Ci-joint une étude sur les effets physiologiques de la décompression datée de 1978 (obtenue des USA) :
Effect of Rapid Decompression and Associated Hypoxic Phenomena in Euthanasia of animals : A Review
Nicholas H. Booth.

- document en PDF en anglais
- fichier en html pour ceux qui ne lisent pas l'anglais - (traduction automatique par logiciel)
unité US : un foot ft (pied) = 30,48 cm

Dont la conclusion est que l'induction de la perte de connaissance et la mort par décompression hypoxique n'est pas douloureuse à condition que le vide soit fait lentement : 4000 pieds (d'altitude par rapport au niveau de la mer) par minute pendant 10 minutes (soit 1220 mètres par minute). Donc une euthanasie par décompression devrait durer au moins 10 minutes

Mais les caissons à vide, en France, font le vide instantanément (- 5 secondes) et mettent pour tuer entre 30 secondes et une minute. La mort étant provoquée non par l'asphyxie mais par les effets physiologiques provoqués par la décompression explosive.

Ce procédé est interdit dans la majorité des états aux U.S.A.
voir ici pour le Missouri et le New Jersey

Selon PETA USA :
les gaz emprisonnés dans les sinus, les oreilles moyennes, et les intestins des animaux se dilatent rapidement. Ce qui provoque un grand malaise avec une grande souffrance. Quelques animaux arrivent à survivre au premier passage dans la chambre de décompression et sont de nouveau décompressés à cause d'un dysfonctionnement de l'appareil, d'une erreur de l'opérateur ou parce que les animaux arrivent à survivre dans des poches d'air et ils sont repassés dans le dispositif douloureux une seconde fois.

the gases in animals' sinuses, middle ears, and intestines expand quickly, causing considerable discomfort to severe pain. Some animals survive the first go-round in decompression chambers and are recompressed because of malfunctioning equipment or the operator's mistake or because animals get trapped in air pockets. They are then put through the painful procedure all over again.

Le rapport 2000 de l'American Veterinary Medical Association indique, à la fin, dans sa liste des agents et méthodes inacceptables pour euthanasier que :

La décompression est inacceptable pour l'euthanasie en raison de nombreux inconvénients.
(1) Beaucoup de chambres sont conçues pour produire un vide à une vitesse 15 à 60 fois plus rapide que ce qui est recommandé comme optimum pour les animaux, avec pour résultat la douleur et une détresse attribuable aux gaz qui se dilatent et qui sont emprisonnés dans les cavités du corps.
(2) les animaux immatures résistent à l'hypoxie, et de plus longues périodes de vide sont exigées avant que la respiration cesse.
(3) la récompression accidentelle, avec le rétablissement des animaux blessés peut se produire.
(4) des boursouflages, des saignements, des vomissements, des convulsions, de l'urination, et de la défécation, qui sont esthétiquement désagréables, peuvent se développer chez les animaux sans connaissance.

Decompression is unacceptable for euthanasia because of numerous disadvantages.

(1) Many chambers are designed to produce decompression at a rate 15 to 60 times faster than that recommended as optimum for animals, resulting in pain and distress attributable to expanding gases trapped in body cavities.
(2) Immature animals are tolerant of hypoxia, and longer periods of decompression are required before respiration ceases.
(3) Accidental recompression, with recovery of injured animals, can occur.
(4) Bleeding, vomiting, convulsions, urination, and defecation, which are aesthetically unpleasant, may develop in unconscious animals.

Dans les GUIDELINES FOR HUMANE EUTHANASIA OF ANIMALS de l'American Veterinary Medical Association on retrouve, repris du rapport 2000, ce même procédé considéré comme inacceptable (donc cruel).

L'arrêté du 12 décembre 1997 relatif aux procédés d'immobilisation, d'étourdissement et de mise à mort des animaux et aux conditions de protection animale dans les abattoirs indique dans son annexe IV "mise à mort des animaux - "3. Caisson à vide" que :
Les animaux doivent être mis en caisson étanche où le vide est rapidement réalisé par une pompe électrique puissante.
Ici on demande que le vide soit rapidement réalisé, or la grande vitesse de décompression est justement dénoncée comme facteur de souffrance par le rapport 2000 de l'AVMA.

L'Organisation des Nations Unies pour l'alimentation et l'agriculture (généralement appelée FAO : Food and Agriculture Organization, une organisation spécialisée de l'ONU) a publié en 2001 une brochure intitulée : "MANUAL ON PROCEDURES FOR DISEASE ERADICATION BY STAMPING OUT"

Dans le chapitre 3 méthodes d'abattage, aux autres moyens physiques, la décompression est mentionnée.
On y lit que la décompression est maintenant considérée comme inacceptable.

OTHER PHYSICAL METHODS - Decompression - This method is now regarded as unacceptable.

D'autre part l'office international des épizooties (OIE) publie des lignes directrices pour l'abattage d'animaux à des fins de consommation humaine où il n'est pas fait mention de ce procédé comme méthode de mise à mort acceptable.

Effet de la décompression rapide et des phénomènes hypoxiques associés dans l'euthanasie des animaux : Une revue

Cabine de Nicholas H. DVM. PhD

SOMMAIRE

La documentation dans la littérature indique que la mort est en tant qu'indolore suivant l'induction de l'hypoxie de la décompression rapide comme d'autres méthodes qui mènent à l'hypoxie, telle que l'exposition à l'altitude élevée, à l'oxyde de carbone, et aux gaz inertes (azote, xénon, et krypton). Plusieurs des signes et des symptômes de l'hypoxie sont identiques que ceux pour l'intoxication alcoolique et la narcose de gaz inerte. D'ailleurs, il y a de bonne évidence que les rapports ou les mécanismes analogues peuvent exister pour l'hypoxie, la narcose de gaz inerte, et l'anesthésie.

EN 1972 et 1978, les rapports du panneau d'AVMA sur l'euthanasie [1,2] ont inclus l'utilisation des procédures hypoxiques dans l'euthanasie des animaux. Les rapports ont couvert les effets de l'oxyde de carbone, du gaz d'azote, de la décompression rapide, et des concentrations paralysantes respiratoires des anesthésiques, qui resuit dans la mort en induisant une hypoxie aiguë ou une insuffisance aiguë de l'oxygène.

La polémique a surgi concernant le humaneness d'employer des méthodes hypoxiques d'induire l'euthanasie chez les animaux, particulièrement ceux comportant l'utilisation du gaz rapide de décompression ou d'azote. En conséquence, quelques villes et états ont passé la législation interdisant l'utilisation de la décompression ou du gaz d'azote. En raison de l'intérêt croissant des individus désirant l'information documentée sur si la décompression et d'autres méthodes hypoxiques soyez des procédures humanitaires de tuer des animaux, la littérature appropriée a été assemblée et est passée en revue ici.

Effets comparatifs de la décompression, de l'intoxication alcoolique, et de la narcose de gaz inerte

AJOURNEZ 1—Altitude et rapports de pression barométrique au-dessus de niveau de la mer

Altitude (pi au-dessus de niveau de la mer)	Pression barométrique (millimètre d'hectogramme)
0	760
2000	707
6000	609
10000	522
14000	446
18000	380 *
22000	321
26000	270
30000	226
34000	187
38000	155
42000	128
46000	106
50000	87
54000	72
58000	60
63000	47 **

* Équivalent à un demi- de la pression au niveau de la mer. Altitude de ** que l'ebullition se produit, ou équivalent à la pression de vapeur d'eau dans des poumons.

La décompression produit des effets hypoxiques semblables à ceux observés pendant la montée en escaladant de hautes montagnes ou en volant aux altitudes élevées dans aircraft.3 non-pressurisé plus est haute l'altitude plus la pression ambiante sont inférieure et plus l'hypoxie est plus grave. La composition de pourcentage des divers gaz de l'atmosphère, cependant, demeure la même qu'au niveau de la mer .4 par exemple, le pourcentage de l'O2 au niveau de la mer et à n'importe quelle altitude indiquée au-dessus de niveau de la mer est 20.96.4 au niveau de la mer, l'ambiant ou la pression barométrique est de 760 millimètres d'hectogramme, tandis qu'à 55 000 pi au-dessus de niveau de la mer, la pression est de 68.8 millimètres d'hectogramme. Ainsi, la pression partielle de 02 au niveau de la mer est de 760 x 0.2096 ou 159 millimètres d'hectogramme. À 55.000 pi, la pression partielle de l'O2 est de 68.8 x 0.2096 ou seulement 14 millimètres d'hectogramme (tableau 1). Le sang artériel moyen du chien ou de l'homme a normalement une tension O2 (P02,) d'environ 95 millimètres de Hg.5 à 55.000 pi, le P02 (14 millimètres d'hectogramme) est considérablement au-dessous du niveau de physiologie nécessaire de l'oxygénation appropriée des tissus. Ce bas ou déficient P02 a comme conséquence l'hypoxie grave, l'inconscience, et la mort rapide.

'

AJOURNEZ les pouvoirs 2—Comparative des gaz inertes et des anesthésiques de gaz qui produisent les niveaux équivalents de l'anesthésie ou de la dépression de Neurologie chez les êtres humains et les animaux

Gaz	Pression anesthésique (ATA) *
Hélium	>261
Néon	88
Azote	29
L'argon	20
Kryton	2.9
Protoxyde d'azote	0.9
Xénon	0.85
Éther diéthylique	0.02
Chloroforme	0.015
Halothane	0.008

Données de Millers et autres .59 et de Saidman et autres .60

ATA = les atmosphères absolues.

La montée à l'altitude élevée et à l'hypoxie résultante peut induire de divers effets tels que l'excitation, joie de vivre, et euphorisme suivie de mal de tête, de lassitude, de matité sensorielle, d'affaiblissement visuel, de faiblesse neuromusculaire, de dyspnée, et de perte de consciousness.4 il est bien connu que les pilotes d'avion volant à l'altitude élevée et exposés à un environnement du bas 02 développeront ces symptômes hypoxiques. L'hypoxie peut être si aiguë que la perte de conscience se produise rapidement sans warning.6 antérieur [7]

Toutes les manifestations observées dans l'intoxication alcoolique telle que le mal de tête, la somnolence, la dépression respiratoire grave et l'insuffisance O2 associée, la vision altérée, l'incoordination neuromusculaire, et l'échec dans les essais mentaux également ont été observées dans les êtres humains soumis à hypoxia4 aigu ou l'exposition à decompression.8 dans tous les exemples, ces eîects sont induites par un PO2 insuffisant, au cerveau. L'hypoxie ou un PO2 déficient ne devrait pas être confondue avec l'étouffement, l'étranglement, ou l'asphyxie dans laquelle une insuffisance en O2 est combinée avec une tension C02 accrue (hypercapnie) en tant que cela vue après l'action du succinylcholine ou du [d-tubocurarine1] dans la paralysie de la musculature respiratoire (les muscles et le diaphragme de membrure intermédiaire). L'hypercapnie ou l'étouffement n'est pas un facteur dans la montée à l'altitude élevée ou pendant la décompression.

Intéressant, plusieurs des signes et les symptômes de l'hypoxie décrits ici sont identiques que ceux pour la compression en air et pour la narcose du gaz inerte narcosis.9 induite dans les êtres humains par leur compression en air ont été rapportés dès le du siècle dernier. Des symptômes ressemblant à l'intoxication alcoolique ont été observés en 1835 par Junod.9 cet effet nuisible sur le perceptivity mental et sur la capacité de l'être humain d'exécuter dans la gamme à air comprimé de bidon de l'euphorisme d'abord observée dans des ouvriers de caisson, à l'amnésie, au hyperconfidence dangereux, à la difficulté dans la prise de décision, et aux fautes dans la conscience dans divers.9 en 1935, on l'a appris que cette intoxication à air comprimé était due à la teneur en azote de l'effet narcotique d'air.10 A se produit dans les êtres humains en air aux 3 atmosphères et plus grand. L'euphorisme, le retardement des processus mentaux plus élevés, et la fonction neuromusculaire altérée sont observed.10 l'étude de Behnke et autres [10] menés à la réalisation que la narcose d'azote était juste un exemple d'une caractéristique plus générale de phénomène également de l'autre gases.11-12 inerte que la différence entre les actions narcotiques de ces gaz est principalement un pouvoir impliquant plutôt que la nature des symptômes ils elicit.9 selon les collines et le rayon, [9 le] meilleur index pour doser cette différence est probablement fournis par « la pression partielle equinarcotic » et peut être prolongés pour inclure les anesthésiques gazeux.

Les valeurs sont disponibles pour un assortiment des gaz et fournissent une base comparative pour leurs pouvoirs narcotiques relatifs (tableau 2). Le gaz inerte plus efficace exige de la plus petite pression partielle afin d'obtenir le même degré de narcosis.9 qu'une telle comparaison implique que l'anesthésie inhalant est une prolongation de narcose de gaz inerte ; en fait, il y a de bonne évidence qu'un rapport ou un mécanisme analogue existe dans les deux conditions.13

Semblable aux symptômes induits par la décompression ou l'intoxication d'alcoolique, des manifestations de narcose de gaz inerte ou narcose à air comprimé incluez l'euphorisme, loquacité, l'hallucination, perte provisoire de la mémoire, difficulté en assimilant des faits ou en prenant des décisions, la suffisance, réponse retardée aux stimulus visuels, auditifs, olfactifs, et tactiles, et à la coordination neuromusculaire altérée menant à la stupéfaction et à la perte de l'exposition consciousness.9 à à air comprimé à l'absolu des 2 atmosphères (ATA) * ou 2 x 760 millimètres de résultats d'hectogramme dans l'activité de delta de l'EEG.13 chez 7 ATA, signes et les symptômes de la « narcose d'azote » sont évidents dans un grand nombre d'individus, accompagnés d'une légère diminution de l'amplitude de l'alpha rythme. Chez 10 ATA, cette diminution est plus marquée et les signes de la narcose sont plus graves. Si la pression est augmentée plus loin, l'un-consciousness occurs.13

* ATA = unité de l'égale de la pression (760 millimètres d'hectogramme) à la pression d'air au niveau de la mer à 0 C.

Effets principaux observés après exposition à la décompression

Les effets de la décompression sur le chien sont récapitulés comme foliows [14 18] : Juste après l'exposition à une pression ambiante de 30 millimètres d'hectogramme, la respiration devient profonde et rapide. Cette hyperventilation dure une question des secondes. Le distention abdominal marqué se produit immédiatement. C'est dû à l'expansion des gaz actuels dans l'appareil gastro-intestinal. L'animal s'effondre en environ 8 secondes. Les convulsions se produisent généralement dedans de 10 à 12 secondes et durent pendant plusieurs secondes. On peut également observer la rigidité de Decerebrate. Elle se produit chez les animaux suivant la récompression ou le retour à pressure.15 atmosphérique normal suivant une saisie convulsive, l'animal est tranquille excepté les halètements respiratoires occasionnels qui sont inefficaces en aérant les poumons. Habituellement le lacrimation, la salivation, et l'urination se produisent.

Chez le singe, le contenu gastrique est soudainement et de force éjecté lorsque l'animal est décomprimé aux altitudes au-dessus de 55.000 ft.19 trente à 40 secondes après la réduction de pression, le gonflement secondaire commence. Ce gonflement se produit d'abord dans les membres arrière et l'abdomen inférieur et progresse headward. Les animaux survivront et récupéreront complètement si l'exposition à 30 millimètres d'hectogramme a lieu pendant moins de 90 secondes. Les expositions de 2 minutes ou sont plus longtemps habituellement mortelles.

Dans l'être humain, la douleur de l'expansion de gaz dans l'intestin a été rare pendant la montée dans l'altitude, bien que la plupart des sujets notent une sensation « de ébullition » dans l'abdomen.3 quelques individus pour s'être plaints de la douleur vraisemblablement par l'origine oesophagienne suivant des tentatives négligentes à l'eruct pendant la montée. En plus de la douleur abdominale avant l'inconscience, la douleur de coffre généralisée a été rapportée par les sujets humains quelques secondes avant la perte de consciousness.20

Influence de Neurologie de la décompression

Des tissus dans le corps, le tissu nerveux est le mineur capable de résister aux effets de hypoxia.4 dans l'être humain, l'hypoxie aiguë ressemble à l'intoxication alcoolique en raison de l'insuffisance O2 marquée et de la dépression respiratoire qui se développe. Les symptômes sont mal de tête, désorientation mentale, somnolence, activité respiratoire diminuée, faiblesse neuromusculaire, et incoordination.21 selon Van Liere, [21] « personne d'A exposée à une tension à faible teneur en oxygène traverse souvent une première étape d'euphorisme, accompagnée par un sentiment de fatuité et un sens de puissance. L'oxygène veulent des stimulâtes le système nerveux central de sorte que le sujet puisse devenir hilare et chanter ou crier, et d'autres perturbations émotives se manifestent souvent. »

À mesure que l'exposition aux niveaux PO2 bas est augmentée, la perte de conscience se produit. Un pilote d'avion exposé soudainement à une altitude de 45.000 pi au-dessus de niveau de la mer deviendra sans connaissance dans l'inconscience 13 à 16 seconds.22 peut seulement être évité si l'O2 de 100% est inspiré dans 5 à 7 secondes. Les pilotes soumis à 33.000 pi et à O2 de respiration de 100% et immédiatement exposés à 52.500 pi pendant moins de 6 secondes et recompressed alors à 33.000 pi ne perdent pas consciousness.23 si l'exposition est plus longue que 6 en second lieu, inconscience se produiront même tout en respirant l'O2 de 100%.

Dans l'être humain, l'arrestation provisoire de la circulation au cerveau sans affecter la région respiratoire a été accomplie au moyen de réactions caractéristiques cervicales gonflables particulièrement conçues de la pression un cuff.24 résultant de l'arrestation aiguë de la circulation au cerveau pendant 5 à 10 secondes sont fixation des globes oculaires, flou de la vision, perte de conscience, et convulsions hypoxiques. La perte de conscience précède la convulsion hypoxique. Les saisies convulsives sont d'un type tonique et clonique généralisé. Puisque la convulsion est précédée par la perte de conscience, la personne reste sans connaissance dans toute la saisie et n'a aucune mémoire de elle. Les enregistrements d'Electroencephalographic indiquent l'aspect soudain des grandes vagues lentes (vagues de delta) qui sont étroitement corrélées avec la fixation des yeux ou de la perte de conscience. En outre, EEG et d'autres enregistrements électriques ont été faits pour les sujets humains rendus hypoxiques en respirant l'azote, [23-25] concentrations du bas 02, [26] et dans ceux décomprimées aux altitudes simulées de 45.000 ft.22 chez les animaux, l'activité corticale électrique du cerveau a été enregistrée après le hypoxemia [27] et decompression.28

La circulation cérébrale a été arrêtée pour tant que 100 secondes dans humain tous les sujets beings.24 regagnent la conscience dans 30 à 40 secondes après restauration de circulation. Pendant l'arrestation, la perte de conscience, les convulsions, la cyanose marquée, l'urination et la défécation involontaire, la bradycardie, et la dilatation des pupilles sont ces signes observed.24 sont comparables à ceux ont observé chez les animaux suivant l'induction de l'hypoxie de la décompression.

Chez le chien, l'arrestation de la circulation de cerveau pendant 6 minutes ou moins récupèrent la fonction de neurologie, tandis que ceux soumis aux périodes de l'arrestation circulatoire pendant 8 minutes ou ont plus longtemps habituellement le cerveau permanent damage.29 Urination se produit fréquemment pendant la première minute de l'arrestation circulatoire. L'activité respiratoire cesse 15 à 20 secondes après arrestation de circulation de cerveau chez la plupart des animaux que ceci a comme conséquence le développement de l'hypoxie grave.

Pendant une période désignée sous le nom du coma hyperactif suivant l'arrestation circulatoire, il y a les mouvements courants rapides de tous les membres, souvent accompagnés de la salivation et du vocalization. Ceux-ci coordonnés et les mouvements rhythmiques avec le vocalization se produisent avec le mensonge de chien sans connaissance sur son side.29 tôt dans la période du coma hyperactif, rigidité d'extenseur est vus, habituellement exprimé comme des opisthotonos avec les mâchoires fermées étroitement. Pendant les intervalles entre les mouvements courants, il y a de rigidité modérée d'extenseur principalement dans les manifestations forelimbs.29 des signes observés chez les chiens pendant la période du coma hyperactif sont presque, sinon identique, à ce que l'auteur a vu chez quelques chiens soumis à la période tôt de la décompression ou de l'exposition rapide ; concentrations mortelles d'oxyde de carbone.

Selon Kabat et autres, [29] mouvements courants pendant la période du coma hyperactif sont semblables à ceux qui se produisent pendant le rétablissement de l'anesthésie de barbiturate. Des vétérinaires sont bien mis au courant de ces mouvements courants et le vocalization pendant la période de démence pendant le rétablissement du sodium anesthesia.30 de pentobarbital l'animal est comateux ou sans connaissance pendant cette période qui est caractéristique de stage-2 anesthesia.30

Influences pulmonaires et cardiovasculaires de la décompression

La réponse la plus conformée et la plus exceptionnelle observée chez la décompression suivante d'animaux (chat, chien, rat, lapin, et cobaye) est le développement du distention distention.31 abdominal abdominal est la plus grande chez le cobaye et le lapin dus aux quantités de gaz relativement grandes normalement actuelles dans les appareils gastro-intestinaux de ces animaux. À mesure que le distention augmente, le diaphragme est forcé vers le haut dans la position expiratoire, alors que le thorax est soulevé dans la position inspiratoire. Chez le lapin et le cobaye, ces effets peuvent être si en avant quant à interfèrent sérieusement, ou empêchent réellement, les mouvements respiratoires. Cette accumulation de distention et de pression interfère inévitablement le sang retournant au coeur par la veine cave caudale. Il doit être suffisante interférer une pression intra-abdominale positive de la grandeur observée à une altitude simulée de 55.000 pi le retour veineux à la réduction heart.32 marquée par A des résultats de retour veineux d'une diminution de débit cardiaque et de l'abaissement prompt de la pression artérielle. Ceci réduit la période latente de la réponse hypoxique puisque, en outre, la pression artérielle et le sang coulent dans le cerveau et le coeur également sont réduits. L'hypoxie altère le coeur comme pompe circulatoire. La dépression vasculaire cardiovasculaire est comme message de sollicitation et hypoxie en tant que décompression suivante complète à 55.000 pi comme à un plus haut altitudes.32 simulé

Chez les chiens exposés à la décompression, il y a une baisse rapide dans pressure.31 artériel systémique en outre, chez les chiens décomprimés à 30 millimètres d'hectogramme (IE, d'équivalent à une altitude de 72.000 pi), circulation est complètement arrêté en moins de 16 secondes après que decompression.16 des résultats circulatoires de cette arrestation de la vapeur ou des bulles dues à l'expansion des gaz de sang dans le coeur ou le lit vasculaire et correspond à quel ingénieur se réfère comme serrure de vapeur. La brève arrestation de l'écoulement de sang au cerveau du chien d'adulte produit le coma pendant 12 à 18 heures ; après 6 minutes, pendant 24 heures ou plus longtemps et ; après 8 minutes ou plus, coma est permanent.29

Il y a plus de 40 ans, Lennox et autres [33] ont rapporté cela dans les êtres humains que la perte de conscience se produit quand la saturation O2 du sang veineux jugulaire chute à 24 % ou ci-dessous. Les bas de saturation du pourcentage 02 déterminé chez le chien 30 secondes suivant la décompression à la diverse diminution pressures.34 barométrique de la saturation de pourcentage ne se produit pas jusqu'à ce que des pressions moins de 510 millimètres d'hectogramme soient atteintes. La saturation de l'oxygène diminue brusquement aux pressions barométriques entre 510 millimètres d'hectogramme et 50 millimètres d'hectogramme. La saturation de pourcentage est zéro à 50 millimètres de pression ambiante d'hectogramme. À une pression ambiante moins de 52 millimètres d'hectogramme intravasculaire, des bulles sont une conclusion fréquente chez le chien mais des bulles ne sont pas trouvées à un plus haut pressures.34

L'évaporation des fluides de corps peut abaisser la température orale au-dessous de zéro et peut également abaisser la température de corps interne plusieurs degrés en moins de 2 minutes chez les chiens soumis au vide proche (1 millimètre d'hectogramme) conditions.35

Les réponses cardiovasculaires des chiens à l'azote respirant au niveau du sol et à l'hypoxie à 55 millimètres d'absolu d'hectogramme sont tout à fait similar.36 les chutes de pression artériel systémiques, et augmentations pulmonic de pression artérielle dues à l'hypoxie produite par l'azote ou la décompression. Les augmentations veineuses de pression suivant la décompression [37] mais les restes dans une marge normale dans tout l'épisode hypoxique pendant l'azote breathing.36 Apnea se produit plus tôt pendant la décompression à 55 millimètres d'hectogramme dans une moyenne d'environ 60 secondes comparées à environ 80 secondes pour des chiens respirant l'azote. La bradycardie se produit suivant les épisodes hypoxiques produits par l'azote respirant et décompression à 55 millimètres d'hectogramme. Cependant, la fréquence cardiaque diminue plus tôt et tombe aux niveaux plus bas suivant la décompression comparée aux animaux respirant l'azote.

La décompression des chiens anesthésiés au vide proche (4 millimètres d'hectogramme) pour la réduction grave de 60 causes de secondes d'effets hémodynamiques artériels du sang flow.38 a produit à 4 millimètres d'hectogramme sont attribuable en grande partie à l'obstruction mécanique du système cardiovasculaire par des pressions extra-vasculaires accrues, résultant de l'expansion de gaz et particulièrement de la vaporisation de l'eau.

Les effets de l'hypoxie produits par la décompression à une altitude simulée de 30.000 pieds pendant 90 minutes s étudié dans à résultat conformé non anesthésié de dogs.39 A de la décompression étaient une diminution marquée de concentration en plasma-potassium. La concentration en sodium de plasma demeure sans changement.

Influence d'Otologic de la décompression

L'effet de la décompression sur l'oreille moyenne du singe a été studied.40 au cours de la décompression à un taux lent (50 millimètres d'hectogramme/minute), le tube eustachian ouvert périodiquement pour maintenir la pression tympanic ouverte de pression ambiante. L'ouverture périodique du tube eustachian s'est produite seulement quand le taux de décompression était lent. Quand le taux de décompression est plus haut que 120 millimètres d'hectogramme par minute, un état ouvert soutenu du tube eustachian résulte. Même aux taux excessifs de décompression, tels que vu pendant la décompression explosive, la pression d'oreille moyenne revient très rapidement à celle de la pression ambiante.

La décompression explosive se produit à un taux beaucoup de fois plus rapidement que cela a employé dans la décompression rapide. Par exemple, la décompression explosive peut se produire en environ 12 à 40 millisecondes avec une baisse dans la pression barométrique de 740 millimètres d'hectogramme à 25 millimètres d'hectogramme ou la décompression less.15.17 rapide peut changer à temps de 10.000 millisecondes et upward.3

L'évidence indique que l'hémorragie et la douleur tympanic sont provoquées par la pression négative (> 600 millimètres de l'hectogramme) qui se développe dans l'oreille moyenne pendant la récompression si le dernier est progressif ou l'hémorragie explosive.40 dans les sinus frontaux des chiens a été observé et également attribué à recompression.41 rapide

Myringopuncture peut empêcher le développement de la pression négative et peut donc empêcher la production des lésions barotraumatic à l'oreille. La piqûre des deux tambours d'oreille élimine également complètement la bradycardie pendant la récompression du singe non anesthésié apporté vers le bas de 42.000 pi à une vitesse plus rapide que librement fall.19 apparemment la bradycardie qui se produit pendant la récompression est due au unequalized la pression négative de moyen-oreille et est négocié par réflexes par le nerf de vagus. On l'a suggéré que les impulsions des récepteurs, fassent souffrir probablement des récepteurs, dans la moyen-oreille ou la membrane tympanic, ou tous les deux, lancent ce réflexe.

Dans les êtres humains, le malaise d'oreille et la douleur grave ont été observés principalement pendant la récompression ou en descendant à un altitude.22.42.43 inférieur il y a des cas rares où le barotrauma impliquant les oreilles ou les sinus se produisent pendant l'ascent.44 A prédisposant le facteur dans tous tels cas était infection respiratoire supérieure. Ceci n'étonne pas, parce que on le sait que l'inflammation du mucosa de région respiratoire peut interférer la ventilation de l'oreille moyenne et du sinuses.44 paranasal

Effets de Pathologie après la décompression

Les lésions brutes de pathologie vues chez les chiens suivant la décompression sont hémorragiques dans nature.45 Petechial aux hémorragies ecchymotic dans les poumons se produisent. Les dommages cardiaques se produisent également avec des hémorragies ecchymotic sur les valvules mitrales de quelques animaux. L'hémorragie d'Ecchymotic se produit également sous le mater de dura entourant le sinus sagittal du cerveau.

Des lésions hémorragiques suivant la décompression du type explosif sont trouvées principalement dans les poumons, cerveau, et heart.45 de ces derniers, les lésions pulmonaires sont la plupart des common.45.46 on le pense que que ces lésions se produisent comme resuit de l'augmentation soudaine de la pression intrapulmonary pendant la décompression. L'expansion rapide soudaine des poumons avec l'étirage des murs alvéolaires a probablement comme conséquence la déchirure de ces structures.

Des changements histopathologiques résiduels du système nerveux central des chiens ont été décrits après la décompression rapide à 1 millimètre d'hectogramme pour 120 seconds.47

Effet de la décompression et d'autres épisodes hypoxiques le temps de survie

L'inconscience ou l'effondrement chez des chiens d'adulte exposés aux altitudes simulées entre 50.000 et 55.000 pi, si respirant l'air ou l'O2 de 100 %, se produit dans moins de 9 ou 10 secondes suivant exposure.14 « anoxie complète » ou « hypoxie complète » se produit donc à ces altitudes (IE, 52.500 pi) chez les animaux respirant ou l'air ou 100% 02.32.48.49 dans les êtres humains, l'hypoxie potentiellement grave produite au-dessus de 50.000 pi commence à devenir efficacement renversé au niveau de 50.000 pi, s'améliorant rapidement avec la récompression continue à 40.000 pi ou lower.8

Les études chez les animaux ont prouvé que le temps de survie diminue avec l'augmentation de l'altitude comme sévérité de l'hypoxie increases.50 cependant, les extensions de temps de survie par minimum et restes constants indépendamment d'un accroissement plus ultérieur d'altitude. La période de survie minimale des animaux exposés à la décompression rapide a été étudiée en O2 et en air par Lutz.51 chez les animaux respirant 02, Lutz a constaté qu'un temps de survie minimal de 25 secondes a été atteint quand des animaux ont été décomprimés à une altitude simulée de 52.000 pi après le même procédé aux altitudes en-dessous de 52.000 pi que les temps de survie étaient plus longs, et aux altitudes au-dessus de 52.000 pi les temps de survie ne sont pas devenus sensiblement plus courts mais a été resté approximativement 25 secondes. Chez les animaux respirant l'air, Lutz a observé qu'un temps de survie minimal de 25 secondes a été atteint sur la décompression à 43.000 pi ou en haut.

La période de survie des animaux non anesthésiés (rats) après la décompression en air, quand le cessation de la respiration est employé comme point final, est constante pour toutes les altitudes finales simulées de décompression au-dessus de 52.000 ft.49 chez le rat, aux altitudes simulées de 52.000 et en haut, respiration rhythmique cessée sur la moyenne de 17.8 secondes après la décompression en air. Les études sur les effets de la décompression des chiens et des rats du niveau de la mer à 30 millimètres d'hectogramme (IE, 72.000 pi) ont indiqué que la respiration a cessé à environ 30 secondes. En outre, elle est d'intérêt et remarquable que la respiration chez les chiens cesse en 15 à 20 secondes après soudain accomplissez l'arrestation du circulation.29 cérébral

À mesure que l'exposition à l'altitude élevée et à l'environnement hypoxique de accompagnement augmente, la résistance ou la tolérance à l'hypoxie devient la tolérance less.50 à l'altitude élevée ou la décompression semble changer avec de diverses espèces animales. Comparé au cobaye, le chat et le chien sont plus tolerant.52 de chats, lapins, cavies, hamsters, rats, et les souris ne survivent pas une décompression de 100 millimètres d'hectogramme (IE, 47.000 pi) pour 3 minutes.53

Le centre respiratoire est le plus résistant à l'hypoxie à la naissance, puis diminue par le 4ème mois de la vie dans la résistance dog.54 à l'hypoxie induite par l'azote à la naissance change de 28 minutes dans l'écureuil moulu, à 16 minutes chez le chat, à 6 minutes chez le cobaye .55 que l'origine de la résistance hypoxique dans les mammifères n'a pas été identifiée.

Les lapins d'adulte peuvent tolérer une atmosphère d'anoxie de l'azote de 100 % pendant seulement 1.5 minutes avant la mort, tandis que le lapin nouveau-né peut survivre pour tant que 27 minutes.56 chez le chien d'adulte, occlusion aiguë de la circulation cérébrale et hypoxie résultante produisent le cessation de la respiration spontanée après seulement des 20 à 30 secondes ; dans les 8 - à de dix jours - le vieux chiot, cet effet se produit en 5 minutes, et dans l'animal nouveau-né, se produit en 27 minutes. Les reptiles et les amphibies peuvent tolérer la privation O2 jusqu'à un degré beaucoup plus grand que les espèces mammifères ; par exemple, la tortue peut tolérer l'anoxie produite de l'azote de 100 % pendant plusieurs heures et une dose de cyanure 50 fois plus grandes que qui toxique au mammal.56 [57]

L'exposition du chien à un environnement proche de vide (moins de 2 millimètres de l'absolu d'hectogramme) indique que les chiens exposés pendant moins de 120 secondes sont capables de la survie sur la récompression à 35.000 pi tout en respirant O2.14 chez de tels animaux, effondrement se produit dans 9 à 10 secondes après la décompression avec un spasticity généralisé de muscle, quelques halètements, saisies convulsives momentanées, apnea, et gonflement brut du corps et des extrémités.

Considérations humanitaires de la décompression

La technique rapide de décompression pour produire l'hypoxie (pas la méthode explosive de décompression) a été employée pour l'euthanasie d'animals.53.58 là ont été beaucoup d'études pathophysiologiques comportant l'utilisation des animaux soumis à la décompression. Les la plupart ont été conduites par altitude élevée ou espacent des laboratoires de recherches, des vols spatiaux ainsi équipés ont pu être accomplies avec un minimum de risque. L'évidence suffisante comme indiquée par des enregistrements d'EEG ont indiqué que l'hypoxie induit rapidement l'inconscience dans les deux animaux et homme soumis à l'altitude élevée simulée par l'utilisation des chambres de décompression ou l'inhalation des gaz inertes. On ne le connaît pas ce qui être les perceptions subjectives d'un animal dans une chambre peuvent mais une fois correctement faite, la décompression est un procédé indolore pour toute la décompression des espèces [58] au taux de 4.000 pi par minute pendant 10 minutes, de ce fait créant une altitude simulée de 40.000 pi (141 millimètres d'hectogramme), et maintenant ce presure jusqu'à ce que la respiration cesse sont considérés optimal pour un dog.58 mûr pour des adultes d'autres espèces telles que des chats, lapins, cavies, hamsters, rats, et souris, une décompression de 100 millimètres d'hectogramme (IE, 47.000 pi au-dessus de lel de mer) pendant 3 minutes est proportionné pour l'induction de l'euthanasie suivant un taux de décompression de 15 millimètres d'hectogramme par minute.53

Comme souligné dans le panneau de 1978 AVMA sur le rapport d'euthanasie, [2] l'utilisation réussie des chambres de décompression est affirmés sur le fonctionnement approprié et l'entretien de l'équipement. Le personnel actionnant l'équipement doit être habile et bien informé dans son utilisation comme comprenez les réactions esthétiquement désagréables manifestées par des animaux pendant la période du coma ou de l'inconscience hyperactif avant la mort. Les chiens au-dessous de 4 mois d'âge sont plus tolérants à l'hypoxie et ont besoin de de plus longues périodes de la décompression avant des animaux de la respiration ceases.54 avec des complications respiratoires et particulièrement ceux avec des médias d'otitis ne devraient pas être soumis à la décompression en raison de la possibilité du développement de la douleur de unequalized la pression positive de moyen-oreille.

Références

Le 1 Conseil d'AVMA sur la recherche : Rapport du panneau d'AVMA sur l'euthanasie. JAVMA 160:761 - 772, 1972.

Le Conseil de 2 AVMA sur la recherche : Rapport du panneau d'AVMA sur l'euthanasie. JAVMA 173:59 - 72, 1978.

3. Bryan CA, GT d'épuisement : Effets de Physiologie d'échec de pression de carlingue dans l'avion de passager d'altitude élevée. *Med aérospatial* 31:267 - 275, 1960.

4 Van Liere EJ, Stickney JC : *Hypoxie*. Chicago, université de la pression de Chicago, 1963, pp 1-381.

5. SM de Tenney : Respiration dans les mammifères (chapitre 15), dans Swenson, J Melvin (ED) : *Physiology de ducs des animaux domestiques*, ED 9. Ithaca, NY, associés de édition de Constock, Division de la pression d'université de Cornell, 1977, p 186.

6. Bonnet de fourrure De, Higgins ea, GE de Funkhouser : Effet d'activité physique des préposés de vol de ligne aérienne leur période de conscience utile dans une décompression rapide. *L'espace d'Aviat entourent Med* 47:117 - 120, 1976.

7. Bonnet de fourrure De, Higgins ea, GE de Funkhouser : Protection des préposés de vol de ligne aérienne contre l'hypoxie suivant la décompression rapide. *L'espace d'Aviat entourent Med* 47:942 - 944, 1976.

8. Bancroft RW, dg de Simmons : Décompressions rapides jusqu'à 60.000 pieds portant le masque d'oxygène standard. *Med aérospatial* 35:203 - 211, 1964.

9. Collines BA, rayon De : Narcose de gaz inerte. *Pharmacol là* [B] 3:99 - 111, 1977.

10. Behnke AR, Thomson RM, PE bariolé : Les effets psychologiques de l'air de respiration à la pression des 4 atmosphères. *AM J Physiol* 112:554 - 558, 1935.

11. Behnke AR, Yarbrough OD : Résistance respiratoire, solubilité de la huile-eau, et effets mentaux de l'argon, comparés à l'hélium et à l'azote. *AM J Physiol* 126:409 - 415, 1939.

12. Lawrence JH, Loomis WF, Tobias CA, et autres : Observations préliminaires sur l'effet narcotique du xénon avec un examen des valeurs pour des solubilités des gaz en eau et pétroles. *J Physiol* 105:197 - 204, 1946.

13. PB de Bennett, mb en verre : Electroencephalographic et d'autres changements induits par des pressions partielles élevées de l'azote. *Electroencephalogr Clin Neurophysiol* 13:91 - 98, 1961.

14. Bancroft RW, Dunn JE II : Décompressions d'animal d'expérience à un environnement proche de vide. *Med aérospatial* 36:720 - 725, 1965.

15. Edelmann A, Hitchcock fa : Observations sur des chiens exposés à une pression ambiante de 30 millimètres hectogramme. *J APPL Physiol* 4:807 - 812, 1952.

16. Hitchcock fa, Kemph J : L'ébullition des liquides de corps aux altitudes extrêmement élevées. *Aviat Med* 26:289 - 297, 1955.

17. Kemph JP, Beman FM, Hitchcock fa : Pression sous-cutanée développée chez les chiens suivant la décompression explosive à 25 ou 30 millimètres hectogramme. *AM J Physiol* 168:601 - 604, 1952.

18. Kemph JP, Hitchcock fa : D'autres études des effets « de la pression intrapulmonic d'igh sur des chiens à 30 millimètres hectogramme. *Aviat Med* 25:227 - 234, 1954.

19. Gelfan S : Décompression explosive des altitudes et de récompression extrêmes des singes W de macaque aux taux de chute libre. *J APPL Physiol* 3:254 - 281, 1950.

20. Holmstrom FMG : Effondrement pendant la décompression rapide. Rapport de trois cas. *J Aviat Med* 29:91 - 96, 1958.

21. Van Liere EJ : *Anoxie. Son effet sur le corps*. Chicago, université de la pression de Chicago, 1942, pp 1-269.

22. Ci de Barron, cuisinier TJ : Effets des décompressions variables à 45.000 pieds. *Med aérospatial* 36:425 - 430, 1965.

23. Luft UC, Clamann hectogramme, Opitz E : La latence de l'hypoxie au contact de l'altitude au-dessus de 50.000 pieds. *J Aviat Med* 22 117-136, 1951.

24. Rossen R, Kabat H, Andersen JP : Arrestation aiguë de circulation de cerclai chez l'homme. *Voûte d'AMA Neurol Psychiatr* 50:510 - S28, 1943.

Sust I. 1978

25. Gibbs fa, Davis H : Changements de l'électroencéphalogramme humain lié à la perte de conscience. *AM J Physiol* 113:49 - 50, 1935.

26. PA de Davis, Davis H, Thompson JW : Changements de progressif de l'électroencéphalogramme humain sous la tension à faible teneur en oxygène. *AM J Physiol* 123:51 - 52, 1938.

27. Sucre O, Gerard RW : Anoxie et potentiel de cerveau. *J Neurophysiol* 1:558 - 571, 1938.

28. Stephens LM, Hartman JL, Lewis DE, et autres : Physiologie électro- des chimpanzés pendant la décompression rapide. *Med aérospatial* 38:694 - 698, 1967.

29. Kabat H, Dennis C, Baker ab : Rétablissement de l'arrestation suivante de fonction de la circulation de cerveau. *AM J Physiol* 132:737 - 747, 1941.

30. Cabine NH : Intravenous et tout autre anestlietics parentéral, en Jones LM, cabine NH, McDonald le (ED) : *Pharmacologie et thérapeutique vétérinaires.* Ames, pression d'université de l'Etat de lowa, 1977, pp 241-306.

31. Whitehorn WV, Lein A, Edelmann A : La tolérance générale et les réponses cardiovasculaires des animaux à la décompression explosive. *AM J Physiol* 147:289 - 298, 1946.

32. Gelfan S, Werner AY : Réponses cardiovasculaires suivant la décompression explosive des singes de macaque aux altitudes d'extrême. *J APPL Physiol* 4:280 - 310, 1951.

