Premedication of Dogs with Acepromazine or Pentazocine Before Euthanasia with Carbon Monoxide

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ABSTRACT

Euthanasia of unwanted or sick animals should always be done in a humane manner. This study involving two groups of 12 dogs evaluated a two step method of euthanasia using first acepromazine or pentazocine then inhalation of carbon monoxide.

During the experiment, behavioral reactions (anxiety, agitation, vocalization and sphincter relaxation) and physiological parameters (electroencephalogram, electrocardiogram, arterial blood pressure, respiratory and heart rates and serum cortisol) were monitored.

The results showed that both drugs modified many behavioral reactions and physiological changes associated with administration of carbon monoxide. Acepromazine and pentazocine reduced by 25% and 20% respectively the number of dogs that showed vocalization and agitation. In acepromazine premedicated dogs, the duration of these signs was significantly diminished and sphincter relaxation did not occur in more than 50% of cases. Furthermore, with the use of acepromazine, no significant peaks or drastic drops were noticed in the heart and respiratory rates and in the arterial blood pressure. These manifestations are usually related to stress.

In light of these results, it is recommended to premedicate dogs with acepromazine before submitting them to euthanasia by carbon monoxide inhalation.

Key words: Acepromazine, pentazocine, behavioral reactions, physiological parameters, carbon monoxide, euthanasia, dog.

RÉSUMÉ

L'euthanasie d'animaux abandonnés ou malades doit toujours s'effectuer d'une façon humanitaire. Cette étude impliquait deux groupes de 12 chiens et elle visait à évaluer une méthode d'euthanasie qui comportait d'abord l'injection intramusculaire d'acépromazine ou de pentazocine et ensuite l'inhalation de monoxyde de carbone.

Au cours de l'expérience, les auteurs évaluèrent certains changements de comportement, entre autres : l'anxiété, l'agitation, la vocalisation et le relâchement des sphincters; ils surveillèrent aussi les paramètres physiologiques suivants: l'électroencéphalogramme, l'électrocardiogramme, la pression artérielle, les fréquences cardiaque et respiratoire, ainsi que la teneur du sérum en cortisol.

L'expérience permit de constater que les deux drogues précitées modifiaient plusieurs aspects du comportement et certains changements physiologiques, reliés à l'inhalation de monoxyde de carbone. L'acépromazine et la pentazocine réduisirent respectivement de 25% et 20% le nombre de chiens qui manifestèrent de la vocalisation et de l'agitation. Chez ceux qui avaient reçu de l'acépromazine, la durée de ces deux manifestations accusa une réduction appréciable et le relâchement des sphincters ne se produisit que dans environ 50% des cas. De plus, l'utilisation d'acépromazine n'engendra ni sommet ni chute drastique des fréquences cardiaque ou respiratoire et de la pression artérielle, manifestations ordinairement reliées au stress. Seule la teneur du sérum en cortisol subit une hausse, lors des manipulations prélables à l'inhalation de monoxyde de carbone.

Les résultats de cette expérience permettent par conséquent de recommander l'administration intramusculaire d'acépromazine aux chiens, avant de les soumettre à l'euthanasie par inhalation de monoxyde de carbone.

Mots clés: acépromazine, pentazocine, changements de comportement, paramètres physiologiques, monoxyde de carbone, euthanasie, chien.

INTRODUCTION

The two step method of euthanasia, using a sedative, a tranquilizer or an analgesic followed by a euthanizing agent, is widely used in European countries (1) and is becoming more common in North America. This two phase method could also be applied when carbon monoxide (CO) is used to submit dogs to euthanasia.

A previous study evaluating CO euthanasia in dogs has shown that a period of anxiety, agitation and vocalization occurred when the electroencephalogram (EEG) showed abnormal cortical function characterized by a medium frequency/high amplitude pattern. During that period, there still remained a grey zone of about 3 to 8 s when a state of consciousness or unconsciousness could not be established (2). Since carbon monoxide is largely used for euthanasia of small mammals (3,4,5,6), is it important to make this method as humane as possible.

