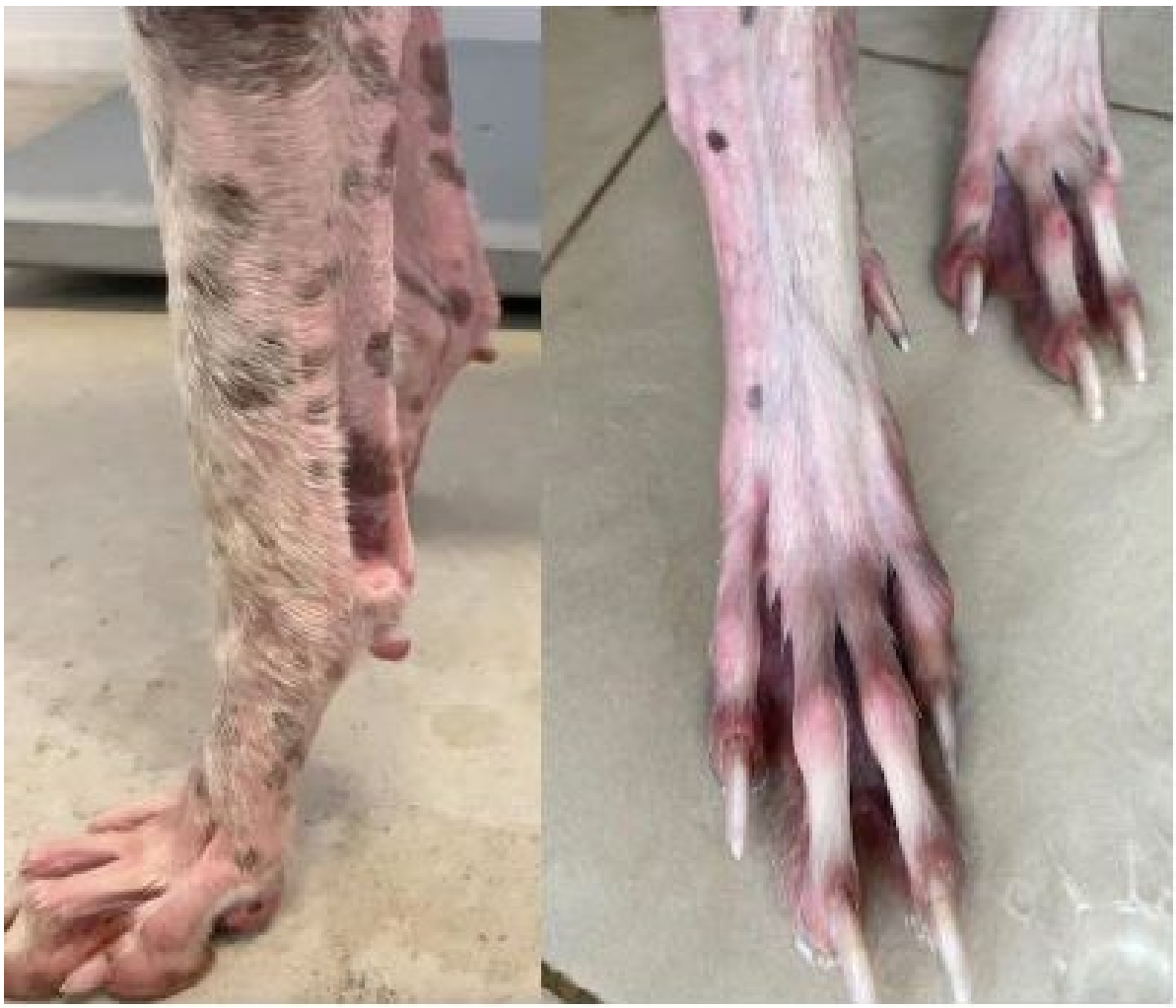


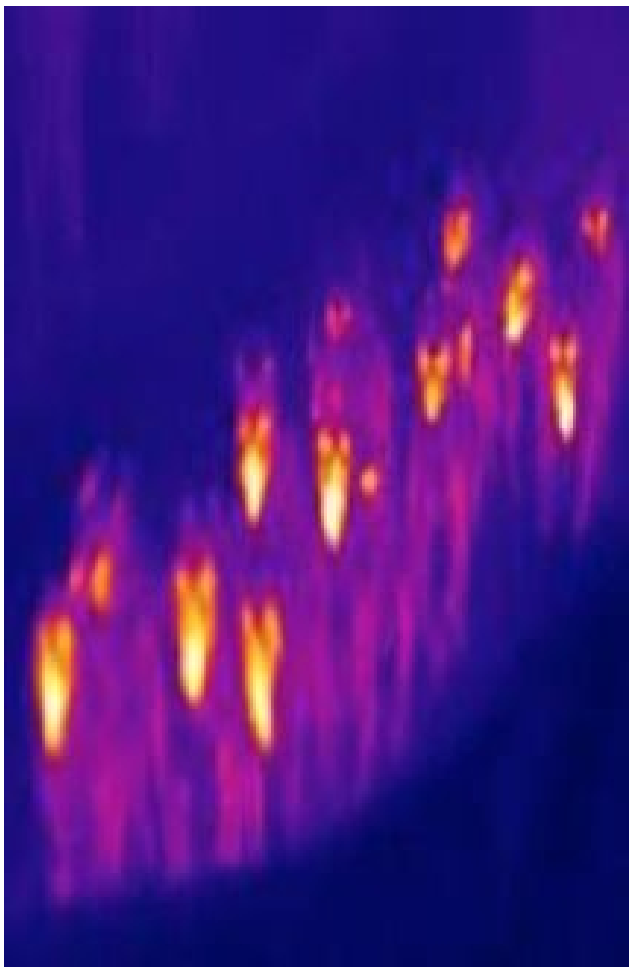
Figure 11: A thermogram from a Beagle which has been exercised on a high-speed treadmill for 35 minutes and then allowed to recover. This pictorial representation of IRT 'hot spots' has important ramifications for cooling techniques in all dogs. (*Source of thermogram: Soroko et al., [61] Open access*).

Figures 12 and 13: Note the hyperaemia of the buccal mucous membranes and internal surface of the ear in the immediate post-race period (*Source: M Brownlow)*



Figures 14 and 15: Note the hyperaemia evident in the skin of the lower extremities of the legs and feet. Note the large diameter of the veins (*Source: M Brownlow*)

Figure 16: A most important 'hot' spot' for cooling is the area of the chest wall adjacent to the elbow on both sides (*Source: M Brownlow*).



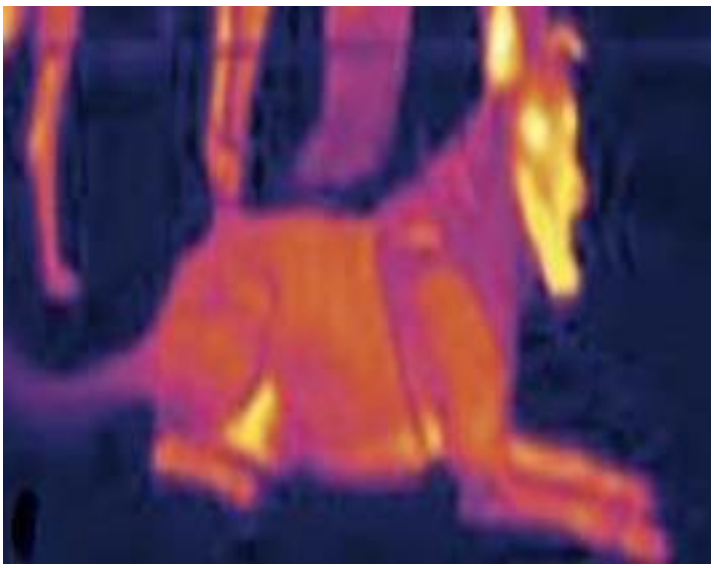


Figure 17 and 18: Infrared thermometry of sled dogs, showing heat dissipating from 'hot spot' areas. Note the dominance of the head, especially the eyes and ears, and the very brightly glowing large tongue. In the dog lying down, note the thermal emissions in the chest area adjacent to the elbow and inguinal area. The paw pads are not as visible because they are vasoconstricted to conserve heat when dogs are running on snow (*Photos with permission of Dr. Dennis Grahn, Stanford University*).