33. GT de Lennox, Gibbs fa, EL de Gibbs : Relation d'un-consciousness avec l'écoulement cérébral de sang et à l'anoxemia. *Voûte Neurol Psychiatr* 34:1001 - 1013, 1935 d'*AMA*.

34. Kemph JP, Hitchcock fa : Changements de sang et de circulation des chiens suivant la décompression explosive à de basses pressions barométriques. *AM J Physiol* 168:592 - 600, 1952.

35. Cooke JP, Bancroft RW : Quelques réponses cardiovasculaires chez les chiens anesthésiés pendant des décompressions répétées à un vide proche. *Med aérospatial* 37:1148 - 1152, 1966.

36. Bancroft RW, Cooke JP, SM de gain : Comparaison de l'anoxie avec et sans l'ebullism. *J APPL Physiol* 25:230 - 237, 1968.

37. Cooke JP, SM de Caïn, Bancroft RW : Pressions veineuses élevées pendant l'exposition des chiens aux états proches de vide. *Med aérospatial* 38:1021 - 1024, 1967.

38. Pratt AJ, HL en pierre, à haute fréquence de Stegall, et autres : Affaiblissement circulatoire pendant l'exposition aux pressions ambiantes de 4 millimètres hectogramme et de 55 millimètres hectogramme. *J APPL Physiol* 29:177 - 180, 1970.

39. Point de gel de Ferguson, C.C de Smith : Effets d'effort aigu de décompression sur des électrolytes de plasma et de fonction rénale chez les chiens. *AM J Physiol* 173:503 - 510, 1953.

40. Chang H-T, Margaria R, Gelfan S : Changements et barotrauma de pression résultant de la décompression et de la récompression de l'oreille moyenne des singes. *Voûte Otolaryngol* 51:378 - 399, 1950.

41. CR de Cole, Chamberlain DM, Burch BH, et autres : Effets pathologiques de la décompression explosive à 30 millimètres hectogramme. *J APPL Physiol* 6:96 - 104, 1953.

42. Ci de Barron, Collier DR Jr, cuisinier TJ : Observations sur des décompressions en 12 secondes simulées à 32.000 pieds. *Aviat Med* 29:563 - 574, 1958.

43. Idicula J : Cas confondant de barotrauma maxillaire de sinus. *Med aérospatial* 43:891 - 892, 1972.

44. Rue de Lewis : Barotrauma dans des accidents/incidents de l'Armée de l'Air des Etats-Unis. *Med aérospatial* 44:1059 - 1061, 1973.

45. Edelman A, Whitehorn WV, Lein A, et autres : Lésions pathologiques produites par la décompression explosive. *Aviat Med* 17-596-612, 1946.

46. Dunn JE II, Bancroft RW, Haymaker W, et autres : Décompressions d'animal d'expérience moins de 2 à l'absolu du millimètre hectogrammes (effets de pathologie). *Med aérospatial* 36:725 - 732, 1965.

47. Casey HW, Bancroft RW, Cooke JP : Changements résiduels de pathologie du système nerveux central d'un chien suivant la décompression rapide à 1 millimètre hectogramme. *Med aérospatial* 37:713 - 718, 1966.

48. Gelfan S, Nims LF, RB de Livingston : Cause de la mort de la décompression explosive à l'altitude élevée (résumé). *Fédéral Proc* 6:110, 1947.

49. Gelfan S, Nims LF, RB de Livingston : Décompression explosive à l'altitude élevée. AM J Physiol 162:37 - 53, 1950.

50. Armstrong hectogramme : Anoxie dans l'aviation. *Aviat Med* 9:84 - 91, 1938.

51. Luft UC, Clamann hectogramme, à haute fréquence d'Adler : Gaz alvéolaires dans la décompression rapide aux altitudes élevées. *J APPL Physiol* 2:37 - 48, 1949.

52. Whitehorn WV, Lein A, Hitchcock fa : L'effet de la décompression explosive sur l'occurrence des bulles intravasculaires. *Aviat Med* 18:392 - 394, 1947.

53. BR de coiffeur : Utilisation d'un autoclave standard pour l'euthanasie de décompression. *Institut Anim Technol* 23:106 - 110, 1972 de *J*.

54. Kabat H : La résistance plus grande des animaux très jeunes à l'arrestation de la circulation *AM J Physiol* 130:588 - 599, 1940 de cerveau.

55. Adolph E-F : Règlements pendant la survie sans oxygène dans les mammifères infantiles. *Respir Physiol* 7:356 - 368, 1969.

56. Cohen PJ : La fonction métabolique de l'oxygène et des lésions biochimiques de l'hypoxie. *Anesthesiology* 37:148 - 177, 1972.

57. Bellamy D, Peterson JA : Anaerobiosis et la toxicité du cyanure dans les tortues. *Biochimie Physiol* 24:543 - 548, 1968 d'*élém*.

58. C.C de Smith : Euthanasla d'ot de méthodes et disposition des animaux de laboratoire, dans WI gai (ED) : *Méthodes d'expérimentation animale*. New York, pression d'Academie, 1965, vol. I, pp 167wi-

59. Kilowatt de Miller, WDM de Paton, Smith eb : Emplacement de l'action des anesthésiques généraux. *Nature* 206:574 - 577, 1965.

60. Saidman LJ, EL II, Munson es d'Eger, et autres : Concentrations alvéolaires minimum de methoxyflurane, halothane, éther, cyclopropane chez l'homme : Corrélation avec des théories d'anesthésie ? *Anesthesiology* 28:994 - 1002, 1967.

TAB F SECTION 20
GUIDELINES FOR HUMANE EUTHANASIA OF ANIMALS

Information from the 2000 Report of the AVMA Panel on Euthanasia
Excerpted from the *Journal of the American Veterinary Medical Association*,
Vol. 218, No. 5, Pages 669-696

INTRODUCTION

The practice of veterinary medicine is complex and involves diverse animal species. Whenever possible, a veterinarian experienced with the species in question should be consulted when selecting the method of euthanasia, particularly when little species-specific euthanasia research has been done.

The recommendations in this report are intended to serve as guidelines for veterinarians who must then use professional judgment in applying them to the various settings where animals are to be euthanatized.

In the context of this report, euthanasia is the act of inducing humane death in an animal. It is our responsibility as veterinarians and human beings to ensure that if an animal's life is to be taken, it is done with the highest degree of respect, and with an emphasis on making the death as painless and distress free as possible. Euthanasia techniques should result in rapid loss of consciousness followed by cardiac or respiratory arrest and the ultimate loss of brain function. In addition, the technique should minimize distress and anxiety experienced by the animal prior to loss of consciousness. The absence of pain and distress cannot always be achieved. This report attempts to balance the ideal of minimal pain and distress with the reality of the many environments in which euthanasia is performed.

It is imperative that death be verified after euthanasia and before disposal of the animal. An animal in deep narcosis following administration of an injectable or inhalant agent may appear dead, but might eventually recover. Death must be confirmed by examining the animal for cessation of vital signs, and consideration given to the animal species and method of euthanasia when determining the criteria for confirming death.

GENERAL CONSIDERATIONS

In evaluating methods of euthanasia, the 2000 AVMA panel on euthanasia used the following criteria: (1) ability to induce loss of consciousness and death without causing pain, distress, anxiety, or apprehension; (2) time required to induce loss of consciousness; (3) reliability; (4) safety of personnel; (5) irreversibility; (6) compatibility with requirement and purpose; (7) emotional effect on observers or operators; (8) compatibility with subsequent evaluation, examination, or use of tissue; (9) drug availability and human abuse potential; (10) compatibility with species, age, and health status; (11) ability to maintain equipment in proper working order; and (12) safety for predators/scavengers should the carcass be consumed.

PHYSICAL METHODS

Physical methods of euthanasia include captive bolt, gunshot, cervical dislocation, decapitation, electrocution, microwave irradiation, kill traps, thoracic compression, exsanguination, stunning, and pithing. When properly used by skilled personnel with well-maintained equipment, physical methods of euthanasia may result in less fear and anxiety and be more rapid, painless, humane, and practical than other forms of euthanasia. Exsanguination, stunning, and pithing are not recommended as a sole means of euthanasia, but should be considered adjuncts to other agents or methods.

Some consider physical methods of euthanasia aesthetically displeasing. There are occasions, however, when what is perceived as aesthetic and what is most humane are in conflict. Physical methods may be the most appropriate method for euthanasia and rapid relief of pain and suffering in certain situations. Personnel performing physical methods of euthanasia must be well trained and monitored for each type of physical technique performed. That person must also be sensitive to the aesthetic implications of the method and inform onlookers about what they should expect when possible.

Since most physical methods involve trauma, there is inherent risk for animals and humans. Extreme care and caution should be used. Skill and experience of personnel is essential. If the method is not performed correctly, animals and personnel may be injured. Inexperienced persons should be trained by experienced persons and should practice on carcasses or anesthetized animals to be euthanatized until they are proficient in performing the method properly and humanely. When done appropriately, the panel considers most physical methods conditionally acceptable for euthanasia.

Penetrating captive bolt

A penetrating captive bolt is used for euthanasia of ruminants, horses, swine, rabbits, and dogs.[8] Its mode of action is concussion and trauma to the cerebral hemisphere and brainstem.[9,10] Captive bolt guns are powered by gunpowder or compressed air and must provide sufficient energy to penetrate the skull of the species on which they are being used.[9] Adequate restraint is important to ensure proper placement of the captive bolt. A cerebral hemisphere and the brainstem must be sufficiently disrupted by the projectile to induce sudden loss of consciousness and subsequent death. Accurate placement of captive bolts for various species has been described.[9-12] A multiple projectile has been suggested as a more effective technique, especially for large cattle.[9]

A nonpenetrating captive bolt only stuns animals and should not be used as a sole means of euthanasia.

Advantage—The penetrating captive bolt is an effective method of euthanasia for use on the farm when use of drugs is inappropriate.

Disadvantages—(1) It is aesthetically displeasing. (2) Death may not occur if equipment is not maintained and used properly.

Recommendations—Use of the penetrating captive bolt is an acceptable and practical method of euthanasia for horses, ruminants, and swine. It is conditionally acceptable in other appropriate species. The nonpenetrating captive bolt must not be used as a sole method of euthanasia.

Gunshot

A properly placed gunshot can cause immediate insensibility and humane death. In some circumstances, a gunshot may be the only practical method of euthanasia. Shooting should only be performed by highly skilled personnel trained in the use of firearms and only in jurisdictions that allow for legal firearm use. Personnel, public, and nearby animal safety should be considered. The procedure should be performed outdoors and away from public access.

For use of a gunshot to the head as a method of euthanasia in captive animals, the firearm should be aimed so that the projectile enters the brain, causing instant loss of consciousness.[3,12-14] This must take into account differences in brain position and skull conformation between species, as well as the energy requirement for skull bone and sinus penetration.[9,15] Accurate targeting for a gunshot to the head in various species has been described.[14,16-19] For wildlife and other freely roaming animals, the preferred target area should be the head. The appropriate firearm should be selected for the situation, with the goal being penetration and destruction of brain tissue without emergence from the contralateral side of the head.[20] A gunshot to the heart or neck does not immediately render animals unconscious and thus is not considered to meet the panel's definition of euthanasia.[21]

Advantages—(1) Loss of consciousness is instantaneous if the projectile destroys most of the brain. (2) Given the need to minimize stress induced by handling and human contact, gunshot may at times be the most practical and logical method of euthanasia of wild or free-ranging species.

Disadvantages—(1) Gunshot may be dangerous to personnel. (2) It is aesthetically unpleasant. (3) Under field conditions, it may be difficult to hit the vital target area. (4) Brain tissue may not be able to be examined for evidence of rabies infection or chronic wasting disease when the head is targeted.

Recommendations—When other methods cannot be used, an accurately delivered gunshot is a conditionally acceptable method of euthanasia.[14,22-25] When an animal can be appropriately restrained, the penetrating captive bolt is preferred to a gunshot. Prior to shooting, animals accustomed to the presence of humans should be treated in a calm and reassuring manner to minimize anxiety. In the case of wild animals, gunshots should be delivered with the least amount of prior human contact necessary. Gunshot should not be used for routine euthanasia of animals in animal control situations, such as municipal pounds or shelters.

Cervical dislocation

Cervical dislocation is a technique that has been used for many years and, when performed by well-trained individuals, appears to be humane. However, there are few scientific studies to confirm this observation. This technique is used to euthanatize poultry, other small birds, mice, and immature rats and rabbits. For mice and rats, the thumb and index finger are placed on either side of the neck at the base of the skull or, alternatively, a rod is pressed at the base of the skull. With the other hand, the base of the tail or the hind limbs are quickly pulled, causing separation of the cervical vertebrae from the skull. For immature rabbits, the head is held in one hand and the hind limbs in the other. The animal is stretched and the neck is hyperextended and dorsally twisted to separate the first cervical vertebra from the skull.[4,11] For poultry, cervical dislocation by stretching is a common method for mass euthanasia, but loss of consciousness may not be instantaneous.[32]

Data suggest that electrical activity in the brain persists for 13 seconds following cervical dislocation,[26] and unlike decapitation, rapid exsanguination does not contribute to loss of consciousness.[27,28]

Advantages—(1) Cervical dislocation is a technique that may induce rapid loss of consciousness.[6,26] (2) It does not chemically contaminate tissue. (3) It is rapidly accomplished.

Disadvantages—(1) Cervical dislocation may be aesthetically displeasing to personnel. (2) Cervical dislocation requires mastering technical skills to ensure loss of consciousness is rapidly induced. (3) Its use is limited to poultry, other small birds, mice, and immature rats and rabbits.

Recommendations—Manual cervical dislocation is a humane technique for euthanasia of poultry, other small birds, mice, rats weighing < 200 g, and rabbits weighing < 1 kg when performed by individuals with a demonstrated high degree of technical proficiency. In lieu of demonstrated technical competency, animals must be sedated or anesthetized prior to cervical dislocation. The need for technical competency is greater in heavy rats and rabbits, in which the large muscle mass in the cervical region makes manual cervical dislocation physically more difficult.[29]

Those responsible for the use of this technique must ensure that personnel performing cervical dislocation techniques have been properly trained and consistently apply it humanely and effectively.

Decapitation

Decapitation can be used to euthanatize rodents and small rabbits. It provides a means to recover tissues and body fluids that are chemically uncontaminated. It also provides a means of obtaining anatomically undamaged brain tissue for study.[30]

Although it has been demonstrated that electrical activity in the brain persists for 13 to 14 seconds following decapitation,[31] more recent studies and reports indicate that this activity does not infer the ability to perceive pain, and in fact conclude that loss of consciousness develops rapidly.[26-28]

Guillotines that are designed to accomplish decapitation in adult rodents and small rabbits in a uniformly instantaneous manner are commercially available. Guillotines are not commercially available for neonatal rodents, but sharp blades can be used for this purpose.

Advantages—(1) Decapitation is a technique that appears to induce rapid loss of consciousness.[26-28] (2) It does not chemically contaminate tissues. (3) It is rapidly accomplished.

Disadvantages—(1) Handling and restraint required to perform this technique may be distressful to animals.[5] (2) The interpretation of the presence of electrical activity in the brain following decapitation has created controversy and its importance may still be open to debate.[26-28,31] (3) Personnel performing this technique should recognize the inherent danger of the guillotine and take adequate precautions to prevent personal injury. (4) Decapitation may be aesthetically displeasing to personnel performing or observing the technique.

Recommendations—The equipment used to perform decapitation should be maintained in good working order and serviced on a regular basis to ensure sharpness of blades. The use of plastic cones to restrain animals appears to reduce distress from handling, minimizes the chance of injury to personnel, and improves positioning of the animal in the guillotine.

Those responsible for the use of this technique must ensure that personnel who perform decapitation techniques have been properly trained to do so.

SPECIAL CONSIDERATIONS

Equine euthanasia

Pentobarbital or a pentobarbital combination is the best choice for equine euthanasia. Because a large volume of solution must be injected, use of an intravenous catheter placed in the jugular vein will facilitate the procedure. To facilitate catheterization of an excitable or fractious animal, a tranquilizer such as acepromazine, or an alpha-2 adrenergic agonist can be administered, but these drugs may prolong time to loss of consciousness because of their effect on circulation and may result in varying degrees of muscular activity and agonal gasping. Opioid agonists or agonist/antagonists in conjunction with alpha-2 adrenergic agonists may further facilitate restraint.

In certain emergency circumstances, it may be difficult to restrain a dangerous horse or other large animal for intravenous injection. The animal might cause injury to itself or to bystanders before a sedative could take effect. In such cases, the animal can be given a neuromuscular blocking agent such as succinylcholine, but the animal must be euthanatized with an appropriate technique as soon as the animal can be controlled. Succinylcholine alone or without sufficient anesthetic must not be used for euthanasia.

Physical methods, including gunshot, are considered conditionally acceptable techniques for equine euthanasia. The penetrating captive bolt is acceptable with appropriate restraint.

Animals intended for human or animal food

In euthanasia of animals intended for human or animal food, chemical agents that result in tissue residues cannot be used, unless they are approved by the US Food and Drug Administration.[33] Carbon dioxide is the only chemical currently used for euthanasia of food animals (primarily swine) that does not result in tissue residues. Physical techniques are commonly used for this reason. Carcasses of animals euthanatized by barbituric acid derivatives or other chemical agents may contain potentially harmful residues. These carcasses should be disposed of in a manner that will prevent them from being consumed by human beings or animals.

Selection of a proper euthanasia technique for free-ranging wildlife must take into account the possibility of consumption of the carcass of the euthanatized animal by nontarget predatory or scavenger species. Numerous cases of toxicosis and death attributable to ingestion of pharmaceutically contaminated carcasses in predators and scavengers have been reported.[7] Proper carcass disposal must be a part of any euthanasia procedure under free-range conditions where there is potential for consumption toxicity. When carcasses are to be left in the field, a gunshot to the head, penetrating captive bolt, or injectable agents that are nontoxic (potassium chloride in combination with a nontoxic general anesthetic) should be used so that the potential for scavenger or predator toxicity is lessened.

Euthanasia of nonconventional species: zoo, wild, aquatic, and ectothermic animals

Compared with objective information on companion, farm, and laboratory animals, euthanasia of species such as zoo, wild, aquatic, and ectothermic animals has been studied less, and guidelines are more limited. Irrespective of the unique or unusual features of some species, whenever it becomes necessary to euthanatize an animal, death must be induced as painlessly and quickly as possible.

When selecting a means of euthanasia for these species, factors and criteria in addition to those previously discussed must be considered. The means selected will depend on the species, size, safety aspects, location of the animals to be euthanatized, and experience of personnel. Whether the animal to be euthanatized is in the wild, in captivity, or free-roaming are major considerations. Anatomic differences must be considered. For example, amphibians, fish, reptiles, and marine mammals differ anatomically from domestic species. Veins may be difficult to locate. Some species have a carapace or other defensive anatomic adaptations (eg, quills, scales, spines). For physical methods, access to the central nervous system may be difficult because the brain may be small and difficult to locate by inexperienced persons.

ZOO ANIMALS

For captive zoo mammals and birds with related domestic counterparts, many of the means described previously are appropriate. However, to minimize injury to persons or animals, additional precautions such as handling and physical or chemical restraint are important considerations.[2]

WILDLIFE

For wild and feral animals, many recommended means of euthanasia for captive animals are not feasible. The panel recognizes there are situations involving free-ranging wildlife when euthanasia is not possible from the animal or human safety standpoint, and killing may be necessary. Conditions found in the field, although more challenging than those that are controlled, do not in any way reduce or minimize the ethical obligation of the responsible individual to reduce pain and distress to the greatest extent possible during the taking of an animal's life. Because euthanasia of wildlife is often performed by lay personnel in remote settings, guidelines are needed to assist veterinarians, wildlife biologists, and wildlife health professionals in developing humane protocols for euthanasia of wildlife.

In the case of free-ranging wildlife, personnel may not be trained in the proper use of remote anesthesia, proper delivery equipment may not be available, personnel may be working alone in remote areas where accidental exposure to potent anesthetic medications used in wildlife capture would present a risk to human safety, or approaching the animal within a practical darting distance may not be possible. In these cases, the only practical means of animal collection may be gunshot and kill trapping.[1,34-38] Under these conditions, specific methods chosen must be as age-, species-, or taxonomic/class-specific as possible. The firearm and ammunition should be appropriate for the species and purpose. Personnel should be sufficiently skilled to be accurate, and they should be experienced in the proper and safe use of firearms, complying with laws and regulations governing their possession and use.

Behavioral responses of wildlife or captive nontraditional species (zoo) in close human contact are very different from those of domestic animals. These animals are usually frightened and distressed. Thus,

minimizing the amount, degree, and/or cognition of human contact during procedures that require handling is of utmost importance. Handling these animals often requires general anesthesia, which provides loss of consciousness and which relieves distress, anxiety, apprehension, and perception of pain. Even though the animal is under general anesthesia, minimizing auditory, visual, and tactile stimulation will help ensure the most stress-free euthanasia possible. With use of general anesthesia, there are more methods for euthanasia available.

A 2-stage euthanasia process involving general anesthesia, tranquilization, or use of analgesics, followed by intravenous injectable pharmaceuticals, although preferred, is often not practical. Injectable anesthetics are not always legally or readily available to those working in nuisance animal control, and the distress to the animal induced by live capture, transport to a veterinary facility, and confinement in a veterinary hospital prior to euthanasia must be considered in choosing the most humane technique for the situation at hand. Veterinarians providing support to those working with injured or live-trapped, free-ranging animals should take capture, transport, handling distress, and possible carcass consumption into consideration when asked to assist with euthanasia. Alternatives to 2-stage euthanasia using anesthesia include a squeeze cage with intraperitoneal injection of sodium pentobarbital, inhalant agents (CO_2 chamber, CO chamber), and gunshot. In cases where preeuthanasia anesthetics are not available, intraperitoneal injections of sodium pentobarbital, although slower in producing loss of consciousness, should be considered preferable over intravenous injection, if restraint will cause increased distress to the animal or danger to the operator.

Wildlife species may be encountered under a variety of situations. Euthanasia of the same species under different conditions may require different techniques. Even in a controlled setting, an extremely fractious large animal may threaten the safety of the practitioner, bystanders, and itself. When safety is in question and the fractious large animal, whether wild, feral, or domestic, is in close confinement, neuromuscular blocking agents may be used immediately prior to the use of an acceptable form of euthanasia. For this technique to be humane, the operator must ensure they will gain control over the animal and perform euthanasia before distress develops. Succinylcholine is not acceptable as a method of restraint for use in free-ranging wildlife because animals may not be retrieved rapidly enough to prevent neuromuscular blocking agent-induced respiratory distress or arrest.[39]

DISEASED, INJURED, OR LIVE-CAPTURED WILDLIFE OR FERAL SPECIES

Euthanasia of diseased, injured, or live-trapped wildlife should be performed by qualified professionals. Certain cases of wildlife injury (eg, acute, severe trauma from automobiles) may require immediate action, and pain and suffering in the animal may be best relieved most rapidly by physical methods including gunshot or penetrating captive bolt followed by exsanguination.

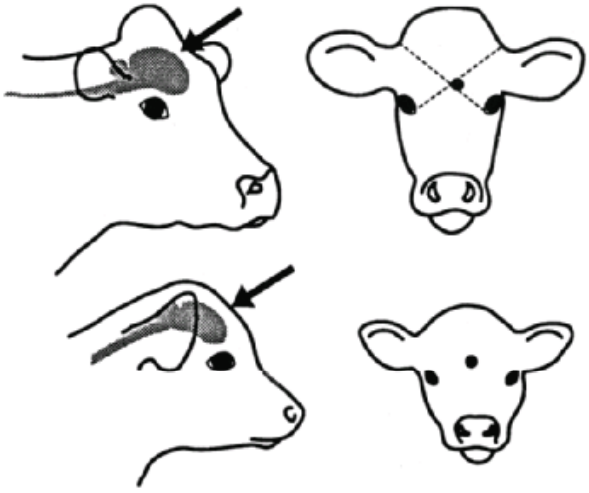
BIRDS

Many techniques discussed previously in this report are suitable for euthanasia of captive birds accustomed to human contact. Free-ranging birds may be collected by a number of methods, including nets and live traps, with subsequent euthanasia. For collection by firearm, shotguns are recommended. The bird should be killed outright by use of ammunition loads appropriate for the species to be collected. Wounded birds should be killed quickly by appropriate techniques previously described. Large birds should be anesthetized prior to euthanasia, using general anesthetics.

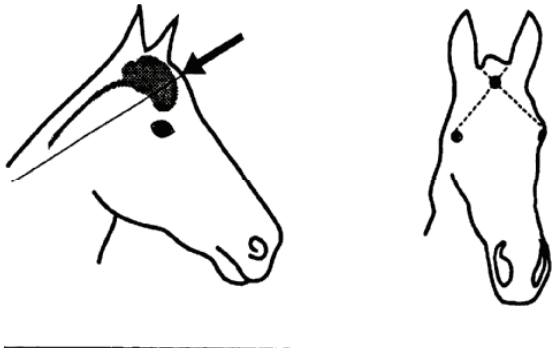
Mass euthanasia

Under unusual conditions, such as disease eradication and natural disasters, euthanasia options may be limited. In these situations, the most appropriate technique that minimizes human and animal health concerns must be used. These options include, but are not limited to, CO_2 and physical methods such as gunshot, penetrating captive bolt, and cervical dislocation.

CORRECT LOCATION FOR EUTHANASIA OF LIVESTOCK WITH A FIREARM OR CAPTIVE BOLT GUN
(Figures reprinted from Can Vet J 1991; 32: 724-726 with the permission of the Canadian Veterinary Medical Association)

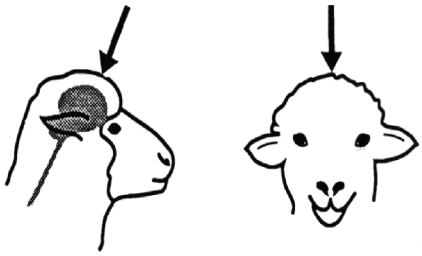


Cow/Calf



Horse/Donkey

Sheep (with horns)



Sheep (without horns)

Pig

References

1. Cooper JE, Ewbank R, Platt C, et al. *Euthanasia of amphibians and reptiles*. London: UFAW/WSPA, 1989.
2. Fowler ME, Miller RE, eds. *Zoo and wild animal medicine: current therapy 4*. Philadelphia: WB Saunders Co, 1999;1–747.
3. *Humane killing of animals*. Preprint of 4th ed. South Mimms, Potters Bar, Herts, England: Universities Federation for Animal Welfare, 1988;16–22.
4. Hughes HC. Euthanasia of laboratory animals. In: Melby EC, Altman NH, eds. *Handbook of laboratory animal science*. Vol 3. Cleveland, Ohio: CRC Press, 1976;553–559.
5. Urbanski HF, Kelly SF. Sedation by exposure to gaseous carbon dioxide-oxygen mixture: application to studies involving small laboratory animal species. *Lab Anim Sci* 1991;41:80–82.
6. Iwarsson K, Rehbinder C. A study of different euthanasia techniques in guinea pigs, rats, and mice. Animal response and postmortem findings. *Scand J Lab Anim Sci* 1993;20:191–205.
7. Barbiturates. In: Ciganovich E, ed. *Field manual of wildlife diseases*. US Department of the Interior/US Geological Survey, Biological Resources Division, Information and Technical Report 1999-2001.
8. Dennis MB, Dong WK, Weisbrod KA, et al. Use of captive bolt as a method of euthanasia in larger laboratory animal species. *Lab Anim Sci* 1988;38:459–462.
9. Blackmore DK. Energy requirements for the penetration of heads of domestic stock and the development of a multiple projectile. *Vet Rec* 1985;116:36–40.
10. Daly CC, Whittington PE. Investigation into the principal determinants of effective captive bolt stunning of sheep. *Res Vet Sci* 1989;46:406–408.
11. Clifford DH. Preanesthesia, anesthesia, analgesia, and euthanasia. In: Fox JG, Cohen BJ, Loew FM, eds. *Laboratory animal medicine*. New York: Academic Press Inc, 1984;528–563.
12. Australian Veterinary Association. Guidelines on humane slaughter and euthanasia. *Aust Vet J* 1987;64:4–7.
13. Carding T. Euthanasia of dogs and cats. *Anim Reg Stud* 1977;1:5–21.
14. Longair JA, Finley GG, Laniel M-A, et al. Guidelines for euthanasia of domestic animals by firearms. *Can Vet J* 1991;32: 724–726.
15. Finnie JW. Neuroradiological aspects of experimental traumatic missle injury in sheep. *N Z Vet J* 1994;42:54–57.
16. Blackmore DK, Madie P, Bowling MC, et al. The use of a shotgun for euthanasia of stranded cetaceans. *N Z Vet J* 1995; 43:158–159.
17. Blackmore DK, Bowling MC, Madie, P, et al. The use of a shotgun for emergency slaughter or euthanasia of large mature pigs. *N Z Vet J* 1995;43:134–137.
18. Denicola AJ. Non-traditional techniques for management of overabundant deer populations. *Wildl Soc Bull* 1997;25:496–499.
19. McAninch JB, ed. Urban deer: a manageable resource? in *Proceedings*. Symp 55th Midwest Fish Wildl Conf 1993;1–175.
20. Finnie JW. Traumatic head injury in ruminant livestock. *Aust Vet J* 1997;75:204–208.
21. Blackmore DK, Daly CC, Cook CJ. Electroencephalographic studies on the nape shooting of sheep. *N Z Vet J* 1995;43:160–163.
22. *On-farm euthanasia of swine—options for the producer*. Perry, Iowa: American Association of Swine Practitioners and Des Moines, Iowa: National Pork Producers, 1997.
23. *Practical euthanasia of cattle: considerations for the producer, livestock market operator, livestock transporter, and veterinarian*. Rome, Ga: American Association of Bovine Practitioners, 1999.
24. *The emergency euthanasia of horses*. Sacramento: California Department of Food and Agriculture and Davis, Calif: University of California's Veterinary Medical Extension, 1999.
25. *The emergency euthanasia of sheep and goats*. Sacramento: California Department of Food and Agriculture and Davis, Calif: University of California's Veterinary Medical Extension, 1999.
26. Vanderwolf CH, Buzak DP, Cain RK, et al. Neocortical and hippocampal electrical activity following decapitation in the rat. *Brain Res* 1988;451:340–344.
27. Derr RF. Pain perception in decapitated rat brain. *Life Sci* 1991;49:1399–1402.
28. Holson RR. Euthanasia by decapitation: evidence that this technique produces prompt, painless unconsciousness in laboratory rodents. *Neurotoxicol Teratol* 1992;14:253–257.
29. Keller GL. Physical euthanasia methods. *Lab Anim* 1982;11:20–26.
30. Feldman DB, Gupta BN. Histopathologic changes in laboratory animals resulting from various methods of euthanasia. *Lab Anim Sci* 1976;26:218–221.
31. Mikeska JA, Klemm WR. EEG evaluation of humaneness of asphyxia and decapitation euthanasia of the laboratory rat. *Lab Anim Sci* 1975;25:175–179.
32. Lambooy E, van Voorst N. Electrocution of pigs with notifiable diseases. *Vet Q* 1986;8:80–82.
33. Booth NH. Drug and chemical residues in the edible tissues of animals. In: Booth NH, McDonald LE, eds. *Veterinary pharmacology and therapeutics*. 6th ed. Ames, Iowa: Iowa State University Press, 1988;1149–1205.
34. Acceptable field methods in mammalogy: preliminary guidelines approved by the American Society of Mammalogists. *J Mammal* 1987;68(Suppl 4):1–18.
35. American Ornithologists' Union. Report of committee on use of wild birds in research. *Auk* 1988;105(Suppl):1A–41A.

36. American Society of Ichthyologists and Herpetologists, Herpetologist League, Society for the Study of Amphibians and Reptiles. Guidelines for the use of live amphibians and reptiles in field research. *J Herpetol* 1987;21(suppl 4):1–14.
37. American Society of Ichthyologists and Herpetologists, American Fisheries Society, American Institute of Fisheries Research Biologists. Guidelines for use of fishes in field research. *Copeia Suppl* 1987;1–12.
38. Cailliet GM. *Fishes: a field guide and laboratory manual on their structure, identification, and natural history*. Belmont, Calif: Wadsworth, 1986.
39. Schwartz JA, Warren R, Henderson D, et al. Captive and field tests of a method for immobilization and euthanasia of urban deer. *Wildl Soc Bull* 1997;25:532–541.

Appendix 1

Agents and methods of euthanasia by species

Species	Acceptable*	Conditionally acceptable†
Amphibians	Barbiturates, inhalant anesthetics (in appropriate species), CO_2, CO, tricaine methane sulfonate (TMS, MS 222), benzocaine hydrochloride, double pithing	Penetrating captive bolt, gunshot, stunning and decapitation, decapitation and pithing.
Birds	Barbiturates, inhalant anesthetics, CO_2, CO, gunshot (free-ranging only)	N_2, Ar, cervical dislocation, decapitation, thoracic compression (small, free-ranging only)
Cats	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar
Dogs	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, penetrating captive bolt, electrocution
Fish	Barbiturates, inhalant anesthetics, CO_2, tricaine methane sulfonate (TMS, MS 222), benzocaine hydrochloride, 2-phenoxyethanol	Decapitation and pithing, stunning and decapitation/pithing
Horses	Barbiturates, potassium chloride in conjunction with general anesthesia, penetrating captive bolt	Chloral hydrate (IV, after sedation), gunshot, electrocution
Marine mammals	Barbiturates, etorphine hydrochloride	Gunshot (cetaceans < 4 meters long)
Mink, fox, and other mammals produced for fur	Barbiturates, inhalant anesthetics, CO_2 (mink require high concentrations for euthanasia without supplemental agents), CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, electrocution followed by cervical dislocation
Nonhuman primates	Barbiturates	Inhalant anesthetics, CO_2, CO, N_2, Ar
Rabbits	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, cervical dislocation (< 1 kg), decapitation, penetrating captive bolt
Reptiles	Barbiturates, inhalant anesthetics (in appropriate species), CO_2 (in appropriate species)	Penetrating captive bolt, gunshot, decapitation and pithing, stunning and decapitation
Rodents and other small mammals	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia, microwave irradiation	Methoxyflurane, ether, N_2, Ar, cervical dislocation (rats < 200 g), decapitation
Ruminants	Barbiturates, potassium chloride in conjunction with general anesthesia, penetrating captive bolt	Chloral hydrate (IV, after sedation), gunshot, electrocution
Swine	Barbiturates, CO_2, potassium chloride in conjunction with general anesthesia, penetrating captive bolt	Inhalant anesthetics, CO, chloral hydrate (IV, after sedation), gunshot, electrocution, blow to the head (< 3 weeks of age)
Zoo animals	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, penetrating captive bolt, gunshot
Free-ranging wildlife	Barbiturates IV or IP, inhalant anesthetics, potassium chloride in conjunction with general anesthesia	CO_2, CO, N_2, Ar, penetrating captive bolt, gunshot, kill traps (scientifically tested)

*Acceptable methods are those that consistently produce a humane death when used as the sole means of euthanasia.
†Conditionally acceptable methods are those techniques that by the nature of the technique or because of greater potential for operator error or safety hazards might not consistently produce humane death or are methods not well documented in the scientific literature.

Appendix 2

Some **unacceptable** agents and methods of euthanasia

AGENT OR METHOD	COMMENTS
Air embolism	Air embolism may be accompanied by convulsions, opisthotonos and vocalization. If used, it should be done only in anesthetized animals.
Blow to the head	Unacceptable for most species.
Burning	Chemical or thermal burning of an animal is not an acceptable method of euthanasia.
Chloral hydrate	Unacceptable in dogs, cats, and small mammals.
Chloroform	Chloroform is a known hepatotoxin and suspected carcinogen, and therefore extremely hazardous to personnel.
Cyanide	Cyanide poses an extreme danger to personnel and the manner of death is aesthetically objectionable.
Decompression	Decompression is unacceptable for euthanasia because of numerous disadvantages. (1) Many chambers are designed to produce decompression at a rate 15 to 60 times faster than that recommended as optimum for animals, resulting in pain and distress attributable to expanding gases trapped in body cavities. (2) Immature animals are tolerant of hypoxia, and longer periods of decompression are required before respiration ceases. (3) Accidental recompression, with recovery of injured animals can occur. (4) Bloating, bleeding, vomiting, convulsions, urination, and defecation, which are aesthetically unpleasant, may develop in unconscious animals.
Drowning	Drowning is not a means of euthanasia and is inhumane.
Exsanguination	Because of the anxiety associated with extreme hypovolemia, exsanguination should be done only in sedated, stunned, or anesthetized animals.
Formalin	Direct immersion of an animal into formalin, as a means of euthanasia, is inhumane.
Household Products and Solvents	Acetone, quaternary compounds (including CCl_4), laxatives, clove oil, dimethylketone, quaternary ammonium products,[a] acids, and other commercial and household products or solvents are not acceptable agents for euthanasia.
Hypothermia	Hypothermia is not an appropriate method of euthanasia.
Neuromuscular blocking agents (nicotine, magnesium sulfate, potassium chloride, all curariform agents)	When used alone, these drugs all cause respiratory arrest before unconsciousness, so the animal may perceive pain and distress after it is immobilized.
Rapid freezing	Rapid freezing as a sole means of euthanasia is not considered to be humane. If used, animals should be anesthetized prior to freezing.
Strychnine	Strychnine causes violent convulsions and painful muscle contractions.
Stunning	Stunning may render an animal unconscious, but it is not a method of euthanasia (except for neonatal animals with thin craniums). If used, it must be immediately followed by a method that ensures death.
Tricaine methane sulfonate (TMS, MS 222)	Should not be used for euthanasia of animals intended as food.
*Roccal D Plus, Pharmacia & Upjohn, Kalamazoo, Michigan	

2000 Report of the AVMA Panel on Euthanasia

2000
Report of the
AVMA Panel on Euthanasia

Members of the AVMA Panel

Bonnie V. Beaver, DVM, MS, DACVB, (Chair) Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, Texas A&M University, 4474 TAMU, College Station, TX 77843-4474, representing the AVMA Executive Board.

Willie Reed, DVM, PhD, DACVP, DACPV, Animal Health Diagnostic Laboratory, College of Veterinary Medicine, Michigan State University, B646 W. Fee Hall-AHDL, East Lansing, MI 48824-1316, representing the AVMA Council on Research.

Steven Leary, DVM, DACLAM, Division of Comparative Medicine, Washington University, Box 8061, St Louis, MO 63110, representing the AVMA Animal Welfare Committee.

Brendan McKiernan, DVM, DACVIM, Denver Veterinary Specialists, 3695 Kipling St, Wheat Ridge, CO 80033, representing the American Animal Hospital Association.

Fairfield Bain, DVM, DACVIM, DACVP, DACVECC, Hagyard-Davidson-McGee Associates PSC, 4250 Iron Works Pike, Lexington, KY 40511-8412, representing the American Association of Equine Practitioners.

Roy Schultz, DVM, MS, DABVP, 1114 N Frost Ave, Avoca, IA 51521, representing the American Board of Veterinary Practitioners.

B. Taylor Bennett, DVM, PhD, DACLAM, Biologic Resources Laboratory (MC533), University of Illinois at Chicago, 1840 W Taylor St, Chicago, IL 60612-7348, representing the American College of Laboratory Animal Medicine.

Peter Pascoe, BVSc, DVA, DACVA, DECVA, Department of Surgical and Radiological Sciences, School of Veterinary Medicine, University of California, Davis, CA 95616-8745, representing the American College of Veterinary Anesthesiologists.

Elizabeth Shull, DVM, DACVB, DACVIM (Neurology), Veterinary Specialty Consultation Services, 1505 Bob Kirby Rd, Knoxville, TN 37931, representing the American College of Veterinary Behaviorists.

Linda C. Cork, DVM, PhD, DACVP, Department of Comparative Medicine, School of Medicine, Stanford University, MSOB Building, Room X347, Stanford, CA 94305-5415, representing the American College of Veterinary Pathologists.

Ruth Francis-Floyd, DVM, MS, DACZM, Department of Large Animal Clinical Sciences, College f Veterinary Medicine, University of Florida, Box 100136, Gainesville, FL 32510-0136, representing the International Association of Aquatic Animal Medicine.

Keith D. Amass, DVM, Safe-Capture International Inc, PO Box 206, Mount Horeb, WI 53572, representing wildlife regulatory/conservation agencies.

Richard Johnson, PhD, Department of Physiological Sciences, College of Veterinary Medicine, University of Florida, Box 100144, Gainesville, FL 32610-0144, representing the Society for Neuroscience.

Robert H. Schmidt, MS, PhD, Department of Fisheries and Wildlife, Utah State University, Logan UT 84322-5210, representing the wildlife damage management profession.

Wendy Underwood, DVM, MS, DACVIM, Lilly Corporate Center, Eli Lilly and Co, Indianapolis, IN 46285, representing the National Institute for Animal Agriculture Euthanasia Task Force.

Gus W. Thornton, DVM, DACVIM, Massachusetts Society for the Prevention of Cruelty to Animals (MSPCA), American Humane Education Society (AHES), 350 S Huntington Ave, Boston, MA 02130, representing an animal protection agency.

Barbara Kohn, DVM, USDA/APHIS/Animal Care, 4700 River Road, Unit 84, Riverdale, MD 20737-1234, representing the USDA/APHIS.

PREFACE

At the request of the AVMA Council on Research, the Executive Board of the AVMA convened a Panel on Euthanasia in 1999 to review and make necessary revisions to the fifth Panel Report, published in 1993.[1] In this newest version of the report, the panel has updated information on euthanasia of animals in research and animal care and control facilities; expanded information on ectothermic, aquatic, and fur-bearing animals; added information on horses and wildlife; and deleted methods or agents considered unacceptable. Because the panel's deliberations were based on currently available scientific information, some euthanasia methods and agents are not discussed.

Welfare issues are increasingly being identified in the management of free-ranging wildlife, and the need for humane euthanasia guidelines in this context is great. Collection of animals for scientific investigations, euthanasia of injured or diseased wildlife species, removal of animals causing damage to property or threatening human safety, and euthanasia of animals in excess population are drawing more public attention. These issues are acknowledged in this report and special considerations are described for handling animals under free-ranging conditions, where their needs are far different from those of their domestic counterparts.