The first objective of this study was to verify the usefulness of premedication before CO euthanasia for the alle-

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Supported by a grant from the Canadian Veterinary Research Trust Fund.

Submitted March 22, 1984.

viation or the suppression of signs of anxiety, vocalization and agitation, associated with CO inhalation euthanasia in dogs. Dogs premedicated with acepromazine or pentazocine have been used. Physiological and behavioral parameters, including brain and cardiac activities, arterial blood pressure, respiratory rate, serum cortisol levels and behavioral reactions were evaluated. The second objective was to compare the efficiency of acepromazine and pentazocine for premedication of these animals.

MATERIALS AND METHODS

Two groups of 12 mongrel dogs, male and female, ranging in age from one to ten years (mean 1.8 years) were used in this study. A physical examination was performed on each animal upon arrival at the laboratory to establish its health status. Only healthy dogs were used. For recording the EEG, electrodes were implanted over the cerebral cortex as previously described (2,7,8). The experimental procedure was performed four days after surgery. The dogs were conditioned to be placed in the restraint apparatus before the day of the experiment to minimize stress. Approximately 20 min before CO inhalation began, twelve dogs received acepromazine maleate (Atravet, Ayerst Laboratories Inc., Montréal, Québec, Canada) at a dose of 0.3 mg/kg of body weight IM, while the other twelve dogs received pentazocine (Talwin, Winthrop Laboratories, Div. Sterling Drug Ltd., Aurora, Ontario, Canada) at a dose of 0.35 mg/kg of body weight IM. Thereafter, a mixture of compressed air and pure carbon monoxide from cylinders was given by inhalation at a concentration of 6% and at a rate of 6.4 L/min. A fiberglass head cage, part of the restraint apparatus, connected to a modified anesthesia machine enabled controlled inhalation of CO (2). To record electrocardiogram (ECG) and respiratory rate, impedance electrodes (Narco Scientific Ltd., Downsview, Ontario, Canada) were placed on the skin on each side of the thorax. A catheter was placed in the left saphenous vein to draw blood for measurement of serum cortisol level. Blood samples were taken before the experiment, when the animal was placed in the restraint apparatus, at the onset of CO inhalation and when the EEG was flat. All electrode leads were connected to a polygraph (Narco Scientific Ltd., Downsview, Ontario, Canada) and monitoring was done throughout the experiment. Onset of CO inhalation and all reactions (anxiety, agitation, vocalization, sphincter relaxation, respiratory spasms) were identified on the recordings. To monitor the arterial blood pressure, an ultrasonic doppler flow detector (Parks Electronics Laboratories, Beaverton, Oregon, USA) was used (2). Readings were taken every 30 s until the blood pressure reached 0 mm Hg. To establish statistical significance of $P < 0.05$, analysis of variance was used. All results were expressed as mean ± standard error.

RESULTS

Good recordings for EEG, ECG and respiratory rate were obtained from all dogs during this experiment. Serum cortisol levels and arterial blood pressure were also measured on all dogs.

A — DOGS PREMEDICATED WITH ACEPROMAZINE

1 — EEG Modifications

The EEG leads used were: frontal/temporal lead, four dogs (33.3%); frontal/occipital lead, three dogs (25.1%); and temporal/occipital lead, five dogs (41.6%). In all dogs, after the injection of acepromazine, high frequency/high amplitude waves were noted intermittently amongst the high frequency/low amplitude waves normally found prior to the onset of CO inhalation (Fig. 1). The higher amplitudes were seen most commonly in the cortical occipital area, then in the cortical temporal and frontal areas.

The changes in the EEG appeared at an average of 1 min 47 s ± 12 s after the onset of CO inhalation. The EEG became isoelectric at 2 min 47 s ± 13 s (Fig. 6). The average duration of these changes was 59.8 ± 13.93 s. On analysis of each lead, we note that the first EEG modifications appeared (1 min 25 s ± 9 s) at the level of the frontal cortical area. The modifications also lasted for the longest period of time (an average of 1 min 16 s ± 22.17 s) in that area of the cerebral cortex. For the temporal and occipital leads, the average time of onset and the duration of EEG modifications were 1 min 53 s ± 16 s and 49 ± 13.03 s; 2 min 2 s ± 15 s and 57 ± 16.65 s respectively.