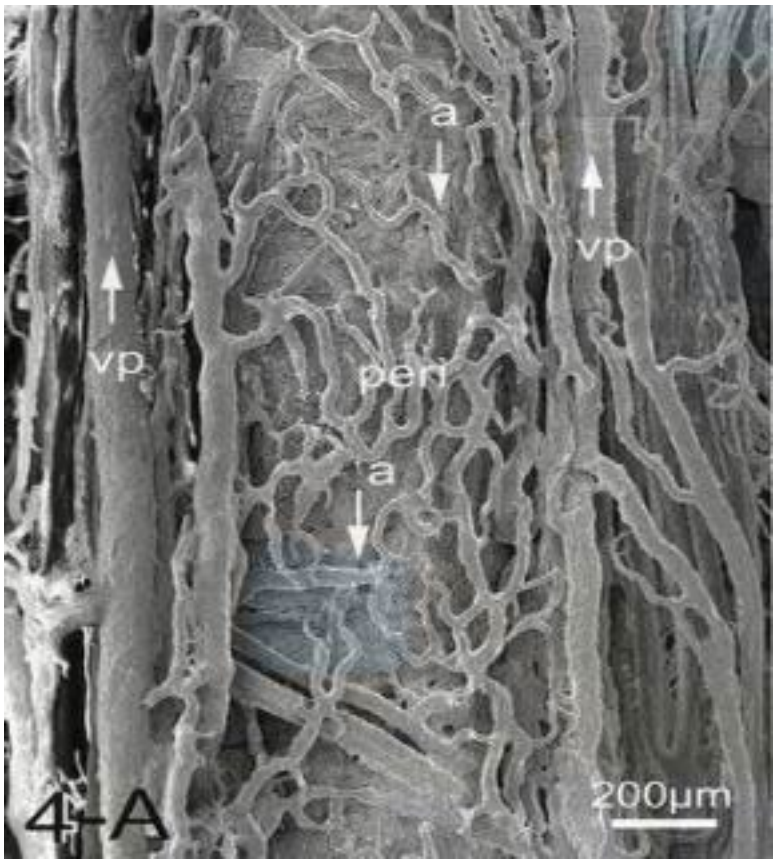


Figure 19: Scanning electron microscopy of a vein-artery-vein triad in the canine foot. Note that the capillaries and venules run parallel around the artery, forming a counter-current heat exchanger. The paw pads have a high surface area to volume ratio, so they can lose or conserve heat very easily. They are a highly uninsulated structure and should be a focus of cooling strategies for dogs racing in adverse heat stress conditions. *Key: a - artery; peri – periarterial venous network; vp – venous plexus; (Source: Ninomiya et al. [64] – Open Access).*

PART 3: COOLING STRATEGIES IN THE RACING GREYHOUND

3.1: Specialised surfaces for heat exchange: the concept of 'thermal windows'

The metaphorical term, 'thermal windows', has been used by some authors to describe specific areas that act as 'hot spots' for heat exchange with the environment.[54] These are generally species-specific and anatomically distinct, with relatively large surface areas characterised by the presence of rich vascular beds containing abundant AVAs or a dense packing of subcutaneous venous structures (retia venosa), with significant vasomotor control. Heat loss is regulated by the autonomic control of blood flow, through vasodilation and vasoconstriction.[55] These 'thermal windows' have different anatomical placement in different species. For example, in humans, heat exchange units have a regional distribution and are concentrated in the non-insulated glabrous skin regions, such as the palms of the hands, the soles of the feet, the ears, and the hairless regions of the face.[55] They also exist in the ears of jackrabbits,[56] the tails of rodents,[57] the ears of elephants,[58] the toucan's bill,[59] the paw pads, lachrymal and auricular regions, and the tongues of dogs.[60] Recently, infrared thermography (IRT) has been used to validate the presence of 'thermal windows' in a wide range of animal species, detecting heat emitted from the body in areas characterised by a high density of near-surface blood vessels that can regulate heat exchange with the environment by modifying blood flow.[61]

Soroko[62] used IRT to measure the changes in body surface temperature associated with intensive treadmill exercise in Beagles and has provided a thermogram of body temperature for the post-exercise recovery period *(see Figure 11*). From that study, areas revealed as specialised 'hot spots' were the paw pads and distal limbs of all four legs, the head and ears, a small area of the back of the neck, the chest adjacent to the elbow, and the inguinal area. Areas in blue had the lowest temperatures, i.e., the neck, back, and croup. This thermogram correlates well with the clinical picture of areas of increased blood flow in the post-race GH *(See Figures 12 to 18*) and the redistribution of cardiac output as described by Hales and Dampney.[21]

3.2: The redistribution of cardiac output associated with environmentally heat-stressed greyhounds

In a seminal study, Hales and Dampney[21] used radioactive techniques to measure changes to the distribution of cardiac output when GHs were subjected to extreme levels of environmental heat. They reported that there was a 74% increase in CO, with 4-6% of it passing through AVAs, compared to only 1% under thermoneutral conditions. There was increased blood flow to the structures of the ear, nose, mouth, and tongue, as expected with panting. There was also increased blood flow to the extremities of all four limbs, which identified these as focus areas for heat transfer. The authors confirmed that the diversion of large volumes of blood through AVAs was an important heat loss mechanism and overall, the results of this study validated the concept of 'thermal windows' as hot spots for heat exchange.

3.3: Specialised 'radiator-like' vascular structures for heat exchange in the foot pads of dogs

Ninomiya[63] studied the functional anatomy of the paw pad in dogs, using scanning electron microscopy and techniques which delineated its vascularity. These showed that it was a highly vascularized, relatively uninsulated structure, and represented an ideal surface for heat loss when required. The structure *(see Figure 19*) incorporated an intricate counter-current mechanism where arteries and veins are enmeshed together in an anatomical arrangement considered to be a functional analogue of the canine tongue. The veins surround an artery and run parallel to form a vein-artery-vein triad, so that the arterial blood flows into the pad surface in the opposite direction to the venous blood

flowing out. Venules are in intimate contact with each other, so that there is a constant temperature gradient between arteries and veins, making an effective counter-current heat exchanger. Venous transporters need only to carry the returning blood a very short distance up the limb extremities directly to the right heart, where it mixes with heated blood from the core and contributes to general body cooling.[55]

4.0: DISCUSSION

4.1: The primary misconception concerning diagnosis of EHI/HS in racing GHs involves the reliance on a benchmark body temperature that has been extrapolated from human HS victims

Research from racing GHs, military, sporting, and working dogs has suggested that veterinarians need to reconsider what is defined as a 'normal' post-exercise temperature for dogs involved in strenuous physical activities.[64] In human subjects, a temperature of 41.5ºC has been historically used in the diagnosis of EHI/HS and extrapolated to other species, notably the horse and the dog. This has created confusion because elevated post-exercise temperatures considerably greater than 41.0ºC have been routinely recorded in dogs[5,6,65,66] and horses[30] without adverse effects. To put this in perspective, a recent systemic review[67] defined a mean resting rectal temperature in human subjects of 36.32ºC, while dogs have a much higher normal resting level of 38.8ºC.[42] This is a difference of almost 2.5 °C between species. If a typical HS temperature for human subjects of 41.5ºC is accepted (knowing that temperature is unreliable for heat related illness diagnosis), it represents an elevation of almost 5.0ºC from the resting normal for humans, but an elevation of only 2.0ºC if applied to the resting dog. On the other hand, if we apply the 5.0 °C to the dog, we arrive at a temperature level of greater than 43.0ºC for heat related illness, which correlates with the Shapiro study[34] as the level above which EHI/HS manifestations occurred.