This report is intended for use by members of the

veterinary profession who carry out or oversee the euthanasia of animals. Although the report may be interpreted and understood by a broad segment of the general population, a veterinarian should be consulted in the application of these recommendations. The practice of veterinary medicine is complex and involves diverse animal species. Whenever possible, a veterinarian experienced with the species in question should be consulted when selecting the method of euthanasia, particularly when little species-specific euthanasia research has been done. Although interpretation and use of this report cannot be limited, the panel's overriding commitment is to give veterinarians guidance in relieving pain and suffering of animals that are to be euthanatized. The recommendations in this report are intended to serve as guidelines for veterinarians who must then use professional judgment in applying them to the various settings where animals are to be euthanatized.

INTRODUCTION

The term euthanasia is derived from the Greek terms *eu* meaning good and *thanatos* meaning death.[2] A "good death" would be one that occurs with minimal pain and distress. In the context of this report, euthanasia is the act of inducing humane death in an animal. It is our responsibility as veterinarians and human beings to ensure that if an animal's life is to be taken, it is done with the highest degree of respect, and with an emphasis on making the death as painless and distress free as possible. Euthanasia techniques should result in rapid loss of consciousness followed by cardiac or respiratory arrest and the ultimate loss of brain function. In addition, the technique should minimize distress and anxiety experienced by the animal prior to loss of consciousness. The panel recognized that the absence of pain and distress cannot always be achieved. This report attempts to balance the ideal of minimal pain and distress with the reality of the many environments in which euthanasia is performed. A veterinarian with appropriate training and expertise for the species involved should be consulted to ensure that proper procedures are used.

Criteria for painless death can be established only after the mechanisms of pain are understood. Pain is that sensation (perception) that results from nerve impulses reaching the cerebral cortex via ascending neural pathways. Under normal circumstances, these pathways are relatively specific, but the nervous system is sufficiently plastic that activation of nociceptive pathways does not always result in pain and stimulation of other (non-nociceptive) peripheral and central neurons can give rise to pain. The term nociceptive is derived from the word *noci* meaning to injure and *ceptive* meaning to receive, and is used to describe neuronal input caused by noxious stimuli, which threaten to, or actually do, destroy tissue. These noxious stimuli initiate nerve impulses by acting at primary nociceptors and other sensory nerve endings that respond to noxious and non-noxious stimuli from mechanical, thermal, or chemical activity. Endogenous chemical substances such as hydrogen ions, potassium ions, ATP, serotonin, histamine, bradykinin, and prostaglandins, as well as electrical currents, are capable of generating nerve impulses in nociceptor nerve fibers. Activity in nociceptive pathways can also be triggered in normally silent receptors that become sensitized by chronic pain conditions.[3,4]

Nerve impulse activity generated by nociceptors is conducted via nociceptor primary afferent fibers to the spinal cord or the brainstem where it is transmitted to two general sets of neural networks. One set is related to nociceptive reflexes (eg, withdrawal and flexion reflexes) that are mediated at the spinal level, and the second set consists of ascending pathways to the reticular formation, hypothalamus, thalamus, and cerebral cortex (somatosensory cortex and limbic system) for sensory processing. It is important to understand that ascending nociceptive pathways are numerous, often redundant, and are capable of considerable plasticity under chronic conditions (pathology or injury). Moreover, even the transmission of nociceptive neural activity in a given pathway is highly variable. Under certain conditions, both the nociceptive reflexes and the ascending pathways may be suppressed, as, for example, in epidural anesthesia. Under another set of conditions, nociceptive reflex actions may occur, but activity in the ascending pathways is suppressed; thus, noxious stimuli are not perceived as pain. It is incorrect to use the term pain for stimuli, receptors, reflexes, or pathways because the term implies perception, whereas all the above may be active without consequential pain perception.[5,6]

Pain is divided into two broad categories: (1) sensory-discriminative, which indicates the site of origin and the stimulus giving rise to the pain; and (2) motivational-affective in which the severity of the stimulus is perceived and the animal's response is determined. Sensory-discriminative processing of nociceptive impulses is most likely to be accomplished by subcortical and cortical mechanisms similar to those used for processing other sensory-discriminative input that provides the individual with information about the intensity, duration, location, and quality of the stimulus. Motivational-affective processing involves the ascending reticular formation for behavioral and cortical arousal. It also involves thalamic input to the forebrain and the limbic system for perceptions such as discomfort, fear, anxiety, and depression. The motivational-affective neural networks also have strong inputs to the limbic system, hypothalamus and the autonomic nervous system for reflex activation of the cardiovascular, pulmonary, and pituitary-adrenal systems. Responses activated by these systems feed back to the forebrain and enhance perceptions derived via motivational-affective inputs. On the basis of neurosurgical experience in humans, it is possible to separate the sensory-discriminative components from the motivational-affective components of pain.[7]

For pain to be experienced, the cerebral cortex and subcortical structures must be functional. If the cerebral cortex is nonfunctional because of hypoxia, depression by drugs, electric shock, or concussion, pain is not experienced. Therefore, the choice of the euthanasia agent or method is less critical if it is to be used on an animal that is anesthetized or unconscious, provided that the animal does not regain consciousness prior to death.

An understanding of the continuum that represents stress and distress is essential for evaluating techniques that minimize any distress experienced by an animal being euthanatized. Stress has been defined as the effect of physical, physiologic, or emotional factors (stressors) that induce an alteration in an animal's homeostasis or adaptive state.[8] The response of an animal to stress represents the adaptive process that is necessary to restore the baseline mental and physiologic state. These responses may involve changes in an animal's neuroendocrinologic system, autonomic nervous system, and mental status that may result in overt behavioral changes. An animal's response varies according to its experience, age, species, breed, and current physiologic and psychologic state.[9]

Stress and the resulting responses have been divided into three phases.[10] Eustress results when harmless stimuli initiate adaptive responses that are beneficial to the animal. Neutral stress results when the animal's response to stimuli causes neither harmful nor beneficial effects to the animal. Distress results when an animal's response to stimuli interferes with its well-being and comfort.[11]

As with many other procedures involving animals, some methods of euthanasia require physical handling of the animal. The amount of control and kind of restraint required will be determined by the animal's species, breed, size, state of domestication, degree of taming, presence of painful injury or disease, degree of excitement, and method of euthanasia. Proper handling is vital to minimize pain and distress in animals, to ensure safety of the person performing euthanasia, and, often, to protect other people and animals.

An in-depth discussion of euthanasia procedures is beyond the scope of this report; however, personnel who perform euthanasia must have appropriate certification and training, experience with the techniques to be used, and experience in the humane restraint of the species of animal to be euthanatized, to ensure that animal pain and distress are minimized during euthanasia. Training and experience should include familiarity with the normal behavior of the species being euthanatized, an appreciation of how handling and restraint affects that behavior, and an understanding of the mechanism by which the selected technique induces loss of consciousness and death. Prior to being assigned full responsibility for performing euthanasia, all personnel must have demonstrated proficiency in the use of the technique in a closely supervised environment. References provided at the end of this document may be useful for training personnel.[12-21]

Selection of the most appropriate method of euthanasia in any given situation depends on the species of animal involved, available means of animal restraint, skill of personnel, number of animals, and other considerations. Available information focuses primarily on domestic animals, but the same general considerations should be applied to all species.

This report includes four appendices that summarize information from the text. Appendix 1 lists acceptable and conditionally acceptable methods of euthanasia, categorized by species. Appendices 2 and 3 provide summaries of characteristics for acceptable and conditionally acceptable methods of euthanasia. Appendix 4 provides a summary of some unacceptable euthanasia agents and methods. Criteria used for acceptable, conditionally acceptable, and unacceptable methods are as follows: acceptable methods are those that consistently produce a humane death when used as the sole means of euthanasia; conditionally acceptable methods are those techniques that by the nature of the technique or because of greater potential for operator error or safety hazards might not consistently produce humane death or are methods not well documented in the scientific literature; and unacceptable techniques are those methods deemed inhumane under any conditions or that the panel found posed a substantial risk to the human applying the technique. The report also includes discussion of several adjunctive methods, which are those methods that cannot be used as the sole method of euthanasia, but that can be used in conjunction with other methods to produce a humane death.

GENERAL CONSIDERATIONS

In evaluating methods of euthanasia, the panel used the following criteria: (1) ability to induce loss of consciousness and death without causing pain, distress, anxiety, or apprehension; (2) time required to induce loss of consciousness; (3) reliability; (4) safety of personnel; (5) irreversibility; (6) compatibility with requirement and purpose; (7) emotional effect on observers or operators; (8) compatibility with subsequent evaluation, examination, or use of tissue; (9) drug availability and human abuse potential; (10) compatibility with species, age, and health status; (11) ability to maintain equipment in proper working order; and (12) safety for predators/scavengers should the carcass be consumed.

The panel discussed the definition of euthanasia used in this report as it applies to circumstances when the degree of control over the animal makes it difficult to ensure death without pain and distress. Slaughter of animals for food, fur, or fiber may represent such situations. However, the same standards for euthanasia should be applied to the killing of animals for food, fur, or fiber, and wildlife or feral animals. Animals intended for food should be slaughtered humanely, taking into account any special requirements of the US Department of Agriculture.[22] Painless death can be achieved by properly stunning the animal, followed immediately by exsanguination. Handling of animals prior to slaughter should be as stress free as possible. Electric prods or other devices should not be used to encourage movement of animals and are not needed if chutes and ramps are properly designed to enable animals to be moved and restrained without undue stress.[23-27] Animals must not be restrained in a painful position before slaughter.

Ethical considerations that must be addressed when euthanatizing healthy and unwanted animals reflect professional and societal concerns.[28,29] These issues are complex and warrant thorough consideration by the profession and all those concerned with the welfare of animals. Whereas the panel recognizes the need for those responsible for the euthanasia of ani-

mals to be cognizant of these issues, it does not believe that this report is the appropriate forum for an in-depth discussion of this topic.

It is the intent of the panel that euthanasia be performed in accordance with applicable federal, state, and local laws governing drug acquisition and storage, occupational safety, and methods used for euthanasia and disposal of animals. However, space does not permit a review of current federal, state, and local regulations.

The panel is aware that circumstances may arise that are not clearly covered by this report. Whenever such situations arise, a veterinarian experienced with the species should use professional judgment and knowledge of clinically acceptable techniques in selecting an appropriate euthanasia technique. Professional judgment in these circumstances will take into consideration the animal's size and its species-specific physiologic and behavioral characteristics. In all circumstances, the euthanasia method should be selected and used with the highest ethical standards and social conscience.

It is imperative that death be verified after euthanasia and before disposal of the animal. An animal in deep narcosis following administration of an injectable or inhalant agent may appear dead, but might eventually recover. Death must be confirmed by examining the animal for cessation of vital signs, and consideration given to the animal species and method of euthanasia when determining the criteria for confirming death.

ANIMAL BEHAVIORAL CONSIDERATIONS

The need to minimize animal distress, including fear, anxiety, and apprehension, must be considered in determining the method of euthanasia. Gentle restraint (preferably in a familiar and safe environment), careful handling, and talking during euthanasia often have a calming effect on animals that are used to being handled. Sedation and/or anesthesia may assist in achieving the best conditions for euthanasia. It must be recognized that any sedatives or anesthetics given at this stage that change circulation may delay the onset of the euthanasia agent. Preparation of observers should also be taken into consideration.

Animals that are wild, feral, injured, or already distressed from disease pose another challenge. Methods of pre-euthanasia handling suitable for domestic animals may not be effective for them. Because handling may stress animals unaccustomed to human contact (eg, wildlife, zoo, and feral species), the degree of restraint required to perform any euthanasia procedure should be considered when evaluating various methods. When handling these animals, calming may be accomplished by minimizing visual, auditory, and tactile stimulation. When struggling during capture or restraint may cause pain, injury, or anxiety to the animal or danger to the operator, the use of tranquilizers, analgesics, and/or anesthetics may be necessary. A route of injection should be chosen that causes the least distress in the animal for which euthanasia must be performed. Various techniques for oral delivery of sedatives to dogs and cats have been described that may be useful under these circumstances.[30,31]

Facial expressions and body postures that indicate various emotional states of animals have been described for some species.[32-37] Behavioral and physiologic responses to noxious stimuli include distress vocalization, struggling, attempts to escape, defensive or redirected aggression, salivation, urination, defecation, evacuation of anal sacs, pupillary dilatation, tachycardia, sweating, and reflex skeletal muscle contractions causing shivering, tremors, or other muscular spasms. Unconscious as well as conscious animals are capable of some of these responses. Fear can cause immobility or "playing dead" in certain species, particularly rabbits and chickens. This immobility response should not be interpreted as loss of consciousness when the animal is, in fact, conscious. Distress vocalizations, fearful behavior, and release of certain odors or pheromones by a frightened animal may cause anxiety and apprehension in other animals. Therefore, for sensitive species, it is desirable that other animals not be present when individual animal euthanasia is performed.

HUMAN BEHAVIORAL CONSIDERATIONS

When animals must be euthanatized, either as individuals or in larger groups, moral and ethical concerns dictate that humane practices be observed. Human psychologic responses to euthanasia of animals need to be considered, with grief at the loss of a life as the most common reaction.[38] There are six circumstances under which we are most aware of the effects of animal euthanasia on people.

The first of these is the veterinary clinical setting where owners have to make decisions about whether and when to euthanatize. Although many owners rely heavily on their veterinarian's judgment, others may have misgivings about making their own decision. This is particularly likely if an owner feels responsible for allowing an animal's medical or behavioral problem to go unattended so that euthanasia becomes necessary. When owners choose to be present during euthanasia, they should be prepared for what will happen. What drugs are being used and how the animal could respond should be discussed. Behaviors such as vocalization, muscle twitches, failure of the eyelids to close, urination, or defecation can be distressing. Counseling services for grieving owners are now available in some communities[39] and telephone counseling is available through some veterinary schools.[40,41] Owners are not the only people affected by euthanasia of animals. Veterinarians and their staffs may also become attached to patients they have known and treated for many years and may continue to struggle with the ethical implications of ending an animal's life.

The second is animal care and control facilities where unwanted, homeless, diseased, and injured animals must be euthanatized in large numbers. Distress may develop among personnel directly involved in performing euthanasia repeatedly. Emotional uneasiness, discomfort, or distress experienced by people involved with euthanasia of animals may be minimized. The person performing euthanasia must be technically proficient, use humane handling methods, understand the reasons for euthanasia, and be familiar with the

method of euthanasia being employed (ie, what is going to happen to the animal). When the person is not knowledgeable about what to expect, he or she may mistakenly interpret any movement of animals as consciousness and a lack of movement as loss of consciousness. Methods that preclude movement of animals are more aesthetically acceptable to most technical staff even though lack of movement is not an adequate criterion for evaluating euthanasia techniques. Constant exposure to, or participation in, euthanasia procedures can cause a psychologic state characterized by a strong sense of work dissatisfaction or alienation, which may be expressed by absenteeism, belligerence, or careless and callous handling of animals.[42] This is one of the principal reasons for turnover of employees directly involved with repeated animal euthanasia. Management should be aware of potential personnel problems related to animal euthanasia and determine whether it is necessary to institute a program to prevent, decrease, or eliminate this problem. Specific coping strategies can make the task more tolerable. Some strategies include adequate training programs so that euthanasia is performed competently, peer support in the workplace, professional support as necessary, focusing on animals that are successfully adopted or returned to owners, devoting some work time to educational activities, and providing time off when workers feel stressed.

The third setting is the laboratory. Researchers, technicians, and students may become attached to animals that must be euthanatized.[43] The same considerations afforded pet owners or shelter employees should be provided to those working in laboratories.

The fourth situation is wildlife control. Wildlife biologists, wildlife managers, and wildlife health professionals are often responsible for euthanatizing animals that are injured, diseased, in excessive number, or that threaten property or human safety. Although relocation of some animals is appropriate and attempted, relocation is often only a temporary solution to a larger problem. People who must deal with these animals, especially under public pressure to save the animals rather than destroy them, can experience extreme distress and anxiety.

The fifth setting is livestock and poultry slaughter facilities. The large number of animals processed daily can take a heavy toll on employees physically and emotionally. Federal and state agricultural employees may also be involved in mass euthanasia of poultry and livestock in the face of disease outbreaks, bioterrorism, and natural disasters.

The last situation is public exposure. Because euthanasia of zoo animals, animals involved in roadside or racetrack accidents, stranded marine animals, nuisance or injured wildlife, and others can draw public attention, human attitudes and responses should be considered whenever animals are euthanatized. Natural disasters and foreign animal disease programs also present public challenges. These considerations, however, should not outweigh the primary responsibility of using the most rapid and painless euthanasia method possible under the circumstances.

MODES OF ACTION OF EUTHANATIZING AGENTS

Euthanatizing agents cause death by three basic mechanisms: (1) hypoxia, direct or indirect; (2) direct depression of neurons necessary for life function; and (3) physical disruption of brain activity and destruction of neurons necessary for life.

Agents that induce death by direct or indirect hypoxia can act at various sites and can cause loss of consciousness at different rates. For death to be painless and distress-free, loss of consciousness should precede loss of motor activity (muscle movement). Loss of motor activity, however, cannot be equated with loss of consciousness and absence of distress. Thus, agents that induce muscle paralysis without loss of consciousness are not acceptable as sole agents for euthanasia (eg, depolarizing and nondepolarizing muscle relaxants, strychnine, nicotine, and magnesium salts). With other techniques that induce hypoxia, some animals may have motor activity following loss of consciousness, but this is reflex activity and is not perceived by the animal.

A second group of euthanatizing agents depress nerve cells of the brain, inducing loss of consciousness followed by death. Some of these agents release inhibition of motor activity during the first stage of anesthesia, resulting in a so-called excitement or delirium phase, during which there may be vocalization and some muscle contraction. These responses do not appear to be purposeful. Death follows loss of consciousness, and is attributable to cardiac arrest and/or hypoxemia following direct depression of respiratory centers.

Physical disruption of brain activity, caused by concussion, direct destruction of the brain, or electrical depolarization of neurons, induces rapid loss of consciousness. Death occurs because of destruction of midbrain centers controlling cardiac and respiratory activity or as a result of adjunctive methods (eg, exsanguination) used to kill the animal. Exaggerated muscular activity can follow loss of consciousness and, although this may disturb some observers, the animal is not experiencing pain or distress.

INHALANT AGENTS

Any gas that is inhaled must reach a certain concentration in the alveoli before it can be effective; therefore, euthanasia with any of these agents takes some time. The suitability of a particular agent depends on whether an animal experiences distress between the time it begins to inhale the agent and the time it loses consciousness. Some agents may induce convulsions, but these generally follow loss of consciousness. Agents inducing convulsions prior to loss of consciousness are unacceptable for euthanasia.

Certain considerations are common to all inhalant agents. (1) In most cases, onset of loss of consciousness is more rapid, and euthanasia more humane, if the animal is rapidly exposed to a high concentration of the agent. (2) The equipment used to deliver and maintain this high concentration must be in good working order and in compliance with state and federal regulations. Leaky or faulty equipment may lead to

slow, distressful death and be hazardous to other animals and to personnel. (3) Most of these agents are hazardous to personnel because of the risk of explosions (eg, ether), narcosis (eg, halothane), hypoxemia (eg, nitrogen and carbon monoxide), addiction (eg, nitrous oxide), or health effects resulting from chronic exposure (eg, nitrous oxide and carbon monoxide). (4) Alveolar concentrations rise slowly in an animal with decreased ventilation, making agitation more likely during induction. Other noninhalant methods of euthanasia should be considered for such animals. (5) Neonatal animals appear to be resistant to hypoxia, and because all inhalant agents ultimately cause hypoxia, neonatal animals take longer to die than adults. Glass et al,[44] reported that newborn dogs, rabbits, and guinea pigs survived a nitrogen atmosphere much longer than did adults. Dogs, at 1 week old, survived for 14 minutes compared with a 3-minute survival time after a few weeks of age. Guinea pigs survived for 4.5 minutes at 1 day old, compared with 3 minutes at 8 days or older. Rabbits survived for 13 minutes at 6 days old, 4 minutes at 14 days, and 1.5 minutes at 19 days and older. The panel recommends that inhalant agents not be used alone in animals less than 16 weeks old except to induce loss of consciousness, followed by the use of some other method to kill the animal. (6) Rapid gas flows can produce a noise that frightens animals. If high flows are required, the equipment should be designed to minimize noise. (7) Animals placed together in chambers should be of the same species, and, if needed, should be restrained so that they will not hurt themselves or others. Chambers should not be overloaded and need to be kept clean to minimize odors that might distress animals subsequently euthanatized. (8) Reptiles, amphibians, and diving birds and mammals have a great capacity for holding their breath and anaerobic metabolism. Therefore, induction of anesthesia and time to loss of consciousness when using inhalants may be greatly prolonged. Other techniques may be more appropriate for these species.

Inhalant anesthetics

Inhalant anesthetics (eg, ether, halothane, methoxyflurane, isoflurane, sevoflurane, desflurane, and enflurane) have been used to euthanatize many species.[45] Halothane induces anesthesia rapidly and is the most effective inhalant anesthetic for euthanasia. Enflurane is less soluble in blood than halothane, but, because of its lower vapor pressure and lower potency, induction rates may be similar to those for halothane. At deep anesthetic planes, animals may seizure. It is an effective agent for euthanasia, but the associated seizure activity may be disturbing to personnel. Isoflurane is less soluble than halothane, and it should induce anesthesia more rapidly. However, it has a slightly pungent odor and animals often hold their breath, delaying onset of loss of consciousness. Isoflurane also may require more drug to kill an animal, compared with halothane. Although isoflurane is acceptable as a euthanasia agent, halothane is preferred. Sevoflurane is less soluble than halothane and does not have an objectionable odor. It is less potent than isoflurane or halothane and has a lower vapor pressure. Anesthetic concentrations can be achieved and maintained rapidly. Desflurane is currently the least soluble potent inhalant anesthetic, but the vapor is quite pungent, which may slow induction. This drug is so volatile that it could displace oxygen (O_2) and induce hypoxemia during induction if supplemental O_2 is not provided. Methoxyflurane is highly soluble, and slow anesthetic induction with its use may be accompanied by agitation. It is a conditionally acceptable agent for euthanasia in rodents.[46] Ether has high solubility in blood and induces anesthesia slowly. It is irritating to the eyes and nose, poses serious risks associated with its flammability and explosiveness, and has been used to create a model for stress.[47-50]

With inhalant anesthetics, the animal can be placed in a closed receptacle containing cotton or gauze soaked with an appropriate amount of the anesthetic,[51] or the anesthetic can be introduced from a vaporizer. The latter method may be associated with a longer induction time. Vapors are inhaled until respiration ceases and death ensues. Because the liquid state of most inhalant anesthetics is irritating, animals should be exposed only to vapors. Also, sufficient air or O_2 must be provided during the induction period to prevent hypoxemia.[51] In the case of small rodents placed in a large container, there will be sufficient O_2 in the chamber to prevent hypoxemia. Larger species placed in small containers may need supplemental air or O_2.[51]

Nitrous oxide (N_2O) may be used with other inhalants to speed the onset of anesthesia, but alone it does not induce anesthesia in animals, even at 100% concentration. When used by itself, N_2O produces hypoxemia before respiratory or cardiac arrest. As a result, animals may become distressed prior to loss of consciousness.

Occupational exposure to inhalant anesthetics constitutes a human health hazard. Spontaneous abortion and congenital abnormalities have been associated with exposure of women to trace amounts of inhalation anesthetic agents during early stages of pregnancy.[52] Regarding human exposure to inhalant anesthetics, the concentrations of halothane, enflurane, and isoflurane should be less than 2 ppm, and less than 25 ppm for nitrous oxide.[52] There are no controlled studies proving that such concentrations of anesthetics are safe, but these concentrations were established because they were found to be attainable under hospital conditions. Effective procedures must be used to protect personnel from anesthetic vapors.

Advantages—(1) Inhalant anesthetics are particularly valuable for euthanasia of smaller animals (< 7 kg) or for animals in which venipuncture may be difficult. (2) Halothane, enflurane, isoflurane, sevoflurane, desflurane, methoxyflurane, and N_2O are nonflammable and nonexplosive under ordinary environmental conditions.

Disadvantages—(1) Animals may struggle and become anxious during induction of anesthesia because anesthetic vapors may be irritating and can induce excitement. (2) Ether is flammable and explo-

sive. Explosions have occurred when animals, euthanatized with ether, were placed in an ordinary (not explosion proof) refrigerator or freezer and when bagged animals were placed in an incinerator. (3) Induction with methoxyflurane is unacceptably slow in some species. (4) Nitrous oxide will support combustion. (5) Personnel and animals can be injured by exposure to these agents. (6) There is a potential for human abuse of some of these drugs, especially N_2O.

Recommendations—In order of preference, halothane, enflurane, isoflurane, sevoflurane, methoxyflurane, and desflurane, with or without nitrous oxide, are acceptable for euthanasia of small animals (< 7 kg). Ether should only be used in carefully controlled situations in compliance with state and federal occupational health and safety regulations. It is conditionally acceptable. Nitrous oxide should not be used alone, pending further scientific studies on its suitability for animal euthanasia. Although acceptable, these agents are generally not used in larger animals because of their cost and difficulty of administration.

Carbon dioxide

Room air contains 0.04% carbon dioxide (CO_2), which is heavier than air and nearly odorless. Inhalation of CO_2 at a concentration of 7.5% increases the pain threshold, and higher concentrations of CO_2 have a rapid anesthetic effect.[53-58]

Leake and Waters[56] reported the experimental use of CO_2 as an anesthetic agent for dogs. At concentrations of 30% to 40% CO_2 in O_2, anesthesia was induced within 1 to 2 minutes, usually without struggling, retching, or vomiting. For cats, inhalation of 60% CO_2 results in loss of consciousness within 45 seconds, and respiratory arrest within 5 minutes.[59] Signs of effective CO_2 anesthesia are those associated with deep surgical anesthesia, such as loss of withdrawal and palpebral reflexes.[60] Time to loss of consciousness is decreased by use of higher concentrations of CO_2 with an 80 to 100% concentration providing anesthesia in 12 to 33 seconds in rats and 70% CO_2 in O_2 inducing anesthesia in 40 to 50 seconds.[61,62] Time to loss of consciousness will be longer if the concentration is increased slowly rather than immersing the animal in the full concentration immediately.

Several investigators have suggested that inhalation of high concentrations of CO_2 may be distressing to animals,[63-66] because the gas dissolves in moisture on the nasal mucosa. The resulting product, carbonic acid, may stimulate nociceptors in the nasal mucosa. Some humans exposed to concentrations of around 50% CO_2 report that inhaling the gas is unpleasant and that higher concentrations are noxious.[67,68] A brief study of swine examined the aversive nature of CO_2 exposure[69] and found that 90% CO_2 was aversive to pigs while 30% was not. For rats, exposure to increasing concentrations of CO_2 (33% achieved after 1 minute) in their home cage produced no evident stress as measured by behavior and ACTH, glucose, and corticosterone concentrations in serum.[70]

Carbon dioxide has been used to euthanatize groups of small laboratory animals, including mice, rats, guinea pigs, chickens, and rabbits,[5,71-76] and to render swine unconscious before humane slaughter.[22,63, 64] The combination of 40% CO_2 and approximately 3% CO has been used experimentally for euthanasia of dogs.[65] Carbon dioxide has been used in specially designed chambers to euthanatize individual cats[77,78] and other small laboratory animals.[51,72,79]

Studies of 1-day-old chickens have revealed that CO_2 is an effective euthanatizing agent. Inhalation of CO_2 caused little distress to the birds, suppressed nervous activity, and induced death within 5 minutes.[73] Because respiration begins during embryonic development, the unhatched chicken's environment may normally have a CO_2 concentration as high as 14%. Thus, CO_2 concentrations for euthanasia of newly hatched chickens and neonates of other species should be especially high. A CO_2 concentration of 60% to 70% with a 5-minute exposure time appears to be optimal.[73]

In studies of mink, high concentrations of CO_2 would kill them quickly, but a 70% CO_2 concentration induced loss of consciousness without killing them.[80] Some burrowing animals, such as rabbits of the species *Oryctolagus,* also have prolonged survival times when exposed to CO_2.[81] Some burrowing and diving animals have physiologic mechanisms for coping with hypercapnia. Therefore, it is necessary to have a sufficient concentration of CO_2 to kill the animal by hypoxemia following induction of anesthesia with CO_2.

Advantages—(1) The rapid depressant, analgesic, and anesthetic effects of CO_2 are well established. (2) Carbon dioxide is readily available and can be purchased in compressed gas cylinders. (3) Carbon dioxide is inexpensive, nonflammable, nonexplosive, and poses minimal hazard to personnel when used with properly designed equipment. (4) Carbon dioxide does not result in accumulation of tissue residues in food-producing animals. (5) Carbon dioxide euthanasia does not distort murine cholinergic markers[82] or corticosterone concentrations.[83]

Disadvantages—(1) Because CO_2 is heavier than air, incomplete filling of a chamber may permit animals to climb or raise their heads above the higher concentrations and avoid exposure. (2) Some species, such as fish and burrowing and diving mammals, may have extraordinary tolerance for CO_2. (3) Reptiles and amphibians may breathe too slowly for the use of CO_2. (4) Euthanasia by exposure to CO_2 may take longer than euthanasia by other means.[61] (5) Induction of loss of consciousness at lower concentrations (< 80%) may produce pulmonary and upper respiratory tract lesions.[67,84] (6) High concentrations of CO_2 may be distressful to some animals.

Recommendations—Carbon dioxide is acceptable for euthanasia in appropriate species (**Tables 1 and 2**). Compressed CO_2 gas in cylinders is the only recommended source of carbon dioxide because the inflow to the chamber can be regulated precisely. Carbon dioxide generated by other methods such as from dry ice, fire extinguishers, or chemical means (eg, antacids) is unacceptable. Species should be separated and cham-

bers should not be overcrowded. With an animal in the chamber, an optimal flow rate should displace at least 20% of the chamber volume per minute.[85] Loss of consciousness may be induced more rapidly by exposing animals to a CO_2 concentration of 70% or more by prefilling the chamber for species in which this has not been shown to cause distress. Gas flow should be maintained for at least 1 minute after apparent clinical death.[86] It is important to verify that an animal is dead before removing it from the chamber. If an animal is not dead, CO_2 narcosis must be followed with another method of euthanasia. Adding O_2 to the CO_2 may or may not preclude signs of distress.[67,87] Additional O_2 will, however, prolong time to death and may complicate determination of consciousness. There appears to be no advantage to combining O_2 with carbon dioxide for euthanasia.[87]

Nitrogen, argon

Nitrogen (N_2) and argon (Ar) are colorless, odorless gases that are inert, nonflammable, and nonexplosive. Nitrogen comprises 78% of atmospheric air, whereas Ar comprises less than 1%.

Euthanasia is induced by placing the animal in a closed container that has been prefilled with N_2 or Ar or into which the gas is then rapidly introduced. Nitrogen/Ar displaces O_2, thus inducing death by hypoxemia.

In studies by Herin et al,[88] dogs became unconscious within 76 seconds when a N_2 concentration of 98.5% was achieved in 45 to 60 seconds. The electroencephalogram (EEG) became isoelectric (flat) in a mean time of 80 seconds, and arterial blood pressure was undetectable at 204 seconds. Although all dogs hyperventilated prior to loss of consciousness, the investigators concluded that this method induced death without pain. Following loss of consciousness, vocalization, gasping, convulsions, and muscular tremors developed in some dogs. At the end of a 5-minute exposure period, all dogs were dead.[88] These findings were similar to those for rabbits[89] and mink.[80,90]

With N_2 flowing at a rate of 39% of chamber volume per minute, rats collapsed in approximately 3 minutes and stopped breathing in 5 to 6 minutes. Regardless of flow rate, signs of panic and distress were evident before the rats collapsed and died.[85] Insensitivity to pain under such circumstances is questionable.[91]

Tranquilization with acepromazine, in conjunction with N_2 euthanasia of dogs, was investigated by Quine et al.[92] Using ECG and EEG recordings, they found these dogs had much longer survival times than dogs not given acepromazine before administration of N_2. In one dog, ECG activity continued for 51 minutes. Quine also addressed distress associated with exposure to N_2 by removing cats and dogs from the chamber following loss of consciousness and allowing them to recover. When these animals were put back into the chamber, they did not appear afraid or apprehensive.

Investigations into the aversiveness of Ar to swine and poultry have revealed that these animals will tolerate breathing 90% Ar with 2% O_2.[69,71] Swine voluntarily entered a chamber containing this mixture, for a food reward, and only withdrew from the chamber as they became ataxic. They reentered the chamber immediately to continue eating. Poultry also entered a chamber containing this mixture for a food reward and continued eating until they collapsed.[71] When Ar was used to euthanatize chickens, exposure to a chamber prefilled with Ar, with an O_2 concentration of < 2%, led to EEG changes and collapse in 9 to 12 seconds. Birds removed from the chamber at 15 to 17 seconds failed to respond to comb pinching. Continued exposure led to convulsions at 20 to 24 seconds. Somatosensory-evoked potentials were lost at 24 to 34 seconds, and the EEG became isoelectric at 57 to 66 seconds. Convulsion onset was after loss of consciousness (collapse and loss of response to comb pinch), so this would appear to be a humane method of euthanasia for chickens.[93] Despite the availability of some information, there is still much about the use of N_2/Ar that needs to be investigated.

Advantages—(1) Nitrogen and Ar are readily available as compressed gases. (2) Hazards to personnel are minimal.

Disadvantages—(1) Loss of consciousness is preceded by hypoxemia and ventilatory stimulation, which may be distressing to the animal. (2) Reestablishing a low concentration of O_2 (ie, 6% or greater) in the chamber before death will allow immediate recovery.[69]

Recommendations—Nitrogen and Ar can be distressful to some species (eg, rats).[85] Therefore, this technique is conditionally acceptable only if O_2 concentrations < 2% are achieved rapidly, and animals are heavily sedated or anesthetized. With heavy sedation or anesthesia, it should be recognized that death may be delayed. Although N_2 and Ar are effective, other methods of euthanasia are preferable.

Carbon monoxide

Carbon monoxide (CO) is a colorless, odorless gas that is nonflammable and nonexplosive unless concentrations exceed 10%. It combines with hemoglobin to form carboxyhemoglobin and blocks uptake of O_2 by erythrocytes, leading to fatal hypoxemia.

In the past, mass euthanasia has been accomplished by use of 3 methods for generating CO: (1) chemical interaction of sodium formate and sulfuric acid, (2) exhaust fumes from idling gasoline internal combustion engines, and (3) commercially compressed CO in cylinders. The first 2 techniques are associated with problems such as production of other gases, achieving inadequate concentrations of carbon monoxide, inadequate cooling of the gas, and maintenance of equipment. Therefore, the only acceptable source is compressed CO in cylinders.

In a study by Ramsey and Eilmann,[94] 8% CO caused guinea pigs to collapse in 40 seconds to 2 minutes, and death occurred within 6 minutes. Carbon monoxide has been used to euthanatize mink[80,90] and chinchillas. These animals collapsed in 1 minute, breathing ceased in 2 minutes, and the heart stopped beating in 5 to 7 minutes.

In a study evaluating the physiologic and behavioral characteristics of dogs exposed to 6% CO in air, Chalifoux and Dallaire[95] could not determine the precise time of loss of consciousness. Electroencephalographic recordings revealed 20 to 25 seconds of abnormal cortical function prior to loss of consciousness. It was during this period that the dogs became agitated and vocalized. It is not known whether animals experience distress; however, humans in this phase reportedly are not distressed.[96] Subsequent studies have revealed that tranquilization with acepromazine significantly decreases behavioral and physiologic responses of dogs euthanatized with CO.[97]

In a comparative study, CO from gasoline engine exhaust and 70% CO_2 plus 30% O_2 were used to euthanatize cats. Euthanasia was divided into 3 phases. Phase I was the time from initial contact to onset of clinical signs (eg, yawning, staggering, or trembling). Phase II extended from the end of phase I until recumbency, and phase III from the end of phase II until death.[54] The study revealed that signs of agitation before loss of consciousness were greatest with CO_2 plus O_2. Convulsions occurred during phases II and III with both methods. However, when the euthanasia chamber was prefilled with CO (ie, exhaust fumes), convulsions did not occur in phase III. Time to complete immobilization was greater with CO_2 plus O_2 (approximately 90 seconds) than with CO alone (approximately 56 seconds).[54] In neonatal pigs, excitation was more likely to precede loss of consciousness if the pigs were exposed to a rapid rise in CO concentration. This agitation was reduced at lower flow rates, or when CO was combined with nitrogen.[98]

In people, the most common symptoms of early CO toxicosis are headache, dizziness, and weakness. As concentrations of carboxyhemoglobin increase, these signs may be followed by decreased visual acuity, tinnitus, nausea, progressive depression, confusion, and collapse.[99] Because CO stimulates motor centers in the brain, loss of consciousness may be accompanied by convulsions and muscular spasms.

Carbon monoxide is a cumulative poison.[96] Distinct signs of CO toxicosis are not evident until the CO concentration is 0.05% in air, and acute signs do not develop until the CO concentration is approximately 0.2% in air. In humans, exposure to 0.32% CO and 0.45% CO for one hour will induce loss of consciousness and death, respectively.[100] Carbon monoxide is extremely hazardous for personnel because it is highly toxic and difficult to detect. Chronic exposure to low concentrations of carbon monoxide may be a health hazard, especially with regard to cardiovascular disease and teratogenic effects.[101-103] An efficient exhaust or ventilatory system is essential to prevent accidental exposure of humans.

Advantages—(1) Carbon monoxide induces loss of consciousness without pain and with minimal discernible discomfort. (2) Hypoxemia induced by CO is insidious, so that the animal appears to be unaware. (3) Death occurs rapidly if concentrations of 4 to 6% are used.

Disadvantages—(1) Safeguards must be taken to prevent exposure of personnel. (2) Any electrical equipment exposed to CO (eg, lights and fans) must be explosion proof.

Recommendations—Carbon monoxide used for individual animal or mass euthanasia is acceptable for dogs, cats, and other small mammals, provided that commercially compressed CO is used and the following precautions are taken: (1) personnel using CO must be instructed thoroughly in its use and must understand its hazards and limitations; (2) the CO chamber must be of the highest quality construction and should allow for separation of individual animals; (3) the CO source and chamber must be located in a well-ventilated environment, preferably out of doors; (4) the chamber must be well lit and have view ports that allow personnel direct observation of animals; (5) the CO flow rate should be adequate to rapidly achieve a uniform CO concentration of at least 6% after animals are placed in the chamber, although some species (eg, neonatal pigs) are less likely to become agitated with a gradual rise in CO concentration;[98] and (6) if the chamber is inside a room, CO monitors must be placed in the room to warn personnel of hazardous concentrations. It is essential that CO use be in compliance with state and federal occupational health and safety regulations.

NONINHALANT PHARMACEUTICAL AGENTS

The use of injectable euthanasia agents is the most rapid and reliable method of performing euthanasia. It is the most desirable method when it can be performed without causing fear or distress in the animal. When the restraint necessary for giving an animal an intravenous injection would impart added distress to the animal or pose undue risk to the operator, sedation, anesthesia, or an acceptable alternate route of administration should be employed. Aggressive, fearful, wild, or feral animals should be sedated or given a nonparalytic immobilizing agent prior to intravenous administration of the euthanasia agent.

When intravenous administration is considered impractical or impossible, intraperitoneal administration of a nonirritating euthanasia agent is acceptable, provided the drug does not contain neuromuscular blocking agents. Intracardiac injection is acceptable only when performed on heavily sedated, anesthetized, or comatose animals. It is not considered acceptable in awake animals, owing to the difficulty and unpredictability of performing the injection accurately. Intramuscular, subcutaneous, intrathoracic, intrapulmonary, intrahepatic, intrarenal, intrasplenic, intrathecal, and other nonvascular injections are not acceptable methods of administering injectable euthanasia agents.

When injectable euthanasia agents are administered into the peritoneal cavity, animals may be slow to pass through stages I and II of anesthesia. Accordingly, they should be placed in small cages in a quiet area to minimize excitement and trauma.

Barbituric acid derivatives

Barbiturates depress the central nervous system in descending order, beginning with the cerebral cortex,

with loss of consciousness progressing to anesthesia. With an overdose, deep anesthesia progresses to apnea, owing to depression of the respiratory center, which is followed by cardiac arrest.

All barbituric acid derivatives used for anesthesia are acceptable for euthanasia when administered intravenously. There is a rapid onset of action, and loss of consciousness induced by barbiturates results in minimal or transient pain associated with venipuncture. Desirable barbiturates are those that are potent, long-acting, stable in solution, and inexpensive. Sodium pentobarbital best fits these criteria and is most widely used, although others such as secobarbital are also acceptable.

Advantages—(1) A primary advantage of barbiturates is speed of action. This effect depends on the dose, concentration, route, and rate of the injection. (2) Barbiturates induce euthanasia smoothly, with minimal discomfort to the animal. (3) Barbiturates are less expensive than many other euthanasia agents.

Disadvantages—(1) Intravenous injection is necessary for best results and requires trained personnel. (2) Each animal must be restrained. (3) Current federal drug regulations require strict accounting for barbiturates and these must be used under the supervision of personnel registered with the US Drug Enforcement Administration (DEA). (4) An aesthetically objectionable terminal gasp may occur in unconscious animals. (5) These drugs tend to persist in the carcass and may cause sedation or even death of animals that consume the body.

Recommendations—The advantages of using barbiturates for euthanasia in small animals far outweigh the disadvantages. Intravenous injection of a barbituric acid derivative is the preferred method for euthanasia of dogs, cats, other small animals, and horses. Intraperitoneal injection may be used in situations when an intravenous injection would be distressful or even dangerous. Intracardiac injection must only be used if the animal is heavily sedated, unconscious, or anesthetized.

Pentobarbital combinations

Several euthanasia products are formulated to include a barbituric acid derivative (usually sodium pentobarbital), with added local anesthetic agents or agents that metabolize to pentobarbital. Although some of these additives are slowly cardiotoxic, this pharmacologic effect is inconsequential. These combination products are listed by the DEA as Schedule III drugs, making them somewhat simpler to obtain, store, and administer than Schedule II drugs such as sodium pentobarbital. The pharmacologic properties and recommended use of combination products that combine sodium pentobarbital with lidocaine or phenytoin are interchangeable with those of pure barbituric acid derivatives.

A combination of pentobarbital with a neuromuscular blocking agent is not an acceptable euthanasia agent.