2 — Behavioral Reactions

The average periods of agitation and vocalization varied from 1 min 47 s ± 16 s to 2 min 12 s ± 14 s and 1 min 58 s ± 17 s to 2 min 18 s ± 14 s respectively after onset of CO inhalation (Fig. 6). The period of vocaliza-

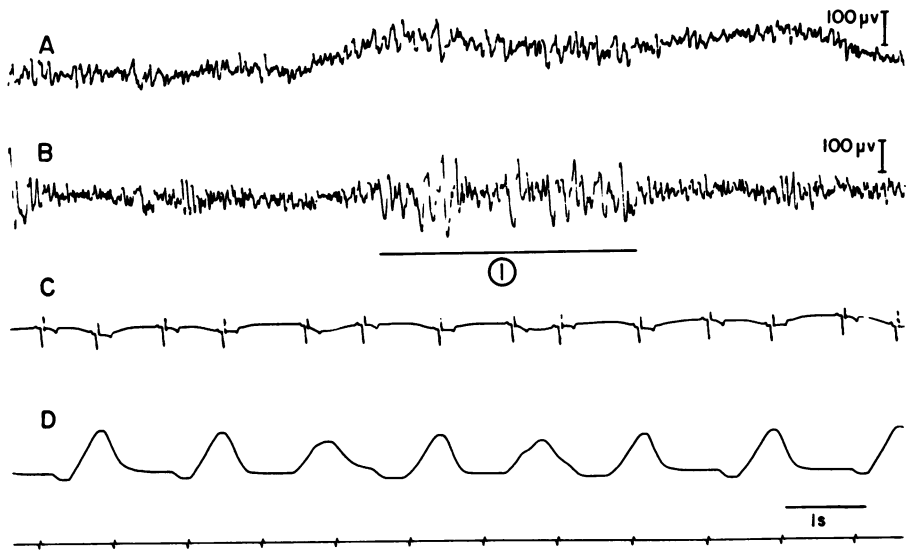

Fig. 1. Modifications of the EEG following the administration of acepromazine. A and B — respectively temporal and occipital EEG, C — ECG and D — respiration. Time is 15 minutes after injection of acepromazine.
① Note spindle-like waves in occipital lead.

tion lasted an average of 21.0 ± 6.93 s in the eight dogs that showed this behavior, while agitation was observed for 23.5 ± 4.9 s in the ten dogs manifesting this reaction. Two dogs did not show signs of vocalization or agitation and two others did not vocalize.

An overall analysis revealed that the average vocalization and agitation period was from 1 min 47 s ± 16 s and extended to 2 min 16 s ± 24 s after onset of CO inhalation with an average duration of 28.8 ± 5 s. These behavioral manifestations appeared at different times compared to the onset of EEG modifications. In 58.3% of the dogs agitation started before or simultaneously with the EEG modifications, while in the remaining dogs, it started after modifications of EEG or was not observed. Vocalization began before or simultaneously with the EEG modifications in 25% of the dogs, while in the remaining 75% it started after the onset of EEG modifications or did not occur (Table I).

Furthermore, anal and urinary sphincters relaxed in only two dogs (17%) at a mean time of 2 min 44 s ± 13 s after onset of CO inhalation (Fig. 6). In six dogs, these behavioral manifestations did not occur (50%) and in four dogs only micturition occurred (33%).

3 — Heart Rate

The heart rate increased slightly, 0.5 fold, to reach 120 beats/min at 1 min 30 s after the onset of CO inhalation. Thereafter, the rate diminished but showed another peak at 9 min 0 s (Fig. 2). The heart beat stopped on average at 15 min 32 s ± 269 s (Fig. 6). In four dogs, heart beats persisted for more than 20 min.

4 — Respiratory Rate

The respiratory rate did not vary significantly during the first 3.5 min after the onset of CO inhalation. Thereafter, we noticed a slow fall in this physiological parameter (Fig. 3). Respiration ceased at 4 min 2 s ± 14 s (Fig. 6). Respiratory spasms were seen at 5 min 54 s ± 16 s and at 8 min 13 s ± 21 s (Fig. 6).