4.2: What causes the CNS manifestations in the EHI/HS condition

A consensus view from experts in the EHI/HS field in human subjects emphasises that reliance should not be placed upon a certain temperature level for a diagnosis of the condition and that the presence of CNS signs should be regarded as the most sensitive indicator of the condition, unless proven otherwise. As described previously (*see Section 3.6:*), Sharma and colleagues,[41,48,49] who used a dog and rat heat stroke model to investigate the effects of hyperthermia, showed that alteration to levels of certain neurotransmitters, particularly serotonin, appeared to be involved in the molecular mechanisms of blood-brain-barrier disruption, oedema formation, and cell injury. This was considered causal for the brain dysfunction that resulted in the associated behavioural deficits. It is of interest that although there was a documented reduction in cerebral blood flow following experimentally induced heat stress, there was no evidence that brain hypoxemia was of importance as a driver of the condition, and it was considered unrelated to BBB disruption in any region of the brain.[48 pg:516]

4.3: Effective Treatment for thermoregulatory dysfunction in the racing GH is dependent upon early detection, rapid assessment, efficient cooling strategies, and advance planning

4.3a: Early detection is enabled by the education of trainers, handlers and OTVs in recognising the earliest clinical signs of an EHI event. This is achieved with the use of a case definition descriptor, or grading system, based on signs of CNS dysfunction, which are considered the most sensitive indicators of an EHI event (*see Figure 8*). The grading system herein originates from a consensus of experienced professional trainers and OTVs working at the racetrack. In line with expert opinion from the human medical field, the use of a body temperature measurement as a diagnostic criterion can no longer be supported,[22,28,31,32,33,44] and studies from the canine and horse racing heat-illness field also confirm that view.[42,30]

It is difficult to compare 'The VetCompass clinical grading tool' proposed by Hall and colleagues[42] for heat-related illness in the general dog population to that proposed in this article for the racing GH. Firstly, theirs is a retrospective analysis of clinical records of dogs presented to primary care practices and the researchers have themselves identified that descriptive data may be incomplete or inaccurate. The tool describes only three grades, mild, moderate and severe, and the type of causal heat-related event cannot be defined. There is also a mixture of dog breeds, each probably represented by a complicated array of predisposing risk factors, and the researchers conceded that there was probably an under-reporting of the first stages of the heat illness episode because owners did not present their dogs until cases were more advanced along the illness spectrum.[42]

In contradistinction, the proposed clinical grading tool for racing GHs is applicable immediately after race completion, allowing the earliest observations by a range of professional trainers, stewards and OTVs. This has made the 'Minor CNS dysfunction' case definition possible, and other more severe case descriptors to be categorised accordingly. It is important to note that the grading tool represents a progressive disorder and the distinction between grades is not necessarily distinct. Most cases will be classified as minor or mild, with some progressing to moderate, but only a very low incidence of cases demonstrating severe manifestations. Further research is required in Australia nationally to gain data on the incidence of heat illness It is considered that the grading system provides a breed and sport specific tool to assist in the triage of racing GHs, enabling the optimisation of their care following a heat-related event.

When dogs are heat stressed in their natural environment, they will seek a body of water and stand immersed to the demarcation line from shoulder to hip, which covers all distinct anatomical areas for heat exchange, except for the head (*see Figure 28*). At the racetrack, the immersion technique can be applied using plastic tubs (*see Figures 29 and 30*), which should ideally be located somewhere near the catching pen, in case of a heat-related incident at that location. This supports the concept given priority in human athletes, that any heat affected individual needs to be 'cooled first and transported second'.[33] Time lost getting the dog to a veterinary room for assessment can mean possible tissue damage due to thermal injury. Additional placement of immersion tanks in the vicinity of the wash bays in the kennelling area allows veterinary scrutiny as dogs return from competition. Cooling can be ceased when there are no abnormal CNS signs, when panting has significantly diminished in intensity, and ear temperature using the IRT device has dropped substantially.

4.3b: Rapid Assessment

Studies of the facial region of the dog have validated the internal surface of the ear as a thermal window.[68] The first author has demonstrated in the TB racehorse that its skin surface, also a thermal window, could be used in conjunction with a hand-held IRT device to measure its temperature relative to other horses within the post-race group, enabling rapid assessment and prioritisation of any horse requiring an immediate cooling intervention.[69]

Similarly, in a pilot study of racing GHs by the first author, the IRT device was used in a no-touch ear canal technique to screen the entire post-race group of eight dogs as they arrived at the wash-bay and to prioritise individuals for a cooling intervention (*see Figure 20*). During kennelling, ear temperatures were usually 38.8°C. Immediately after racing, when dogs had raced in thermoneutral conditions and over short sprints, ear temperatures were usually 41.0°C. However, when environmental conditions were adverse and with longer distance racing (600-700m), ear temperatures tended to increase up to 45.0°C without clinical signs of EHI. Such

dogs were prioritised immediately for an immersion tank cooling intervention and were continually monitored until their ear canal temperatures had decreased to an acceptable level. This can occur very rapidly, with ear canal temperatures decreasing to 39.0°C within 2 to 3 minutes, and the dog's panting efforts decreasing accordingly. Such dogs would need further observation, however, for changes to their condition and were walked, and immersed again if required, before being placed back in their kennels.