Chloral hydrate

Chloral hydrate depresses the cerebrum slowly; therefore, restraint may be a problem for some animals. Death is caused by hypoxemia resulting from progressive depression of the respiratory center, and may be preceded by gasping, muscle spasms, and vocalization.

Recommendations—Chloral hydrate is conditionally acceptable for euthanasia of large animals only when administered intravenously, and only after sedation to decrease the aforementioned undesirable side effects. Chloral hydrate is not acceptable for dogs, cats, and other small animals because the side effects may be severe, reactions can be aesthetically objectionable, and other products are better choices.

T-61

T-61 is an injectable, nonbarbiturate, non-narcotic mixture of 3 drugs used for euthanasia. These drugs provide a combination of general anesthetic, curariform, and local anesthetic actions. T-61 has been withdrawn from the market and is no longer manufactured or commercially available in the United States. It is available in Canada and other countries. T-61 should be used only intravenously and at carefully monitored rates of injection, because there is some question as to the differential absorption and onset of action of the active ingredients when administered by other routes.[1]

Tricaine methane sulfonate (MS 222, TMS)

MS 222 is commercially available as tricaine methane sulfonate (TMS), which can be used for the euthanasia of amphibians and fish. Tricaine is a benzoic acid derivative and, in water of low alkalinity (< 50 mg/L as $CaCo_3$); the solution should be buffered with sodium bicarbonate.[104] A 10 g/L stock solution can be made, and sodium bicarbonate added to saturation, resulting in a pH between 7.0 and 7.5 for the solution. The stock solution should be stored in a dark brown bottle, and refrigerated or frozen if possible. The solution should be replaced monthly and any time a brown color is observed.[105] For euthanasia, a concentration ≥ 250 mg/L is recommended and fish should be left in this solution for at least 10 minutes following cessation of opercular movement.[104] In the United States, there is a 21-day withdrawal time for MS 222; therefore, it is not appropriate for euthanasia of animals intended for food.

Potassium chloride in conjunction with prior general anesthesia

Although unacceptable and condemned when used in unanaesthetized animals, the use of a supersaturated solution of potassium chloride injected intravenously or intracardially in an animal under general anesthesia is an acceptable method to produce cardiac arrest and death. The potassium ion is cardiotoxic, and rapid intravenous or intracardiac administration of 1 to 2 mmol/kg of body weight will cause cardiac arrest. This is a preferred injectable technique for euthanasia of livestock or wildlife species to reduce the risk of toxicosis for predators or scavengers in situations where carcasses of euthanatized animals may be consumed.[106,107]

Advantages—(1) Potassium chloride is not a controlled substance. It is easily acquired, transported, and mixed in the field. (2) Potassium chloride, when used with appropriate methods to render an animal unconscious, results in a carcass that is potentially less toxic for scavengers and predators in cases where carcass disposal is impossible or impractical.

Disadvantage—Rippling of muscle tissue and clonic spasms may occur on or shortly after injection.

Recommendations—It is of utmost importance that personnel performing this technique are trained and knowledgeable in anesthetic techniques, and are competent in assessing anesthetic depth appropriate for administration of potassium chloride intravenously. Administration of potassium chloride intravenously requires animals to be in a surgical plane of anesthesia characterized by loss of consciousness, loss of reflex muscle response, and loss of response to noxious stimuli. Saturated potassium chloride solutions are effective in causing cardiac arrest following rapid intracardiac or intravenous injection. Residual tissue concentrations of general anesthetics after anesthetic induction have not been documented. Whereas no scavenger toxicoses have been reported with potassium chloride in combination with a general anesthetic, proper carcass disposal should always be attempted to prevent possible toxicosis by consumption of a carcass contaminated with general anesthetics.

Unacceptable injectable agents

When used alone, the injectable agents listed in **Appendix 4** (strychnine, nicotine, caffeine, magnesium sulfate, potassium chloride, cleaning agents, solvents, disinfectants and other toxins or salts, and all neuromuscular blocking agents) are unacceptable and are absolutely condemned for use as euthanasia agents.

PHYSICAL METHODS

Physical methods of euthanasia include captive bolt, gunshot, cervical dislocation, decapitation, electrocution, microwave irradiation, kill traps, thoracic compression, exsanguination, stunning, and pithing. When properly used by skilled personnel with well-maintained equipment, physical methods of euthanasia may result in less fear and anxiety and be more rapid, painless, humane, and practical than other forms of euthanasia. Exsanguination, stunning, and pithing are not recommended as a sole means of euthanasia, but should be considered adjuncts to other agents or methods.

Some consider physical methods of euthanasia aesthetically displeasing. There are occasions, however, when what is perceived as aesthetic and what is most humane are in conflict. Physical methods may be the most appropriate method for euthanasia and rapid relief of pain and suffering in certain situations. Personnel performing physical methods of euthanasia must be well trained and monitored for each type of physical technique performed. That person must also be sensitive to the aesthetic implications of the method and inform onlookers about what they should expect when possible.

Since most physical methods involve trauma, there is inherent risk for animals and humans. Extreme care and caution should be used. Skill and experience of personnel is essential. If the method is not performed correctly, animals and personnel may be injured. Inexperienced persons should be trained by experienced persons and should practice on carcasses or anesthetized animals to be euthanatized until they are proficient in performing the method properly and humanely. When done appropriately, the panel considers most physical methods conditionally acceptable for euthanasia.

Penetrating captive bolt

A penetrating captive bolt is used for euthanasia of ruminants, horses, swine, laboratory rabbits, and dogs.[108] Its mode of action is concussion and trauma to the cerebral hemisphere and brainstem.[109,110] Captive bolt guns are powered by gunpowder or compressed air and must provide sufficient energy to penetrate the skull of the species on which they are being used.[109] Adequate restraint is important to ensure proper placement of the captive bolt. A cerebral hemisphere and the brainstem must be sufficiently disrupted by the projectile to induce sudden loss of consciousness and subsequent death. Accurate placement of captive bolts for various species has been described.[109-112] A multiple projectile has been suggested as a more effective technique, especially for large cattle.[109]

A nonpenetrating captive bolt only stuns animals and should not be used as a sole means of euthanasia (see "Stunning" under "Adjunctive Methods").

Advantage—The penetrating captive bolt is an effective method of euthanasia for use in slaughterhouses, in research facilities, and on the farm when use of drugs is inappropriate.

Disadvantages—(1) It is aesthetically displeasing. (2) Death may not occur if equipment is not maintained and used properly.

Recommendations—Use of the penetrating captive bolt is an acceptable and practical method of euthanasia for horses, ruminants, and swine. It is conditionally acceptable in other appropriate species. The nonpenetrating captive bolt must not be used as a sole method of euthanasia.

Euthanasia by a blow to the head

Euthanasia by a blow to the head must be evaluated in terms of the anatomic features of the species on which it is to be performed. A blow to the head can be a humane method of euthanasia for neonatal animals with thin craniums, such as young pigs, if a single sharp blow delivered to the central skull bones with sufficient force can produce immediate depression of the central nervous system and destruction of brain tissue. When properly performed, loss of consciousness is rapid. The anatomic features of neonatal calves, however, make a blow to the head in this species unacceptable. Personnel performing euthanasia by use of a blow to the head must be properly trained and monitored for proficiency with this method of euthanasia, and they must be aware of its aesthetic implications.

Gunshot

A properly placed gunshot can cause immediate insensibility and humane death. In some circumstances, a gunshot may be the only practical method of euthanasia. Shooting should only be performed by highly skilled personnel trained in the use of firearms and only in jurisdictions that allow for legal firearm use. Personnel, public, and nearby animal safety should be considered. The procedure should be performed outdoors and away from public access.

For use of a gunshot to the head as a method of euthanasia in captive animals, the firearm should be aimed so that the projectile enters the brain, causing instant loss of consciousness.[51,112-114] This must take into account differences in brain position and skull conformation between species, as well as the energy requirement for skull bone and sinus penetration.[109,115] Accurate targeting for a gunshot to the head in various species has been described.[114,116-119] For wildlife and other freely roaming animals, the preferred target area should be the head. The appropriate firearm should be selected for the situation, with the goal being penetration and destruction of brain tissue without emergence from the contralateral side of the head.[120] A gunshot to the heart or neck does not immediately render animals unconscious and thus is not considered to meet the panel's definition of euthanasia.[121]

Advantages—(1) Loss of consciousness is instantaneous if the projectile destroys most of the brain. (2) Given the need to minimize stress induced by handling and human contact, gunshot may at times be the most practical and logical method of euthanasia of wild or free-ranging species.

Disadvantages—(1) Gunshot may be dangerous to personnel. (2) It is aesthetically unpleasant. (3) Under field conditions, it may be difficult to hit the vital target area. (4) Brain tissue may not be able to be examined for evidence of rabies infection or chronic wasting disease when the head is targeted.

Recommendations—When other methods cannot be used, an accurately delivered gunshot is a conditionally acceptable method of euthanasia.[114,122-125] When an animal can be appropriately restrained, the penetrating captive bolt is preferred to a gunshot. Prior to shooting, animals accustomed to the presence of humans should be treated in a calm and reassuring manner to minimize anxiety. In the case of wild animals, gunshots should be delivered with the least amount of prior human contact necessary. Gunshot should not be used for routine euthanasia of animals in animal control situations, such as municipal pounds or shelters.

Cervical dislocation

Cervical dislocation is a technique that has been used for many years and, when performed by well-trained individuals, appears to be humane. However, there are few scientific studies to confirm this observation. This technique is used to euthanatize poultry, other small birds, mice, and immature rats and rabbits. For mice and rats, the thumb and index finger are placed on either side of the neck at the base of the skull or, alternatively, a rod is pressed at the base of the skull. With the other hand, the base of the tail or the hind limbs are quickly pulled, causing separation of the cervical vertebrae from the skull. For immature rabbits, the head is held in one hand and the hind limbs in the other. The animal is stretched and the neck is hyperextended and dorsally twisted to separate the first cervical vertebra from the skull.[72,111] For poultry, cervical dislocation by stretching is a common method for mass euthanasia, but loss of consciousness may not be instantaneous.[134]

Data suggest that electrical activity in the brain persists for 13 seconds following cervical dislocation,[127] and unlike decapitation, rapid exsanguination does not contribute to loss of consciousness.[128,129]

Advantages—(1) Cervical dislocation is a technique that may induce rapid loss of consciousness.[84,127] (2) It does not chemically contaminate tissue. (3) It is rapidly accomplished.

Disadvantages—(1) Cervical dislocation may be aesthetically displeasing to personnel. (2) Cervical dislocation requires mastering technical skills to ensure loss of consciousness is rapidly induced. (3) Its use is limited to poultry, other small birds, mice, and immature rats and rabbits.

Recommendations—Manual cervical dislocation is a humane technique for euthanasia of poultry, other small birds, mice, rats weighing < 200 g, and rabbits weighing < 1 kg when performed by individuals with a demonstrated high degree of technical proficiency. In lieu of demonstrated technical competency, animals must be sedated or anesthetized prior to cervical dislocation. The need for technical competency is greater in heavy rats and rabbits, in which the large muscle mass in the cervical region makes manual cervical dislocation physically more difficult.[130] In research settings, this technique should be used only when scientifically justified by the user and approved by the Institutional Animal Care and Use Committee.

Those responsible for the use of this technique must ensure that personnel performing cervical dislocation techniques have been properly trained and consistently apply it humanely and effectively.

Decapitation

Decapitation can be used to euthanatize rodents and small rabbits in research settings. It provides a means to recover tissues and body fluids that are chemically uncontaminated. It also provides a means of obtaining anatomically undamaged brain tissue for study.[131]

Although it has been demonstrated that electrical activity in the brain persists for 13 to 14 seconds following decapitation,[132] more recent studies and reports indicate that this activity does not infer the ability to perceive pain, and in fact conclude that loss of consciousness develops rapidly.[127-129]

Guillotines that are designed to accomplish decapitation in adult rodents and small rabbits in a uniformly instantaneous manner are commercially available.

Guillotines are not commercially available for neonatal rodents, but sharp blades can be used for this purpose.

Advantages—(1) Decapitation is a technique that appears to induce rapid loss of consciousness.[127-129] (2) It does not chemically contaminate tissues. (3) It is rapidly accomplished.

Disadvantages—(1) Handling and restraint required to perform this technique may be distressful to animals.[83] (2) The interpretation of the presence of electrical activity in the brain following decapitation has created controversy and its importance may still be open to debate.[127-129,132] (3) Personnel performing this technique should recognize the inherent danger of the guillotine and take adequate precautions to prevent personal injury. (4) Decapitation may be aesthetically displeasing to personnel performing or observing the technique.

Recommendations—This technique is conditionally acceptable if performed correctly, and it should be used in research settings when its use is required by the experimental design and approved by the Institutional Animal Care and Use Committee. The equipment used to perform decapitation should be maintained in good working order and serviced on a regular basis to ensure sharpness of blades. The use of plastic cones to restrain animals appears to reduce distress from handling, minimizes the chance of injury to personnel, and improves positioning of the animal in the guillotine. Decapitation of amphibians, fish, and reptiles is addressed elsewhere in this report.

Those responsible for the use of this technique must ensure that personnel who perform decapitation techniques have been properly trained to do so.

Electrocution

Electrocution, using alternating current, has been used as a method of euthanasia for species such as dogs, cattle, sheep, swine, foxes, and mink.[113,133-138] Electrocution induces death by cardiac fibrillation, which causes cerebral hypoxia.[135,137,139] However, animals do not lose consciousness for 10 to 30 seconds or more after onset of cardiac fibrillation. It is imperative that animals be unconscious before being electrocuted. This can be accomplished by any acceptable means, including electrical stunning.[25] Although an effective, 1-step stunning and electrocution method has been described for use in sheep and hogs, euthanasia by electrocution in most species remains a 2-step procedure.[25,63,140]

Advantages—(1) Electrocution is humane if the animal is first rendered unconscious. (2) It does not chemically contaminate tissues. (3) It is economical.

Disadvantages—(1) Electrocution may be hazardous to personnel. (2) When conventional single-animal probes are used, it may not a useful method for mass euthanasia because so much time is required per animal. (3) It is not a useful method for dangerous, intractable animals. (4) It is aesthetically objectionable because of violent extension and stiffening of the limbs, head, and neck. (5) It may not result in death in small animals (< 5 kg) because ventricular fibrillation and circulatory collapse do not always persist after cessation of current flow.

Recommendations—Euthanasia by electrocution requires special skills and equipment that will ensure passage of sufficient current through the brain to induce loss of consciousness and cardiac fibrillation in the 1-step method for sheep and hogs, or cardiac fibrillation in the unconscious animal when the 2-step procedure is used. Although the method is conditionally acceptable if the aforementioned requirements are met, its disadvantages far outweigh its advantages in most applications. Techniques that apply electric current from head to tail, head to foot, or head to moistened metal plates on which the animal is standing are unacceptable.

Microwave irradiation

Heating by microwave irradiation is used primarily by neurobiologists to fix brain metabolites *in vivo* while maintaining the anatomic integrity of the brain.[141] Microwave instruments have been specifically designed for use in euthanasia of laboratory mice and rats. The instruments differ in design from kitchen units and may vary in maximal power output from 1.3 to 10 kw. All units direct their microwave energy to the head of the animal. The power required to rapidly halt brain enzyme activity depends on the efficiency of the unit, the ability to tune the resonant cavity and the size of the rodent head.[142] There is considerable variation among instruments in the time required for loss of consciousness and euthanasia. A 10 kw, 2,450 MHz instrument operated at a power of 9 kw will increase the brain temperature of 18 to 28 g mice to 79 C in 330 ms, and the brain temperature of 250 to 420 g rats to 94 C in 800 ms.[143]

Advantages—(1) Loss of consciousness is achieved in less than 100 ms, and death in less than 1 second. (2) This is the most effective method to fix brain tissue *in vivo* for subsequent assay of enzymatically labile chemicals.

Disadvantages—(1) Instruments are expensive. (2) Only animals the size of mice and rats can be euthanatized with commercial instruments that are currently available.

Recommendations—Microwave irradiation is a humane method for euthanatizing small laboratory rodents if instruments that induce rapid loss of consciousness are used. Only instruments that are designed for this use and have appropriate power and microwave distribution can be used. Microwave ovens designed for domestic and institutional kitchens are absolutely unacceptable for euthanasia.

Thoracic (cardiopulmonary, cardiac) compression

Thoracic (cardiopulmonary, cardiac) compression is used to euthanatize small- to medium-sized free-ranging birds when alternate techniques described in this report are not practical.[144]

Advantages—(1) This technique is rapid. (2) It is apparently painless. (3) It maximizes carcass use for analytical/contaminant studies.

Disadvantages—(1) It may be considered aesthetically unpleasant by onlookers. (2) The degree of distress is unknown.

Recommendations—Thoracic (cardiopulmonary, cardiac) compression is a physical technique for avian euthanasia that has applicability in the field when other methods cannot be used. It is accomplished by bringing the thumb and forefinger of one hand under the bird's wing from the posterior and placing them against the ribs.[144] The forefinger of the other hand is placed against the ventral edge of the sternum, just below the furculum. All fingers are brought together forcefully and held under pressure to stop the heart and lungs. Loss of consciousness and death develop quickly. Proper training is needed in the use of this technique to avoid trauma to the bird. Cardiopulmonary compression is not appropriate for laboratory settings, for large or diving birds,[144] or for other species.

Kill traps

Mechanical kill traps are used for the collection and killing of small, free-ranging mammals for commercial purposes (fur, skin, or meat), scientific purposes, to stop property damage, and to protect human safety. Their use remains controversial, and the panel recognizes that kill traps do not always render a rapid or stress-free death consistent with criteria for euthanasia found elsewhere in this document. For this reason, use of live traps followed by other methods of euthanasia is preferred. There are a few situations when that is not possible or when it may actually be more stressful to the animals or dangerous to humans to use live traps. Although newer technologies are improving kill trap performance in achieving loss of consciousness quickly, individual testing is recommended to be sure the trap is working properly.[145] If kill traps must be used, the most humane available must be chosen,[146-148] as evaluated by use of International Organization for Standardization (ISO) testing procedures,[149] or by the methods of Gilbert,[150] Proulx et al,[151,152] or Hiltz and Roy.[153]

To reach the required level of efficiency, traps may need to be modified from manufacturers production standards. In addition, as specified in scientific studies, trap placement (ground versus tree sets), bait type, set location, selectivity apparatus, body placement modifying devices (eg, sidewings, cones), trigger sensitivity, and trigger type, size, and conformation are essential considerations that could affect a kill trap's ability to reach these standards.

Several kill traps, modifications, and set specifics have been scientifically evaluated and found to meet the aforereferenced standards for various species.[151,152,154-167]

Advantage—Free-ranging small mammals may be killed with minimal distress associated with handling and human contact.

Disadvantages—(1) Traps may not afford death within acceptable time periods. (2) Selectivity and efficiency is dependent on the skill and proficiency of the operator.

Recommendations—Kill traps do not always meet the panel's criteria for euthanasia. At the same time, it is recognized that they can be practical and effective for scientific animal collection when used in a manner that ensures selectivity, a swift kill, no damage to body parts needed for field research, and minimal potential for injury of nontarget species.[168,169] Traps need to be checked at least once daily. In those instances when an animal is wounded or captured but not dead, the animal must be killed quickly and humanely. Kill traps should be used only when other acceptable techniques are impossible or have failed. Traps for nocturnal species should not be activated during the day to avoid capture of diurnal species.[168] Trap manufacturers should strive to meet their responsibility of minimizing pain and suffering in target species.

Adjunctive methods

Stunning and pithing, when properly done, induce loss of consciousness but do not ensure death. Therefore, these methods must be used only in conjunction with other procedures,[123] such as pharmacologic agents, exsanguination, or decapitation to euthanatize the animal.

Exsanguination

Exsanguination can be used to ensure death subsequent to stunning, or in otherwise unconscious animals. Because anxiety is associated with extreme hypovolemia, exsanguination must not be used as a sole means of euthanasia.[170] Animals may be exsanguinated to obtain blood products, but only when they are sedated, stunned, or anesthetized.[171]

Stunning

Animals may be stunned by a blow to the head, by use of a nonpenetrating captive bolt, or by use of electric current. Stunning must be followed immediately by a method that ensures death. With stunning, evaluating loss of consciousness is difficult, but it is usually associated with a loss of the menace or blink response, pupillary dilatation, and a loss of coordinated movements. Specific changes in the electroencephalogram and a loss of visually evoked responses are also thought to indicate loss of consciousness.[60,172]

Blow to the head—Stunning by a blow to the head is used primarily in small laboratory animals with thin craniums.[9,173-175] A single sharp blow must be delivered to the central skull bones with sufficient force to produce immediate depression of the central nervous system. When properly done, consciousness is lost rapidly.

Nonpenetrating captive bolt—A nonpenetrating captive bolt may be used to induce loss of consciousness in ruminants, horses, and swine. Signs of effective stunning by captive bolt are immediate collapse and a several second period of tetanic spasm, followed by slow hind limb movements of increasing frequency.[60,176]

Other aspects regarding use of the nonpenetrating captive bolt are similar to the use of a penetrating captive bolt, as previously described.

Electrical stunning—Alternating electrical current has been used for stunning species such as dogs, cattle, sheep, goats, hogs, fish and chickens.[133,134,140,177,178] Experiments with dogs have identified a need to direct the electrical current through the brain to induce rapid loss of consciousness. In dogs, when electricity passes only between fore- and hind limbs or neck and feet, it causes the heart to fibrillate but does not induce sudden loss of consciousness.[139] For electrical stunning of any animal, an apparatus that applies electrodes to opposite sides of the head, or in another way directs electrical current immediately through the brain, is necessary to induce rapid loss of consciousness. Attachment of electrodes and animal restraint can pose problems with this form of stunning. Signs of effective electrical stunning are extension of the limbs, opisthotonos, downward rotation of the eyeballs, and tonic spasm changing to clonic spasm, with eventual muscle flaccidity.

Electrical stunning should be followed promptly by electrically induced cardiac fibrillation, exsanguination, or other appropriate methods to ensure death. Refer to the section on electrocution for additional information.

PITHING

In general, pithing is used as an adjunctive procedure to ensure death in an animal that has been rendered unconscious by other means. For some species, such as frogs, with anatomic features that facilitate easy access to the central nervous system, pithing may be used as a sole means of euthanasia, but an anesthetic overdose is a more suitable method.

SPECIAL CONSIDERATIONS

Equine euthanasia

Pentobarbital or a pentobarbital combination is the best choice for equine euthanasia. Because a large volume of solution must be injected, use of an intravenous catheter placed in the jugular vein will facilitate the procedure. To facilitate catheterization of an excitable or fractious animal, a tranquilizer such as acepromazine, or an alpha-2 adrenergic agonist can be administered, but these drugs may prolong time to loss of consciousness because of their effect on circulation and may result in varying degrees of muscular activity and agonal gasping. Opioid agonists or agonist/antagonists in conjunction with alpha-2 adrenergic agonists may further facilitate restraint.

In certain emergency circumstances, such as euthanasia of a horse with a serious injury at a racetrack, it may be difficult to restrain a dangerous horse or other large animal for intravenous injection. The animal might cause injury to itself or to bystanders before a sedative could take effect. In such cases, the animal can be given a neuromuscular blocking agent such as succinylcholine, but the animal must be euthanatized with an appropriate technique as soon as the animal can be controlled. Succinylcholine alone or without sufficient anesthetic must not be used for euthanasia.

Physical methods, including gunshot, are considered conditionally acceptable techniques for equine euthanasia. The penetrating captive bolt is acceptable with appropriate restraint.

Animals intended for human or animal food

In euthanasia of animals intended for human or animal food, chemical agents that result in tissue residues cannot be used, unless they are approved by the US Food and Drug Administration.[179] Carbon dioxide is the only chemical currently used for euthanasia of food animals (primarily swine) that does not result in tissue residues. Physical techniques are commonly used for this reason. Carcasses of animals euthanatized by barbituric acid derivatives or other chemical agents may contain potentially harmful residues. These carcasses should be disposed of in a manner that will prevent them from being consumed by human beings or animals.

Selection of a proper euthanasia technique for free-ranging wildlife must take into account the possibility of consumption of the carcass of the euthanatized animal by nontarget predatory or scavenger species. Numerous cases of toxicosis and death attributable to ingestion of pharmaceutically contaminated carcasses in predators and scavengers have been reported.[107] Proper carcass disposal must be a part of any euthanasia procedure under free-range conditions where there is potential for consumption toxicity. When carcasses are to be left in the field, a gunshot to the head, penetrating captive bolt, or injectable agents that are nontoxic (potassium chloride in combination with a nontoxic general anesthetic) should be used so that the potential for scavenger or predator toxicity is lessened.

Euthanasia of nonconventional species: zoo, wild, aquatic, and ectothermic animals

Compared with objective information on companion, farm, and laboratory animals, euthanasia of species such as zoo, wild, aquatic, and ectothermic animals has been studied less, and guidelines are more limited. Irrespective of the unique or unusual features of some species, whenever it becomes necessary to euthanatize an animal, death must be induced as painlessly and quickly as possible.

When selecting a means of euthanasia for these species, factors and criteria in addition to those previously discussed must be considered. The means selected will depend on the species, size, safety aspects, location of the animals to be euthanatized, and experience of personnel. Whether the animal to be euthanatized is in the wild, in captivity, or free-roaming are major considerations. Anatomic differences must be considered. For example, amphibians, fish, reptiles, and marine mammals differ anatomically from domestic species. Veins may be difficult to locate. Some species have a carapace or other defensive anatomic adaptations (eg, quills, scales, spines). For physical methods, access to the central nervous system may be difficult because the brain may be small and difficult to locate by inexperienced persons.

Zoo Animals

For captive zoo mammals and birds with related domestic counterparts, many of the means described previously are appropriate. However, to minimize injury to persons or animals, additional precautions such as handling and physical or chemical restraint are important considerations.[16]

Wildlife

For wild and feral animals, many recommended means of euthanasia for captive animals are not feasible. The panel recognizes there are situations involving free-ranging wildlife when euthanasia is not possible from the animal or human safety standpoint, and killing may be necessary. Conditions found in the field, although more challenging than those that are controlled, do not in any way reduce or minimize the ethical obligation of the responsible individual to reduce pain and distress to the greatest extent possible during the taking of an animal's life. Because euthanasia of wildlife is often performed by lay personnel in remote settings, guidelines are needed to assist veterinarians, wildlife biologists, and wildlife health professionals in developing humane protocols for euthanasia of wildlife.

In the case of free-ranging wildlife, personnel may not be trained in the proper use of remote anesthesia, proper delivery equipment may not be available, personnel may be working alone in remote areas where accidental exposure to potent anesthetic medications used in wildlife capture would present a risk to human safety, or approaching the animal within a practical darting distance may not be possible. In these cases, the only practical means of animal collection may be gunshot and kill trapping.[13,180-184] Under these conditions, specific methods chosen must be as age-, species-, or taxonomic/class-specific as possible. The firearm and ammunition should be appropriate for the species and purpose. Personnel should be sufficiently skilled to be accurate, and they should be experienced in the proper and safe use of firearms, complying with laws and regulations governing their possession and use.

Behavioral responses of wildlife or captive nontraditional species (zoo) in close human contact are very different from those of domestic animals. These animals are usually frightened and distressed. Thus, minimizing the amount, degree, and/or cognition of human contact during procedures that require handling is of utmost importance. Handling these animals often requires general anesthesia, which provides loss of consciousness and which relieves distress, anxiety, apprehension, and perception of pain. Even though the animal is under general anesthesia, minimizing auditory, visual, and tactile stimulation will help ensure the most stress-free euthanasia possible. With use of general anesthesia, there are more methods for euthanasia available.

A 2-stage euthanasia process involving general anesthesia, tranquilization, or use of analgesics, followed by intravenous injectable pharmaceuticals, although preferred, is often not practical. Injectable anesthetics are not always legally or readily available to those working in nuisance animal control, and the distress to the animal induced by live capture, transport to a veterinary facility, and confinement in a veterinary hospital prior to euthanasia must be considered in choosing the most humane technique for the situation at hand. Veterinarians providing support to those working with injured or live-trapped, free-ranging animals should take capture, transport, handling distress, and possible carcass consumption into consideration when asked to assist with euthanasia. Alternatives to 2-stage euthanasia using anesthesia include a squeeze cage with intraperitoneal injection of sodium pentobarbital, inhalant agents (CO_2 chamber, CO chamber), and gunshot. In cases where preeuthanasia anesthetics are not available, intraperitoneal injections of sodium pentobarbital, although slower in producing loss of consciousness, should be considered preferable over intravenous injection, if restraint will cause increased distress to the animal or danger to the operator.

Wildlife species may be encountered under a variety of situations. Euthanasia of the same species under different conditions may require different techniques. Even in a controlled setting, an extremely fractious large animal may threaten the safety of the practitioner, bystanders, and itself. When safety is in question and the fractious large animal, whether wild, feral, or domestic, is in close confinement, neuromuscular blocking agents may be used immediately prior to the use of an acceptable form of euthanasia. For this technique to be humane, the operator must ensure they will gain control over the animal and perform euthanasia before distress develops. Succinylcholine is not acceptable as a method of restraint for use in free-ranging wildlife because animals may not be retrieved rapidly enough to prevent neuromuscular blocking agent-induced respiratory distress or arrest.[185]

Diseased, Injured, or Live-Captured Wildlife or Feral Species

Euthanasia of diseased, injured, or live-trapped wildlife should be performed by qualified professionals. Certain cases of wildlife injury (eg, acute, severe trauma from automobiles) may require immediate action, and pain and suffering in the animal may be best relieved most rapidly by physical methods including gunshot or penetrating captive bolt followed by exsanguination.

Birds

Many techniques discussed previously in this report are suitable for euthanasia of captive birds accustomed to human contact. Free-ranging birds may be collected by a number of methods, including nets and live traps, with subsequent euthanasia. For collection by firearm, shotguns are recommended. The bird should be killed outright by use of ammunition loads appropriate for the species to be collected. Wounded birds should be killed quickly by appropriate techniques previously described. Large birds should be anesthetized prior to euthanasia, using general anesthetics.

Amphibians, Fish, and Reptiles

Euthanasia of ectothermic animals must take into account differences in their metabolism, respiration, and tolerance to cerebral hypoxia. In addition, it is often more difficult to ascertain when an animal is dead. Some unique aspects of euthanasia of amphibians, fishes, and reptiles have been described.[13,51,186,187]

Injectable agents—Sodium pentobarbital (60 to 100 mg/kg of body weight) can be administered intravenously, intraabdominally, or intrapleuroperitoneally in most ectothermic animals, depending on anatomic features. Subcutaneous lymph spaces may also be used in frogs and toads. Time to effect may be variable, with death occurring in up to 30 minutes.[1,187,188] Barbiturates other than pentobarbital can cause pain on injection.[189]

Clove oil—Because adequate and appropriate clinical trials have not been performed on fish to evaluate its effects, use of clove oil is not acceptable.

External or topical agents—Tricaine methane sulfonate (TMS, MS-222) may be administered by various routes to euthanatize. For fish and amphibians, this chemical may be placed in water.[190-193] Large fish may be removed from the water, a gill cover lifted, and a concentrated solution from a syringe flushed over the gills. MS 222 is acidic and in concentrations ≥ 500 mg/L should be buffered with sodium bicarbonate to saturation resulting in a solution pH of 7.0 to 7.5.[105] MS 222 may also be injected into lymph spaces and pleuroperitoneal cavities.[194] These are effective but expensive means of euthanasia.

Benzocaine hydrochloride, a compound similar to TMS, may be used as a bath or in a recirculation system for euthanasia of fish[184] or amphibians.[13] Benzocaine is not water soluble and therefore is prepared as a stock solution (100 g/L), using acetone or ethanol, which may be irritating to fish tissues. In contrast, benzocaine hydrochloride is water soluble and can be used directly for anesthesia or euthanasia.[105] A concentration ≥ 250 mg/L can be used for euthanasia. Fish should be left in the solution for at least 10 minutes following cessation of opercular movement.[104]

The anesthetic agent 2-phenoxyethanol is used at concentrations of 0.5 to 0.6 ml/L or 0.3 to 0.4 mg/L for euthanasia of fish. Death is caused by respiratory collapse. As with other agents, fish should be left in solution for 10 minutes following cessation of opercular movement.[195,196]

Inhalant agents—Many reptiles and amphibians, including chelonians, are capable of holding their breath and converting to anaerobic metabolism, and can survive long periods of anoxia (up to 27 hours for some species).[197-202] Because of this ability to tolerate anoxia, induction of anesthesia and time to loss of consciousness may be greatly prolonged when inhalants are used. Death in these species may not occur even after prolonged inhalant exposure.[203] Lizards, snakes, and fish do not hold their breath to the same extent and can be euthanatized by use of inhalant agents.

Carbon dioxide—Amphibians,[1] reptiles,[1] and fish[203-205] may be euthanatized with CO_2. Loss of consciousness develops rapidly, but exposure times required for euthanasia are prolonged. This technique is more effective in active species and those with less tendency to hold their breath.

Physical methods—Line drawings of the head of various amphibians and reptiles, with recommended locations for captive bolt or firearm penetration, are available.[13] Crocodilians and other large reptiles can also be shot through the brain.[51]

Decapitation with heavy shears or a guillotine is effective for some species that have appropriate anatomic features. It has been assumed that stopping blood supply to the brain by decapitation causes rapid loss of consciousness. Because the central nervous system of reptiles, fish, and amphibians is tolerant to hypoxic and hypotensive conditions,[13] decapitation must be followed by pithing.[188]

Two-stage euthanasia procedures—Propofol and ultrashort-acting barbiturates may be used for these species to produce rapid general anesthesia prior to final administration of euthanasia.

In zoos and clinical settings, neuromuscular blocking agents are considered acceptable for restraint of reptiles if given immediately prior to administration of a euthanatizing agent.

Most amphibians, fishes, and reptiles can be euthanatized by cranial concussion (stunning) followed by decapitation, pithing, or some other physical method.

Severing the spinal cord behind the head by pithing is an effective method of killing some ectotherms. Death may not be immediate unless both the brain and spinal cord are pithed. For these animals, pithing of the spinal cord should be followed by decapitation and pithing of the brain or by another appropriate procedure. Pithing requires dexterity and skill and should only be done by trained personnel. The pithing site in frogs is the foramen magnum, and it is identified by a slight midline skin depression posterior to the eyes with the neck flexed.[187]

Cooling—It has been suggested that, when using physical methods of euthanasia in ectothermic species, cooling to 4 C will decrease metabolism and facilitate handling, but there is no evidence that whole body cooling reduces pain or is clinically efficacious.[206] Local cooling in frogs does reduce nociception, and this may be partly opioid mediated. [207] Immobilization of reptiles by cooling is considered inappropriate and inhumane even if combined with other physical or chemical methods of euthanasia. Snakes and turtles, immobilized by cooling, have been killed by subsequent freezing. This method is not recommended.[13] Formation of ice crystals on the skin and in tissues of an animal may cause pain or distress. Quick freezing of deeply anesthetized animals is acceptable.[208]

Marine Mammals

Barbiturates or potent opioids (eg, etorphine hydrochloride [M 99] and carfentanil) are the agents of choice for euthanasia of marine mammals,[209] although it is recognized their use is not always possible and can

be potentially dangerous to personnel. An accurately placed gunshot may also be a conditionally acceptable method of euthanasia for some species and sizes of stranded marine mammals.[51,209,210]

For stranded whales or other large cetaceans or pinnipeds, succinylcholine chloride in conjunction with potassium chloride, administered intravenously or intraperitoneally, has been used.[211] This method, which is not an acceptable method of euthanasia as defined in this report, leads to complete paralysis of the respiratory musculature and eventual death attributable to hypoxemia.[209] This method may be more humane than allowing the stranded animal to suffocate over a period of hours or days if no other options are available.

Euthanasia of animals raised for fur production

Animals raised for fur are usually euthanatized individually at the location where they are raised. Although any handling of these species constitutes a stress, it is possible to minimize this by euthanatizing animals in or near their cages. For the procedures described below, please refer to previous sections for more detailed discussion.

Carbon monoxide—For smaller species, CO appears to be an adequate method for euthanasia. Compressed CO is delivered from a tank into an enclosed cage that can be moved adjacent to holding cages. Using the apparatus outside reduces the risk to humans; however, people using this method should still be made aware of the dangers of CO. Animals introduced into a chamber containing 4% CO lost consciousness in 64 ± 14 seconds and were dead within 215 ± 45 seconds.[80] In a study involving electroencephalography of mink being euthanatized with 3.5% CO, the mink were comatose in 21 ± 7 seconds.[212] Only 1 animal should be introduced into the chamber at a time, and death should be confirmed in each case.

Carbon dioxide—Administration of CO_2 is also a good euthanasia method for smaller species and is less dangerous than CO for personnel operating the system. When exposed to 100% CO_2, mink lost consciousness in 19 ± 4 seconds and were dead within 153 ± 10 seconds. When 70% CO_2 was used with 30% O_2, mink were unconscious in 28 seconds, but they were not dead after a 15-minute exposure.[80] Therefore, if animals are first stunned by 70% CO_2, they should be killed by exposure to 100% CO_2 or by some other means. As with carbon monoxide, only one animal should be introduced into the chamber at a time.

Barbiturates—Barbiturate overdose is an acceptable procedure for euthanasia of many species of animals raised for fur. The drug is injected intraperitoneally and the animal slowly loses consciousness. It is important that the death of each animal be confirmed following barbiturate injection. Barbiturates will contaminate the carcass; therefore the skinned carcass cannot be used for animal food.

Electrocution—Electrocution has been used for killing foxes and mink.[135] The electric current must pass through the brain to induce loss of consciousness before electricity is passed through the rest of the body. Electrical stunning should be followed by euthanasia, using some other technique. Cervical dislocation has been used in mink and other small animals and should be done within 20 seconds of electrical stunning.[213] Use of a nose-to-tail or nose-to-foot method[135] alone may kill the animal by inducing cardiac fibrillation, but the animal may be conscious for a period of time before death. Therefore, these techniques are unacceptable.

Prenatal and neonatal euthanasia

When ovarian hysterectomies are performed, euthanasia of feti should be accomplished as soon as possible after removal from the dam. Neonatal animals are relatively resistant to hypoxia.[44,214]

Mass euthanasia

Under unusual conditions, such as disease eradication and natural disasters, euthanasia options may be limited. In these situations, the most appropriate technique that minimizes human and animal health concerns must be used. These options include, but are not limited to, CO_2 and physical methods such as gunshot, penetrating captive bolt, and cervical dislocation.

POSTFACE

This report summarizes contemporary scientific knowledge on euthanasia in animals and calls attention to the lack of scientific reports assessing pain, discomfort, and distress in animals being euthanatized. Many reports on various methods of euthanasia are either anecdotal, testimonial narratives, or unsubstantiated opinions and are, therefore, not cited in this report. The panel strongly endorses the need for well-designed experiments to more fully determine the extent to which each procedure meets the criteria used for judging methods of euthanasia.

Each means of euthanasia has advantages and disadvantages. It is unlikely that, for each situation, any means will meet all desirable criteria. It is also impractical for this report to address every potential circumstance in which animals are to be euthanatized. Therefore, the use of professional judgment is imperative.

Failure to list or recommend a means of euthanasia in this report does not categorically condemn its use. There may occasionally be special circumstances or situations in which other means may be acceptable. For research animals, these exceptions should be carefully considered by the attending veterinarian and the Institutional Animal Care and Use Committee. In other settings, professional judgment should be used.

The panel discourages the use of unapproved products for euthanasia, unless the product has a clearly understood mechanism of action and pharmacokinetics, and studies published in the literature that scientifically verify and justify its use. Those responsible for euthanasia decisions have a critically important responsibility to carefully assess any new technique, method, or device, using the panel's criteria. In the absence of definitive proof or reasonable expectation, the best interest of the animal should guide the decision process.

References cited in this report do not represent a comprehensive bibliography on all methods of euthanasia. Persons interested in additional information on a particular aspect of animal euthanasia are encouraged to contact the Animal Welfare Information Center, National Agricultural Library, 10301 Baltimore Blvd, Beltsville, MD 20705.

The Panel on Euthanasia is fully committed to the concept that, whenever it becomes necessary to kill any animal for any reason whatsoever, death should be induced as painlessly and quickly as possible. It has been our charge to develop workable guidelines for veterinarians needing to address this problem, and it is our sincere desire that these guidelines be used conscientiously by all animal care providers. We consider this report to be a work in progress with new editions warranted as results of more scientific studies are published.

Acknowledgment: The panel acknowledges the assistance of Ms. Julie Horvath and Dr. David Granstrom in coordinating the preparation and circulation of various drafts of the report. The panel also acknowledges and thanks Dr. Laurence Roy, Dr. Leah Greer, and the many other individuals and organizations that provided valuable review, criticism, and input to the panel through the many drafts of the report. The research and humane communities were especially helpful in shaping important changes and additions to the report.

References

1. Andrews EJ, Bennet BT, Clark JD, et al. 1993 Report on the AVMA panel on euthanasia. *J Am Vet Med Assoc* 1993;202:230–247.

2. *Webster's ninth new collegiate dictionary*. Springfield: Merriam-Webster Inc, 1990.

3. Wall PD. Defining pain in animals. In: Short CE, Poznak AV, eds. *Animal pain*. New York: Churchill-Livingstone Inc, 1992;63–79.

4. Vierck CJ, Cooper BY, Ritz LA, et al. Inference of pain sensitivity from complex behaviors of laboratory animals. In: Chapman CR, Loeser JD, eds. *Issues in pain measurement*. New York: Raven Press, 1989;93–115.

5. Breazile JE, Kitchell RL. Euthanasia for laboratory animals. *Fed Proc* 1969;28:1577–1579.

6. Zimmerman M. Neurobiological concepts of pain, its assessment and therapy. In: Bromm B, ed. *Pain measurement in man: neurophysiological correlates of pain*. Amsterdam: Elsevier Publishing Co, 1984;15–35.

7. Kitchell RL, Erickson NH, Carstens E, et al, eds. *Animal pain: perception and alleviation*. Bethesda: American Physiological Society, 1983.

8. Kitchen N, Aronson AL, Bittle JL, et al. Panel report on the colloquium on recognition and alleviation of animal pain and distress. *J Am Vet Med Assoc* 1987;191:1186–1191.