5 — Arterial Blood Pressure

The arterial blood pressure did not change significantly during the first 3 min of the experimental process. Thereafter, it slowly diminished to reach 0 mm Hg at 8 min 10 s ± 57 s (Fig. 4).

6 — Serum Cortisol

The serum cortisol levels showed a significant increase from 30.35 ± 9.66 n mol/L to 165.54 ± 28.77 n mol/L during the preexperimental manipulations, before the onset of CO inhalation. During CO inhalation the level of serum cortisol dropped slightly from 226.24 ± 36.42 n mol/L to 183.20 ± 30.62 n mol/L (Fig. 5).

TABLE I. Time of Onset of Behavioral Reactions versus EEG Modifications in Dogs Pretreated with Acepromazine

Onset of agitation versus EEG modifications Agitation	Before	After	Same	None
Number of dogs	4	3	3	2
$\bar{X}$ time (s)	17.25	13.67	0	N/A
Onset of vocalization versus EEG modifications **Vocalization**	**Before**	**After**	**Same**	**None**
Number of dogs	2	5	1	4
$\bar{X}$ time (s)	16.00	20.40	0	N/A

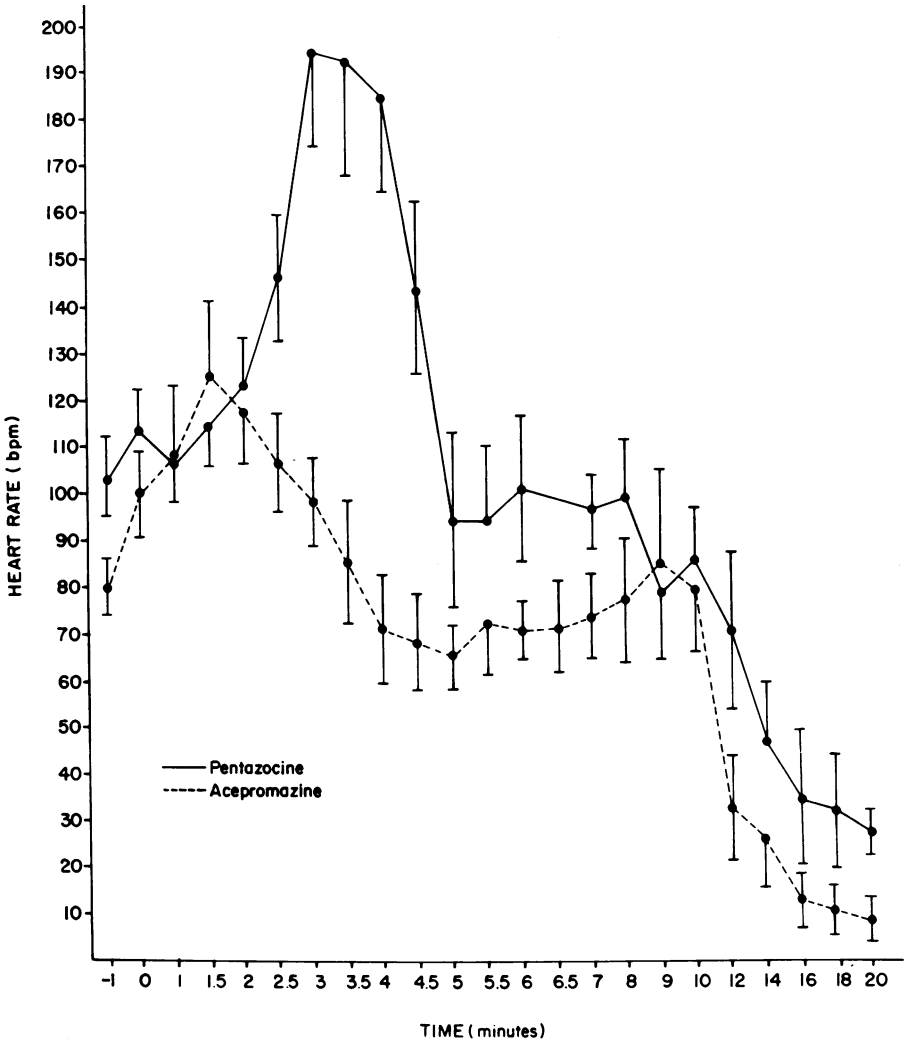

Fig. 2. Modifications of the heart rate during CO euthanasia of dogs pretreated with acepromazine or pentazocine. With acepromazine the heart rate gradually diminished to 0 bpm. With pentazocine the heart rate increased significantly approximately one minute before cerebral death, then gradually decreased to 0 bpm.