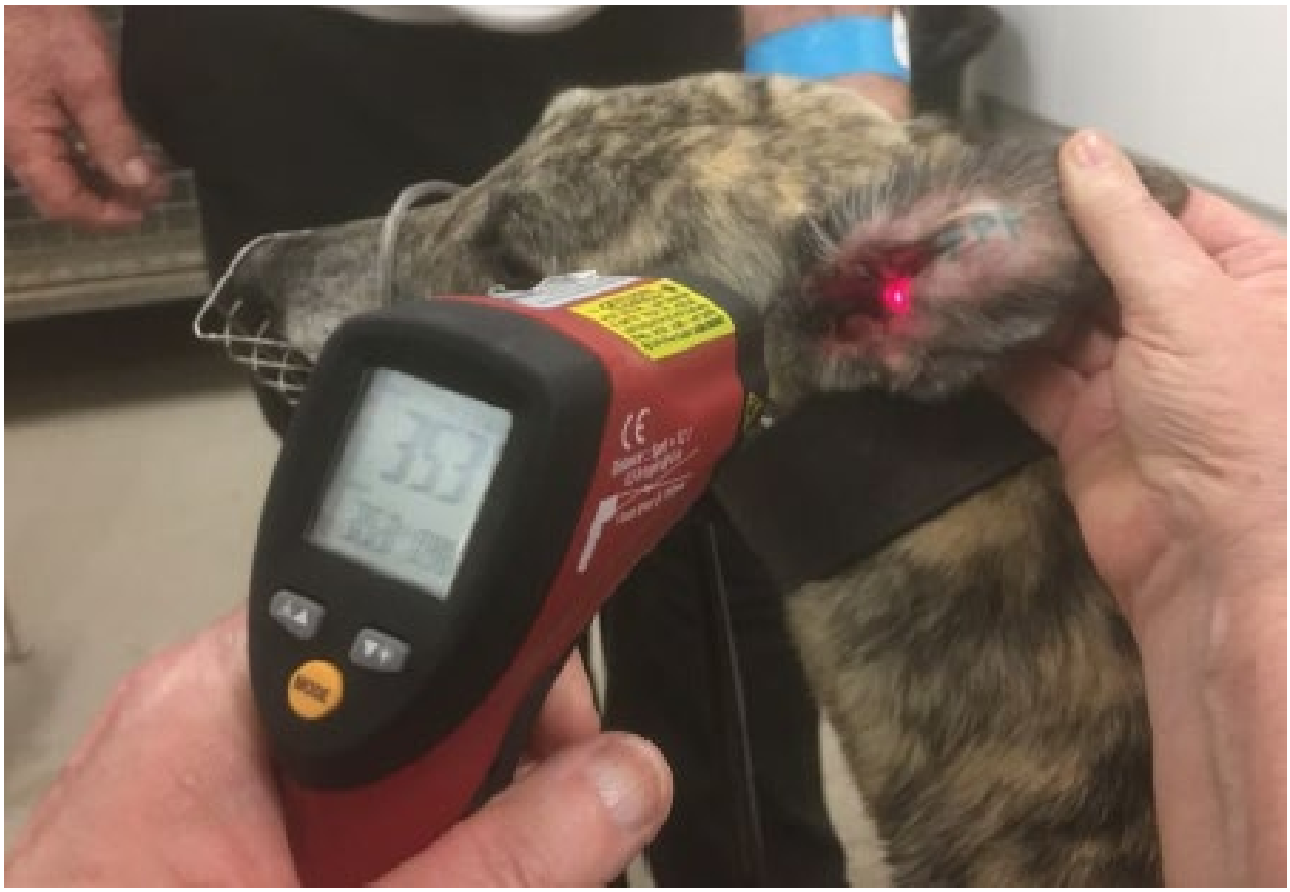


Figure 20: A hand-held infrared thermometer being used to measure temperature within the ear canal of a racing GH. In this picture the usual distance of the IRT from the ear canal has been reduced for the purpose of this illustration. Use of the IRT allows early detection and rapid assessment of an impending EHI condition and is essential to the prioritisation of those GHs requiring a cooling intervention. The IRT* - Digitech Dual IR infrared thermometer. Instrument Choice – South Australia (*Source; Meg Brownlow*).

4.3c: The technique for cooling in racing GHs

Air versus water. The thermal conductivity of water is approximately 24 times that of air, which means that heat will transfer from skin to water more readily than from skin to air.

Cooling can be divided into (1) a passive technique, where no intervention is used, and the dog must cool by panting and walking in the prevailing environmental conditions after it has raced. A wind speed above 1m/sec will aid cooling, but lack of wind and a high level of humidity will impose a greater level of thermal strain, possibly predisposing the dog to a heat illness episode. A study has shown that dogs subjected to the passive or no-active-cooling technique had slow and ineffective reductions in core body temperature, which in some cases continued to rise after racing, compared to other dogs who had a cooling intervention.[70,71]

The second cooling technique (2), which is mostly used at racetracks in NSW, is a targeted, active cooling by hosing, where

the handler is familiar with the position of key areas for heat exchange, described as 'thermal windows' (*see Section 3.1*), and the stream of water is concentrated on these areas accordingly. Thirdly, and most importantly, is (3) the cold-water immersion technique, considered to be the "gold standard" for human EHI/HS subjects[22,31-33,44] and which has been endorsed as best practice for dogs.[37,72,73,74]

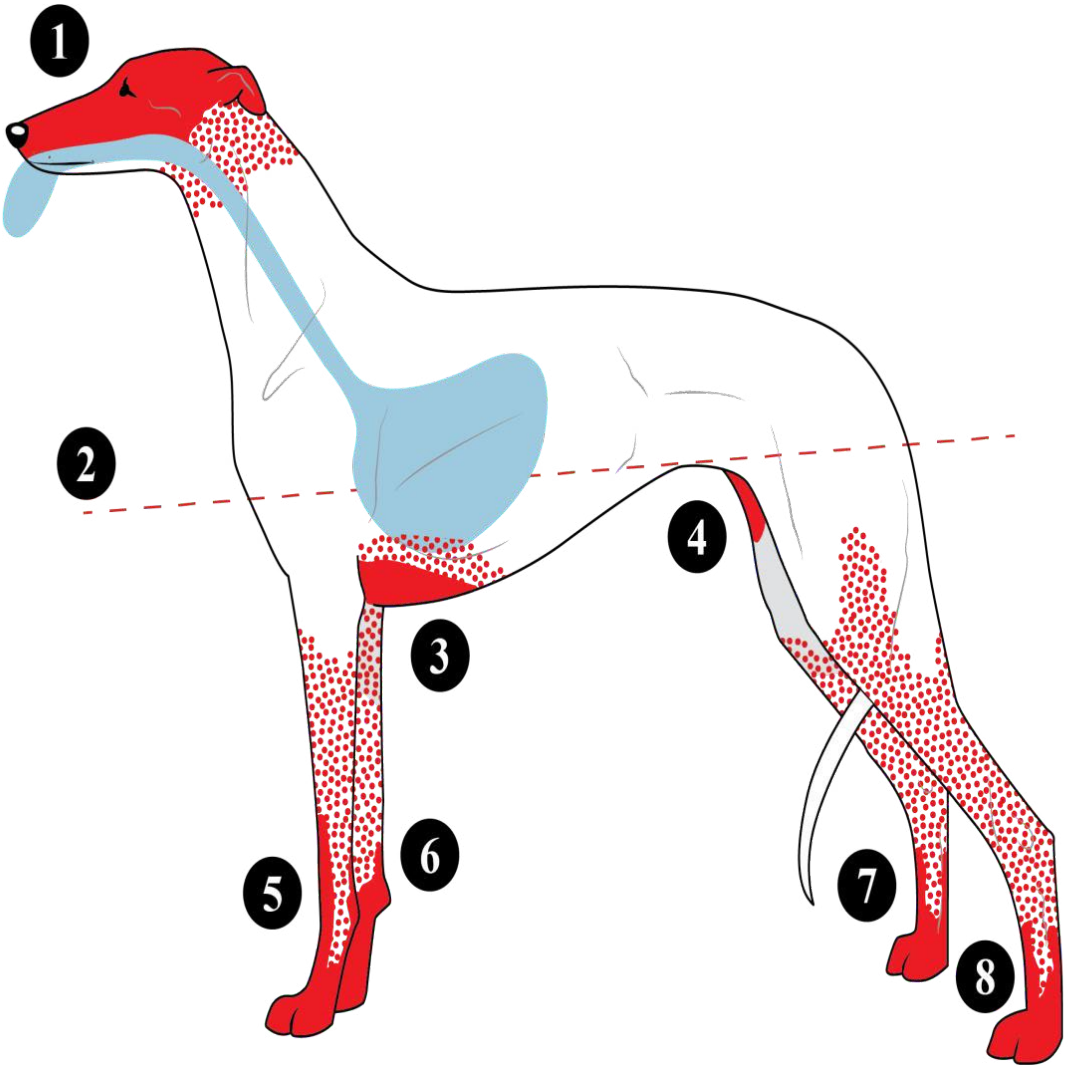


Figure 21 shows the method for targeted active cooling and the following **Figures 22** to **27** illustrate the process in photographic details.

Figure 21: A diagrammatic representation showing the anatomical location of 'thermal windows' or 'hot spots' for heat exchange. Note that the tongue and respiratory tract are shaded blue and represent the pathway for the respiratory evaporative heat loss mechanism. The line labelled **2** runs from the point of the shoulder to the hip joint. Above this line there are minimal vascularised areas for heat exchange. On the thermogram *(see Figure 11)* the lower neck, shoulder, back and croup are cool even after strenuous exercise. Below that line, however, are the most important 'hot spots', especially the chest **3** and inguinal areas **4,** and the lower extremities of the limbs **and** paw pads **5,6,7,8.** Pass the hose along this line many times on one side then turn the dog to concentrate on the same areas on the other side. The back can be left dry.