9. National Research Council. *Recognition and alleviation of pain and distress in laboratory animals*. Washington, DC: National Academy Press, 1992.

10. Breazile JE. Physiologic basis and consequences of distress in animals. *J Am Vet Med Assoc* 1987;191:1212–1215.

11. McMillan FD. Comfort as the primary goal in veterinary medical practice. *J Am Vet Med Assoc* 1998;212:1370–1374.

12. Grier RL, Clovin TL. *Euthanasia guide (for animal shelters)*. Ames, Iowa: Moss Creek Publications, 1990.

13. Cooper JE, Ewbank R, Platt C, et al. *Euthanasia of amphibians and reptiles*. London: UFAW/WSPA, 1989.

14. Greyhavens T. *Handbook of pentobarbital euthanasia*. Salem, Ore: Humane Society of Willamette Valley, 1989;1–126.

15. *Operational guide for animal care and control agencies*. Denver: American Humane Association, 1988.

16. Fowler ME, Miller RE, eds. *Zoo and wild animal medicine: current therapy 4*. Philadelphia: WB Saunders Co, 1999;1–747.

17. Clark R, Jessup DA. *Wildlife restraint series*. Salinas, Calif: International Wildlife Veterinary Services Inc, 1992.

18. Kreeger T. *Handbook of wildlife chemical immobilization*. Laramie, Wyo: Wildlife Veterinary Services Inc, 1996.

19. Nielsen L. *Chemical immobilization of wild and exotic animals*. Ames, Iowa: Iowa State University Press, 1999.

20. McKenzie A, ed. *The capture and care manual*. South Africa: Wildlife Decision Support Services/The South African Veterinary Foundation, 1993.

21. Amass K, Neilsen L, Brunson D. *Chemical immobilization of animals*. Mount Horeb, Wis: Safe-Capture International Inc, 1999.

22. Humane slaughter regulations. *Fed Reg* 1979;44: 68809–68817.

23. Grandin T. Observations of cattle behavior applied to design of cattle-handling facilities. *Appl Anim Ethol* 1980;6:19–31.

24. Grandin T. Pig behavior studies applied to slaughter-plant design. *Appl Anim Ethol* 1982;9:141–151.

25. Grandin T. Farm animal welfare during handling, transport, and slaughter. *J Am Vet Med Assoc* 1994;204:372–377.

26. Grandin T. Objective scoring of animal handling and stunning practices at slaughter plants. *J Am Vet Med Assoc* 1998;212: 36–39.

27. Grandin T. Effect of animal welfare audits of slaughter plants by a major fast food company on cattle handling and slaughter practices. *J Am Vet Med Assoc* 2000;216:848–851.

28. Tannenbaum J. Issues in companion animal practice. In: *Veterinary ethics*. Baltimore: The Williams & Wilkins Co, 1989;208–225.

29. Rollin BE. Ethical question of the month. *Can Vet J* 1992;33:7–8.

30. Ramsey EC, Wetzel RW. Comparison of five regimens for oral administration of medication to induce sedation in dogs prior to euthanasia. *J Am Vet Med Assoc* 1998;213:240–242.

31. Wetzel RW, Ramsay EC. Comparison of four regimens for oral administration of medication to induce sedation in cats prior to euthanasia. *J Am Vet Med Assoc* 1998;213:243–245.

32. Beaver BV. *Feline behavior: a guide for veterinarians*. Philadelphia: WB Saunders Co, 1992;1–276.

33. Houpt KA. *Domestic animal behavior for veterinarians and animal scientists*. 3rd ed. Ames, Iowa: Iowa State University Press, 1998;1–495.

34. Hart BL. *The behavior of domestic animals*. New York: WH Freeman & Co, 1985;1–390.

35. Beaver BV. *Canine behavior: a guide for veterinarians*. Philadelphia: WB Saunders Co, 1999;1–355.

36. Beaver BV. *The veterinarian's encyclopedia of animal behavior*. Ames, Iowa: Iowa State University Press, 1994;1–307.

37. Schafer M. *The language of the horse: habits and forms of expression*. New York: Arco Publishing Co, 1975;1–187.

38. Hart LA, Hart BL, Mader B. Humane euthanasia and companion animal death: caring for the animal, the client, and the veterinarian. *J Am Vet Med Assoc* 1990;197:1292–1299.

39. Neiburg HA, Fischer A. *Pet loss, a thoughtful guide for adults and children*. New York: Harper & Row, 1982.

40. Hart LA, Mader B. Pet loss support hotline: the veterinary students' perspective. *Calif Vet* 1992;Jan-Feb:19–22.

41. Pet loss support hotlines (grief counseling). *J Am Vet Med Assoc* 1999;215:1804.

42. Arluke A. Coping with euthanasia: a case study of shelter culture. *J Am Vet Med Assoc* 1991;198:1176–1180.

43. Wolfle TL. Laboratory animal technicians: their role in stress reduction and human-companion animal bonding. *Vet Clin North Am Small Anim Pract* 1985;15:449–454.

44. Glass HG, Snyder FF, Webster E. The rate of decline in resistance to anoxia of rabbits, dogs, and guinea pigs from the onset of viability to adult life. *Am J Physiol* 1944;140:609–615.

45. Booth NH. Inhalant anesthetics. In: Booth NH, McDonald LE, eds. *Veterinary pharmacology and therapeutics*. 6th ed. Ames, Iowa: Iowa State University Press, 1988;181–211.

46. Wixon SK, Smiler KL. Anesthesia and analgesia in rodents. In: Kohn DF, Wixson SK, White WJ, et al, eds. *Anesthesia and analgesia in laboratory animals*. New York: Academic Press Inc, 1997;165–203.

47. Knigge U, Soe-Jensen P, Jorgensen H, et al. Stress-induced release of anterior pituitary hormones: effect of H3 receptor-mediat-

ed inhibition of histaminergic activity or posterior hypothalamic lesion. *Neuroendocrin* 1999;69:44–53.

48. Tinnikov AA. Responses of serum conticosterone and corticosteroid-binding globulin to acute and prolonged stress in the rat. *Endocrine* 1999;11:145–150.

49. Zelena D, Klem DT, Barna I, et al. Alpha 2-adrenoreceptor subtypes regulate ACTH and beta-endorphon secretions during stress in the rat. *Psychoneuroendocrin* 1999;24:333–343.

50. Van Herck H, Baumans V, DeBoer SF, et al. Endocrine stress response in rats subjected to singular orbital puncture while under diethyl-ether anaesthesia. *Lab Anim* 1991;25:325–329.

51. *Humane killing of animals*. Preprint of 4th ed. South Mimms, Potters Bar, Herts, England: Universities Federation for Animal Welfare, 1988;16–22.

52. *Occupational exposure to waste anesthetic gases and vapors*. No. 77-140. Washington, DC: Department of Health, Education, and Welfare (National Institute for Occupational Safety and Health), 1977.

53. Lecky JH, ed. *Waste anesthetic gases in operating room air: a suggested program to reduce personnel exposure*. Park Ridge, Ill: The American Society of Anesthesiologists, 1983.

54. Simonsen HB, Thordal-Christensen AA, Ockens N. Carbon monoxide and carbon dioxide euthanasia of cats: duration and animal behavior. *Br Vet J* 1981;137:274–278.

55. Klemm WR. Carbon dioxide anesthesia in cats. *Am J Vet Res* 1964;25:1201–1205.

56. Leake CD, Waters RM. The anesthetic properties of carbon dioxide. *Curr Res Anesthesiol Analg* 1929;8:17–19.

57. Mattsson JL, Stinson JM, Clark CS. Electroencephalographic power—spectral changes coincident with onset of carbon dioxide narcosis in rhesus monkey. *Am J Vet Res* 1972;33:2043–2049.

58. Woodbury DM, Rollins LT, Gardner MD, et al. Effects of carbon dioxide on brain excitability and electrolytes. *Am J Physiol* 1958;192:79–90.

59. Glen JB, Scott WN. Carbon dioxide euthanasia of cats. *Br Vet J* 1973;129:471–479.

60. Blackmore DK, Newhook JC. The assessment of insensibility in sheep, calves and pigs during slaughter. In: Eikelenboom G, ed. *Stunning of animals for slaughter*. Boston: Martinus Nijhoff Publishers, 1983;13–25.

61. Coenen AML, Drinkenburg WHIM, Hoenderken R, et al. Carbon dioxide euthanasia in rats: oxygen supplementation minimizes signs of agitation and asphyxia. *Lab Anim* 1995;29:262–268.

62. Kohler I, Meier R, Busato A, et al. Is carbon dioxide (CO_2) a useful short acting anaesthetic for small laboratory animals? *Lab Anim* 1998;33:155–161.

63. Hoenderken R. Electrical and carbondioxide stunning of pigs for slaughter. In: Eikelenboom G, ed. *Stunning of animals for slaughter*. Boston: Martinus Nijhoff Publishers, 1983;59–63.

64. Gregory NG, Moss BW, Leeson RH. An assessment of carbon dioxide stunning in pigs. *Vet Rec* 1987;121:517–518.

65. Carding AH. Mass euthanasia of dogs with carbon monoxide and/or carbon dioxide: preliminary trials. *J Small Anim Pract* 1968;9:245–259.

66. Britt DP. The humaneness of carbon dioxide as an agent of euthanasia for laboratory rodents. In: *Euthanasia of unwanted, injured or diseased animals for educational or scientific purposes*. Potters Bar, UK: UFAW, 1987;19–31.

67. Danneman PJ, Stein S, Walshaw SO. Humane and practical implications of using carbon dioxide mixed with oxygen for anesthesia or euthanasia of rats. *Lab Anim Sci* 1997;47:376–385.

68. Anton F, Euchner I, Handwerker HO. Psycophysical examination of pain induced by defined CO_2 pulses applied to nasal mucosa. *Pain* 1992;49:53–60.

69. Raj ABM, Gregory NG. Welfare implications of gas stunning pigs 1. Determination of aversion to the initial inhalation of carbon dioxide or argon. *Anim Welfare* 1995;4:273–280.

70. Hackbarth H, Kppers N, Bohnet W. Euthanasia of rats with carbon dioxide-animal welfare aspects. *Lab Anim* 2000;34:91–96.

71. Raj ABM, Gregory NG. Investigation into the batch stunning/killing of chickens using carbon dioxide or argon-induced hypoxia. *Res Vet Sci* 1990;49:364–366.

72. Hughes HC. Euthanasia of laboratory animals. In: Melby EC, Altman NH, eds. *Handbook of laboratory animal science*. Vol 3. Cleveland, Ohio: CRC Press, 1976;553–559.

73. Jaksch W. Euthanasia of day-old male chicks in the poultry industry. *Int J Stud Anim Prob* 1981;2:203–213.

74. Kline BE, Peckham V, Hesic HE. Some aids in handling large numbers of mice. *Lab Anim Care* 1963;13:84–90.

75. Kocula AW, Drewniak EE, Davis LL. Experimentation with in-line carbon dioxide immobilization of chickens prior to slaughter. *Poult Sci* 1961;40:213–216.

76. Stone WS, Amiraian K, DueIl C, et al. Carbon dioxide anesthetization of guinea pigs to increase yields of blood and serum. *Proc Care Panel* 1961;11:299–303.

77. Euthanasia (carbon dioxide). In: *Report and accounts 1976-1977*. South Mimms, Potters Bar, Herts, England: Universities Federation for Animal Welfare, 1977;13–14.

78. Hall LW. The anaesthesia and euthanasia of neonatal and juvenile dogs and cats. *Vet Rec* 1972;90:303–306.

79. Blackshaw JK, Fenwick DC, Beattie AW, et al. The behaviour of chickens, mice and rats during euthanasia with chloroform, carbon dioxide and ether. *Lab Anim* 1988;22:67–75.

80. Hansen NE, Creutzberg A. Simonsen HB. Euthanasia of mink (*Mustela vison*) by means of carbon dioxide (CO_2), carbon monoxide (CO) and nitrogen (N_2). *Br Vet J* 1991;147:140–146.

81. Hayward JS, Lisson PA. Carbon dioxide tolerance of rabbits and its relation to burrow fumigation. *Aust Wildl Res* 1978;5:253–261.

82. Bereger-Sweeney J, Berger UV, Sharma M, et al. Effects of carbon dioxide-induced anesthesia on cholinergic parameters in rat brain. *Lab Anim Sci* 1994;44:369–371.

83. Urbanski HF, Kelly SF. Sedation by exposure to gaseous carbon dioxide-oxygen mixture: application to studies involving small laboratory animal species. *Lab Anim Sci* 1991;41:80–82.

84. Iwarsson K, Rehbinder C. A study of different euthanasia techniques in guinea pigs, rats, and mice. Animal response and postmortem findings. *Scand J Lab Anim Sci* 1993;20:191–205.

85. Hornett TD, Haynes AP. Comparison of carbon dioxide/air mixture and nitrogen/air mixture for the euthanasia of rodents: design of a system for inhalation euthanasia. *Anim Technol* 1984;35: 93–99.

86. Smith W, Harrap SB. Behavioral and cardiovascular responses of rats to euthanasia using carbon dioxide gas. *Lab Anim* 1997; 31:337–346.

87. Hewett TA, Kovacs MS, Artwohl JE, et al. A comparison of euthanasia methods in rats, using carbon dioxide in prefilled and fixed flow rate filled chambers. *Lab Anim Sci* 1993;43:579–582.

88. Herin RA, Hall P, Fitch JW. Nitrogen inhalation as a method of euthanasia in dogs. *Am J Vet Res* 1978;39:989–991.

89. Noell WK, Chinn HI. Time course of failure of the visual pathway in rabbits during anoxia. *Fed Proc* 1949;8:119.

90. Vinte FJ. *The humane killing of mink*. London: Universities Federation for Animal Welfare, 1957.

91. Stonehouse RW, Loew FM, Quine JP, et al. The euthanasia of dogs and cats: a statement of the humane practices committee of the Canadian Veterinary Medical Association. *Can Vet J* 1978;19: 164–168.

92. Quine JP, Buckingham W, Strunin L. Euthanasia of small animals with nitrogen; comparison with intravenous pentobarbital. *Can Vet J* 1988;29:724–726.

93. Raj ARM, Gregory NG, Wotton SR. Changes in the somatosensory evoked potentials and spontaneous electroencephalogram of hens during stunning in Argon-induced anoxia. *Br Vet J* 1991;147:322–330.

94. Ramsey TL, Eilmann HJ. Carbon monoxide acute and chronic poisoning and experimental studies. *J Lab Clin Med* 1932; 17:415–427.

95. Chalifoux A, Dallaire A. Physiologic and behavioral evaluation of CO euthanasia of adult dogs. *Am J Vet Res* 1983;44: 2412–2417.

96. Haldane J. The action of carbonic oxide in man. *J Physiol* 1895;18:430–462.

97. Dallaire A, Chalifoux A. Premedication of dogs with acepromazine or pentazocine before euthanasia with carbon monoxide. *Can J Comp Med* 1985;49:171–178.

98. Lambooy E, Spanjaard W. Euthanasia of young pigs with carbon monoxide. *Vet Rec* 1980;107:59–61.

99. Lowe-Ponsford FL, Henry JA. Clinical aspects of carbon monoxide poisoning. *Adverse Drug React Acute Poisoning Rev* 1989;8:217–240.

100. Bloom JD. Some considerations in establishing divers' breathing gas purity standards for carbon monoxide. *Aerosp Med* 1972;43:633–636.

101. Norman CA, Halton DM. Is carbon monoxide a workplace teratogen? A review and evaluation of the literature. *Ann Occup Hyg* 1990;34:335–347.

102. Eechter LD. Neurotoxicity of prenatal carbon monoxide exposure. Research report. *Health Effects Inst* 1987;Vol:3–22.

103. Wojtczak-Jaroszowa J, Kubow S. Carbon monoxide, carbon disulfide, lead and cadmium—four examples of occupational toxic agents linked to cardiovascular disease. *Med Hypotheses* 1989;30: 141–150.

104. Noga E. *Fish disease: diagnosis and treatment*. St. Louis: CV Mosby, 1996;1–367.

105. Stoskopf MK. Anaesthesia. In: Brown LA, ed. *Aquaculture for veterinarians: fish husbandry and medicine*. Oxford, UK: Pergamon Press, 1993;161–167.

106. Lumb W. Euthanasia by noninhalant pharmacologic agents. *J Am Vet Med Assoc* 1974;165:851–852.

107. Barbiturates. In: Ciganovich E, ed. *Field manual of wildlife diseases*. US Department of the Interior/US Geological Survey, Biological Resources Division, Information and Technical Report 1999-2001.

108. Dennis MB, Dong WK, Weisbrod KA, et al. Use of captive bolt as a method of euthanasia in larger laboratory animal species. *Lab Anim Sci* 1988;38:459–462.

109. Blackmore DK. Energy requirements for the penetration of heads of domestic stock and the development of a multiple projectile. *Vet Rec* 1985;116:36–40.

110. Daly CC, Whittington PE. Investigation into the principal determinants of effective captive bolt stunning of sheep. *Res Vet Sci* 1989;46:406–408.

111. Clifford DH. Preanesthesia, anesthesia, analgesia, and euthanasia. In: Fox JG, Cohen BJ, Loew FM, eds. *Laboratory animal medicine*. New York: Academic Press Inc, 1984;528–563.

112. Australian Veterinary Association. Guidelines on humane slaughter and euthanasia. *Aust Vet J* 1987;64:4–7.

113. Carding T. Euthanasia of dogs and cats. *Anim Reg Stud* 1977;1:5–21.

114. Longair JA, Finley GG, Laniel M-A, et al. Guidelines for euthanasia of domestic animals by firearms. *Can Vet J* 1991;32: 724–726.

115. Finnie JW. Neuroradiological aspects of experimental traumatic missle injury in sheep. *N Z Vet J* 1994;42:54–57.

116. Blackmore DK, Madie P, Bowling MC, et al. The use of a shotgun for euthanasia of stranded cetaceans. *N Z Vet J* 1995; 43:158–159.

117. Blackmore DK, Bowling MC, Madie, P, et al. The use of a shotgun for emergency slaughter or euthanasia of large mature pigs. *N Z Vet J* 1995;43:134–137.

118. Denicola AJ. Non-traditional techniques for management of overabundant deer populations. *Wildl Soc Bull* 1997;25:496–499.

119. McAninch JB, ed. Urban deer: a manageable resource? in *Proceedings*. Symp 55th Midwest Fish Wildl Conf 1993;1–175.

120. Finnie JW. Traumatic head injury in ruminant livestock. *Aust Vet J* 1997;75:204–208.

121. Blackmore DK, Daly CC, Cook CJ. Electroencephalographic studies on the nape shooting of sheep. *N Z Vet J* 1995;43:160–163.

122. *On-farm euthanasia of swine—options for the producer*. Perry, Iowa: American Association of Swine Practitioners and Des Moines, Iowa: National Pork Producers, 1997.

123. *Practical euthanasia of cattle: considerations for the producer, livestock market operator, livestock transporter, and veterinarian*. Rome, Ga: American Association of Bovine Practitioners, 1999.

124. *The emergency euthanasia of horses*. Sacramento: California Department of Food and Agriculture and Davis, Calif: University of California's Veterinary Medical Extension, 1999.

125. *The emergency euthanasia of sheep and goats*. Sacramento: California Department of Food and Agriculture and Davis, Calif: University of California's Veterinary Medical Extension, 1999.

126. Gregory NG, Wotton SB. Comparison of neck dislocation and percussion of the head on visual evoked responses in the chicken's brain. *Vet Rec* 1990;126:570–572.

127. Vanderwolf CH, Buzak DP, Cain RK, et al. Neocortical and hippocampal electrical activity following decapitation in the rat. *Brain Res* 1988;451:340–344.

128. Derr RF. Pain perception in decapitated rat brain. *Life Sci* 1991;49:1399–1402.

129. Holson RR. Euthanasia by decapitation: evidence that this technique produces prompt, painless unconsciousness in laboratory rodents. *Neurotoxicol Teratol* 1992;14:253–257.

130. Keller GL. Physical euthanasia methods. *Lab Anim* 1982;11:20–26.

131. Feldman DB, Gupta BN. Histopathologic changes in laboratory animals resulting from various methods of euthanasia. *Lab Anim Sci* 1976;26:218–221.

132. Mikeska JA, Klemm WR. EEG evaluation of humaneness of asphyxia and decapitation euthanasia of the laboratory rat. *Lab Anim Sci* 1975;25:175–179.

133. Warrington R. Electrical stunning, a review of the literature. *Vet Bull* 1974;44:617–628.

134. Lambooy E, van Voorst N. Electrocution of pigs with notifiable diseases. *Vet Q* 1986;8:80–82.

135. Loftsgard G, Rraathen S, Helgebostad A. Electrical stunning of mink. *Vet Rec* 1972;91:132–134.

136. Hatch RC. Euthanatizing agents. In: Booth NH and McDonald LE, eds. *Veterinary pharmacology and therapeutics*.6th ed. Ames, Iowa: Iowa State University Press, 1988;1143–1148.

137. Croft PG, Hume CW. Electric stunning of sheep. *Vet Rec* 1956;68:318–321.

138. Roberts TDM. Electrocution cabinets. *Vet Rec* 1974;95:241–242.

139. Roberts TDM. Cortical activity in electrocuted dogs. *Vet Rec* 1954;66:561–567.

140. Anil MH, McKinstry JL. Reflexes and loss of sensibility following head-to-back electrical stunning in sheep. *Vet Rec* 1991;128:106–107.

141. Stavinoha WR. Study of brain neurochemistry utilizing rapid inactivation of brain enzyme activity by heating and mirowave irradiation. In: Black CL, Stavinoha WB, Marvyama Y, eds. *Microwave irradiation as a tool to study labile metabolites in tissue*. Elmsford, NY: Pergamon Press, 1983;1–12.

142. Stavinoha WB, Frazer J, Modak AT. Microwave fixation for the study of acetylcholine metabolism. In: Jenden DJ, ed. *Cholinergic mechanisms and psychopharmacology*. New York: Plenum Publishing Corp, 1978;169–179.

143. Ikarashi Y, Marvyama Y, Stavinoha WB. Study of the use of the microwave magnetic field for the rapid inactivation of brain enzymes. *Jpn J Pharmacol* 1984;35:371–387.

144. Gaunt AS, Oring LW. *Guidelines to the use of wild birds in research*. Washington DC: The Ornithological Council, 1997;1–52.

145. Federal Provincial Committee for Humane Trapping. *Final report: committee of the federal provincial wildlife conference*. Ottawa: Canadian Wildl Service, 1981;1–172.

146. *Agreement on international humane trapping standards*. The European Community, the Government of Canada, and the Government of the Russian Federation. Department of Foreign Affairs and International Trade, 1997;1–32.

147. Canadian General Standards Board. *Animal (mammal) traps—mechanically powered, trigger-activated killing traps for use on land*. No. CAN/CGSB-144.1-96. Ottawa: Canadian General Standards Board, 1996;1–36.

148. Nolan JW, Barrett MW. *Description and operation of the humane trapping research facility at the Alberta Environmental Centre, AECV90-R3*. Vegreville, AB: Alberta Environmental Centre, 1990.

149. *Animal (mammal) traps-part 4: methods for testing killing trap systems used on land or underwater, TC 191, ISO/DIS 10990-4E*. International Standardization Organization, 2000;1–15.

150. Gilbert FF. Assessment of furbearer response to trapping devices. In: Chapman JA, Pursley D, eds. *Worldwide furbearer conference proceedings*. Frostburg, Md: 1981;1599–1611.

151. Proulx G, Barrett MW. Evaluation of the Bionic Trap to quickly kill mink (*Mustela vison*) in simulated natural environments. *J Wildl Dis* 1991;27:276–280.

152. Proulx G, Barrett MW. Field testing of the C120 magnum trap for mink. *Wildl Soc Bull* 1993;21:421–426.

153. Hiltz M, Roy LD. Rating killing traps against humane trapping standards using computer simulations, in *Proceedings*. 19th Vertebrate Pest Conf 2000.

154. Proulx G, Barret M. Evaluation of the Bionic Trap to quickly kill fisher (*Martes pennanti*) in simulated natural environments. *J Wildl Dis* 1993;29:310–316.

155. Proulx G, Pawlina IM, Wong RK. Re-evaluation of the C120 magnum and bionic traps to humanely kill mink. *J Wildl Dis* 1993;29:184.

156. Proulx G, Barrett MW, Cook SR. The C120 Magnum with pan trigger: a humane trap for mink (*Mustela vison*). *J Wildl Dis* 1990;26:511–517.

157. Proulx G, Kolenosky AJ, Cole PJ. Assessment of the Kania trap to humanely kill red squirrels (*Tamiasciurus hudsonicus*) in enclosures. *J Wildl Dis* 1993;29:324–329.

158. Proulx G, Kolenosky AJ, Badry MJ, et al. Assessment of the Savageau 2001-8 trap to effectively kill arctic fox. *Wildl Soc Bull* 1993;21:132–135.

159. Proulx G, Kolenosky AJ, Cole PJ, et al. A humane killing trap for lynx (*Felis lynx*): the Conibear 330 with clamping bars. *J Wildl Dis* 1995;1:57–61.

160. Proulx G, Barret MW, Cook SR. The C120 Magnum: an effective kill trap for marten. *Wildl Soc Bull* 1989;17:294–298.

161. Proulx G, Cook SR, Barrett MW. Assessment and preliminary development of the rotating jaw Conibear 120 trap to effectively kill marten (*Martes americana*). *Can J Zool* 1989;67:1074–1079.

162. Naylor BJ, Novak M. Catch efficiency and selectivity of various traps and sets used for capturing American martens. *Wildl Soc Bull* 1994;22:489–496.

163. Hill EP. *Evaluation of improved traps and trapping techniques*. Alabama Department of Conservation and Natural Resources P-R Project Report W-44-5 Job IV-B:1-19.

164. King CM. The effects of two types of steel traps upon captured stoats (*Mustela erminea*). *J Zool (Lond)* 1995;553–554.

165. Cooper JE, Ewbank R, Platt C, et al. *Euthanasia of amphibians and reptiles*. London: UFAQ/WSPA, 1989.

166. Twitchell C, Roy LD, Gilbert FF, et al. Effectiveness of rotating-jaw killing traps for beaver (*Castor canadensis*), in *Proceedings*. North Am Aquatic Furbearer Symp 1999.

167. Warburton B, Hall JV. Impact momentum and clamping force thresholds for developing standards for possum kill traps. *N Z J Zool* 1995;22:39–44.

168. Guidelines for the capture, handling, and care of mammals as approved by the American Society of Mammalogists. *J Mammal* 1998;79:1416–1431.

169. *Improving animal welfare in US trapping programs*. Washington, DC: International Association of Fish and Wildlife Agencies, 1997.

170. Blackmore DK. Differences in behaviour between sheep and cattle during slaughter. *Res Vet Sci* 1984;37:223–226.

171. Gregory NG, Wotton SB. Time to loss of brain responsiveness following exsanguination in calves. *Res Vet Sci* 1984;37:141–143.

172. Blackmore DK. Non-penetrative percussion stunning of sheep and calves. *Vet Rec* 1979;105:372–375.

173. Canadian Council on Animal Care. *Guide to the care and use of experimental animals*. Vol 1. Ottawa: Canadian Council on Animal Care, 1980.

174. Green CJ. Euthanasia. In: *Animal anaesthesia*. London: Laboratory Animals Ltd, 1979;237-241.

175. Clifford DH. Preanesthesia, anesthesia, analgesia, and euthanasia. In: Fox JG, Cohen BJ, Loew FM, eds. *Laboratory animal medicine*. Orlando: Academic Press Inc, 1984;527–562.

176. Finnie JW. Neuropathologic changes produced by non-penetrating percussive captive bolt stunning of cattle. *N Z Vet J* 1995;43:183–185.

177. Gregory NG, Wotton SB. Effect of slaughter on spontaneous and evoked activity of the brain. *Br Poult Sci* 1986;27:195–205.

178. Eikelenboom G, ed. *Stunning of animals for slaughter*. Boston: Martinus Nijhoff Publishers, 1983;1–227.

179. Booth NH. Drug and chemical residues in the edible tissues of animals. In: Booth NH, McDonald LE, eds. *Veterinary pharmacology and therapeutics*. 6th ed. Ames, Iowa: Iowa State University Press, 1988;1149–1205.

180. Acceptable field methods in mammalogy: preliminary guidelines approved by the American Society of Mammalogists. *J Mammal* 1987;68(Suppl 4):1–18.

181. American Ornithologists' Union. Report of committee on use of wild birds in research. *Auk* 1988;105(Suppl):1A–41A.

182. American Society of Ichthyologists and Herpetologists, Herpetologist League, Society for the Study of Amphibians and Reptiles. Guidelines for the use of live amphibians and reptiles in field research. *J Herpetol* 1987;21(suppl 4):1–14.

183. American Society of Ichthyologists and Herpetologists, American Fisheries Society, American Institute of Fisheries Research Biologists. Guidelines for use of fishes in field research. *Copeia Suppl* 1987;1–12.

184. Cailliet GM. *Fishes: a field guide and laboratory manual on their structure, identification, and natural history*. Belmont, Calif: Wadsworth, 1986.

185. Schwartz JA, Warren R, Henderson D, et al. Captive and field tests of a method for immobilization and euthanasia of urban deer. *Wildl Soc Bull* 1997;25:532–541.

186. Zwart P, deVries HR, Cooper JE. The humane killing of fishes, amphibia, reptiles and birds. *Tijdsehr Diergeneeskd* 1989;114:557–565.

187. Burns R. Considerations in the euthanasia of reptiles, fish and amphibians, in *Proceedings*. AAZV, WDA, AAWV Joint Conference 1995;243–249.

188. National Research Committee on Pain and Distress in Laboratory Animals. *Recognition of pain and distress in laboratory animals*. Washington DC: National Academy Press, 1992.

189. Heard DJ. Principles and techniques of anesthesia and analgesia for exotic practice. *Vet Clin North Am Small Anim Pract* 1993;23:1301–1327.

190. Canadian Council on Animal Care. *Guide to the use and care of experimental animals*. Vol 2. Ottawa: Association of Universities and Colleges of Canada, 1984;1–16.

191. Harrell L. Handling euthanasia in production facilities. In: Schaeffer DO, Kleinow KM, Krulisch L, eds. *The care and use of amphibians, reptiles and fish in research*. Bethesda, Md: Scientists Center for Animal Welfare, 1992;129.

192. Ferguson HW. *Systemic pathology of fish*. Ames, Iowa: Iowa State University Press, 1989.

193. Letcher J. Intracelomic use of tricaine methane sulfonate for anesthesia of bullfrogs (*Rana catesbeiana*) and leopard frogs (*Rana pipens*). *Zoo Biol* 1992;11:242–251.

194. Brown LA. Anesthesia in fish. *Vet Clin North Am Small Anim Pract* 1988;18:317–330.

195. Josa A, Espinosa E, Cruz JI, et al. Use of 2-phenoxyethanol as an anesthetic agent in goldfish (*Cyprinus carpio*). *Vet Rec* 1992;131:468.

196. Noga EJ. *Fish disease. Diagnosis and treatment*. St Louis: Mosby, 1996.

197. Brannian RE, Kirk E, Williams D. Anesthetic induction of kinosternid turtles with halothane. *J Zoo Anim Med* 1987;18:115–117.

198. Calderwood HW. Anesthesia for reptiles. *J Am Vet Med Assoc* 1971;159:1618–1625.

199. Jackson OF, Cooper JE. Anesthesia and surgery. In: Cooper JE, Jackson OF, eds. *Diseases of the reptilia*. Vol. 2. New York: Academic Press Inc, 1981;535–549.

200. Johlin JM, Moreland FB. Studies of the blood picture of the turtle after complete anoxia. *J Biol Chem* 1933;103:107–114.

201. Moberly WR. The metabolic responses of the common iguana, *Iguana iguana*, to walking and diving. *Comp Biochem Physiol* 1968;27:21–32.

202. Storey KB. Life in a frozen state: adaptive strategies for natural freeze tolerance in amphibians and reptiles. *Am J Physiol* 1990;258:R559–R568.

203. Burns R, McMahan B. Euthanasia methods for ectothermic vertebrates. In: Bonagura JD, ed. *Continuing veterinary therapy XII*. Philadelphia: WB Saunders Co, 1995;1379–1381.

204. Cooper JE, Ewbank R, Platt C, et al. *Euthanasia of amphib-*

ians and reptiles. London: Universities Federation for Animal Welfare and World Society for the Protection of Animals, 1989.

205. Zwart P, deVries HR, Cooper JE. Humane methods of killing fish, amphibians and birds. *Tijdschr Diergeneedkd* 1989;114:557–565.

206. Martin B. Evaluation of hypothermia for anesthesia in reptiles and amphibians. *ILAR News* 1995;37:186–190.

207. Suckow MA, Terril LA, Grigdesby CF, et al. Evaluation of hypothermia-induced analgesia and influence of opioid antagonists in Leopard frogs (*Rana pipiens*). *Pharmacol Biochem Behav* 1999;63: 39–43.

208. Schaffer DO. Anesthesia and analgesia in nontraditional laboratory animal species. In: Kohn DF, Wixson SK, White WJ, et al. eds. *Anesthesia and analgesia in laboratory animals*. San Diego: Academic Press Inc, 1997;337–378.

209. Greer LL, Rowles T. Euthanasia. In: Dierauf LA, ed. *CRC handbook of marine mammal medicine: health, disease, and rehabilitation*. 2nd ed. Boca Raton, Fla: CRC Press, in press.

210. Blackmore DK, Madie P, Bowling MC, et al. The use of a shotgun for euthanasia of stranded cetaceans. *N Z Vet J* 1995;43:158–159.

211. Hyman J. Euthanasia in marine animals. In: Dierauf LA, ed. *CRC handbook of marine mammal medicine: health, disease, and rehabilitation*. Boca Raton, Fla: CRC Press, 1990;265–266.

212. Lambooy E, Roelofs JA, Van Voorst N. Euthanasia of mink with carbon monoxide. *Vet Rec* 1985;116:416.

213. *Recommended code of practice for the care and handling of mink*. Ottawa: Agriculture Canada, 1988;1–17.

214. Singer D. Neonatal tolerance to hypoxia: a comparative-physiological approach. *Comp Biochem Physiol* 1999;123:221–234.

215. Ludders JW, Schmidt RH, Dein J, et al. Drowning is not euthanasia. *Wildlife Soc Bull* 1999;27(3):1.

Appendix 1

Agents and methods of euthanasia by species (refer to Appendix 4 for unacceptable agents and methods.)

Species	Acceptable* (refer to Appendix 2 and text for details)	Conditionally acceptable† (refer to Appendix 3 and text for details)
Amphibians	Barbiturates, inhalant anesthetics (in appropriate species), CO_2, CO, tricaine methane sulfonate (TMS, MS 222), benzocaine hydrochloride, double pithing	Penetrating captive bolt, gunshot, stunning and decapitation, decapitation and pithing
Birds	Barbiturates, inhalant anesthetics, CO_2, CO, gunshot (free-ranging only)	N_2, Ar, cervical dislocation, decapitation, thoracic compression (small, free-ranging only)
Cats	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar
Dogs	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, penetrating captive bolt, electrocution
Fish	Barbiturates, inhalant anesthetics, CO_2, tricaine methane sulfonate (TMS, MS 222), benzocaine hydrochloride, 2-phenoxyethanol	Decapitation and pithing, stunning and decapitation/pithing
Horses	Barbiturates, potassium chloride in conjunction with general anesthesia, penetrating captive bolt	Chloral hydrate (IV, after sedation), gunshot, electrocution
Marine mammals	Barbiturates, etorphine hydrochloride	Gunshot (cetaceans < 4 meters long)
Mink, fox, and other mammals produced for fur	Barbiturates, inhalant anesthetics, CO_2 (mink require high concentrations for euthanasia without supplemental agents), CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, electrocution followed by cervical dislocation
Nonhuman primates	Barbiturates	Inhalant anesthetics, CO_2, CO, N_2, Ar
Rabbits	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, cervical dislocation (< 1 kg), decapitation, penetrating captive bolt
Reptiles	Barbiturates, inhalant anesthetics (in appropriate species), CO_2 (in appropriate species)	Penetrating captive bolt, gunshot, decapitation and pithing, stunning and decapitation
Rodents and other small mammals	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia, microwave irradiation	Methoxyflurane, ether, N_2, Ar, cervical dislocation (rats < 200 g), decapitation
Ruminants	Barbiturates, potassium chloride in conjunction with general anesthesia, penetrating captive bolt	Chloral hydrate (IV, after sedation), gunshot, electrocution
Swine	Barbiturates, CO_2, potassium chloride in conjunction with general anesthesia, penetrating captive bolt	Inhalant anesthetics, CO, chloral hydrate (IV, after sedation), gunshot, electrocution, blow to the head (< 3 weeks of age)
Zoo animals	Barbiturates, inhalant anesthetics, CO_2, CO, potassium chloride in conjunction with general anesthesia	N_2, Ar, penetrating captive bolt, gunshot
Free-ranging wildlife	Barbiturates IV or IP, inhalant anesthetics, potassium chloride in conjunction with general anesthesia	CO_2, CO, N_2, Ar, penetrating captive bolt, gunshot, kill traps (scientifically tested)

*Acceptable methods are those that consistently produce a humane death when used as the sole means of euthanasia. †Conditionally acceptable methods are those that by the nature of the technique or because of greater potential for operator error or safety hazards might not consistently produce humane death or are methods not well documented in the scientific literature.

Continued on next page.