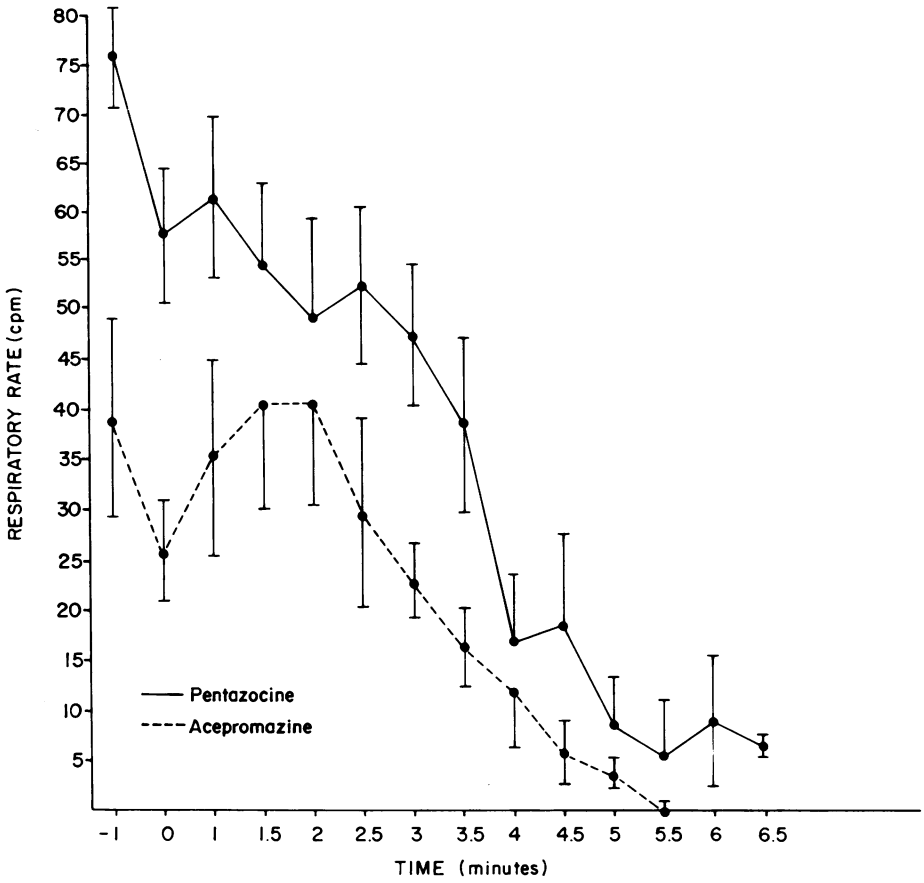

Fig. 3. Modifications of the respiratory rate during CO euthanasia of dogs pretreated with acepromazine or pentazocine. The respiratory rate is significantly decreased with the use of acepromazine before onset of CO inhalation. The respiratory rate decreased gradually during the euthanasia process with the use of either drugs.