The solid red portions represent specialised, anatomically distinct, vascular heat-exchange structures, where the temperature difference between the skin surface and cooling medium dictates the efficiency of heat loss. The dotted red portions are anatomically regions which may become involved if thermoregulatory demands intensify (*Created by Meg Brownlow; Artwork S. Feeney*)

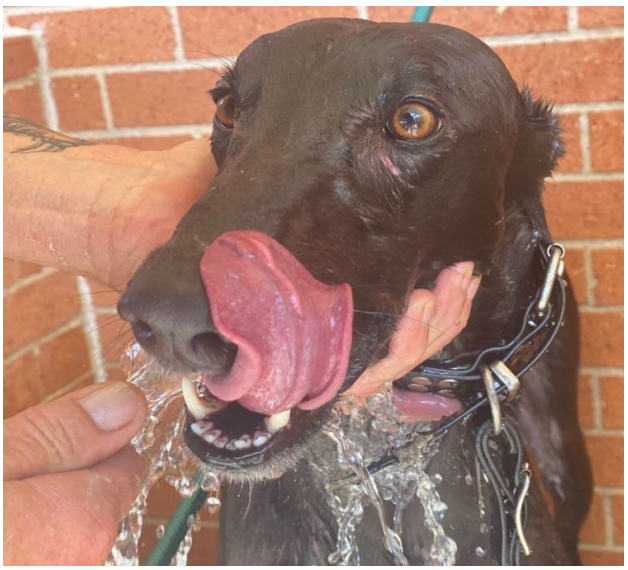


Figure 22: Strategy 1 – First, a drink. Never force a dog to drink post exercise but allow it to lap freely.

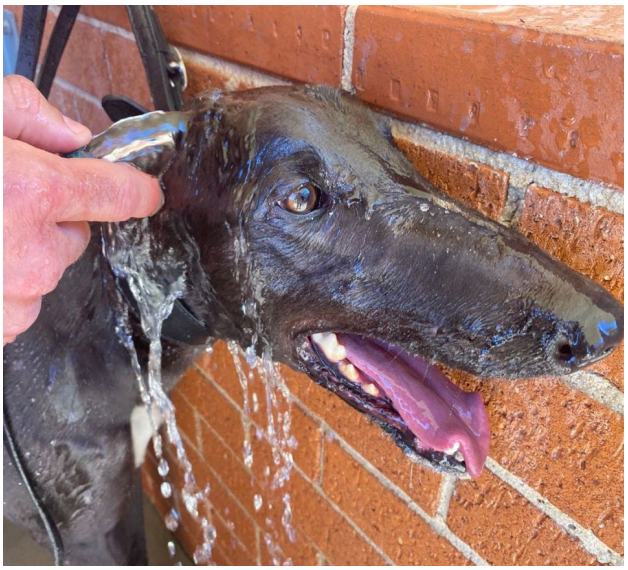
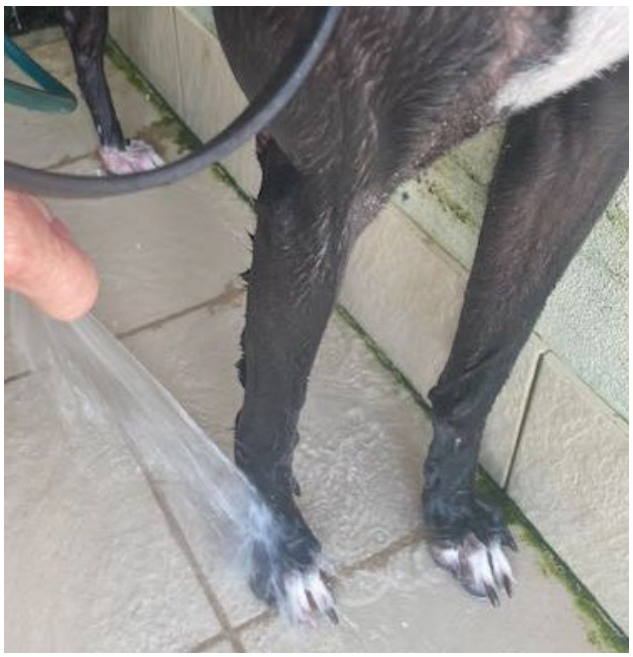


Figure 23: Strategy 2- Gently drizzle water over the area of the head and neck. Dogs appear to get great relief from thermal strain when water is applied to the back of the neck.

Figure 24: Strategy 3 - hosing along a line from shoulder to hip includes most of the thermal windows. Traverse along this line for some minutes and then pass to the other side.

Figure 25: Strategy 4 - Hosing the inguinal area to cool blood in the major femoral arteries.

Figure 26: Strategy 5 - Cooling the chest area. Some trainers maintain that this is the most important of all thermal windows and handlers should concentrate the stream of water on that area preferentially.



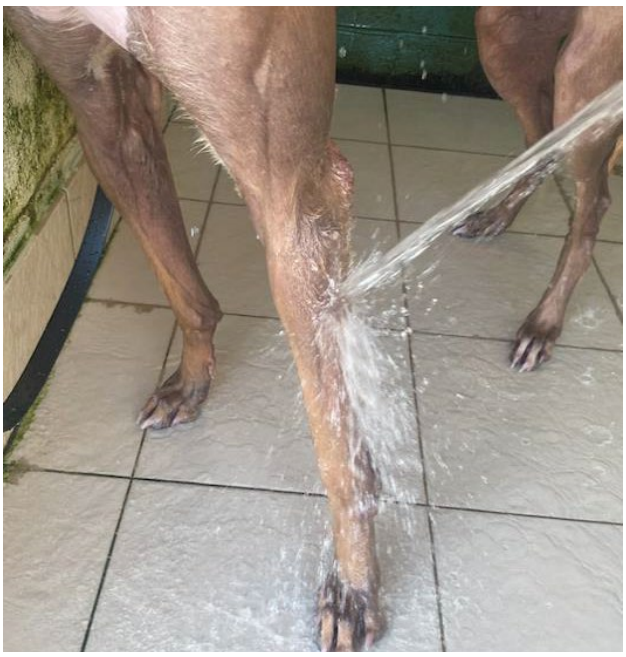


Figure 27: Strategy 6 - The distal limb extremities and footpads 5, 6, 7, and 8, are very important. They have been shown to contain specialized 'radiator-like' vascular structures for rapid heat dissipation. They do not involve any evaporative heat loss but rely on 'dry heat loss', where the temperature difference between the skin surface and the water used for cooling dictates the efficiency of heat dissipation. Use the stream of water to traverse up and down each limb, concentrating on an open-toed approach if possible. Cooling between the toes will achieve the maximum benefit.

Figure 28 – The first author's own dog, Mila the Dobermann, who had a very high prey drive and would chase kangaroos (without success) on the family farm until she became ataxic and collapsed. She always managed however, to take herself to one of the many dams on the property and would stand immersed in the water up to the line from point of shoulder to hip until her panting ceased, and then she was ready to go again.

Figure 29: A simple plastic immersion tub used for cooling GHs at a track in NSW. Moving the water around the dog whilst it is in the tub will increase conductive cooling. Most dogs seem to really enjoy the experience and stop panting very quickly. Total immersion time is usually fairly brief - only 3 to 4 minutes.

Figure 30: This is a four-dog immersion tub used at a GH race meeting in country NSW. There are usually eight dogs in races in NSW and racecourse managers ay choose one large tub rather than multiple smaller tubs where it may be difficult to maintain the supply of water and/or ice.

When dogs are heat stressed in their natural environment, they will seek a body of water and stand immersed to the demarcation line from shoulder to hip, which covers all distinct anatomical areas for heat exchange, except for the head (see Figure 28). At the racetrack, the immersion technique can be applied using plastic tubs (see Figures 29 and 30), which should ideally be located somewhere near the catching pen, in case of a heat-related incident at that location. This supports the concept given priority in human athletes, that any heat affected individual needs to be 'cooled first and transported second'.33 Time lost getting the dog to a veterinary room for assessment can mean possible tissue damage due to thermal injury. Additional placement of immersion tanks in the vicinity of the wash bays in the kennelling area allows veterinary scrutiny as dogs return from competition. Cooling can be ceased when there are no abnormal CNS signs, when panting has significantly diminished in intensity, and ear temperature using the IRT device has dropped substantially.