Appendix 2

Acceptable agents and methods of euthanasia—characteristics and modes of action (refer to text for details)

Agent	Classification	Mode of action	Rapidity	Ease of performance	Safety for personnel	Species suitability	Efficacy and comments
Barbiturates	Hypoxia attributable to depression of vital centers	Direct depression of cerebral cortex, subcortical structures, and vital centers; direct depression of heart muscle	Rapid onset of anesthesia	Animal must be restrained; personnel must be skilled to perform IV injection	Safe except human abuse potential; DEA-controlled substance	Most species	Highly effective when appropriately administered; acceptable IP in small animals and IV
Benzocaine hydrochloride	Hypoxia attributable to depression of vital centers	Depression of CNS	Very rapid, depending on dose	Easily used	Safe	Fish, amphibians	Effective but expensive
Carbon dioxide (bottled gas only)	Hypoxia attributable to depression of vital centers	Direct depression of cerebral cortex, subcortical structures, and vital centers; direct depression of heart muscle	Moderately rapid	Used in closed container	Minimal hazard	Small laboratory animals, birds, cats, small dogs, rabbits, mink (high concentrations required), zoo animals, amphibians, fish, some reptiles, swine	Effective, but time required may be prolonged in immature and neonatal animals
Carbon monoxide (bottled gas only)	Hypoxia	Combines with hemoglobin, preventing its combination with oxygen	Moderate onset time, but insidious so animal is unaware of onset	Requires appropriately maintained equipment	Extremely hazardous, toxic, and difficult to detect	Most small species including dogs, cats, rodents, mink, chinchillas, birds, reptiles, amphibians, zoo animals, rabbits	Effective; acceptable only when equipment is properly designed and operated
Inhalant anesthetics	Hypoxia attributable to depression of vital centers	Direct depression of cerebral cortex, subcortical structures, and vital centers	Moderately rapid onset of anesthesia, excitation may develop during induction	Easily performed with closed container; can be administered to large animals by means of a mask	Must be properly scavenged or vented to minimize exposure to personnel	Some amphibians, birds, cats, dogs, furbearing animals, rabbits, some reptiles, rodents and other small mammals, zoo animals, fish, free-ranging wildlife	Highly effective provided that subject is sufficiently exposed; either is conditionally acceptable
Microwave irradiation	Brain enzyme inactivation	Direct inactivation of brain enzymes by rapid heating of brain	Very rapid	Requires training and highly specialized equipment	Safe	Mice, rats	Highly effective for special needs
Penetrating captive bolt	Physical damage to brain	Direct concussion of brain tissue	Rapid	Requires skill, adequate restraint, and proper placement of captive bolt	Safe	Horses, ruminants, swine	Instant loss of consciousness, but motor activity may continue
2-Phenoxyethanol	Hypoxia attributable to depression of vital centers	Depression of CNS	Very rapid, depending on dose	Easily used	Safe	Fish	Effective but expensive
Potassium chloride (intracardially or intravenously in conjunction with general anesthesia only)	Hypoxia	Direct depression of cerebral cortex, subcortical structures, and vital centers secondary to cardiac arrest.	Rapid	Requires training and specialized equipment for remote injection anesthesia, and ability to give IV injection of potassium chloride	Anesthetics may be hazardous with accidental human exposure	Most species	Highly effective, some clonic muscle spasms may be observed
Tricaine methane sulfonate (TMS, MS 222)	Hypoxia attributable to depression of vital centers	Depression of CNS	Very rapid, depending on dose	Easily used	Safe	Fish, amphibians	Effective but expensive

Appendix 3

Conditionally acceptable agents and methods of euthanasia—characteristics and modes of action (refer to text for details)

Agent	Classification	Mode of action	Rapidity	Ease of performance	Safety	Species suitability	Efficacy and comments
Blow to the head	Physical damage to brain	Direct concussion of brain tissue	Rapid	Requires skill, adequate restraint, and appropriate force	Safe	Young pigs < 3 weeks old	Must be properly applied to be humane and effective
Carbon dioxide (bottled gas only)	Hypoxia due to depression of vital centers	Direct depression of cerebral cortex, subcortical structures and vital centers; direct depression of heart muscle	Moderately rapid	Used in closed container	Minimal hazard	Nonhuman primates, free-ranging wildlife	Effective, but time required may be prolonged in immature and neonatal animals
Carbon monoxide (bottled gas only)	Hypoxia	Combines with hemoglobin, preventing its combination with oxygen	Moderate onset time, but insidious so animal is unaware of onset	Requires appropriately maintained equipment	Extremely hazardous, toxic, and difficult to detect	Nonhuman primates, free-ranging wildlife	Effective; acceptable only when equipment is properly designed and operated
Cervical dislocation	Hypoxia due to disruption of vital centers	Direct depression of brain	Moderately rapid	Requires training and skill	Safe	Poultry, birds, laboratory mice, rats (< 200 g), rabbits (< 1 kg)	Irreversible; violent muscle contractions can occur after cervical dislocation
Chloral hydrate	Hypoxia from depression of respiratory center	Direct depression of brain	Rapid	Personnel must be skilled to perform IV injection	Safe	Horses, ruminants, swine	Animals should be sedated prior to administration
Decapitation	Hypoxia due to disruption of vital centers	Direct depression of brain	Rapid	Requires training and skill	Guillotine poses potential employee injury hazard	Laboratory rodents; small rabbits; birds; some fish, amphibians, and reptiles (latter 3 with pithing)	Irreversible; violent muscle contraction can occur after decapitation
Electrocution	Hypoxia	Direct depression of brain and cardiac fibrillation	Can be rapid	Not easily performed in all instances	Hazardous to personnel	Used primarily in sheep, swine, foxes, mink (with cervical dislocation), ruminants, animals > 5 kg	Violent muscle contractions occur at same time as loss of consciousness
Gunshot	Hypoxia due to disruption of vital centers	Direct concussion of brain tissue	Rapid	Requires skill and appropriate firearm	May be dangerous	Large domestic and zoo animals, reptiles, amphibians, wildlife, cetaceans (< 4 meters long)	Instant loss of consciousness, but motor activity may continue
Inhalant anesthetics	Hypoxia due to depression of vital centers	Direct depression of cerebral cortex, subcortical structures, and vital centers	Moderately rapid onset of anesthesia; excitation may develop during induction	Easily performed with closed container; can be administered to large animals by means of a mask	Must be properly scavenged or vented to minimize exposure to personnel; ether has explosive potential and exposure to ether may be stressful	Nonhuman primates, swine; ether is conditionally acceptable for rodents and small mamals; methoxyflurane is conditionally acceptable for rodents and small mammals.	Highly effective provided that subject is sufficiently exposed
Nitrogen, argon	Hypoxia	Reduces partial pressure of oxygen available to blood	Rapid	Used in closed chamber with rapid filling	Safe if used with ventilation	Cats, small dogs, birds, rodents, rabbits, other small species, mink, zoo animals, nonhuman primates, free-ranging wildlife	Effective except in young and neonates; an effective agent, but other methods are preferable
Penetrating captive bolt	Physical damage to brain	Direct concussion of brain tissue	Rapid	Requires skill, adequate restraint and proper placement of captive bolt	Safe	Dogs, rabbits, zoo animals, reptiles, amphibians, free-ranging wildlife	Instant loss of consciousness but motor activity may continue
Pithing	Hypoxia due to disrution of vital centers, physical damage to brain	Trauma of brain and spinal cord tissue	Rapid	Easily performed but requires skill	Safe	Some ectotherms	Effective, but death not immediate unless brain and spinal cord are pithed
Thoracic compresion	Hypoxia and cardiac arrest	Physical interference with cardiac and respiratory function	Moderately rapid	Requires training	Safe	Small- to medium-sized free-ranging birds	Apparently effective

Appendix 4

Some unacceptable agents and methods of euthanasia (refer to text for details)

Agent or method	Comments
Air embolism	Air embolism may be accompanied by convulsions, opisthotonos, and vocalization. If used, it should be done only in anesthetized animals.
Blow to the head	Unacceptable for most species.
Burning	Chemical or thermal burning of an animal is not an acceptable method of euthanasia.
Chloral hydrate	Unacceptable in dogs, cats, and small mammals.
Chloroform	Chloroform is a known hepatotoxin and suspected carcinogen and, therefore, is extremely hazardous to personnel.
Cyanide	Cyanide poses an extreme danger to personnel and the manner of death is aesthetically objectionable.
Decompression	Decompression is unacceptable for euthanasia because of numerous disadvantages. (1) Many chambers are designed to produce decompression at a rate 15 to 60 times faster than that recommended as optimum for animals, resulting in pain and distress attributable to expanding gases trapped in body cavities. (2) Immature animals are tolerant of hypoxia, and longer periods of decompression are required before respiration ceases. (3) Accidental recompression, with recovery of injured animals, can occur. (4) Bleeding, vomiting, convulsions, urination, and defecation, which are aesthetically unpleasant, may develop in unconscious animals.
Drowning	Drowning is not a means of euthanasia and is inhumane.
Exsanguination	Because of the anxiety associated with extreme hypovolemia, exsanguination should be done only in sedated, stunned, or anesthetized animals.
Formalin	Direct immersion of an animal into formalin, as a means of euthanasia, is inhumane.
Household products and solvents	Acetone, quaternary compounds (including CCl_4), laxatives, clove oil, dimethylketone, quaternary ammonium products*, antacids, and other commercial and household products or solvents are not acceptable agents for euthanasia.
Hypothermia	Hypothermia is not an appropriate method of euthanasia.
Neuromuscular blocking agents (nicotine, magnesium sulafte, potassiumchloride, all curariform agents)	When used alone, these drugs all cause respiratory arrest before loss of consciousness, so the animal may perceive pain and distress after it is immobilized.
Rapid freezing	Rapid freezing as a sole means of euthanasia is not considered to be humane. If used, animals should be anesthetized prior to freezing.
Strychnine	Strychnine causes violent convulsions and painful muscle contractions.
Stunning	Stunning may render an animal unconscious, but it is not a method of euthanasia (except for neonatal animals with thin craniums). If used, it must be immediately followed by a method that ensures death.
Tricaine methane sulfonate (TMS, MS 222)	Should not be used for euthanasia of animals intended as food.

*Roccal D Plus, Pharmacia & Upjohn, Kalamazoo, Mich.

Effect of Rapid Decompression and Associated Hypoxic Phenomena in Euthanasia of animals : A Review

Nicholas H. Booth. DVM. PhD

SUMMARY

Documentation in the literature indicates that death is as painless following the induction of hypoxia by rapid decompression as by other methods that lead to hypoxia, such as exposure to high altitude, carbon monoxide, and inert gases (nitrogen, xenon, and krypton). Many of the signs and symptoms of hypoxia are the same as those for alcoholic intoxication and inert gas narcosis. Moreover, there is good evidence that analogous relationships or mechanisms may exist for hypoxia, inert gas narcosis, and anesthesia.

IN 1972 and 1978, reports of the AVMA Panel on Euthanasia [1,2] included the utilization of hypoxic procedures in euthanasia of animals. The reports covered the effects of carbon monoxide, nitrogen gas, rapid decompression, and respiratory paralyzing concentrations of anesthetics, all of which resuit in death by inducing an acute hypoxia or acute oxygen deficiency.

Controversy has arisen regarding the humaneness of using hypoxic methods of inducing euthanasia in animals, especially those involving use of rapid decompression or nitrogen gas. Consequently, some cities and states have passed legislation banning the use of decompression or nitrogen gas. Because of the increasing in-terest of individuals desiring documented information on whether or not decompression and other hypoxic methods are humane procedures of killing animals, relevant literature was assembled and is reviewed here.

Comparative Effects of Decompression, Alcoholic Intoxication, and Inert Gas Narcosis

TABLE 1—Altitude and Barometric Pressure Relationships Above SeaLevel

Altitude (ft above sea level)	Barometric pressure (mm of Hg)
0	760
2000	707
6000	609
10000	522
14000	446
18000	380*
22000	321
26000	270
30000	226
34000	187
38000	155
42000	128
46000	106
50000	87
54000	72
58000	60
63000	47**

* Equivalent to one-half the pressure at sea level. **Altitude that ebullition occurs, or equivalent to water vapor pressure in lungs.

Decompression produces hypoxic effects similar to those observed during ascent in climbing high mountains or in flying at high altitudes in unpressurized aircraft.[3] The higher the altitude the lower the ambient pressure and the more severe the hypoxia. The percentage composition of the various gases of the atmosphere, however, remains the same as at sea level.[4] For example, the percentage of O_2 at sea level and at any given altitude above sea level is 20.96.[4] At sea level, the ambient or barometric pressure is 760 mm of Hg, whereas at 55 000 ft above sea level, the pressure is 68.8 mm of Hg. Thus, the partial pressure of 0_2 at sea level is 760 X 0.2096 or 159 mm of Hg. At 55,000 ft, the partial pressure of O_2 is 68.8 x 0.2096 or only 14 mm of Hg (Table 1). The mean arterial blood of dog or man normally has an O_2 tension (P_{02},) of about 95 mm of Hg.[5] At 55,000 ft, the P_{02} (14 mm of Hg) is considerably below the physiologie level necessary to proper oxygenation of tissues. This low or deficient P_{02} results in severe hypoxia, unconsciousness, and rapid death.

'

TABLE 2—Comparative Potencies of Inert Gases and Gas Anesthetics Which Produce Equivalent Levels of Anesthesia or Neurologie Depression in Human Beings and Animals

Gas	Anesthetic pressure (ATA)*
Helium	>261
Neon	88
Nitrogen	29
Argon	20
Kryton	2,9
Nitrous oxide	0,9
Xenon	0,85
Diethyl ether	0,02
Chloroform	0,015
Halothane	0,008

Data from Miller et al.[59] and Saidman et al.[60]
ATA = Atmospheres absolute.

Ascent to high altitude and the resultant hypoxia may induce various effects such as excitement, exhilaration, and euphoria followed by headache, lassitude, sen-sory dullness, visual impairment, neuromuscular weak-ness, dyspnea, and loss of consciousness.[4] It is well known that aircraft pilots flying at high altitude and exposed to a low 0_2 environment will develop these hypoxic symptoms. Hypoxia may be so acute that loss of consciousness occurs rapidly without prior warning.[6 7]

All manifestations observed in alcoholic intoxication such as headache, drowsiness, severe respiratory depression and the associated O_2 deficiency, impaired vision, neuromuscular incoordination, and failure in mental tests also have been observed in human beings subjected to acute hypoxia[4] or exposure to decompression.[8] In all instances, these effiects are induced by an insufficient P_{O2}, to the brain. Hypoxia or a deficient P_{O2} should not be confused with suffocation, strangulation, or asphyxiation in which a deficiency in O_2 is combined with an increased $C0_2$ tension (hypercapnia) as that seen following the action of succinylcholine or *d*-tubocurarine[1] in paralysis of the respiratory musculature (intercostal muscles and diaphragm). Hypercapnia or suffocation is not a factor in ascent to high altitude or during decompression.

Interestingly, many of the signs and symptoms of hypoxia described here are the same as those for compression in air and for inert gas narcosis.[9] Narcosis induced in human beings by their compression in air was reported as early as the last century. Symptoms resembling alcoholic intoxication were observed in 1835 by Junod.[9] This adverse effect on mental perceptivity and on the ability of the human being to perform in compressed air can range from the euphoria first observed in caisson workers, to amnesia, dangerous hyperconfidence, difficulty in decision making, and lapses in consciousness in divers.[9] In 1935, it was learned that this compressed-air intoxication was due to the nitrogen content of air.[10] A narcotic effect occurs in human beings in air at 3 atmospheres and greater. Euphoria, retardation of the higher mental processes, and impaired neuromuscular function are observed.[10] The study of Behnke et al[10] led to the realization that nitrogen narcosis was just one example of a more general phenomenon also characteristic of other inert gases.[11-12] The difference between the narcotic actions of these gases is primarily one involving potency rather than the nature of the symptoms they elicit.[9] According to Hills and Ray,[9] the best index for quantitating this difference is probably provided by the « equinarcotic partial pressure » and can be extended to include gaseous anesthetics.

Values are available for an assortment of gases and provide a comparative basis for their relative narcotic potencies (Table 2). The more potent inert gas requires the smallest partial pressure in order to elicit the same degree of narcosis.[9] Such a comparison infers that inhalant anesthesia is an extension of inert gas narcosis; in fact, there is good evidence that an analogous relationship or mechanism exists in both conditions.[13]

Similar to the symptoms induced by decompression or alcoholic intoxication, manifestations of inert gas narcosis or compressed air narcosis include euphoria, loquacity, hallucination, temporary loss of memory, difficulty in assimilating facts or in making decisions, overconfidence, delayed response to visual, auditory, olfactory, and tactile stimuli, and impaired neuromuscular coordination leading to stupefaction and loss of consciousness.[9] Exposure to compressed air at 2 atmospheres absolute (ATA)* or 2 X 760 mm of Hg results in delta activity of the EEG.[13] At 7 ATA, signs and symptoms of « nitrogen narcosis » are evident in a large number of individuals, accompanied by a slight decrease in the amplitude of the alpha rhythm. At 10 ATA, this decrease is more marked and the signs of the narcosis are more severe. If the pressure is increased further, un-consciousness occurs.[13]

* ATA = Unit of pressure (760 mm of Hg) equal to the pressure of air at sea level at 0 C.

Major Effects Observed Following Exposure to Decompression

The effects of decompression on the dog are summarized as foliows [14 18]: Immediately after exposure to an ambient pressure of 30 mm of Hg, respiration becomes deep and rapid. This hyperventilation lasts for a matter of seconds. Marked abdominal distention occurs immediately. This is due to the expansion of gases present in the gastrointestinal tract. The animal collapses in about 8 seconds. Convulsions generally occur in from 10 to 12 seconds and last for several seconds. Decerebrate rigidity also may be observed. It occurs in animals following recompression or return to normal atmospheric pressure.[15] Following a convulsive seizure, the animal is quiescent except for occasional respiratory gasps which are ineffective in ventilating the lungs. Usually lacrimation, salivation, and urination occur.

In the monkey, gastric contents are suddenly and forcibly ejected at the time the animal is decompressed to altitudes above 55,000 ft.[19] Thirty to 40 seconds after the reduction of pressure, secondary swelling begins. This swelling occurs first in the rear limbs and lower abdomen and progresses headward. Animals will survive and completely recover if exposure to 30 mm of Hg is for less than 90 seconds. Exposures of 2 minutes or longer are usually fatal.

In the human being, pain from gas expansion in the gut has been uncommon during ascent in altitude, although most subjects notice a « boiling » sensation in the abdomen.[3] Some individuals have complained of pain presumably by esophageal origin following inadvertent attempts to eruct during ascent. In addition to abdominal pain prior to unconsciousness, generalized chest pain has been reported by human subjects a few seconds before loss of consciousness.[20]

Neurologie Influence of Decompression

Of the tissues in the body, nervous tissue is the least capable of withstanding the effects of hypoxia.[4] In the human being, acute hypoxia resembles alcoholic intoxication because of the marked O_2 deficiency and respiratory depression that develops. The symptoms are headache, mental disorientation, drowsiness, depressed res-piratory activity, neuromuscular weakness, and incoordination.[21] According to Van Liere,[21] « A person exposed to a low oxygen tension often passes through an initial stage of euphoria, accompanied by a feeling of self-satisfaction and a sense of power. The oxygen want stimulâtes the central nervous System so that the subject may become hilarious and sing or shout, and other emotional disturbances often manifest themselves. »

As exposure to low P_{O2} levels is increased, loss of consciousness occurs. An aircraft pilot exposed suddenly to an altitude of 45,000 ft above sea level will become unconscious in 13 to 16 seconds.[22] Unconsciousness can only be avoided if 100% O_2 is inspired within 5 to 7 seconds. Pilots subjected to 33,000 ft and breathing 100% O_2 and immediately exposed to 52,500 ft for less than 6 seconds and then recompressed to 33,000 ft do not lose consciousness.[23] If exposure is longer than 6 seconds, unconsciousness will occur even while breathing 100% O_2.

In the human being, temporary arrest of the circulation to the brain without affecting the respiratory tract has been accomplished by means of a specially de-signed inflatable cervical pressure cuff.[24] Characteristic reactions resulting from acute arrest of the circulation to the brain for 5 to 10 seconds are fixation of the eyeballs, blurring of vision, loss of consciousness, and hy-poxic convulsions. Loss of consciousness precedes the hypoxic convulsion. Convulsive seizures are of a generalized tonic and clonic type. Inasmuch as the convulsion is preceded by loss of consciousness, the person remains unconscious throughout the seizure and has no memory of it. Electroencephalographic recordings reveal the sudden appearance of large slow waves (delta waves) that are closely correlated with fixation of the eyes or loss of consciousness. Also, EEG and other electrical recordings have been made for human subjects made hypoxic by breathing nitrogen,[23-25] low 0_2 concentrations,[26] and in those decompressed to simulated altitudes of 45,000 ft.[22] In animals, electrical cortical activity of the brain has been recorded following hypoxemia [27] and decompression.[28]

The cerebral circulation has been arrested for as long as 100 seconds in human beings.[24] All subjects regain consciousness within 30 to 40 seconds after restoration of circulation. During the arrest, loss of consciousness, convulsions, marked cyanosis, involuntary urination and defecation, bradycardia, and dilation of pupils are observed.[24] These signs are comparable to those observed in animals following the induction of hypoxia by decompression.

In the dog, arrest of brain circulation for 6 minutes or less recover neurologie function, whereas those subjected to periods of circulatory arrest for 8 minutes or longer usually have permanent brain damage.[29] Urination frequently occurs during the first minute of circulatory arrest. Respiratory activity ceases 15 to 20 seconds after arrest of brain circulation in most animals This results in development of severe hypoxia.

During a period referred to as hyperactive coma following circulatory arrest, there are rapid running movements of all limbs, often accompanied by salivation and vocalization. These coordinated and rhythmic movements along with vocalization occur with the dog lying unconscious on its side.[29] Early in the period of hyperactive coma, extensor rigidity is seen, usually expressed as opisthotonos with the jaws closed tightly. During intervals between running movements, there is moderate extensor rigidity predominantly in the forelimbs.[29] Manifestations of the signs observed in dogs during the period of hyperactive coma are almost, if not identical, to what the author has seen in some dogs subjected to the early period of rapid decompression or exposure; lethal concentrations of carbon monoxide.

According to Kabat et al,[29] running movements during the period of hyperactive coma are similar to those that occur during recovery from barbiturate anesthesia. Veterinarians are well acquainted with these running movements and vocalization during the delirium period during recovery from pentobarbital sodium anesthesia.[30] The animal is comatose or unconscious during this period which is characteristic of stage-2 anesthesia.[30]

Pulmonary and Cardiovascular Influences of Decompression

The most consistent and outstanding response observed in animals (cat, dog, rat, rabbit, and guinea pig) following decompression is the development of abdominal distention.[31] Abdominal distention is greatest in the guinea pig and rabbit due to the relatively large amounts of gas normally present in the gastrointestinal tracts of these animals. As the distention increases, the diaphragm is forced up into the expiratory position, while the thorax is lifted into the inspiratory position. In the rabbit and guinea pig, these effects may be so prominent as to interfere seriously with, or actually prevent, respiratory movements. This distention and pressure build up inevitably interferes with blood returning to the heart by way of the caudal vena cava. A positive intra-abdominal pressure of the magnitude observed at a simulated altitude of 55,000 ft must be sufficient to interfere with venous return to the heart.[32] A marked reduction in venous return results in a decrease in cardiac output and prompt lowering of arterial pressure. This reduces the latent period of the hypoxic response since, in addition, the arterial pressure and blood flow to the brain and heart also are reduced. Hypoxia impairs the heart as a circulatory pump. Cardiovascular vascular depression is as prompt and the hypoxia as complete following decompression to 55,000 ft as at higher simulated altitudes.[32]

In dogs exposed to decompression, there is a rapid drop in systemic arterial pressure.[31] Also, in dogs decompressed to 30 mm of Hg (ie, equivalent to an altitude of 72,000 ft), circulation is completely stopped in less than 16 seconds after decompression.[16] This circulatory arrest results from vapor or bubbles due to the expansion of blood gases in the heart or vascular bed and corresponds to what an engineer refers to as vapor lock. Brief arrest of blood flow to the brain of the adult dog produces coma for 12 to 18 hours; after 6 minutes, for 24 hours or longer and; after 8 or more minutes, coma is permanent.[29]

More than 40 years ago, Lennox et al [33] reported that in human beings loss of consciousness occurs when O_2 saturation of the jugular venous blood drops to 24 % or below. The percentage 0_2 saturation bas been determined in the dog 30 seconds following decompression to various barometric pressures.[34] Decrease in percentage saturation does not occur until pressures less than 510 mm of Hg are attained. Oxygen saturation decreases sharply at barometric pressures between 510 mm of Hg and 50 mm of Hg. The percentage saturation is zero at 50 mm of Hg ambient pressure. At an ambient pressure less than 52 mm of Hg intravascular, bubbles are a frequent finding in the dog but bubbles are not found at higher pressures.[34]

Evaporation of body fluids may lower the oral temperature below freezing and also may lower the internal body temperature several degrees in less than 2 minutes in dogs subjected to near vacuum (1 mm of Hg) conditions.[35]

Cardiovascular responses of dogs to nitrogen breathing at ground level and to hypoxia at 55 mm of Hg absolute are quite similar.[36] The systemic arterial pressure drops, and pulmonic arterial pressure increases due to the hypoxia produced by nitrogen or decompression. Venous pressure increases following decompression [37] but remains within a normal range throughout the hypoxic episode during nitrogen breathing.[36] Apnea occurs sooner during decompression to 55 mm of Hg within an average of about 60 seconds compared with about 80 seconds for dogs breathing nitrogen. Bradycardia occurs following the hypoxic episodes produced by both nitrogen breathing and decompression to 55 mm of Hg. However, the heart rate decreases sooner and falls to lower levels following decompression compared with animals breathing nitrogen.

Decompression of anesthetized dogs to near vacuum (4 mm of Hg) for 60 seconds causes severe reduction of arterial blood flow.[38] Hemodynamic effects produced at 4 mm of Hg are attributable largely to mechanical obstruction of the cardiovascular system by increased extravascular pressures, resulting from gas expansion and especially vaporization of water.

The effects of hypoxia produced by decompression to a simulated altitude of 30,000 feet for 90 minutes s been studied in unanesthetized dogs.[39] A consistent result of decompression was a marked decrease in plasma-potassium concentration. Plasma sodium concentration remains unchanged.

Otologic Influence of Decompression

The effect of decompression on the middle ear of the monkey has been studied.[40] In the course of decompression at a slow rate (50 mm of Hg/min), the eustachian tube opened periodically to keep the tympanic pressure open to the ambient pressure. Periodic opening of the eustachian tube occurred only when the decompression rate was slow. When the rate of decompression is higher than 120 mm of Hg per minute, a sustained patency of the eustachian tube results. Even at excessive rates of decompression, such as seen during explosive decompression, the middle ear pressure very quickly returns to that of the ambient pressure.

Explosive decompression occurs at a rate many times faster than that used in rapid decompression. For example, explosive decompression can occur in about 12 to 40 msec with a drop in barometric pressure from 740 mm of Hg to 25 mm of Hg or less.[15.17] Rapid decompression may vary in time from 10,000 msec and upward.[3]

Evidence indicates that tympanic hemorrhage and pain are caused by negative pressure (> 600 mm of Hg) that develops in the middle ear during recompression whether the latter is gradual or explosive.[40] Hemorrhage in the frontal sinuses of dogs also has been observed and attributed to rapid recompression.[41]

Myringopuncture can prevent development of negative pressure and therefore can prevent the production of barotraumatic lesions to the ear. Puncture of both ear drums also completely eliminates bradycardia during recompression of the unanesthetized monkey brought down from 42,000 ft at a faster rate than free fall.[19] Apparently the bradycardia that occurs during recompression is due to the unequalized negative middle-ear pressure and is mediated reflexly by the vagus nerve. It has been suggested that impulses from receptors, possibly pain receptors, in the middle-ear or tympanic membrane, or both, initiate this reflex.

In human beings, ear discomfort and severe pain have been observed principally during recompression or upon descending to a lower altitude.[22.42.43] There are rare cases where barotrauma involving the ears or sinuses occur during ascent.[44] A predisposing factor in all such cases was upper respiratory infection. This is not surprising, for it is known that inflammation of the respiratory tract mucosa can interfere with ventilation of the middle ear and paranasal sinuses.[44]

Pathologie Effects Following Decompression

The gross pathologie lesions seen in dogs following decompression are hemorrhagic in nature.[45] Petechial to ecchymotic hemorrhages in the lungs occur. Cardiac damage occurs also with ecchymotic hemorrhages on the mitral valves of some animals. Ecchymotic hemorrhage occurs also under the dura mater encompassing the sagittal sinus of the brain.

Hemorrhagic lesions following decompression of the explosive type are found primarily in the lungs, brain, and heart.[45] Of these, the pulmonary lesions are most common.[45.46] It is thought that these lesions occur as a resuit of the sudden increase in intrapulmonary pressure during decompression. The sudden rapid expansion of the lungs with stretching of the alveolar walls probably results in tearing of these structures.

Residual histopathologic changes in the central nervous system of dogs have been described following rapid decompression to 1 mm of Hg for 120 seconds.[47]

Effect of Decompression and Other Hypoxic Episodes on Survival Time

Unconsciousness or collapse in adult dogs exposed to simulated altitudes between 50,000 and 55,000 ft, whether breathing air or 100 % O_2, occurs in less than 9 or 10 seconds following exposure.[14] « Complete anoxia » or « complete hypoxia » therefore occurs at these altitudes (ie, 52,500 ft) in animals breathing either air or 100% 0_2.[32.48.49] In human beings, the potentially severe hypoxia encountered above 50,000 ft begins to become effectively reversed at the 50,000-ft level, improving rapidly with continued recompression to 40,000 ft or lower.[8]

Studies in animals have shown that survival time decreases with increasing altitude as the severity of hypoxia increases.[50] However, the survival time reaches a minimum and remains constant regardless of further increase in altitude. The minimal survival time of animals exposed to rapid decompression has been studied in O_2 and in air by Lutz.[51] In animals breathing 0_2, Lutz found that a minimal survival time of 25 seconds was attained when animals were decompressed to a simulated altitude of 52,000 ft. Following the same procedure to altitudes below 52,000 ft the survival times were longer, and to altitudes above 52,000 ft the survival times did not become significantly shorter but remained approximately 25 seconds. In animals breathing air, Lutz observed that a minimal survival time of 25 seconds was reached on decompression to 43,000 ft or above.

The survival time of unanesthetized animals (rats) after decompression in air, when cessation of respiration is used as the end point, is constant for all simulated final decompression altitudes above 52,000 ft.[49] In the rat, at simulated altitudes of 52,000 and above, rhythmic respiration ceased on the average of 17,8 seconds after decompression in air. Studies on the effects of decompression of dogs and rats from sea level to 30 mm of Hg (ie, 72,000 ft) revealed that respiration ceased at about 30 seconds. Also, it is of interest and noteworthy that respiration in dogs ceases in 15 to 20 seconds after sudden complete arrest of the cerebral circulation.[29]

As exposure to high altitude and the accompanying hypoxic environment increases, resistance or tolerance to hypoxia becomes less.[50] Tolerance to high altitude or decompression appears to vary with various animal species. Compared with the guinea pig, the cat and dog are more tolerant.[52] Cats, rabbits, cavies, hamsters, rats, and mice fail to survive a decompression of 100 mm of Hg (ie, 47,000 ft) for 3 minutes.[53]

The respiratory center is most resistant to hypoxia at birth, then declines through the 4th month of life in the dog.[54] Resistance to hypoxia induced by nitrogen at birth varies from 28 minutes in the ground squirrel, to 16 minutes in the cat, to 6 minutes in the guinea pig.[55] The origin of hypoxic resistance in mammals has not been identified.

Adult rabbits can tolerate an anoxie atmosphere of 100 % nitrogen for only 1,5 minutes before death, whereas the newborn rabbit can survive for as long as 27 minutes.[56] In the adult dog, acute occlusion of the cerebral circulation and resultant hypoxia produce cessation of spontaneous respiration after only 20 to 30 seconds; in the 8- to 10-day-old puppy, this effect occurs in 5 minutes, and in the newborn animal, occurs in 27 minutes. Reptiles and amphibians can tolerate O_2 deprivation to a much greater extent than the mammalian species; for example, the turtle can tolerate anoxia produced by 100 % nitrogen for several hours and a dose of cyanide 50 times greater than that toxic to the mammal.[56 57]

Exposure of the dog to a near vacuum environment (less than 2 mm of Hg absolute) indicates that dogs exposed for less than 120 seconds are capable of survival upon recompression to 35,000 ft while breathing O_2.[14] In such animals, collapse occurs within 9 to 10 seconds after decompression along with a generalized muscle spasticity, a few gasps, momentary convulsive seizures, apnea, and gross swelling of the body and extremities.

Humane Considerations of Decompression

The rapid decompression technique for producing hypoxia (not the explosive decompression method) has been used for euthanasia of animals.[53.58] There have been many pathophysiologic studies involving the use of animals subjected to decompression. Most were conducted by high altitude or space research laboratories, so manned space flights could be accomplished with a minimum of hazard. Sufficient evidence as indicated by EEG recordings have revealed that hypoxia rapidly induces unconsciousness in both animals and man subjected to high altitude simulated by the use of decompression chambers or inhalation of inert gases. It is not known what the subjective perceptions of an animal in a chamber may be but when properly done, decompression is a painless procedure for all species [58] Decompression at the rate of 4,000 ft per minute for 10 minutes, thus creating a simulated altitude of 40,000 ft (141 mm of Hg), and maintaining this presure until respiration ceases are considered optimal for a mature dog.[58] For adults of other species such as cats, rabbits, cavies, hamsters, rats, and mice, a decompression of 100 mm of Hg (ie, 47,000 ft above sea lel) for 3 minutes is adequate for induction of euthanasia following a decompression rate of 15 mm of Hg per minute.[53]

As emphasized in the 1978 AVMA Panel on Euthanasia report,[2] the successful use of decompression chambers is predicated on the proper operation and maintenance of the equipment. Personnel operating the equipment must be skilled and knowledgeable in its use as well as understand the esthetically unpleasant reactions manifested by animals during the period of hyperactive coma or unconsciousness prior to death. Dogs under 4 months of age are more tolerant to hypoxia and require longer periods of decompression before respiration ceases.[54] Animals with respiratory complications and especially those with otitis media should not be subjected to decompression because of the possibility of the development of pain from unequalized positive middle-ear pressure.

References

1 AVMA Council on Research: Report of the AVMA panel on euthanasia. JAVMA 160:761-772, 1972.

2 AVMA Council on Research: Report of the AVMA panel on euthanasia. JAVMA 173:59-72, 1978.

3. Bryan CA, Leach WG: Physiologie effects of cabin pressure failure in high altitude passenger aircraft. *Aerospace Med* 31:267-275, 1960.

4 Van Liere EJ, Stickney JC: *Hypoxia*. Chicago, University of Chicago Press, 1963, pp 1-381.

5. Tenney SM: Respiration in mammals (chapter 15), in Swenson, J Melvin (ed) : *Dukes' Physiology of Domestic animals*, ed 9. Ithaca, NY, Constock Publishing Associates, Division of Cornell University Press, 1977, p 186.

6. Busby DE, Higgins EA, Funkhouser GE : Effect of physical activity of airline flight attendants on their time of useful consciousness in a rapid decompression. *Aviat Space Environ Med* 47:117-120, 1976.

7. Busby DE, Higgins EA, Funkhouser GE: Protection of airline flight attendants from hypoxia following rapid decompression. *Aviat Space Environ Med* 47:942-944, 1976.

8. Bancroft RW, Simmons DG: Rapid decompressions up to 60,000 feet wearing the standard oxygen mask. *Aerospace Med* 35:203-211, 1964.

9. Hills BA, Ray DE: Inert gas narcosis. *Pharmacol Ther* [B] 3:99-111, 1977.

10. Behnke AR, Thomson RM, Motley EP: The psychologic effects from breathing air at 4 atmospheres pressure. *Am J Physiol* 112:554-558, 1935.

11. Behnke AR, Yarbrough OD: Respiratory resistance, oil-water solubility, and mental effects of argon, compared with helium and nitrogen. *Am J Physiol* 126:409-415, 1939.

12. Lawrence JH, Loomis WF, Tobias CA, et al: Prelim-inary observations on the narcotic effect of xenon with a review of values for solubilities of gases in water and oils. *J Physiol* 105:197-204, 1946.

13. Bennett PB, Glass MB: Electroencephalographic and other changes induced by high partial pressures of nitrogen. *Electroencephalogr Clin Neurophysiol* 13:91-98, 1961.

14. Bancroft RW, Dunn JE II: Experimental animal de-compressions to a near vacuum environment. *Aerospace Med* 36:720-725, 1965.

15. Edelmann A, Hitchcock FA: Observations on dogs exposed to an ambient pressure of 30 mm Hg. *J Appl Physiol* 4:807-812, 1952.

16. Hitchcock FA, Kemph J: The boiling of body liquids at extremely high altitudes. *Aviat Med* 26:289-297, 1955.

17. Kemph JP, Beman FM, Hitchcock FA: Subcutaneous Pressure developed in dogs following explosive decompression to 25 or 30 mm Hg. *Am J Physiol* 168:601-604, 1952.

18. Kemph JP, Hitchcock FA: Further studies of effects of "igh intrapulmonic pressure on dogs at 30 mm Hg. *Aviat Med* 25:227-234, 1954.

19. Gelfan S: Explosive decompression of macaque monkeys w extreme altitudes and recompression at free-fall rates. *J Appl Physiol* 3:254-281, 1950.

20. Holmstrom FMG: Collapse during rapid decompression. Report of three cases. *J Aviat Med* 29:91-96, 1958.

21. Van Liere EJ: *Anoxia. Its Effect on the Body*. Chicago, University of Chicago Press, 1942, pp 1-269.

22. Barron CI, Cook TJ: Effects of variable decompressions to 45,000 feet. *Aerospace Med* 36:425-430, 1965.

23. Luft UC, Clamann HG, Opitz E: The latency of hypoxia on exposure to altitude above 50,000 feet. *J Aviat Med* 22 117-136, 1951.

24. Rossen R, Kabat H, Andersen JP: Acute arrest of cerclai circulation in man. *AMA Arch Neurol Psychiatr* 50:510-S28, 1943.

Sust I. 1978

25. Gibbs FA, Davis H: Changes in the human electroen-cephalogram associated with loss of consciousness. *Am J Physiol* 113:49-50, 1935.

26. Davis PA, Davis H, Thompson JW: Progressive changes in the human electroencephalogram under low oxygen tension. *Am J Physiol* 123:51-52, 1938.

27. Sugar O, Gerard RW: Anoxia and brain potential. *J Neurophysiol* 1:558-571, 1938.

28. Stephens LM, Hartman JL, Lewis OF, et al: Electro-physiology of chimpanzees during rapid decompression. *Aerospace Med* 38:694-698, 1967.

29. Kabat H, Dennis C, Baker AB: Recovery of function following arrest of the brain circulation. *Am J Physiol* 132: 737-747, 1941.

30. Booth NH: Intravenous and other parenteral anestlietics, in Jones LM, Booth NH, McDonald LE (ed) : *Veterinary Phar-macology and Therapeutics*. Ames, lowa State University Press, 1977, pp 241-306.

31. Whitehorn WV, Lein A, Edelmann A: The general tolerance and cardiovascular responses of animals to explosive de-compression. *Am J Physiol* 147:289-298, 1946.

32. Gelfan S, Werner AY: Cardiovascular responses following explosive decompression of macaque monkeys to extrême altitudes. *J Appl Physiol* 4:280-310, 1951.

33. Lennox WG, Gibbs FA, Gibbs EL: Relationship of un-consciousness to cerebral blood flow and to anoxemia. *AMA Arch Neurol Psychiatr* 34:1001-1013, 1935.

34. Kemph JP, Hitchcock FA: Changes in blood and circulation of dogs following explosive decompression to low baro-metric pressures. *Am J Physiol* 168:592-600, 1952.

35. Cooke JP, Bancroft RW: Some cardiovascular responses in anesthetized dogs during repeated decompressions to a near vacuum. *Aerospace Med* 37:1148-1152, 1966.

36. Bancroft RW, Cooke JP, Gain SM: Comparison of anoxia with and without ebullism. *J Appl Physiol* 25:230-237, 1968.

37. Cooke JP, Cain SM, Bancroft RW: High venous pressures during exposure of dogs to near vacuum conditions. *Aerospace Med* 38:1021-1024, 1967.

38. Pratt AJ, Stone HL, Stegall HF, et al: Circulatory im-pairment during exposure to ambient pressures of 4 mm Hg and 55 mm Hg. *J Appl Physiol* 29:177-180, 1970.

39. Ferguson FP, Smith DC: Effects of acute decompression stress upon plasma electrolytes and renal function in dogs. *Am J Physiol* 173:503-510, 1953.

40. Chang H-T, Margaria R, Gelfan S: Pressure changes and barotrauma resulting from decompression and recompression in the middle ear of monkeys. *Arch Otolaryngol* 51:378-399, 1950.

41. Cole CR, Chamberlain DM, Burch BH, et al: Pathological effects of explosive decompression to 30 mm Hg. *J Appl Physiol* 6:96-104, 1953.

42. Barron CI, Collier DR Jr, Cook TJ: Observations on simulated 12-second decompressions to 32,000 feet. *Aviat Med* 29:563-574, 1958.

43. Idicula J: Perplexing case of maxillary sinus barotrauma. *Aerospace Med* 43:891-892, 1972.

44. Lewis ST: Barotrauma in United States Air Force accidents/incidents. *Aerospace Med* 44:1059-1061, 1973.

45. Edelman A, Whitehorn WV, Lein A, et al: Pathological lesions produced by explosive decompression. *Aviat Med* 17-596-612, 1946.

46. Dunn JE II, Bancroft RW, Haymaker W, et al: Experimental animal decompressions to less than 2 mm Hg abso-lute (pathologie effects). *Aerospace Med* 36:725-732, 1965.

47. Casey HW, Bancroft RW, Cooke JP: Residual pathologie changes in the central nervous System of a dog following rapid decompression to 1 mm Hg. *Aerospace Med* 37:713-718, 1966.

48. Gelfan S, Nims LF, Livingston RB: Cause of death from explosive decompression at high altitude (abstr). *Fed Proc* 6: 110, 1947.

49. Gelfan S, Nims LF, Livingston RB: Explosive decompression at high altitude. Am J Physiol 162:37-53, 1950.

50. Armstrong HG: Anoxia in aviation. *Aviat Med* 9:84-91, 1938.

51. Luft UC, Clamann HG, Adler HF: Alveolar gases in rapid decompression to high altitudes. *J Appl Physiol* 2:37-48, 1949.

52. Whitehorn WV, Lein A, Hitchcock FA: The effect of explosive decompression on the occurrence of intravascular bub-bles. *Aviat Med* 18:392-394, 1947.

53. Barber BR: Use of a standard autoclave for decompression euthanasia. *J Institute Anim Technol* 23:106-110, 1972.

54. Kabat H: The greater resistance of very young animals to arrest of the brain circulation *Am J Physiol* 130:588-599, 1940.

55. Adolph EF: Regulations during survival without oxygen in infant mammals. *Respir Physiol* 7:356-368, 1969.

56. Cohen PJ: The metabolic function of oxygen and bio-chemical lesions of hypoxia. *Anesthesiology* 37:148-177, 1972.

57. Bellamy D, Peterson JA: Anaerobiosis and the toxicity of cyanide in turtles. *Comp Biochem Physiol* 24:543-548, 1968.

58. Smith DC: Methods ot euthanasla and disposaL of laboratory animals, in Gay WI (ed) : *Methods of Animal experimentation*. New York, Academie Press, 1965, vol I, pp 167wi-

59. Miller KW, Paton WDM, Smith EB: Site of action of general anesthetics. *Nature* 206:574-577, 1965.

60. Saidman LJ, Eger El II, Munson ES, et al: Minimum alveolar concentrations of methoxyflurane, halothane, ether, cyclopropane in man: Correlation with theories of anesthesia ? *Anesthesiology* 28:994-1002, 1967.

Effect of Rapid Decompression and Associated Hypoxic Phenomena in Euthanasia of Animals: A Review

Nicholas H. Booth, DVM, PhD

SUMMARY

Documentation in the literature indicates that death is as painless following the induction of hypoxia by rapid decompression as by other methods that lead to hypoxia, such as exposure to high altitude, carbon monoxide, and inert gases (nitrogen, xenon, and krypton). Many of the signs and symptoms of hypoxia are the same as those for alcoholic intoxication and inert gas narcosis. Moreover, there is good evidence that analogous relationships or mechanisms may exist for hypoxia, inert gas narcosis, and anesthesia.

In 1972 and 1978, reports of the AVMA Panel on Euthanasia[1,2] included the utilization of hypoxic procedures in euthanasia of animals. The reports covered the effects of carbon monoxide, nitrogen gas, rapid decompression, and respiratory paralyzing concentrations of anesthetics, all of which result in death by inducing an acute hypoxia or acute oxygen deficiency.

Controversy has arisen regarding the humaneness of using hypoxic methods of inducing euthanasia in animals, especially those involving use of rapid decompression or nitrogen gas. Consequently, some cities and states have passed legislation banning the use of decompression or nitrogen gas. Because of the increasing interest of individuals desiring documented information on whether or not decompression and other hypoxic methods are humane procedures of killing animals, relevant literature was assembled and is reviewed here.

Comparative Effects of Decompression, Alcoholic Intoxication, and Inert Gas Narcosis

Decompression produces hypoxic effects similar to those observed during ascent in climbing high mountains or in flying at high altitudes in unpressurized aircraft.[3] The higher the altitude the lower the ambient pressure and the more severe the hypoxia. The percentage composition of the various gases of the atmosphere, however, remains the same as at sea level.[4] For example, the percentage of O_2 at sea level and at any given altitude above sea level is 20.96.[4] At sea level, the ambient or barometric pressure is 760 mm of Hg, whereas at 55,000 ft above sea level, the pressure is 68.8 mm of Hg. Thus, the partial pressure of O_2 at sea level is 760×0.2096 or 159 mm of Hg. At 55,000 ft, the partial pressure of O_2 is 68.8×0.2096 or only 14 mm of Hg (Table 1). The mean arterial blood of dog or man normally has an O_2 tension (P_{O_2}) of about 95 mm of Hg.[5] At 55,000 ft, the P_{O_2} (14 mm of Hg) is considerably below the physiologic level necessary to maintain proper oxygenation of tissues. This low or deficient P_{O_2} results in severe hypoxia, unconsciousness, and rapid death.

Ascent to high altitude and the resultant hypoxia may induce various effects such as excitement, exhilara-

TABLE 1—Altitude and Barometric Pressure Relationships Above Sea Level

Altitude (ft above sea level)	Barometric pressure (mm of Hg)
0	760
2,000	707
6,000	609
10,000	522
14,000	446
18,000	380*
22,000	321
26,000	270
30,000	226
34,000	187
38,000	155
42,000	128
46,000	106
50,000	87
54,000	72
58,000	60
63,000	47†

* Equivalent to one-half the pressure at sea level. † Altitude that ebullition occurs, or equivalent to water vapor pressure in lungs.