4.3d: The modality of the cooling technique dictates cooling rates in C per minute

Definitions: iced water 0 to 4 °C: cold water 5 to 15 °C; warm water 16 to 24 °C:

It has been established that the best outcomes for the treatment of EHI/HS in human subjects requires on-site whole-body immersion cooling and the use of ice-cold water. The latter enables the fastest cooling rates of less than 0.15 °C per minute, enabling the best results for survival without medical complications. It has also been recognised that it is not only the peak of the hyperthermic response but also its duration that determine the extent of tissue injury.[22,28,31-33] So, if the time taken to reduce body temperature is critical, then the efficiency of the cooling modality is an essential consideration.

For racing GHs, the conductive heat exchange method of whole-body immersion has been recognised as best practice but the recommended temperature for cooling requires discussion. GHs attain peak speeds of 70+ km/hr over race distances from 250 metres to 720 metres. Despite these seemingly short distances, the metabolic heat produced at that work intensity is substantial. The Greyhound, however, appears to be adapted to dissipate heat very efficiently. They have a thin body frame, an absence of body fat, and a thin, sparse hair coat. One laboratory-based study[70] showed that conscious dogs with experimentally induced heat illness cooled most rapidly in water temperatures of 10.0º to 16.0ºC, whilst comatose dogs cooled more quickly at 1.0º to 3.0º C. These dogs were not GHs. In the author's view, the immersion tub water does not need to be ice-cold, as we have recommended previously for racehorses,[74] and 12.0º to 16.0ºC would seem adequate for cooling GHs. Although some authors[72] have recommended warm water for cooling hyperthermic working dogs, it must be emphasised that this represents an inefficient cooling modality and will not achieve best patient outcomes if EHI/HS is evident.

4.3e Advance Planning

It has been shown that although EHI occurs mostly in the warmer summer months, it can occur at any time of the year, which means that veterinarians must be prepared at any time to deal with cases of EHI. It is also a commonly held view that the weather is the sole cause of the condition, through the direct effects of heat and/or humidity, but this is not the whole picture. It has been shown that intrinsic or host risk factors contribute to individual variability in the heat stress response (*see Figure 10*); nevertheless, being able to predict the effect of environmental conditions on a given race day is a vital step in risk analysis and will form the basis for planning risk mitigation strategies.

To evaluate environmental conditions in advance, a predictive weather site needs to be consulted so that an estimation of ambient temperature, vapour pressure (absolute

humidity), radiant heat, and wind speed can be made. Vapor pressure levels can be calculated using the Vaisala Humidity app. https://humiditycalculator/com/.[52]

A risk analysis will determine which heat mitigation strategies may need to be instigated. With this information, the OTV can assemble the infrastructure for cooling numbers of dogs if necessary. On race day, most greyhound racetracks in NSW have weather monitoring devices and these can be utilized in conjunction with the scrutiny of dogs as they complete their races. Evidence of an unusual level of distress or CNS dysfunction should signal the potential for heat stress and initiate a supervised cooling intervention. Infrastructure includes multiple wash bays for 8 dogs, which are shaded, have multiple hoses with access to cool water, immersion tubs, and access to ice as required. Because the early signs of EHI are easily missed, extra personnel experienced in the condition may enable rapid assessment of dogs and provide cooling assistance should it be required.

CONCLUSION

The racing GH is the product of selective breeding over millennia for absolute speed. It is anatomically and physiologically adapted for running, capable of explosive acceleration and racing speeds of up to 70 kilometres per hour. To achieve this, the GH's metabolic rate undergoes an estimated 35-fold increase, producing a large quantity of excess heat that elevates core body temperature, occasionally reaching or exceeding 43.0 °C. Although most dogs show no associated adverse effects, under certain circumstances the direct effects of heat can cause a condition referred to as exertional heat illness. If unchecked, this can result in heat stroke characterized by severe central nervous system manifestations, such as seizures, coma, and possibly even death.

Thermoregulation is a complex physiological process requiring inputs from multiple body systems, and exercise in the heat is its ultimate challenge. The GH, however, has a finely tuned thermoregulatory system, which together with the animal's lean body shape, absence of insulating body fat and thin hair coat enables it to deal efficiently with exercise-related heat loads. Dissipation of heat is achieved by the respiratory-evaporative heat loss mechanism, or put more simply, by panting. The upper respiratory tract has a bony turbinate system covered by a heat exchange mucosal surface that is larger in area than the animal's actual body surface and has a specialized vascularity. When there are high demands for heat loss, the tongue becomes the major heat loss effector organ. It becomes large, hangs out of the mouth, and there are variations to breathing patterns and direction of air flow that maximise the heat loss capability.

A panting animal is different. Evaporative heat loss in the dog is restricted to the respiratory system and the tongue. If that is impaired by high levels of absolute humidity and low wind speed, not uncommon in the summer months, heat transfer must be assisted by cooling interventions provided by the handler. Even more complicated is the fact that dogs have certain specific body locations where heat transfer is effected by specialised sub-cutaneous vascular structures, which are referred to as 'thermal windows'. Active targeted cooling by hosing of these areas or body immersion in cool water is recommended. Education for handlers and trainers concerning efficient cooling methods is an important welfare initiative.

Exertional heat illness in the GH has been categorised as minor, mild, moderate, and severe, and a grading tool has been provided by the authors to enable case definition. Cases at the minor-to-mild end of the spectrum, although infrequent, can occur at any time of the year, the dog presenting with a brief onset of ataxia characterised by a high-stepping gait. The moderate-to-severe cases, however, have a less distinct categorisation, and a diverse presentation in terms of CNS

dysfunction. It is possible that such affected animals may have biomarkers of tissue injury, and more research is needed to gain insight into levels of incidence and to screen dogs for indicators of organ dysfunction due to thermal injury.

Scrutiny of the epidemiological triad for EHI, extrapolating from studies in horses and human athletes, suggests that when dealing with cases at the high end of the severity spectrum, OTVs need to consider the impact of intrinsic host factors. Cases of EHI/HS often have a multi-factorial basis, referred to as the 'triple-hit' hypothesis, where extrinsic and intrinsic factors (including any genetic predisposition) may combine to create an EHI event.

Global warming is likely to result in weather conditions with higher-than-average levels of heat and humidity, and heat illness events may become more common amongst racing GHs, as they have in racehorses and human athletes. Preventative strategies have been described and are the best approach to limiting its impact. They should include educational initiatives that focus on early detection of the condition and best practice cooling strategies as described.

Author Contributions: M.B. wrote the article, E.S. collaborated in discussion, debate, and experience. Both authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: The study was conducted in accordance with the declaration of Helsinki and approved by the Head Veterinarian of The Greyhound Welfare and Integrity Commission of NSW. This was an observational study only and did not involve experimentation on animals.

Informed Consent Statement: Consent was obtained from owners and trainers for photographs taken during race day procedures.

Data Availability Statement: See; Brownlow, M.A.; Brotherhood, J.R. An investigation into environmental variables influencing post-race exertional heat illness in Thoroughbred racehorses in temperate eastern Australia. *Aust. Vet. J.* 2021, 99, 433-481.

Acknowledgments: We would like to acknowledge the guidance of many of the GH trainers in sharing their long experience with all things pertaining to the greyhound.

Conflicts of Interest: The authors declare no conflict of interest, but both are employed by the Greyhound Welfare and Integrity Commission of NSW.

REFERENCES

1. Pool, D.H.; Erickson, H.H. Highly athletic terrestrial mammals: Horses and dogs. *Comp Physiol.* 2011;1:1-37.
2. Staaden, R. The exercise physiology of the racing greyhound. PhD thesis. School of Veterinary Studies. Murdock University 1984.
3. Li, WL.; Liu, YH.; Li, JX. et al. Multiple origins and genomic basis of complex traits in sighthounds. *Molec. Biology Evolut.* 2023; 40; 8,158-161.
4. Parker, HG.; Dreger, DL.; Rimbault, M.; Davis, BW.; Mullen, AB.; Ramirez, C.; Ostrander, EA. Genomic analyses reveal the influence of geographic origin, migration and hybridization on modern dogs' breed development. *Cell Reports*, 2017;19;697-208.
5. McNicholl, J.; Howarth. GS.; Hazel, SJ. Influence of the environment on the body temperature of racing Greyhounds. Front.Vet. Sci. 2016; 3:54- 59.
6. Nold, JL; Petersn, LJ.; Fedde, MR.; Physiological changes in the running greyhound (Canis domesticus): influence of race length. *Comp Biochem Physiol* (A).1991;100; 623-627.
7. Vainionpaa, M.; Tienhaara, EP.; Raekallio, M.; Junnila, J.; Snellam, M.; Vainio, O. Thermographic imaging of the superficial temperature in racing Greyhounds before and after the race. World Journal, 2012; Article ID 182749.
8. Jessen, C. Temperature regulation in humans and other mammals. 2000' Springer-Verlag Berlin Heidelberg New York
9. Pieschl, RL.; Toll, PW.; Leith, DE.; Peterson, LJ.; Fedde, MR. Acid-base changes in the running Greyhound: contributing variables. *J. Appl.Physiol.*1992;73;2297-2304.
10. Sawka, MN.; Young, AJ. Physiological systems and their responses to conditions of heat and cold. 2006; In: Tipton, CM. (Editor) *ACSM's Exercise Physiology.* Baltimore: Lippincott, Williams and Wilkins, 26; 535-563.
11. Iwabuchi, T.; General sweating on the hairy skin of the dog and its mechanisms. J Investig. Physiol. 1967;49:1,61-70.
12. 12. Taylor, CR.; Schmidt-Nielsen, K.; Dmi'el R.; Fedak, M. Effect of hyperthermia on heat balance during running in the African hunting dog. *Am. J. Physiol* 1971; 220; 823-827.
13. Robertshaw, D. Mechanisms for the control of respiratory evaporative heat loss in panting animals. *J.Appl.Physiol,* 2006;101: 664-688.
14. Blatt, CM.; Taylor, CR; Habal, MB. The lateral nasal gland: a source of water for evaporative cooling. *Science,*1972;17,4051, 804-805.

15. Adams, DR.; DeYoung, DW.; Griffith R. The lateral nasal gland of the dog: its structure and secretory content. *J. Anat.*1981;132,1,29-37.
16. Baker, MA. A Brain-cooling system in mammals. *Scientific American,* 1979; 240; 5: 130-139.
17. Dawes, JDK; Prichard, MML. Studies of the vascular arrangement of the nose. *J. Anat*. 1953; 87, 311-322.
18. Lung, MA.; Wang, JCC. An anatomical investigation of the nasal venous vascular bed in the dog. *J.Anat.*1989;166, 113-119.
19. Pleschka, K. Control of tongue blood flow in regulation of heat loss in mammals. *Rev. Physiol. Biochem. Pharmacol*. 1984;100,76-120.
20. Goldberg, MB.; Langman, VA.; Taylor ,CR. Panting in dogs: paths of air flow in response to heat and exercise. *Resp. Physiol.*1981; 43, 327-338.
21. Hales, JRS.; Dampney, RAL. The redistribution of cardiac output in the dog during heat stress. *J.Thermal. Biol.* 1975; 1, 29-34.
22. Laitano, O.; Leon, LR.; Roberts, WO.; Sawka, MN. Controversies in exertional heat stroke, diagnosis, prevention and treatment. *J. Appl. Physiol*. 2019;127, 1338-1348.
23. Karamatic, SL.; Anderson, GA.; Parry, BW; Slocombe, RF.; Mansfield, CS. Prevalence and risk factors for medical events following exercise at Australian Greyhound race meetings. *Aust. Vet. J.* 2018; 96, 4,120-126.
24. Hall, EJ.; Carter, AJ.; O'Neill, DG. Dogs don't die just in hot cars – exertional heat-related illness (heatstroke) is a greater threat to UK dogs. *Animals,* 2020;10,1324-1355.
25. Carter, AJ.; Hall, EJ. Investigating factors affecting body temperature of dogs competing in cross country (canicross) races in the UK. *J.Thermal Biol.* 2018;72,33-38.
26. Andress, M.; Goodnight, M.; Heatstroke in a military working dog. *The US Army Med. Dept. J.* 2013; January-March 34-38.
27. O'Brien, C.; Berglund, LG. Predicting recovery from exertional heat strain in military working dogs. *J. Thermal Biol.* 2018;76,45-51.
28. Garcia, CK.; Renteria, LL.; Leite-Santos, G.; Leon, LR.; Laitano, O. Exertional heat stroke: pathophysiology and risk factors. *BMJ Med,* 2022;1,1,e000239.
29. Brownlow MA;, Mizzi, JX. Pathophysiology of exertional heat illness in the Thoroughbred racehorse: Broadening perspective to include an exercise-induced gastrointestinal syndrome in which endotoxaemia and systemic inflammation may contribute to the condition. *Equine Vet. Ed.* 2022,00,1-10
30. Brownlow, MA.; Mizzi, JX. Exertional heat illness in Thoroughbred racehorses – pathophysiology, case definition and treatment rationale. *Equine Vet. Ed.* 2021; 34, 5,259-271.
31. Roberts, WO., Armstrong, LE.; Sawka, MN.; Yeargin, SW.; Heled, Y.; O'Connor, FG.. ACSM expert consensus statement on exertional heat illness: recognition, management and return to activity. *Current Sports Med. Rep.* 2021; 20, 9, 470-484.
32. Pryor, RR.; Pryor, JL.; McDermott, BP. Persistent knowledge gaps regarding exertional heat stroke treatment. *J. Athletic Train.* 2022; 57, 8, 756-759.
33. Racinais,S.; Hosokawa,Y.; Akama,T., Bermon,S.; Bigard, X., Casa, DJ.; Grundstein, A. et al. IOC consensus statement on recommendations and regulations for sports events in the heat. *Br. J. Sports Med.* 2022; 0, 1-18.
34. Shapiro, Y.; Rosenthal, T.; Sohar, E. Experimental Heatstroke: A model in dogs. *Arch. Intern. Med.* 1973;131, 688-692.
35. Kim, KK.; Yoo, TH.; Lee. SH. Neurological manifestations and image findings in patients with exercise induced heat stroke. *Korean J. Med.* 2004; 2, 115-121.
36. Hall EJ., Carter, AJ., Chico, G;, Bradbury, J.; Gentle, LK.; Barfield, D,; O'Neill, DG. Risk factors for severe and fatal heat related illness in UK Dogs - A VetCompass Study, *Vet.Sci.* 2022; 90, 231.
37. Hall, EJ.; Carter, AJ; Bradbury, J.; Beard, S.; Gilbert, S.; Barfield, D.; O'Neill, DG. Cooling methods used to manage heat related illness in dogs presented to a primary care veterinary practice during 2016-2018 in the UK. *Vet. Sci.* 2023, 10, 465-478.
38. Bruchim, Y.; Klemen, E.; Saragusty,J.; Fingeilstein, E.; Kass, P.; Aroach, I. Heat stroke in dogs: a retrospective study of 54 cases (1999-2004) and analysis of risk factors for death. *J. Vet. Intern. Med.* 2006, 20, 38-46.
39. Brownlow MA,; MIzzi, JX. EHI in TB racehorses: Pathophysiology, case definition and treatment rationale. *Equine Vet. Educ*. 2021:,34, 5, 259-271.
40. Kim, KK.; Yoo, TH.; Lee, SH. Neurological manifestations and image findings in patients with exercise induced heat stroke. *Korean J. Med.* 2004; 22, 115-121.
41. Sharma, HS.; Hoopes, PJ. Hyperthermia induced pathophysiology of the central nervous system. *Int. J. Hyperth.* 2003;19 ,3, 325-354.
42. Hall, EJ.; Carter, AJ.; Bradbury, J.; Barfield, D.; O'Neill, DG. Proposing the VetCompass clinical grading tool for heat-related illness in dogs. *Nature Scientific Reports,* 2022; 11, 6828.
43. Arida, R.M. Physical exercise and seizure activity. BBA – *Molecular Basis Disease* 1867, 2021; 165979.
44. O'Connor FG.; Deuster, P.; Casa, DJ.; Bergeron, MF.; Carter, R.; Leon.L American College of Sports Medicine Roundtable on Exertional Heat Sroke – return to duty/return to play: Conference Proceedings. *Curr Sports Med Rep.* 2010; 9, 5, 314-321.
45. Ward, MD.; King, MA.; Gabrial. C.; Kenefick, RW., Leon, LR. Biochemical recovery from exertional heat stroke follows a 16-day time course. PLoS ONE, 2020; 15, 3, e0229616.
46. Bennet, ED.; Parkin, TDH. Fifteen risk factors associated with sudden death in Thoroughbred racehorses in North America (2009-2021). *J.A.V.M.A.* 2022, 260, 15-21.
47. Lyle, CH.; Blissitt, RN,; Kennedy, BC.; Gorum, BC.; Newton, JR.; Parkin, TDH.; Stirk, A. Risk factors for race-associated sudden death in Thoroughbred racehorses in the UK.(2000-2007). *Equine Vet. J*. 2012, 44, 4, 459-465.
48. Sharma, HS. Hyperthermia influences excitatory and inhibitory amino acid neurotransmitters in the central nervous system. *J. Neural Trans.* 2006, 113,4, 497-519.
49. Sharma, HS. Heat-related deaths are largely due to brain damage. *Indian. J. Med. Res.* 2005; 21, 5, 621-623.
50. Westwood, CS.; Fallowfield, JL.; Delves, SK. Individual risk factors associated with exertional heat illness: a systemic review. *Exp. Physiol.* 2021,106,1,191-199.
51. Otani, H.; Goto, T.; Goto, H; Shirato, M. Time-of-day effects of exposure to solar radiation on thermoregulation during outdoor exercise in the heat. *Chronobiol. Int.* 2017; 34,9,1224-1238.
52. Brownlow, MA.; Brotherhood, JR. An investigation into environmental variables influencing post-race exertional heat illness in thoroughbred racehorses in temperate eastern Australia. *Aust.Vet.J.* 2021; 99, 433-481.
53. Budd, GM.; Brotherhood, JR.; Hendrie, AL.' Project Aquarius 9. Relative influence of job demands and personal factors on the energy expenditure, strain and productivity of men supressing wildland fires. *Int.J.Wildland Fire* 1997; 7, 159-166.
54. Weissenbock, NM.; Weiss,CC.; Schwammer, HM.; Krarochi, H. Thermal windows on the body surface of African elephants. Studies by infrared thermography. *J. Thermal Biol.* 2010; 35,182-188.