From the Department of Physiology and Pharmacology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602.

TABLE 2—Comparative Potencies of Inert Gases and Gas Anesthetics Which Produce Equivalent Levels of Anesthesia or Neurologic Depression in Human Beings and Animals

Gas	Anesthetic pressure (ATA)*
Helium	> 261
Neon	88
Nitrogen	29
Argon	20
Krypton	2.9
Nitrous oxide	0.9
Xenon	0.85
Diethyl ether	0.02
Chloroform	0.015
Halothane	0.008

Data from Miller et al[50] and Saidman et al.[60] ATA = Atmospheres absolute.

tion, and euphoria followed by headache, lassitude, sensory dullness, visual impairment, neuromuscular weakness, dyspnea, and loss of consciousness.[4] It is well known that aircraft pilots flying at high altitude and exposed to a low O_2 environment will develop these hypoxic symptoms. Hypoxia may be so acute that loss of consciousness occurs rapidly without prior warning.[6,7]

All manifestations observed in alcoholic intoxication such as headache, drowsiness, severe respiratory depression and the associated O_2 deficiency, impaired vision, neuromuscular incoordination, and failure in mental tests also have been observed in human beings subjected to acute hypoxia[4] or exposure to decompression.[8] In all instances, these effects are induced by an insufficient P_{O_2} to the brain. Hypoxia or a deficient P_{O_2} should not be confused with suffocation, strangulation, or asphyxiation in which a deficiency in O_2 is combined with an increased CO_2 tension (hypercapnia) as that seen following the action of succinylcholine or *d*-tubocurarine[1] in paralysis of the respiratory musculature (intercostal muscles and diaphragm). Hypercapnia or suffocation is not a factor in ascent to high altitude or during decompression.

Interestingly, many of the signs and symptoms of hypoxia described here are the same as those for compression in air and for inert gas narcosis.[9] Narcosis induced in human beings by their compression in air was reported as early as the last century. Symptoms resembling alcoholic intoxication were observed in 1835 by Junod.[9] This adverse effect on mental perceptivity and on the ability of the human being to perform in compressed air can range from the euphoria first observed in caisson workers, to amnesia, dangerous hyperconfidence, difficulty in decision making, and lapses in consciousness in divers.[9] In 1935, it was learned that this compressed-air intoxication was due to the nitrogen content of air.[10] A narcotic effect occurs in human beings in air at 3 atmospheres and greater. Euphoria, retardation of the higher mental processes, and impaired neuromuscular function are observed.[10] The study of Behnke et al[10] led to the realization that nitrogen narcosis was just one example of a more general phenomenon also characteristic of other inert gases.[11,12] The difference between the narcotic actions of these gases is primarily one involving potency rather than the nature of the symptoms they elicit.[9] According to Hills and Ray,[9] the best index for quantitating this difference is probably provided by the "equinarcotic partial pressure" and can be extended to include gaseous anesthetics.

Values are available for an assortment of gases and provide a comparative basis for their relative narcotic potencies (Table 2). The more potent inert gas requires the smallest partial pressure in order to elicit the same degree of narcosis.[9] Such a comparison infers that inhalant anesthesia is an extension of inert gas narcosis; in fact, there is good evidence that an analogous relationship or mechanism exists in both conditions.[13]

Similar to the symptoms induced by decompression or alcoholic intoxication, manifestations of inert gas narcosis or compressed air narcosis include euphoria, loquacity, hallucination, temporary loss of memory, difficulty in assimilating facts or in making decisions, overconfidence, delayed response to visual, auditory, olfactory, and tactile stimuli, and impaired neuromuscular coordination leading to stupefaction and loss of consciousness.[9] Exposure to compressed air at 2 atmospheres absolute (ATA)[a] or 2 × 760 mm of Hg results in delta activity of the EEG.[13] At 7 ATA, signs and symptoms of "nitrogen narcosis" are evident in a large number of individuals, accompanied by a slight decrease in the amplitude of the alpha rhythm. At 10 ATA, this decrease is more marked and the signs of the narcosis are more severe. If the pressure is increased further, unconsciousness occurs.[13]

Major Effects Observed Following Exposure to Decompression

The effects of decompression on the dog are summarized as follows[14-18]: Immediately after exposure to an ambient pressure of 30 mm of Hg, respiration becomes deep and rapid. This hyperventilation lasts for a matter of seconds. Marked abdominal distention occurs immediately. This is due to the expansion of gases present in the gastrointestinal tract. The animal collapses in about 8 seconds. Convulsions generally occur in from 10 to 12 seconds and last for several seconds. Decerebrate rigidity also may be observed. It occurs in animals following recompression or return to normal atmospheric pressure.[15] Following a convulsive seizure, the animal is quiescent except for occasional respiratory gasps which are ineffective in ventilating the lungs. Usually lacrimation, salivation, and urination occur.

In the monkey, gastric contents are suddenly and forcibly ejected at the time the animal is decompressed to altitudes above 55,000 ft.[19] Thirty to 40 seconds after the reduction of pressure, secondary swelling begins. This swelling occurs first in the rear limbs and lower abdomen and progresses headward. Animals will survive and completely recover if exposure to 30 mm of Hg is for less than 90 seconds. Exposures of 2 minutes or longer are usually fatal.

In the human being, pain from gas expansion in the gut has been uncommon during ascent in altitude, although most subjects notice a "boiling" sensation in

[a] ATA = Unit of pressure (760 mm of Hg) equal to the pressure of air at sea level at 0 C.

the abdomen.[3] Some individuals have complained of pain presumably by esophageal origin following inadvertent attempts to eruct during ascent. In addition to abdominal pain prior to unconsciousness, generalized chest pain has been reported by human subjects a few seconds before loss of consciousness.[20]

Neurologic Influence of Decompression

Of the tissues in the body, nervous tissue is the least capable of withstanding the effects of hypoxia.[4] In the human being, acute hypoxia resembles alcoholic intoxication because of the marked O_2 deficiency and respiratory depression that develops. The symptoms are headache, mental disorientation, drowsiness, depressed respiratory activity, neuromuscular weakness, and incoordination.[21] According to Van Liere,[21] "A person exposed to a low oxygen tension often passes through an initial stage of euphoria, accompanied by a feeling of self-satisfaction and a sense of power. The oxygen want stimulates the central nervous system so that the subject may become hilarious and sing or shout, and other emotional disturbances often manifest themselves."

As exposure to low P_{O_2} levels is increased, loss of consciousness occurs. An aircraft pilot exposed suddenly to an altitude of 45,000 ft above sea level will become unconscious in 13 to 16 seconds.[22] Unconsciousness can only be avoided if 100% O_2 is inspired within 5 to 7 seconds. Pilots subjected to 33,000 ft and breathing 100% O_2 and immediately exposed to 52,500 ft for less than 6 seconds and then recompressed to 33,000 ft do not lose consciousness.[23] If exposure is longer than 6 seconds, unconsciousness will occur even while breathing 100% O_2.

In the human being, temporary arrest of the circulation to the brain without affecting the respiratory tract has been accomplished by means of a specially designed inflatable cervical pressure cuff.[24] Characteristic reactions resulting from acute arrest of the circulation to the brain for 5 to 10 seconds are fixation of the eyeballs, blurring of vision, loss of consciousness, and hypoxic convulsions. Loss of consciousness precedes the hypoxic convulsion. Convulsive seizures are of a generalized tonic and clonic type. Inasmuch as the convulsion is preceded by loss of consciousness, the person remains unconscious throughout the seizure and has no memory of it. Electroencephalographic recordings reveal the sudden appearance of large slow waves (delta waves) that are closely correlated with fixation of the eyes or loss of consciousness. Also, EEG and other electrical recordings have been made for human subjects made hypoxic by breathing nitrogen,[23,25] low O_2 concentrations,[26] and in those decompressed to simulated altitudes of 45,000 ft.[22] In animals, electrical cortical activity of the brain has been recorded following hypoxemia[27] and decompression.[28]

The cerebral circulation has been arrested for as long as 100 seconds in human beings.[24] All subjects regain consciousness within 30 to 40 seconds after restoration of circulation. During the arrest, loss of consciousness, convulsions, marked cyanosis, involuntary urination and defecation, bradycardia, and dilation of pupils are observed.[24] These signs are comparable to those observed in animals following the induction of hypoxia by decompression.

In the dog, arrest of brain circulation for 6 minutes or less recover neurologic function, whereas those subjected to periods of circulatory arrest for 8 minutes or longer usually have permanent brain damage.[29] Urination frequently occurs during the first minute of circulatory arrest. Respiratory activity ceases 15 to 20 seconds after arrest of brain circulation in most animals. This results in development of severe hypoxia.

During a period referred to as hyperactive coma following circulatory arrest, there are rapid running movements of all limbs, often accompanied by salivation and vocalization. These coordinated and rhythmic movements along with vocalization occur with the dog lying unconscious on its side.[29] Early in the period of hyperactive coma, extensor rigidity is seen, usually expressed as opisthotonos with the jaws closed tightly. During intervals between running movements, there is moderate extensor rigidity predominantly in the forelimbs.[29] Manifestations of the signs observed in dogs during the period of hyperactive coma are almost, if not identical, to what the author has seen in some dogs subjected to the early period of rapid decompression or exposure to lethal concentrations of carbon monoxide.

According to Kabat et al,[29] running movements during the period of hyperactive coma are similar to those that occur during recovery from barbiturate anesthesia. Veterinarians are well acquainted with these running movements and vocalization during the delirium period during recovery from pentobarbital sodium anesthesia.[30] The animal is comatose or unconscious during this period which is characteristic of stage-2 anesthesia.[30]

Pulmonary and Cardiovascular Influences of Decompression

The most consistent and outstanding response observed in animals (cat, dog, rat, rabbit, and guinea pig) following decompression is the development of abdominal distention.[31] Abdominal distention is greatest in the guinea pig and rabbit due to the relatively large amounts of gas normally present in the gastrointestinal tracts of these animals. As the distention increases, the diaphragm is forced up into the expiratory position, while the thorax is lifted into the inspiratory position. In the rabbit and guinea pig, these effects may be so prominent as to interfere seriously with, or actually prevent, respiratory movements. This distention and the pressure build up inevitably interferes with blood returning to the heart by way of the caudal vena cava. A positive intra-abdominal pressure of the magnitude observed at a simulated altitude of 55,000 ft must be sufficient to interfere with venous return to the heart.[32] A marked reduction in venous return results in a decrease in cardiac output and prompt lowering of arterial pressure. This reduces the latent period of the hypoxic response since, in addition, the arterial pressure and blood flow to the brain and heart also are reduced. Hypoxia impairs the heart as a circulatory pump. Cardiovascular depression is as prompt and the hypoxia as

complete following decompression to 55,000 ft as at higher simulated altitudes.[32]

In dogs exposed to decompression, there is a rapid drop in systemic arterial pressure.[31] Also, in dogs decompressed to 30 mm of Hg (ie, equivalent to an altitude of 72,000 ft), circulation is completely stopped in less than 16 seconds after decompression.[16] This circulatory arrest results from vapor or bubbles due to the expansion of blood gases in the heart or vascular bed and corresponds to what an engineer refers to as vapor lock. Brief arrest of blood flow to the brain of the adult dog produces coma for 12 to 18 hours; after 6 minutes, for 24 hours or longer and; after 8 or more minutes, coma is permanent.[29]

More than 40 years ago, Lennox et al[33] reported that in human beings loss of consciousness occurs when O_2 saturation of the jugular venous blood drops to 24% or below. The percentage O_2 saturation has been determined in the dog 30 seconds following decompression to various barometric pressures.[34] Decrease in percentage saturation does not occur until pressures less than 510 mm of Hg are attained. Oxygen saturation decreases sharply at barometric pressures between 510 mm of Hg and 50 mm of Hg. The percentage saturation is zero at 50 mm of Hg ambient pressure. At an ambient pressure less than 52 mm of Hg intravascular, bubbles are a frequent finding in the dog but bubbles are not found at higher pressures.[34]

Evaporation of body fluids may lower the oral temperature below freezing and also may lower the internal body temperature several degrees in less than 2 minutes in dogs subjected to near vacuum (1 mm of Hg) conditions.[35]

Cardiovascular responses of dogs to nitrogen breathing at ground level and to hypoxia at 55 mm of Hg absolute are quite similar.[36] The systemic arterial pressure drops, and pulmonic arterial pressure increases due to the hypoxia produced by nitrogen or decompression. Venous pressure increases following decompression[37] but remains within a normal range throughout the hypoxic episode during nitrogen breathing.[36] Apnea occurs sooner during decompression to 55 mm of Hg within an average of about 60 seconds compared with about 80 seconds for dogs breathing nitrogen. Bradycardia occurs following the hypoxic episodes produced by both nitrogen breathing and decompression to 55 mm of Hg. However, the heart rate decreases sooner and falls to lower levels following decompression compared with animals breathing nitrogen.

Decompression of anesthetized dogs to near vacuum (4 mm of Hg) for 60 seconds causes severe reduction of arterial blood flow.[38] Hemodynamic effects produced at 4 mm of Hg are attributable largely to mechanical obstruction of the cardiovascular system by increased extravascular pressures, resulting from gas expansion and especially vaporization of water.

The effects of hypoxia produced by decompression to a simulated altitude of 30,000 feet for 90 minutes has been studied in unanesthetized dogs.[39] A consistent result of decompression was a marked decrease in plasma-potassium concentration. Plasma sodium concentration remains unchanged.

Otologic Influence of Decompression

The effect of decompression on the middle ear of the monkey has been studied.[40] In the course of decompression at a slow rate (50 mm of Hg/min), the eustachian tube opened periodically to keep the tympanic pressure open to the ambient pressure. Periodic opening of the eustachian tube occurred only when the decompression rate was slow. When the rate of decompression is higher than 120 mm of Hg per minute, a sustained patency of the eustachian tube results. Even at excessive rates of decompression, such as seen during explosive decompression, the middle ear pressure very quickly returns to that of the ambient pressure.

Explosive decompression occurs at a rate many times faster than that used in rapid decompression. For example, explosive decompression can occur in about 12 to 40 msec with a drop in barometric pressure from 740 mm of Hg to 25 mm of Hg or less.[15,17] Rapid decompression may vary in time from 10,000 msec and upward.[3]

Evidence indicates that tympanic hemorrhage and pain are caused by negative pressure ($>$ 600 mm of Hg) that develops in the middle ear during recompression whether the latter is gradual or explosive.[40] Hemorrhage in the frontal sinuses of dogs also has been observed and attributed to rapid recompression.[41]

Myringopuncture can prevent development of negative pressure and therefore can prevent the production of barotraumatic lesions to the ear. Puncture of both ear drums also completely eliminates bradycardia during recompression of the unanesthetized monkey brought down from 42,000 ft at a faster rate than free fall.[19] Apparently the bradycardia that occurs during recompression is due to the unequalized negative middle-ear pressure and is mediated reflexly by the vagus nerve. It has been suggested that impulses from receptors, possibly pain receptors, in the middle-ear or tympanic membrane, or both, initiate this reflex.

In human beings, ear discomfort and severe pain have been observed principally during recompression or upon descending to a lower altitude.[22,42,43] There are rare cases where barotrauma involving the ears or sinuses occur during ascent.[44] A predisposing factor in all such cases was upper respiratory infection. This is not surprising, for it is known that inflammation of the respiratory tract mucosa can interfere with ventilation of the middle ear and paranasal sinuses.[44]

Pathologic Effects Following Decompression

The gross pathologic lesions seen in dogs following decompression are hemorrhagic in nature.[45] Petechial to ecchymotic hemorrhages in the lungs occur. Cardiac damage occurs also with ecchymotic hemorrhages on the mitral valves of some animals. Ecchymotic hemorrhage occurs also under the dura mater encompassing the sagittal sinus of the brain.

Hemorrhagic lesions following decompression of the explosive type are found primarily in the lungs, brain, and heart.[45] Of these, the pulmonary lesions are most common.[45,46] It is thought that these lesions occur as a result of the sudden increase in intrapulmonary pres-

sure during decompression. The sudden rapid expansion of the lungs with stretching of the alveolar walls probably results in tearing of these structures.

Residual histopathologic changes in the central nervous system of dogs have been described following rapid decompression to 1 mm of Hg for 120 seconds.[47]

Effect of Decompression and Other Hypoxic Episodes on Survival Time

Unconsciousness or collapse in adult dogs exposed to simulated altitudes between 50,000 and 55,000 ft, whether breathing air or 100% O_2, occurs in less than 9 or 10 seconds following exposure.[14] "Complete anoxia" or "complete hypoxia" therefore occurs at these altitudes (ie, 52,500 ft) in animals breathing either air or 100% O_2.[32,48,49] In human beings, the potentially severe hypoxia encountered above 50,000 ft begins to become effectively reversed at the 50,000-ft level, improving rapidly with continued recompression to 40,000 ft or lower.[8]

Studies in animals have shown that survival time decreases with increasing altitude as the severity of hypoxia increases.[50] However, the survival time reaches a minimum and remains constant regardless of further increase in altitude. The minimal survival time of animals exposed to rapid decompression has been studied in O_2 and in air by Lutz.[51] In animals breathing O_2, Lutz found that a minimal survival time of 25 seconds was attained when animals were decompressed to a simulated altitude of 52,000 ft. Following the same procedure to altitudes below 52,000 ft the survival times were longer, and to altitudes above 52,000 ft the survival times did not become significantly shorter but remained approximately 25 seconds. In animals breathing air, Lutz observed that a minimal survival time of 25 seconds was reached on decompression to 43,000 ft or above.

The survival time of unanesthetized animals (rats) after decompression in air, when cessation of respiration is used as the end point, is constant for all simulated final decompression altitudes above 52,000 ft.[49] In the rat, at simulated altitudes of 52,000 and above, rhythmic respiration ceased on the average of 17.8 seconds after decompression in air. Studies on the effects of decompression of dogs and rats from sea level to 30 mm of Hg (ie, 72,000 ft) revealed that respiration ceased at about 30 seconds. Also, it is of interest and noteworthy that respiration in dogs ceases in 15 to 20 seconds after sudden complete arrest of the cerebral circulation.[29]

As exposure to high altitude and the accompanying hypoxic environment increases, resistance or tolerance to hypoxia becomes less.[50] Tolerance to high altitude or decompression appears to vary with various animal species. Compared with the guinea pig, the cat and dog are more tolerant.[52] Cats, rabbits, cavies, hamsters, rats, and mice fail to survive a decompression of 100 mm of Hg (ie, 47,000 ft) for 3 minutes.[53]

The respiratory center is most resistant to hypoxia at birth, then declines through the 4th month of life in the dog.[54] Resistance to hypoxia induced by nitrogen at birth varies from 28 minutes in the ground squirrel, to 16 minutes in the cat, to 6 minutes in the guinea pig.[55] The origin of hypoxic resistance in mammals has not been identified.

Adult rabbits can tolerate an anoxic atmosphere of 100% nitrogen for only 1.5 minutes before death, whereas the newborn rabbit can survive for as long as 27 minutes.[56] In the adult dog, acute occlusion of the cerebral circulation and resultant hypoxia produce cessation of spontaneous respiration after only 20 to 30 seconds; in the 8- to 10-day-old puppy, this effect occurs in 5 minutes, and in the newborn animal, occurs in 27 minutes. Reptiles and amphibians can tolerate O_2 deprivation to a much greater extent than the mammalian species; for example, the turtle can tolerate anoxia produced by 100% nitrogen for several hours and a dose of cyanide 50 times greater than that toxic to the mammal.[56,57]

Exposure of the dog to a near vacuum environment (less than 2 mm of Hg absolute) indicates that dogs exposed for less than 120 seconds are capable of survival upon recompression to 35,000 ft while breathing O_2.[14] In such animals, collapse occurs within 9 to 10 seconds after decompression along with a generalized muscle spasticity, a few gasps, momentary convulsive seizures, apnea, and gross swelling of the body and extremities.

Humane Considerations of Decompression

The rapid decompression technique for producing hypoxia (not the explosive decompression method) has been used for euthanasia of animals.[53,58] There have been many pathophysiologic studies involving the use of animals subjected to decompression. Most were conducted by high altitude or space research laboratories, so manned space flights could be accomplished with a minimum of hazard. Sufficient evidence as indicated by EEG recordings have revealed that hypoxia rapidly induces unconsciousness in both animals and man subjected to high altitude simulated by the use of decompression chambers or inhalation of inert gases. It is not known what the subjective perceptions of an animal in a chamber may be but when properly done, decompression is a painless procedure for all species.[58] Decompression at the rate of 4,000 ft per minute for 10 minutes, thus creating a simulated altitude of 40,000 ft (141 mm of Hg), and maintaining this pressure until respiration ceases are considered optimal for a mature dog.[58] For adults of other species such as cats, rabbits, cavies, hamsters, rats, and mice, a decompression of 100 mm of Hg (ie, 47,000 ft above sea level) for 3 minutes is adequate for induction of euthanasia following a decompression rate of 15 mm of Hg per minute.[53]

As emphasized in the 1978 AVMA Panel on Euthanasia report,[2] the successful use of decompression chambers is predicated on the proper operation and maintenance of the equipment. Personnel operating the equipment must be skilled and knowledgeable in its use as well as understand the esthetically unpleasant reactions manifested by animals during the period of hyperactive coma or unconsciousness prior to death.

Dogs under 4 months of age are more tolerant to hypoxia and require longer periods of decompression before respiration ceases.[54] Animals with respiratory complications and especially those with otitis media should not be subjected to decompression because of the possibility of the development of pain from unequalized positive middle-ear pressure.

References

1. AVMA Council on Research: Report of the AVMA panel on euthanasia. *JAVMA* 160:761–772, 1972.
2. AVMA Council on Research: Report of the AVMA panel on euthanasia. *JAVMA* 173:59–72, 1978.
3. Bryan CA, Leach WG: Physiologic effects of cabin pressure failure in high altitude passenger aircraft. *Aerospace Med* 31:267–275, 1960.
4. Van Liere EJ, Stickney JC: *Hypoxia.* Chicago, University of Chicago Press, 1963, pp 1–381.
5. Tenney SM: Respiration in mammals (chapter 15), in Swenson, J Melvin (ed): *Dukes' Physiology of Domestic Animals,* ed 9. Ithaca, NY, Constock Publishing Associates, Division of Cornell University Press, 1977, p 186.
6. Busby DE, Higgins EA, Funkhouser GE: Effect of physical activity of airline flight attendants on their time of useful consciousness in a rapid decompression. *Aviat Space Environ Med* 47:117–120, 1976.
7. Busby DE, Higgins EA, Funkhouser GE: Protection of airline flight attendants from hypoxia following rapid decompression. *Aviat Space Environ Med* 47:942–944, 1976.
8. Bancroft RW, Simmons DG: Rapid decompressions up to 60,000 feet wearing the standard oxygen mask. *Aerospace Med* 35:203–211, 1964.
9. Hills BA, Ray DE: Inert gas narcosis. *Pharmacol Ther [B]* 3:99–111, 1977.
10. Behnke AR, Thomson RM, Motley EP: The psychologic effects from breathing air at 4 atmospheres pressure. *Am J Physiol* 112:554–558, 1935.
11. Behnke AR, Yarbrough OD: Respiratory resistance, oil-water solubility, and mental effects of argon, compared with helium and nitrogen. *Am J Physiol* 126:409–415, 1939.
12. Lawrence JH, Loomis WF, Tobias CA, et al: Preliminary observations on the narcotic effect of xenon with a review of values for solubilities of gases in water and oils. *J Physiol* 105:197–204, 1946.
13. Bennett PB, Glass MB: Electroencephalographic and other changes induced by high partial pressures of nitrogen. *Electroencephalogr Clin Neurophysiol* 13:91–98, 1961.
14. Bancroft RW, Dunn JE II: Experimental animal decompressions to a near vacuum environment. *Aerospace Med* 36:720–725, 1965.
15. Edelmann A, Hitchcock FA: Observations on dogs exposed to an ambient pressure of 30 mm Hg. *J Appl Physiol* 4:807–812, 1952.
16. Hitchcock FA, Kemph J: The boiling of body liquids at extremely high altitudes. *Aviat Med* 26:289–297, 1955.
17. Kemph JP, Beman FM, Hitchcock FA: Subcutaneous pressure developed in dogs following explosive decompression to 25 or 30 mm Hg. *Am J Physiol* 168:601–604, 1952.
18. Kemph JP, Hitchcock FA: Further studies of effects of high intrapulmonic pressure on dogs at 30 mm Hg. *Aviat Med* 25:227–234, 1954.
19. Gelfan S: Explosive decompression of macaque monkeys to extreme altitudes and recompression at free-fall rates. *J Appl Physiol* 3:254–281, 1950.
20. Holmstrom FMG: Collapse during rapid decompression. Report of three cases. *J Aviat Med* 29:91–96, 1958.
21. Van Liere EJ: *Anoxia, Its Effect on the Body.* Chicago, University of Chicago Press, 1942, pp 1–269.
22. Barron CI, Cook TJ: Effects of variable decompressions to 45,000 feet. *Aerospace Med* 36:425–430, 1965.
23. Luft UC, Clamann HG, Opitz E: The latency of hypoxia on exposure to altitude above 50,000 feet. *J Aviat Med* 22:117–136, 1951.
24. Rossen R, Kabat H, Anderson JP: Acute arrest of cerebral circulation in man. *AMA Arch Neurol Psychiatr* 50:510–528, 1943.
25. Gibbs FA, Davis H: Changes in the human electroencephalogram associated with loss of consciousness. *Am J Physiol* 113:49–50, 1935.
26. Davis PA, Davis H, Thompson JW: Progressive changes in the human electroencephalogram under low oxygen tension. *Am J Physiol* 123:51–52, 1938.
27. Sugar O, Gerard RW: Anoxia and brain potential. *J Neurophysiol* 1:558–571, 1938.
28. Stephens LM, Hartman JL, Lewis OF, et al: Electrophysiology of chimpanzees during rapid decompression. *Aerospace Med* 38:694–698, 1967.
29. Kabat H, Dennis C, Baker AB: Recovery of function following arrest of the brain circulation. *Am J Physiol* 132: 737–747, 1941.
30. Booth NH: Intravenous and other parenteral anesthetics, in Jones LM, Booth NH, McDonald LE (ed): *Veterinary Pharmacology and Therapeutics.* Ames, Iowa State University Press, 1977, pp 241–306.
31. Whitehorn WV, Lein A, Edelmann A: The general tolerance and cardiovascular responses of animals to explosive decompression. *Am J Physiol* 147:289–298, 1946.
32. Gelfan S, Werner AY: Cardiovascular responses following explosive decompression of macaque monkeys to extreme altitudes. *J Appl Physiol* 4:280–310, 1951.
33. Lennox WG, Gibbs FA, Gibbs EL: Relationship of unconsciousness to cerebral blood flow and to anoxemia. *AMA Arch Neurol Psychiatr* 34:1001–1013, 1935.
34. Kemph JP, Hitchcock FA: Changes in blood and circulation of dogs following explosive decompression to low barometric pressures. *Am J Physiol* 168:592–600, 1952.
35. Cooke JP, Bancroft RW: Some cardiovascular responses in anesthetized dogs during repeated decompressions to a near vacuum. *Aerospace Med* 37:1148–1152, 1966.
36. Bancroft RW, Cooke JP, Cain SM: Comparison of anoxia with and without ebullism. *J Appl Physiol* 25:230–237, 1968.
37. Cooke JP, Cain SM, Bancroft RW: High venous pressures during exposure of dogs to near vacuum conditions. *Aerospace Med* 38:1021–1024, 1967.
38. Pratt AJ, Stone HL, Stegall HF, et al: Circulatory impairment during exposure to ambient pressures of 4 mm Hg and 55 mm Hg. *J Appl Physiol* 29:177–180, 1970.
39. Ferguson FP, Smith DC: Effects of acute decompression stress upon plasma electrolytes and renal function in dogs. *Am J Physiol* 173:503–510, 1953.
40. Chang H-T, Margaria R, Gelfan S: Pressure changes and barotrauma resulting from decompression and recompression in the middle ear of monkeys. *Arch Otolaryngol* 51:378–399, 1950.
41. Cole CR, Chamberlain DM, Burch BH, et al: Pathological effects of explosive decompression to 30 mm Hg. *J Appl Physiol* 6:96–104, 1953.
42. Barron CI, Collier DR Jr, Cook TJ: Observations on simulated 12-second decompressions to 32,000 feet. *Aviat Med* 29:563–574, 1958.
43. Idicula J: Perplexing case of maxillary sinus barotrauma. *Aerospace Med* 43:891–892, 1972.
44. Lewis ST: Barotrauma in United States Air Force accidents/incidents. *Aerospace Med* 44:1059–1061, 1973.
45. Edelman A, Whitehorn WV, Lein A, et al: Pathological lesions produced by explosive decompression. *Aviat Med* 17: 596–612, 1946.
46. Dunn JE II, Bancroft RW, Haymaker W, et al: Experimental animal decompressions to less than 2 mm Hg absolute (pathologic effects). *Aerospace Med* 36:725–732, 1965.
47. Casey HW, Bancroft RW, Cooke JP: Residual pathologic changes in the central nervous system of a dog following rapid decompression to 1 mm Hg. *Aerospace Med* 37:713–718, 1966.
48. Gelfan S, Nims LF, Livingston RB: Cause of death from explosive decompression at high altitude (abstr). *Fed Proc* 6: 110, 1947.
49. Gelfan S, Nims LF, Livingston RB: Explosive decompression at high altitude. *Am J Physiol* 162:37–53, 1950.
50. Armstrong HG: Anoxia in aviation. *Aviat Med* 9:84–91, 1938.
51. Luft UC, Clamann HG, Adler HF: Alveolar gases in rapid decompression to high altitudes. *J Appl Physiol* 2:37–48, 1949.
52. Whitehorn WV, Lein A, Hitchcock FA: The effect of explosive decompression on the occurrence of intravascular bubbles. *Aviat Med* 18:392–394, 1947.
53. Barber BR: Use of a standard autoclave for decompression euthanasia. *J Institute Anim Technol* 23:106–110, 1972.

54. Kabat H: The greater resistance of very young animals to arrest of the brain circulation. *Am J Physiol* 130:588–599, 1940.
55. Adolph EF: Regulations during survival without oxygen in infant mammals. *Respir Physiol* 7:356–368, 1969.
56. Cohen PJ: The metabolic function of oxygen and biochemical lesions of hypoxia. *Anesthesiology* 37:148–177, 1972.
57. Bellamy D, Peterson JA: Anaerobiosis and the toxicity of cyanide in turtles. *Comp Biochem Physiol* 24:543–548, 1968.
58. Smith DC: Methods of euthanasia and disposal of laboratory animals, in Gay WI (ed): *Methods of Animal Experimentation*. New York, Academic Press, 1965, vol I, pp 167–195.
59. Miller KW, Paton WDM, Smith EB: Site of action of general anesthetics. *Nature* 206:574–577, 1965.
60. Saidman LJ, Eger EI II, Munson ES, et al: Minimum alveolar concentrations of methoxyflurane, halothane, ether, cyclopropane in man: Correlation with theories of anesthesia. *Anesthesiology* 28:994–1002, 1967.

Canine Mycotoxicosis

Although many mycotoxicoses in domestic and companion animals remain undiagnosed, epizootics have occurred on a regular basis for many decades in the United States and other nations. In the southern United States where the climate is warm and humid, mycotoxicoses are more prevalent than in other regions. A mycotoxicosis in dogs, designated "hepatitis X," was first reported in the southeastern United States in 1952. Subsequent investigations traced the cause to commercial dog food which contained peanut meal as the principal protein source. A relationship also was established between this disease in dogs and "moldy corn poisoning" in cattle and swine.

A review of several epizootic mycotoxicoses occurring in the southeastern United States indicated that aflatoxin and aflatoxigenic strains of *Aspergillus flavus* were in most of the feed samples. Reportedly much of the feed-grade peanut meal purchased during this period on the open market in the United States was contaminated with aflatoxin. However, in the cases of hepatitis X in dogs and moldy corn toxicoses in cattle and swine, other toxigenic fungi, including *Penicillium rubrum*, were isolated. Furthermore, other toxic substances having a synergistic effect with aflatoxin were present in contaminated feeds. While similarities between experimentally induced aflatoxicosis and field cases of hepatitis X are impressive, there are differences, particularly in renal alterations.

Since hepatitis X in dogs generally has been associated with commercial feeds from which *A flavus* and *Penicillium* sp were isolated, a study was initiated to examine the effect of aflatoxin B_1 and rubratoxin B in dogs and to compare the experimental disease with spontaneous hepatitis X observed in field cases. Results indicated that the dog is sensitive to the toxic effects of both mycotoxins. Histologic changes were induced in the dog not only by aflatoxin but by rubratoxin. A striking similarity was observed between induced lesions when the 2 toxins were combined and lesions observed in dogs used in laboratory studies or affected in natural outbreaks of hepatitis X.

Based on these data and earlier reports, it would seem that there is little doubt of an association of hepatitis X and aflatoxin B_1, although it is apparent that the disease probably is not the result of a single toxic factor.—*A. W. Hayes and W. L. Williams in J Environ Pathol Toxicol, 1, (1977): 59.*

AVMA American Veterinary Medical Association

1931 N. Meacham Rd.
Suite 100
Schaumburg, IL
60173-4360
www.avma.org

AVMA American Veterinary Medical Association
1931 N. Meacham Rd., Suite 100
Schaumburg, IL 60173-4360

Pascal Cousin
9, Boulevard de la Liberté
94170 Le Perreux sur Marne
France

ETAT DU MISSOURI USA

Attorney General's Opinion 32-2003

Topics:
Animals.
Municipalities.
Wildlife.
Summary conclusion:

Municipal animal pounds are only authorized to impound or harbor dogs and cats. Decompression is not a recommended method of euthanasia by the American Veterinary Medical Association's Panel on Euthanasia. Therefore, a municipal animal pound is not authorized to use a decompression chamber to euthanize wildlife.

Jan. 31, 2003

Honorable Matt Blunt
Secretary of State
State Capitol Building
201 West Capitol Avenue
Jefferson City, MO 65101

Dear Secretary Blunt:

You have asked this office whether it is legal for a municipal animal pound to use a decompression chamber to euthanize wildlife.

In order to answer your question, we first look to the statutory authority granted to municipal animal shelters. Section 273.325.2(17) [footnote 1] defines "pound" or "dog pound" as "a facility operated by the state or any political subdivision of the state for the purpose of impounding or harboring seized, stray, homeless, abandoned, or unwanted animals[.]" See also 2CSR 30-9.010(2)(JJ).

The provisions of Sections 273.325 to 273.357 deal with regulating privately owned kennels, breeders, dealers, and pet shops, as well as publicly owned pounds. The Department of Agriculture has adopted regulations implementing these provisions. See 2CSR 30-9.010, et seq.

Political subdivisions have such authority as granted to them by the legislature. Harris v. William R. Compton Bond & Mortgage Co., 149 S.W. 603, 609 (Mo. banc 1912). The authority granted municipalities under Section 273.325.2(17) is limited to "impounding or harboring seized, stray, homeless, abandoned, or unwanted animals." "Animals" are defined in Section 273.325.2(4) as "any dog or cat, which is being used, or is intended for use, for research, teaching, testing, breeding, or exhibition purposes, or as a pet." Based upon this definition of "animal," a municipal animal shelter is limited to handling dogs or cats.

"Humane euthanasia" is defined at Section 273.325.2(14) as "the act or practice of putting an animal to death in a humane or instantaneous manner under guidelines and procedures established by rules promulgated by the director [of agriculture.]" The Department of Agriculture has stated "[e]uthanasia means the act of putting an animal to death in a humane manner and shall be accomplished by a method specified as acceptable by the American Veterinary Medical Association Panel on Euthanasia[.]" 2 CSR 30-9.010(2)(V). The

Department has also adopted 2CSR 30-9.020(14)(F)5, which states "All euthanasia of animals shall be accomplished by a method approved by the 1993 edition, or later revisions, of the American Veterinary Medical Association's Panel on Euthanasia."

Section 578.005(7) defines "humane killing" as "the destruction of an animal accomplished by a method approved by the American Veterinary Medical Association's Panel on Euthanasia (JAVMA 173:59-72, 1978); or more recent editions, but animals killed during the feeding of pet carnivores shall be considered humanely killed[.]" In this context, animal is defined more broadly as "every living vertebrate except a human being[.]" Section 578.005(3).

The most recent version of the Journal of the American Veterinary Medical Association's Report from the Panel on Euthanasia, Vol. 218, No. 5, dated March 1, 2001, states at page 696 that:

Decompression is unacceptable for euthanasia because of numerous disadvantages.

(1) Many chambers are designed to produce decompression at a rate 15 to 60 times faster than that recommended as optimum for animals, resulting in pain and distress attributable to expanding gases trapped in body cavities. (2) Immature animals are tolerant of hypoxia, and longer periods of decompression are required before respiration ceases. (3) Accidental recompression, with recovery of injured animals, can occur. (4) Bloating, vomiting, convulsions, urination, and defecation, which are aesthetically unpleasant, may occur in unconscious animals.

From the foregoing, it appears that municipal animal shelters are authorized to impound or harbor seized, stray, homeless, abandoned, or unwanted animals, with "animals" defined as "any dog or cat." See Sections 273.325.2(17) and 273.325.2(4). Use of decompression is not recommended by the American Veterinary Medical Association's Panel on Euthanasia. Therefore, such a method of destroying an animal in a municipal animal shelter is not allowed.

CONCLUSION

Municipal animal pounds are only authorized to impound or harbor dogs and cats. Decompression is not a recommended method of euthanasia by the American Veterinary Medical Association's Panel on Euthanasia. Therefore, a municipal animal pound is not authorized to use a decompression chamber to euthanize wildlife.

Very truly yours,
Jeremiah W. (Jay) Nixon
Attorney General

TRADUCTION AUTOMATIQUE

L'opinion 32-2003 de l'Attorney General

Matières :
Animaux.
Municipalités.
Faune.
Conclusion récapitulative :
Livres animales municipales sont seulement autorisées à confisquer ou chiens et chats de port. La décompression n'est pas une méthode recommandée d'euthanasie par le panneau de l'association médicale vétérinaire américaine sur l'euthanasie. Par conséquent, livre animale municipale n'est pas autorisée pour employer une chambre de décompression euthanize la faune.

31 jan. 2003

Mats honorables émoussent
Secrétaire d'état
Bâtiment de capitol d'état

Avenue occidentale du capitol 201
Ville de Jefferson, MOIS 65101

Cher secrétaire émoussé :

Vous avez demandé que ce bureau s'il est légal pour que livre animale municipale emploie une chambre de décompression euthanize la faune.

Afin de répondre à votre question, nous regardons d'abord à l'autorité statutaire accordée aux abris animaux municipaux. La section 273.325.2 (17) [apostille 1] définit « livre » ou « livre de chien » comme « service actionné par l'état ou n'importe quelle subdivision politique de l'état afin de la confiscation ou de l'hébergement saisi, de la bête perdue, du sans-abri, abandonné, ou des animaux non désirés [.] » Voir également le 2CSR 30-9.010(2) (JJ).

Les dispositions des sections 273.325 273.357 traitent régler les établissements, les sélectionneurs, les revendeurs, et les magasins de bêtes en privé possédés, comme livres publiquement possédées. Le ministère de l'agriculture a adopté des règlements mettant en application ces dispositions. Voir le 2CSR 30-9.010, et seq.

Les subdivisions politiques ont une telle autorité comme accordée à eux par la législature. Lien et hypothèque Cie., 149 S.W. 603, 609 de Harris v. William R. Compton (MOIS. banc 1912). L'autorité a accordé des municipalités sous la section 273.325.2 (17) est « confiscation ou hébergement saisi, bête perdue, sans-abri limité, abandonné, ou animaux non désirés. » Des « animaux » sont définis dans la section 273.325.2(4) en tant que « n'importe quel chien ou chat, qui sont employés, ou sont prévus pour l'usage, pour la recherche, l'enseignement, l'essai, la multiplication, ou l'exposition, ou comme animal de compagnie. » Basé sur cette définition de « animal, » un abri animal municipal est limité à manipuler des chiens ou des chats.

« L'euthanasie humanitaire » est définie à la section 273.325.2 (14) comme « acte ou pratique de mettre un animal à la mort dans une façon humanitaire ou instantanée sous des directives et des procédures établies par des règles promulguées par le directeur [de l'agriculture.] » Le ministère de l'agriculture a énoncé « des moyens de l'uthanasia [e] l'acte de mettre un animal à la mort d'une façon humanitaire et sera accompli par une méthode indiquée comme acceptable par le panneau médical vétérinaire américain d'association sur l'euthanasie [.] » 2 CSR 30-9.010(2) (v). Le département a également adopté 2CSR 30-9.020 (14) (F)5, qui énonce que « toute l'euthanasie des animaux sera accomplie par une méthode approuvée par l'édition 1993, ou des révisions postérieures, du panneau de l'association médicale vétérinaire américaine sur l'euthanasie. »

La section 578.005(7) définit « le massacre humanitaire » comme « destruction d'un animal accompli par une méthode approuvée par le panneau de l'association médicale vétérinaire américaine sur l'euthanasie (JAVMA 173:59 - 72, 1978) ; ou des éditions plus récentes, mais des animaux tués pendant l'alimentation des carnivores d'animal de compagnie seront considérés avec humanité tués [.] » Dans ce contexte, l'animal est défini plus largement en tant que « chaque vertébré vivant excepté un être d'humain [.] » Section 578.005(3).

La version la plus récente du journal du rapport de l'association médicale vétérinaire américaine du panneau sur l'euthanasie, vol. 218, non 5, daté du 1er mars 2001, états à la page 696 cela :

La décompression est inacceptable pour l'euthanasie en raison de nombreux inconvénients.
(1) beaucoup de chambres sont conçues pour produire la décompression à un taux 15 à 60 fois plus rapidement que cela recommandé comme optimum pour des animaux, ayant pour résultat la douleur et pour affliger attribuable aux gaz de extension emprisonnés en cavités de corps. (2) les animaux immatures sont tolérants de l'hypoxie, et de plus longues périodes de la décompression sont exigées avant que la respiration cesse. (3) la récompression accidentelle, avec le rétablissement des animaux blessés, peut se produire. (4) le boursouflage, le vomissement, les convulsions, l'urination, et la défécation, qui sont esthétiquement désagréables, peuvent se produire chez les animaux sans connaissance.