55. Gahn, DA.; Dillon, JL.; Heller, HC. Heat loss through the glabrous skin surfaces of heavily insulated heat stressed individuals. *J. Biomech. Eng*. 2009;131, 071005-1.
56. Hill, RW.; Veghte, JH. Jackrabbit ears: surface temperature and vascular responses. *Science,* 1976; 194, 4263, 436-438.
57. Romanovsky, AA.; Ivanov, AI.; Shimansky, YP. Ambient temperature for experiments in rats: a new method for determining the zone of thermal neutrality. *J. Appl. Physiol*. 2002; 92, 6, 2667-2679.
58. Phillips, PK.; Heath, JE.; Heat exchange by the pinna of the African Elephant (Loxodonta Africana). *Comp. Biochem. Physiol. Comp. Physiol.* 1992,101, 4, 693-699.
59. Tattersall, GJ; Andrade, DV.; Abe, AS. Heat exchange from the Toucan bill reveals a controllable vascular thermal radiator. *Science,* 2009, 325, 468-470.
60. Baker, M.A. Brain cooling in endotherms in heat and exercise. *Ann. Rev. Physiol.* 1982; 44, 85-96.
61. Mota-Rojas, D.; Pereira, AMF.; Wang, D.; Martinez-Burnes, J..; Ghezzi, M. Clinical applications and factors involved in validating thermal windows used in infrared thermography in cattle and river buffalo to assess health and productivity. *Animals*, 2021;11, 2247-2270.
62. Soroko, M.; Gorniak, W.; Howell, K.; Zielinska, P.; Dudek, K. Changes in body surface temperature associated with high-speed treadmill exercise in beagle dogs measured by infrared thermography. *Animals,* 2021;11, 2982.
63. Ninomiya, H.; Yamazaki ,K.; Inomata, T. Comparative anatomy of the vasculature of the dog (Canis familiaris) and domestic cat (Felis catus) paw pad. *Open J. Vet. Med*. 2013; 3,11-14.
64. Baker, JL.; Hollier, PJ.; Miller, L.; Lacy, WA. Rethinking heat injury in the SOF multipurpose canine: A critical review. *J. Special Op. Med*. 2012; Summer Edition.1-15.
65. Gordon, L.E. Hyperthermia and heatstroke in the working canine. Massachusetts Task Force 1, Urban Search and Rescue, *USAR Veterinary Group*, 2017; 1-15.
66. Phillips, CJ.; Coppinger, RP; Schimel, DS. Hyperthermia in running sled dogs. *J. Appl. Physio*l.1981; 51,135-142.
67. Ivayla, IG.; Cuzzo, B.; Fazili. T.; Javaid, W. Normal body temperature: A Systemic Review. *Open Forum Infectious Diseases*, 2019; 6, 4, 2023-2025.
68. Casas-Alvarado. A.; Martinez-Burnes, J.; Mora-Medina, P.; Hernandez-Axalos, I. Thermal and circulatory changes in diverse body regions in dogs and cats by infrared thermography. *Animals*, 2022; 12, 789.
69. Brownlow MA.; Smith T. The use of the hand-held infrared thermometer as an early detection tool for exertional heat illness in Thoroughbred racehorses: a study at racetracks in eastern Australia*. Equine Vet. Ed*. 2020; 33, 296-305.
70. Magazanik, A.; Epstein, Y.; Udassin, R.; Shapiro, Y., Sohar, E. Tap water, an efficient method for cooling heatstroke victims – a model in dogs. *Aviat, Space. Environ. Med.* 1980; 51, 865-866
71. Bradbury, J.; Hall, E; Carter, A.; O'Neill, DG. Canine Heat-Related Illnesses – a new perspective from recent research. *Comp Animals,* 2023, 28, 2-5.
72. Parnes, S.C., Mallikarjun, A.; Ramos, MT,; Stone, TA.; Otto, CM. A randomized cross-over study comparing cooling methods for exercise-induced hyperthermia in working dogs. *Animals,* 2023,13,3673.
73. Davis, MS; Marcellin-Little, DJ; O'Connor E. Comparison of post exercise cooling methods in working dogs*. J. Spec. Oper. Crit. Care* 2019; 19, 56-60.
74. Brownlow, MA.; Mizzi, JX. An overview of exertional heat illness in Thoroughbred racehorses. Pathophysiology, diagnosis and treatment rationale. *Animals,* **2023,** 13, 610-645.