De ce qui précède, il s'avère que des abris animaux municipaux sont autorisés à confisquer ou port saisi,

bête perdue, sans-abri, abandonné, ou animaux non désirés, avec des « animaux » définis en tant que « n'importe quel chien ou chat. » Voir les sections 273.325.2 (17) et 273.325.2(4). L'utilisation de la décompression n'est pas recommandée par le panneau de l'association médicale vétérinaire américaine sur l'euthanasie. Par conséquent, on ne permet pas une telle méthode de détruire un animal dans un abri animal municipal.

CONCLUSION
Livres animales municipales sont seulement autorisées à confisquer ou chiens et chats de port. La décompression n'est pas une méthode recommandée d'euthanasie par le panneau de l'association médicale vétérinaire américaine sur l'euthanasie. Par conséquent, livre animale municipale n'est pas autorisée pour employer une chambre de décompression euthanize la faune.

Nous vous prions d'agréer l'expression de nos salutations distinguées
Jérémie W. (geai) Nixon
Attorney General

ETAT DU NEW JERSEY USA

NEW JERSEY ANIMAL CRUELTY LAWS

NJS 4:22-19 Hypoxia Induced By Decompression Prohibited

A person who shall:

A. Impound or confine, or cause to be impounded or confined, in a pound or other place, a living animal or creature, and shall fail to supply it during such confinement with a sufficient quantity of good and wholesome food and water; or

B. Destroy or cause to be destroyed any such animal by hypoxia induced by decompression or in any other manner, by the administration of a lethal gas other than an inhalant anesthetic, or in any other manner except by a method of euthanasia generally accepted by the veterinary medical profession as being reliable, appropriate to the type of animal upon which it is to be employed, and capable of producing loss of consciousness and death as rapidly and painlessly as possible for such animal shall, in the case of a violation of subsection a., be guilty of a disorderly persons offense and shall be punished as provided in subsection a. of R.S.4:22-17; or, in the case of a violation of subsection b., be subject to a penalty of $25 for the first offense and $50 for each subsequent offense. Each animal destroyed in violation of subsection b. shall constitute a separate offense. The penalty shall be collected in accordance with the "Penalty Enforcement Law of 1999," P.L.1999, c.274 (C.2A:58-10 et seq.) and all money collected shall be remitted to the State.

This section shall apply to kennels, pet shops, shelters and pounds as defined and licensed pursuant to P.L.1941, c.151 (C.4:19-15.1 et seq.); to pounds and places of confinement owned and operated by municipalities, counties or regional governmental authorities; and to every contractual warden or impounding service, any provision to the contrary in this title notwithstanding.

NJS 4:22-19.1 Dismantling Of Devices Mandated

Within 30 days of the effective date of this act, any chamber or device used to induce hypoxia through decompression or in any other manner shall be dismantled and removed from the premises. The owner of any premises on which the chamber or device remains 30 days subsequent to the effective date of this act shall be guilty of a disorderly persons offense.

NJS 4:22-19.2 Dismantling Of Devices Ordered

Within 30 days of the effective date of this act, any chamber or device used to induce hypoxia through decompression or in any other manner and any gas chamber or similar device, except one which is used for the administration of an inhalant anesthetic, shall be dismantled and removed from the premises. The owner of any premises on which the chamber or device remains 30 days subsequent to the effective date of this act shall be guilty of a disorderly persons offense.

TRADUCTION AUTOMATIQUE

LOIS ANIMALES DE CRUAUTÉ DE NEW JERSEY

NJS 4:22 - hypoxie 19 induite par Decompression Prohibited
Une personne qui : A. Confisquez ou confinez, ou causez pour être confisqué ou confiné, en livre ou tout autre endroit, un animal ou une créature vivant, et ne le fournira pas pendant un tel emprisonnement avec une quantité suffisante de bons et sains aliments et d'eau ; ou le B. détruisent ou causent pour être détruits un tel animal par l'hypoxie induite par la décompression ou de n'importe quelle autre façon, par l'administration d'un gaz mortel autre qu'un anesthésique inhalant, ou de n'importe quelle autre façon excepté près une méthode d'euthanasie courante par la profession médicale vétérinaire en tant qu'étant fiable, approprié au type d'animal sur lequel elle doit être utilisée, et capable de produire la perte de conscience et de mort aussi rapidement et sans douleur que possible à un tel animal, dans le cas d'une violation de la sous-section A., être coupable d'une offense désordonnée de personnes et serez puni de la manière prévue en sous-section A. de R.S.4 :22-17 ; ou, dans le cas d'une violation de la sous-section B., soyez sujet à une pénalité de $25 pour la première offense et de $50 pour chaque offense suivante. Chaque animal détruit dans la violation de la sous-section B. constituera une offense séparée. La pénalité sera rassemblée selon « la loi d'application de pénalité de 1999, » P.L.1999, c.274 (C.2A :58-10 et seq.) et tout l'argent rassemblé sera remis à l'état. Cette section s'appliquera aux établissements, aux magasins de bêtes, aux abris et aux livres comme défini et autorisé conformément à P.L.1941, c.151 (C.4 :19-15.1 et seq.) ; à livres et aux endroits de l'emprisonnement possédés et actionnés par des municipalités, des comtés ou des autorités gouvernementales régionales ;

NJS 4:22 - 19.1 démantèlements des dispositifs exigés

Dans les 30 jours de la date efficace de cet acte, n'importe quelle chambre ou dispositif utilisé pour induire l'hypoxie par la décompression ou de n'importe quelle autre façon sera démantelée et enlevée des lieux. Le propriétaire de tous les lieux sur lesquels la chambre ou le dispositif demeure de 30 jours de suivant à la date efficace de cet acte sera coupable d'une offense désordonnée de personnes.

NJS 4:22 - 19.2 démantèlements des dispositifs commandés

Dans les 30 jours de la date efficace de cet acte, n'importe quelle chambre ou dispositif induisait l'-hypoxie par la décompression ou de n'importe quelle autre façon et n'importe quelle chambre de gaz ou dispositif semblable, à moins qu'un qui est employé pour l'administration d'un anesthésique inhalant, sera démantelée et enlevée des lieux. Le propriétaire de tous les lieux sur lesquels la chambre ou le dispositif demeure de 30 jours de suivant à la date efficace de cet acte sera coupable d'une offense

désordonnée de personnes.

Part 1
Destruction of animals

Chapter 1
Introduction

If an outbreak of a transboundary animal disease or other serious disease occurs and a stamping-out policy is adopted for its control and eradication, it may be necessary to destroy a large number of animals. It is essential that these animals are speedily and humanely slaughtered and are indeed dead before disposal of carcasses commences. Speed is of the essence once the disease has been confirmed because, in most situations, the live animals will continue to produce and possibly disseminate the disease pathogen. An experienced veterinarian should be present during destruction. There is likely to be considerable public interest, at least initially, in the destruction of animals. Positive media coverage concerning animal welfare will reflect favourably on staff and increase community support for the eradication campaign.

The destruction of large animals poses the biggest concern in this regard. They may have to be destroyed individually in public view with firearms, humane killers (captive-bolt pistols) or other means.

Officers in charge must be aware of the impact that animal destruction will have on all personnel involved. They must quickly acquaint themselves with the skills and experience of all assistants and brief and train them accordingly. Furthermore, they must be aware that some people will be unable to handle the mentally and physically stressful environment likely to be encountered.

Where possible, the livestock owner and his or her family should not be present during the slaughter process, as they may experience considerable distress. Counselling and welfare should be made available if needed.

The policy regarding compensation for destroyed animals should be communicated clearly to owners before destruction is attempted. Destruction of animals without adequate compensation of owners is likely to meet with serious opposition and at worst result in large-scale illegal movement of animals and/or their products. Payment of compensation at market-related prices is the only way to ensure owner cooperation and the success of the eradication campaign.

What animals should be slaughtered will depend on the disease in question and the epidemiological circumstances. In some non-emergency diseases, e.g. bovine tuberculosis, slaughter of individual infected animals only may be necessary.

For emergency diseases, one of two options is usually selected:

- If animals in the infected zone are not well controlled and there is a serious risk of further rapid spread of the disease or spillover to feral or wild animals or if inadequate resources are available for surveillance and imposition of quarantine and movement controls, it may be expedient to slaughter all animals in the infected zone or in specific areas of the zone.
- If animals are well contained on farms and resources are available for surveillance and imposition of quarantine and movement controls, the best decision would probably be to slaughter only animals on known infected farms and dangerous-contact premises.

This decision will depend on the mode of disease transmission; it will be different for diseases capable of airborne dissemination over distances and those requiring direct contact.

Chapter 2
Organization of destruction

ACTION PLAN

Planning is essential to ensure that the task of destruction is carried out efficiently and not impeded by lack of resources. An action plan should be drawn up in consultation with owners or agents and appropriate officials. The procedures below should be followed.

The veterinary officer should undertake the tasks listed below.

- Discuss the situation with affected farmers and village leaders, briefing them on what is going to happen, including the method of compensation.
- Consult with the farm owner/manager and/or village leaders to establish:
  - farm layout, facilities and equipment;
  - the number, species and location of animals to be destroyed;
  - the destruction technique to be used;
  - the time-frame for commencement and completion of animal destruction.
- Decide on the methods and facilities needed for safe, humane and efficient destruction of the animals.
- Advise the team leader of immediate resources needed to move and secure animals in preparation for destruction.
- Consult with the officer in charge (OIC) of the disposal team if different from the destruction team, determine the disposal method and site; if necessary, identify centrally located carcass disposal sites as close as practicable to the site of destruction.
- Draw up a concise written plan for approval, including:
  - destruction method(s);
  - destruction site(s);
  - order of destruction;

    - personnel required;
    - facilities and equipment needed.
- Make a diagram of the infected property (IP) or dangerous-contact premises (DCP), including details of the destruction operation.
- Make sure that there is a complete inventory of animals to be destroyed on the property, not delaying destruction because there has been no agreement on valuation; where possible, all animals should be valued before destruction; where there is no prior agreement on valuation, provide close supervision to ensure that all the animals are available for destruction.
- Seek authority to destroy in terms of the law(s) pertaining to control of animal diseases when there is a delay in reaching agreement on valuation with the owner or his/her agent; delay may endanger the success of the operation and result in negative perceptions of animal disease control activities.
- Request livestock owners to assemble, confine and restrain their animals the day before the destruction team starts operations.
- Ensure that animals not to be destroyed, including domestic pets, are confined well away from the destruction site.
- Send a team into the surrounding countryside to assess the presence of free-roaming or unrestrained susceptible animals.
- Arrange for teams to be sent to round up, shoot or poison such animals where they are; helicopter shooting by trained marksmen may be the only option, in which case proper disposal of carcasses is essential, as the animals may already be infected.
- Arrange for any necessary support services, such as police and army personnel, to be made available.

Before commencing destruction, the team leader should carry out the tasks below.

- Move animals to the centre of the IP or to areas most remote from other susceptible animals, including wild animals.
- Brief the destruction teams, then supervise and coordinate their activities.
- Ensure that:
    - destruction takes place away from public view if possible;
    - destruction facilities, methods and working conditions are consistent with personal safety;
    - destruction is humane and that no animal is removed for disposal until it is dead;
    - destruction teams receive adequate rest and meal breaks.
- Make every effort to avoid damage to property; damage must be drawn to the attention of the owner/manager, recorded and reported promptly.
- Check all destruction against the authorized inventory, to ensure that all variations are accounted for (e.g. births and natural deaths) and that all animals scheduled for destruction have in fact been destroyed.
- Provide a situation report for the team leader at the end of each day.
- Advise the team leader of resource requirements for the next 48 hours.
- Advise the appropriate officer/s immediately destruction has been completed, so that other tasks, such as disinfection, can be started without delay; carcasses and the destruction area should be sprayed with disinfectant as soon as destruction is complete.

SELECTION OF DESTRUCTION SITE

The factors that need to be considered in selecting a destruction site are:

- facilities available on site;
- additional facilities and equipment required;
- animal security;
- proximity of the disposal site and ease of access;
- safety of personnel;
- acceptability to the owner/manager;
- safe and secure means of transporting carcasses to the destruction site;
- likelihood of damage to property and services;
- protection from public view.

ORDER OF DESTRUCTION

The order of destruction will be determined by the veterinary officer in charge of the operation. Normally the order will be:

- affected animals;
- their direct contacts;
- other susceptible animals in order of epidemiological importance.

In foot-and-mouth disease, pigs should be destroyed before other species.

Fractious and potentially dangerous animals, such as bulls, sows with litters and boars should be destroyed first.

Chapter 3
Methods of destruction

Methods of destruction of animals are set out below. Rabid or suspect rabid animals should be shot in the heart with a firearm to preserve the brain, which is the best diagnostic specimen, and to avoid contamination of personnel with potentially infective brain or saliva. Animals with bovine spongiform encephalopathy (BSE) or scrapie should not be shot through the head, as brain tissue is required for diagnostic testing.

FIREARMS (RIFLES AND GUNS)

Ensure compliance with any firearm licensing requirements, including the use of trained and approved operators for rifles and guns.

Part of the preparation process for an emergency disease outbreak is to ensure that firearms operators experienced in shooting livestock can be contacted at short notice. The following aspects of firearms safety should be considered:

- All firearms are potentially hazardous.
- When shooting at short range in stockyards, relatively low-velocity hollow/soft-point ammunition should be used. Solid-point ammunition should be avoided, because the projectiles can leave the target at high velocity, which is dangerous to personnel in the area. Hollow point ammunition disintegrates when entering the target, more effectively destroying brain tissue. (For details see relevant species in Chapter 4.) When paddock shooting, use high velocity, hollow/soft-point ammunition.

- Persons other than the shooters and assistants should be cleared from the area or should stand well behind the shooters. The line of fire must be chosen to prevent accidents or injury from stray bullets or ricochets.
- To provide maximum impact and the least possibility of misdirection, the range should be as short as circumstances permit.
- Although the humane killer pistol and captive-bolt pistol are designed to be pressed firmly on the head before being discharged, it is not safe to do this with a standard rifle or pistol.
- Always notify police before using firearms near populated areas.

Advantages of using firearms

The advantages of firearms are:

- clean kills in the hands of experienced operators;
- handling individual animals is not necessary;
- destruction of animals from a distance;
- firearms and ammunition are readily available;
- many people are proficient in their use.

Disadvantages of using firearms

The disadvantages of firearms are:

- they are potentially dangerous;
- they are unsuitable for use close to populated areas.

CAPTIVE-BOLT PISTOLS

Captive-bolt pistols are an acceptable alternative to firearms where animals are sufficiently restrained, provided that the team understands that animals may be stunned rather than killed. They must be competent to know when an animal is only stunned and trained and equipped to kill such an animal immediately after stunning.

FIGURE 1

Captive-bolt pistol
Provided that animals are properly restrained and that the slaughter team is aware that animals may be stunned but not killed, the captive-bolt pistol is an alternative to firearms.

Blank cartridges for the captive-bolt pistol are colour coded according to the amount of charge they contain. It is essential that manufactures' recommendations should be followed regarding blank cartridges for different farm animals. The most widely used is the “Cash Special”, a single shot .22 calibre captive bolt similar to a revolver. It uses three different loads:

- pink: 1 grains (weaners);
- purple: 2 grains (sheep);
- green: 3 grains (cattle, boars).

Regular maintenance of the captive-bolt pistol is essential for efficient stunning.

When using a captive-bolt pistol, more than one operator can work in the same area with safety. Spare weapons and parts should be on hand.

Advantages of captive-bolt pistols

The advantages of captive-bolt pistols are:

- operator safety, as there is no free projectile;
- both pistols and ammunition are readily obtainable;
- ease of use;
- operators do not need to be expert shooters; they must, however, be trained in correct placement of the pistol against the head in the different species.

Disadvantages of captive-bolt pistols

The disadvantages of captive-bolt pistols are:

- they usually only stun larger animals such as cattle over one-year old, sows, boars, billy goats and rams, which must then be pithed or bled (see Pithing, below) to ensure death;
- some animals have to be individually restrained;
- they are relatively slow, especially when destroying large numbers of animals.

Humane killers that work on the same principle as the captive bolt but destroy a larger amount of tissue are a better option.

PITHING

Pithing is the process of destroying nervous tissue in the region of the brain stem to ensure the death of the animal. It is usually done by inserting a rod through the hole made by the captive-bolt in the head or by severing the spinal cord between the atlas and axis, the first and second bones of the neck.

Pithing unstunned animals is not an acceptable method of destruction as it is inhumane. It is essential on animals that have been stunned only, for example when captive-bolts are used on larger animals.

Pithing is also a safety measure to prevent workers being struck by the involuntary movements of a stunned animal.

Pithing is preferable to exsanguination, or bleeding out, which could release infectious material and make working conditions slippery and dangerous.

OTHER PHYSICAL METHODS

Dislocation of the neck

This may be suitable for poultry and smaller laboratory animals. Suitable methods are by burdizzo, bone cutters, secateurs or manually. Burdizzos are particularly useful when large numbers of poultry with strong necks, such as geese and duck, are to be destroyed.

Electrocution

Electrocution is used widely in abattoirs but is not suitable for field use.

Decompression

This method is now regarded as unacceptable.

Exsanguination

Exsanguination combined with stunning or neck dislocation is a humane method of destruction of sheep and goats when performed by an experienced operator. It is undesirable, however, because released infectious material makes the destruction site slippery and dangerous.

GASEOUS AGENTS

Carbon dioxide

Carbon dioxide is the method of choice for destroying most poultry species when large numbers are involved and for many laboratory animals.

Animals must be exposed to an atmosphere of at least 30 percent carbon dioxide to ensure loss of consciousness and then at least 70 percent carbon dioxide to ensure death.

To achieve this, animals may be placed in an air-filled container into which carbon dioxide is allowed to flow so that concentration rises to a minimum of 70 percent for at least 3 minutes. An optimum flow rate is one that will displace 20 percent of the chamber volume per minute. Animals may be left in the container until *rigor mortis* ensues or they may be removed once unconscious and killed by cervical dislocation or exsanguination. Exposure of up to 20 minutes may be necessary to ensure death; this will be even longer in neonatal or juvenile animals, which are tolerant of carbon dioxide. They may require 30 minutes exposure or longer.

Alternatively, the container may be filled with the carbon dioxide/air mixture before animals are placed in it, in which case anaesthesia is said to occur more rapidly (20 seconds to unconsciousness, compared to 70 seconds). Some workers have suggested, however, that this technique is more stressful.

If cylinders of carbon dioxide are not available, dry ice may be used. This is placed in the bottom of a deep container under a gauze floor, in such a way that there is no direct contact with the dry ice. Animals are then placed in the container and left there until unconsciousness or death ensues.

The use of a 70 percent carbon dioxide/30 percent oxygen mixture is said to decrease the discomfort of hypoxia before the onset of anaesthesia and narcosis. This will complicate the procedures, however, by requiring additional cylinders of oxygen and reducing valves.

Carbon dioxide is safe and easy to use as long as it is used in a well ventilated area.

Gaseous anaesthetic agents

These agents, which include halothane, enflurane and isoflurane, can be used to produce anaesthesia and death. Halothane at concentrations greater than 4 percent can produce anaesthesia and cardiac arrest in 90 seconds. These agents can be used in exactly the same way as carbon dioxide, piped into a container with a carrier gas such as oxygen or poured onto cotton wool and placed under gauze at the bottom of a deep container. There should be no direct contact between the animal and the liquid anaesthetic.

The major disadvantages are that these agents are expensive and should only be used in a well ventilated room or, preferably, in a fume cupboard. Prolonged exposure, even at low concentrations, may be detrimental to the health of personnel. As with carbon dioxide, animals may be left in the anaesthetic chamber until dead or may be removed once unconscious and killed by one of the physical methods or by injection of an overdose of barbiturate as detailed below.

Ether is not recommended. Induction of anaesthesia is slow and stressful, as the high concentrations of the vapour necessary to produce unconsciousness are irritant to skin and mucous membranes. Ether is also hazardous to personnel because of its explosive properties during use and when disposing of carcasses.

Hydrogen cyanide gas

Hydrogen cyanide gas is a highly effective method of destroying poultry. Human safety considerations restrict its use, however, and it is not recommended.

Carbon monoxide

Carbon monoxide can be used to destroy poultry. It is readily available from car exhaust but unleaded petrol produces less than super petrol and the fumes must be cooled. Human safety considerations restrict its use.

Methyl bromide

Methyl bromide is effective at killing poultry but operator safety requirements restrict its use. There are people trained in its use in all agriculture departments. It is also virucidal. Environmental concerns are now restricting its use.

INJECTABLE AGENTS

An overdose of any of the barbiturates can be used for euthanasia, ideally by the intravenous route in large animals; the intracardiac or intraperitoneal route may be preferable in smaller animals. Destruction of cats, rabbits and some birds by intraperitoneal sodium pentobarbitone may be accompanied by an excitement phase. Animals should be confined and handled with extreme care. Specific euthanasia solutions are available (sodium pentobarbitone 325 mg/kg). This should not be used by the intrathoracic, subcutaneous or intramuscular route as at this concentration it is extremely irritant to tissues. Pentobarbitone at concentrations normally employed for anaesthesia may also be used but larger volumes will be required.

If the animals are excitable or vicious, other drugs can be administered to calm them. These drugs, such as tranquillizers, analgesics or depressants such as ketamine, opioids or xylazine, can be given by the subcutaneous or intramuscular route. An overdose of barbiturate can then be given intravenously to kill the animal.

These agents are restricted by law and must only be used by a veterinarian or under veterinary supervision.

Chapter 4
Destruction of various species

The preferred methods of destruction of various domestic species and the factors that determine the selection are set out below.

CATTLE AND BUFFALO

Under most circumstances, cattle and domesticated (water) buffalo will be mustered into yards and shot. In extensive areas where 100 percent musters cannot be achieved, unmustered animals will be paddock shot, after first mustering as many as possible.

Captive-bolt pistols are most suitable when animals can be adequately restrained (see Captive-bolt pistols, p. 10). Injectable agents may be most suitable for small numbers of calves.

Frontal method

The firearm should be directed at the point of intersection of lines taken from the base of each horn (or equivalent position in polled animals) to the opposite eye, aiming at the spine (Figure 2a). For bulls or older animals, the bullet should enter about 1 cm to the left or right of this point and hard point/jacketed ammunition may be necessary. Small calves may be shot just behind the nuchal crest (poll) in the mid-line, aiming directly at the muzzle (Figure 2c). Alternatively, a captive-bolt pistol using cartridges may be used.

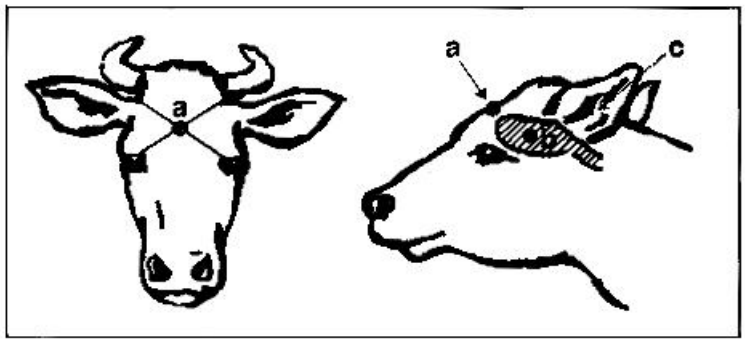


FIGURE 2

Humane destruction of cattle: (a) recommended position for frontal method (suitable for firearm or captive-bolt pistol); (b) recommended position for temporal method (only suitable for firearms); (c) recommended position for small calves.

Temporal method

This is only suitable for firearms. The animal is shot from the side so that the bullet enters the skull midway between the eye and the base of the ear. The bullet should be directed horizontally (Figure 2b).

Shooting in yards

Ideally, only personnel who have had previous experience in this type of work should undertake the task. If such personnel are not available, the task may be allocated to police or army marksmen. They should be fully briefed on humanitarian and safety aspects of destruction before commencing yard shooting. Only hollow/soft point ammunition should be used. The minimum calibre should be .22 magnum; maximum calibre should be. 44 magnum (240 grain) or .375 (250 grain).

Operate from a top rail, preferably in a small yard. It is not practical to shoot in a crush unless dealing with very small numbers and the crush is equipped with a side opening gate, in which case a captive-bolt pistol should be considered.

Paddock/extensive area destruction

Shooting from helicopters is usually the most effective method of destroying unmusterable cattle. Appropriate civil aviation authority approval may be needed before rifles may be used from helicopters. This should be carried out only by experienced, trained personnel with current proficiency in this type of operation. Untrained personnel should undergo a training course and pass a practical and written test at its conclusion before shooting from a helicopter. Minimum recommended calibre is .308 soft point with semi-automatic rifles such as the M14, SLR or MIA.

Shots aimed to destroy the brain are preferred but for practical reasons this is not generally possible with helicopter shooting, in which case heart/lung shots can be used.

The problem of rapid destruction of large numbers of cattle on intensive feedlots is not easy to resolve. The possibility of using a lethal oral agent in water or feed should be considered.

Technique for domesticated (Asian) buffaloes

As for cattle except:

- hard point/jacketed ammunition is preferable for large animals;
- for small numbers, when use of semi-automatic rifles is not critical, use heavier calibre or magnum rifles;
- frontal shooting: check the angle of impact, as a buffalo will often raise its nose.

SHEEP

The preferred method of destruction is by .22 rifles or captive-bolt pistols.

Hornless sheep

The top of the head (centre of upper forehead) is a suitable position, with the firearm or captive-bolt being aimed towards the animal's gullet. Alternatively, the weapon may be placed just behind the poll and aimed in the direction of the animal's muzzle. Both methods are illustrated in Figure 3.

Horned sheep

If using a captive-bolt pistol, the top-of-the-head position used for hornless sheep may not be suitable, in which case the weapon may be placed behind the poll and aimed in the direction of the animal's muzzle (Figure 4a). If using a firearm, shoot at a point in the middle of the face just above the level of the eyes, aiming towards the spine (Figure 4b).

- *Rams*: it may be easier to use .22 magnum rifle, depending on facilities. If captive-bolt is more practical, heavy duty cartridges should be used (see Captive-bolt pistols, p. 10).
- *Wethers/ewes*: sheep must be packed tightly as destruction proceeds. This can be achieved using light portable panels or mesh.
- *Newborn lambs*: these should be separated and given sodium pentobarbitone (intraperitoneal, 3–5 ml through automatic syringes) .

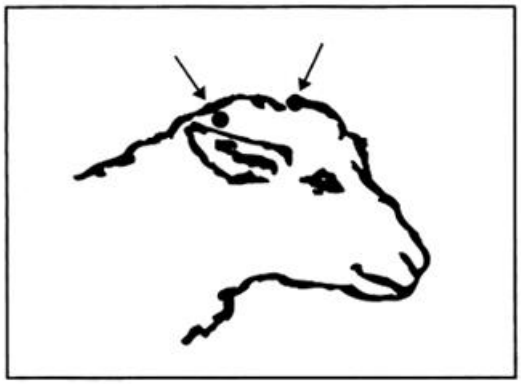


FIGURE 3

Humane destruction of hornless sheep: recommended positions and direction of fire for captive-bolt pistol or firearm.

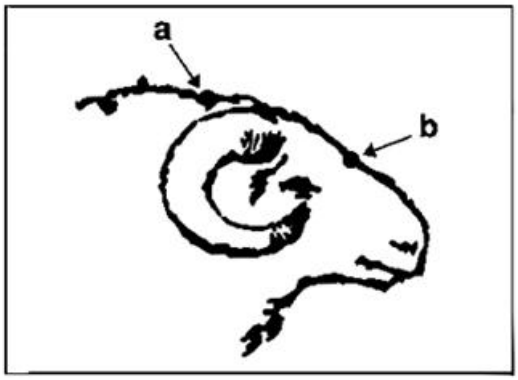


FIGURE 4

Humane destruction of horned sheep: recommended position and direction of fire for (a) captive-bolt pistol or (b) firearm.

PIGS

Pigs are particularly difficult to destroy. Captive-bolt pistols or heavy-calibre humane killers should be used for housed pigs to avoid the danger of ricochets. Housed pigs may be moved outside and destroyed with firearms. Sows with litters are particularly fractious and difficult to handle. Pigs in paddocks can be shot using firearms.

Use sodium pentobarbitone on unweaned pigs. Intraperitoneal injections of 3–5 ml of a suitable product using automatic syringes is satisfactory.

Frontal method

The captive-bolt pistol or firearm should be directed at a point about midway across the forehead and, for adult pigs, about 2 cm above the level of the eyes (Figure 5a).

Temporal method

This is only suitable for firearms. The pig is shot from the side so that the bullet enters the skull at a point midway between the eye and the base of the ear. The bullet should be directed horizontally into the skull (Figure 5b). This method is preferred for adult pigs because of the heavier bone structure of the front of the skull.

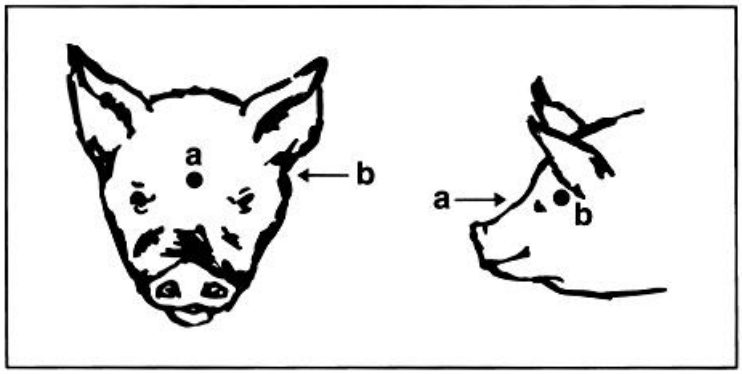


FIGURE 5

Humane destruction of pigs: recommended position and direction of fire for (a) frontal method (suitable for captive-bolt pistol or firearm) or (b) temporal method (suitable for firearm only).

Feed one-third of the normal ration before commencement of destruction. Pigs will stay calmer and therefore be easier to handle. If slaughter is likely to be delayed, ensure sufficient feed is on hand.

- *Growers*: pack in tight; work round perimeter of yard, then climb in to finish balance of group. Pigs usually quieten as destruction progresses.
- *Sows*: do not yard too tightly, as they become upset if jammed and will start climbing on rails; work steadily; do not hurry. Use heavy duty cartridges in captive-bolt pistols (see Captive-bolt pistols, p. 10).
- *Boars*: use heavy-duty cartridges in captive-bolt (see Captive-bolt pistols, p. 10); if this is too difficult, use a .22 magnum rifle.
- *Small pigs*: use standard captive-bolt cartridges (see Captive-bolt pistols, p. 10). It is preferable to have small pigs caught and held over the rail of the yard while destroyed. A wheelbarrow can then be a useful means of conveyance to the front-end loader.

GOATS

Using either a captive-bolt pistol or firearm, aim the weapon to the skull behind the horns as shown in Figure 6. Aim in line with the animal's mouth.

Kids may also be shot from the front, as for cattle. This method is not suitable for mature goats, as the brain is located well back in the skull compared to other livestock. Sodium pentobarbitone is also appropriate.

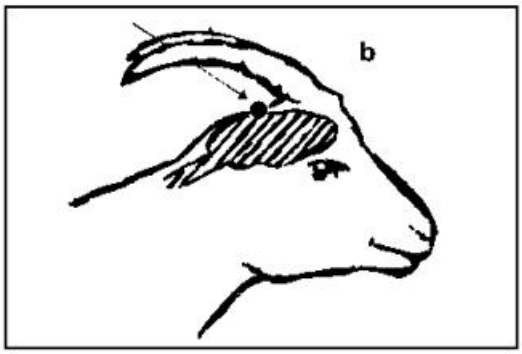


FIGURE 6

Humane destruction of goats: recommended position and direction of fire (captive-bolt pistol or firearm).

Newborn kids should be separated and given sodium pentobarbitone (intraperitoneal, 3–5 ml of a suitable product).

HORSES, DONKEYS AND MULES

These animals can be destroyed either by intravenous injections of euthanasing drug or shooting, as detailed below.

Frontal method

The firearm should be directed at the point of intersection of diagonal lines taken from the base of each ear to the opposite eye, aiming at the spine (Figure 7a).

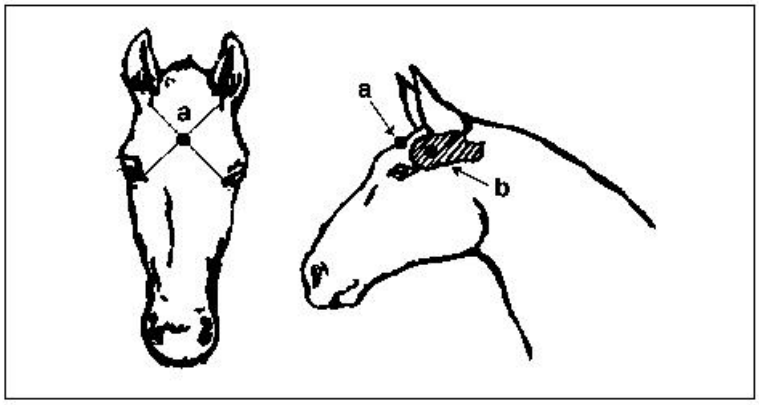


FIGURE 7

Humane destruction of horses: recommended position and direction of fire for (a) frontal method or (b) temporal method.

Temporal method

The horse is shot from the side so that the bullet enters the skull midway between the eye and the base of the ear (Figure 7b). The bullet should be directed horizontally.

Recommended rifles for horses are .22 magnum (hollow point) or .44 magnum. Captive-bolt pistols are not recommended for horses, because some horses rear before the operator can withdraw the bolt or move out of danger. Horses in the public view can be exsanguinated humanely by severing the abdominal aorta *per rectum.*

Paddock/extensive area destruction

As for cattle and buffalo (see Cattle and buffalo, p. 15).

DEER

A firearm or captive-bolt pistol should be directed at the forehead where lines taken from the base of each ear to the opposite eye intersect (Figure 8a). The firearm should be fired horizontally into the forehead. If using a captive-bolt on adult bucks, heavy duty cartridges are necessary.

If the deer are disturbed when approached from the front, an equally effective method is to fire the instrument through the skull just behind the base of the antlers. The weapon should be aimed in line with the animal's muzzle (Figure 8b).

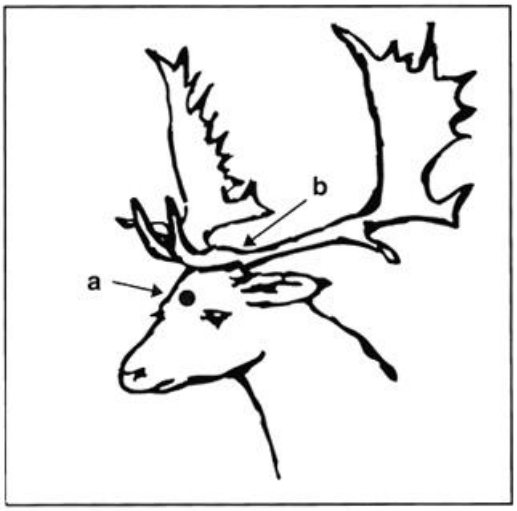


FIGURE 8

Humane destruction of deer: recommended position and direction of fire for (a) firearm or captive-bolt pistol or (b) alternative position for disturbed deer.

BIRDS

For small numbers of birds, for example fancy breeds or pigeons, the preferred methods are dislocation of the neck using burdizzos, bone cutters, secateurs or bare hands or intracardiac or intraperitoneal injection of sodium pentobarbitone.

For large numbers of birds in commercial poultry units, the preferred method is gassing with carbon dioxide. This method involves lining large garbage waste bins (skips) with plastic sheeting that forms a canopy over the top of the bin.

Birds can be caught using teams of 10–15 labourers. Experienced catching teams may be available. Chicks are easily caught under heaters and are transferred to skips in plastic garbage bins. Broilers on the ground are driven with a movable hessian wall to the catching area, where they are caught and placed directly into skips.

Caged birds are more difficult and progress is slower. Each catcher removes three or four birds from cages and carries them by the legs to skips.

Layers on perches are best caught at night or during low light, when they are quiet.

Carbon dioxide is transferred to the bottom of the skips through garden hose fitted to the top of the cylinders. The carbon dioxide should be decanted in bursts of 30–45 seconds. It is essential not to decant too quickly or the bottles will freeze when about half empty.

On average, half a 45 kg cylinder of carbon dioxide is needed for the 3 m^3 skips and three or more cylinders for the 20 m^3 skips. Carbon dioxide should be added at a rate sufficient to ensure that birds succumb before others are placed on top of them. Skips should be three-quarters filled with birds, sealed and transported to the disposal site. Care must be taken to ensure that no bird is still alive when dropped into the burial pit. Should this happen, birds must be immediately caught and humanely killed.

For humane destruction of farmed ostriches, birds should be restrained firmly and dispatched by captive bolt or injection of sodium pentabarbitone into the jugular vein.

DOGS

Injectable agents are the best method for destroying dogs that can be handled. Intravenous sodium pentobarbitone (40 mg/kg) is the ideal method. Intracardiac injections are favoured for puppies and small dogs. Other drugs given subcutaneously or intravenously may be used initially, for example xylazine (2 mg/kg) or ketamine (20 mg/kg), if necessary using a tranquillizer gun. Once the dog is sedated, intravenous barbiturates can be used to kill the animal.

If a tranquillizer gun is not available, injection by any route will be too dangerous for some totally unmanageable dogs and for rapid or suspect rabid dogs. A lasso on a pole may be useful to help catch and control these dogs. Including a sedative (e.g. sodium pentobarbitone) in the food may be an appropriate preliminary to an injectable agent. Dogs may have to be restrained with muzzles or tape before destruction.

Some dogs will have to be shot through the heart.

CATS

Injectable agents are the best method for destroying cats. Intravenous or intracardiac sodium pentobarbitone (40 mg/kg) is the preferred drug. Alphaxalone (Saffan®) may be used as a preliminary. Intraperitoneal injections can cause excitation before death. Tranquillizer guns are not suitable for cats, because cats are small, fast-moving targets.

Animals that are not easy to handle may have to be put in a hessian bag, injected through the bag and left in a cage until dead. Alternatively, they can be placed in a plastic bag or box into which anaesthetic gases (including carbon dioxide) are piped, using oxygen as the carrier gas. Anaesthesia is usually quick and quiet but death may take some time - at least 20 minutes with carbon dioxide but less with some of the other anaesthetic gases. Once the animal is unconscious, it may be removed and killed with an overdose of barbiturate.

RATS, MICE, GUINEA PIGS

Any of the physical or chemical methods described above can be used in a laboratory. The method of choice, however, is carbon dioxide. Newborns are resistant to carbon dioxide and need prolonged exposure or a combination of carbon dioxide and cervical dislocation. If pentobarbitone is used, it should be given by the intraperitoneal route (rats and mice 100 mg/kg, guinea pigs 90 mg/kg).

RABBITS

Physical methods such as cervical dislocation should only be used by skilled personnel and only on rabbits less than 1 kg in weight. The preferred method for laboratory rabbits is intraperitoneal pentobarbitone 60 mg/kg. Intravenous barbiturate injections of the very concentrated barbiturate euthanasia solution into the ear vein are often painful and may be distressing. Standard anaesthetic solutions should therefore be used. Rabbits should be restrained, since an excitement phase may occur, especially if the intraperitoneal or intravenous injection is incorrectly administered.

Induction of anaesthesia with carbon dioxide, as described for birds and cats, is slow and animals appear to become apprehensive before unconsciousness supervenes. The method is therefore not recommended. Overdosing with other inhaled anaesthetic agents may be used.

PRIMATES

Chemical restraint by means of ketamine (20 mg/kg intramuscularly) followed by an overdose of barbiturate given by the intravenous or intracardiac route (50 mg/kg) is recommended for laboratory primates.

FISH

A sharp blow to the head followed by destruction of the brain has been recommended as a physical method of euthanasia. If chemical methods are preferred, an overdose of anaesthetic such as MS222 (tricaine methane sulphonate) can be used or carbon dioxide can be bubbled into the water. This should be followed by destruction of the brain.

CIRCUS AND ZOO ANIMALS

The assistance of a veterinarian with experience of handling and destroying circus and zoo animals should be sought. If none is available, the methods outlined above should be extrapolated to the various species.

Glossary

Ammunition

- Hard point: hard metal ammunition that passes through tissues cleanly but can leave the target at high velocity, causing danger to other people/animals in the area;
- Soft/hollow point: ammunition made of softer metal or with a hollow point that flattens on impact, causing greater damage to tissues; does not exit the target unless it fails to encounter bone or solid muscle.

Burdizzo: castrating pincers.

Captive-bolt pistol: humane animal killer; takes either a blank cartridge that delivers a knockout blow to the skull or a penetrating bolt that is driven a short distance into the brain; the operator does not have to be a marksman as the instrument is pressed firmly against the animal's skull before firing.

FIGURE 9

Movement restriction
Movement into and out of infected premises must be rigorously checked by quarantine and/or road barriers.

Disinfectant: an agent used to destroy micro-organisms outside a living animal.

Disposal: sanitary removal of animal carcasses and other infected material by burial, burning or some other process, so as to prevent the spread of disease.

Exsanguination: severe loss of blood.

Firearm: small arms weapon (gun or rifle).

Infected premises: a defined area, which may be all or part of a property, in which an exotic disease or its infective agent exists or is believed to exist; an infected premises is subject to quarantine and to eradication or control procedures.

Infected premises operations team: team appointed by the local disease control centre (LDCC) controller to coordinate/supervise operations at the infected premises.

Injection sites

- intracardiac: into the heart;
- intraperitoneal: into the peritoneal (abdominal) cavity;
- intramuscular: into muscle (the needle is passed deeply into the substance of a muscle before the fluid is injected);
- intrathoracic: into the thoracic (chest) cavity;
- intravenous: into a vein;
- subcutaneous: under the skin (hypodermic).

Nuchal crest: transverse bony ridge across the back margin of the roof of the vertebrate skull.

Poll: crown of the head.

Quarantine: legal restrictions limiting movement imposed on a place, animal, vehicle or other things.

Susceptible animals: animals that can be infected with the disease.