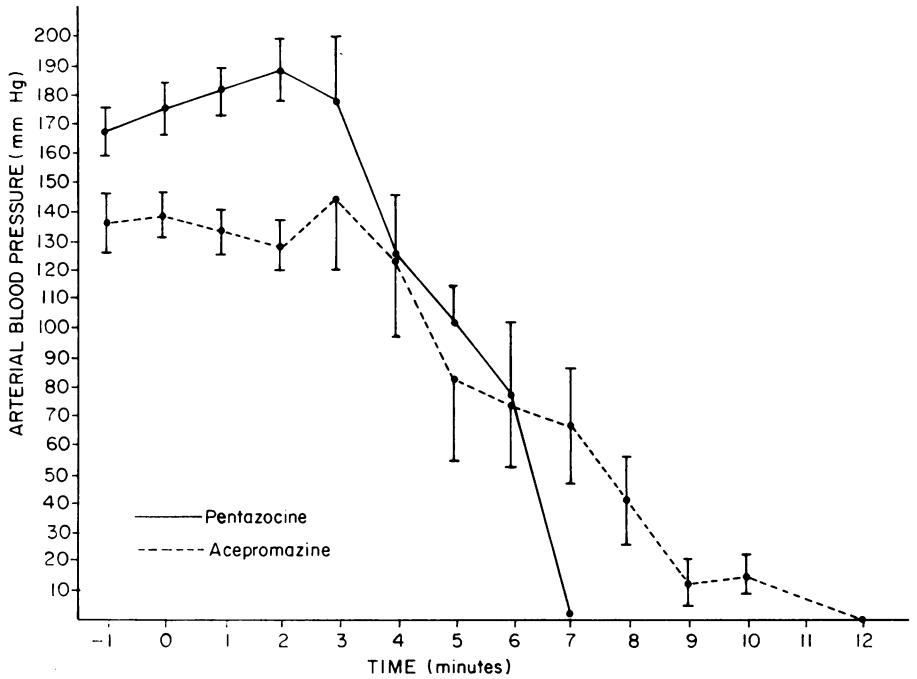

Fig. 4. Modifications of the arterial blood pressure during CO euthanasia of dogs pretreated with acepromazine or pentazocine. With pentazocine, the arterial blood pressure dropped rapidly at the outset of EEG modifications. Within four minutes it reached 0 mm Hg. Acepromazine lowered the arterial blood pressure from the start of the experiment. After cerebral death it gradually decreased to reach 0 mm Hg, twelve minutes after the beginning of CO inhalation.

B — DOGS PREMEDICATED WITH PENTAZOCINE

1 — EEG Modifications

The derivations used were: frontal/temporal lead, five dogs (41.7%), frontal/occipital lead, six dogs (50%) and temporal/occipital lead, one dog (8.3%). The modifications of EEG appeared at an average of 3 min 17 s ± 14 s after the onset of CO inhalation. The EEG became isoelectric at 4 min 7 s ± 14 s (Fig. 6). The average duration of these modifications was 48.6 ± 6 s. An analysis of each lead revealed that the first EEG modifications appeared at 2 min 54 s ± 10 s in the occipital cortical area. The modifications were the longest at that level, averaging 56.0 ± 7 s. For the frontal and temporal leads, the average time of onset and the duration of EEG modifications were respectively: 3 min 24 s ± 15 s and 42.6 ± 5 s; 3 min 44 s ± 21 s and 40.0 ± 7 s.

2 — Behavioral Reactions

The average period of agitation and vocalization varied from 3 min 58 s ± 20 s to 4 min 32 s ± 19 s and 3 min 36 s ± 14 s to 4 min 12 s ± 15 s respectively (Fig. 6) after onset of CO inhalation. The period of agitation lasted an average of 34.3 ± 8 s in the eight dogs that showed that behavior, while vocalization was manifested in 11 dogs for 36.5 ± 6.3 s. Four dogs did not show agitation and one dog did not vocalize.

The overall analysis revealed that the average vocalization and agitation period was from 3 min 35 s ± 14 s until 4 min 12 s ± 15 s after onset of CO inhalation, with an average duration of 36.0 ± 7 s. These behavioral manifestations appeared at different times compared to the onset of EEG modifications. In 16.6% of the dogs, agitation and vocalization started before or simultaneously with EEG modifications, while in the remaining dogs it started after modifications of EEG or was not observed (Table II).

Anal and urinary sphincter relaxation occurred in nine dogs (75%) at an average time of 4 min 2 s ± 14 s (Fig. 6). Micturition was more commonly observed (67% of dogs). In three dogs, these behavioral manifestations did not occur (25%).

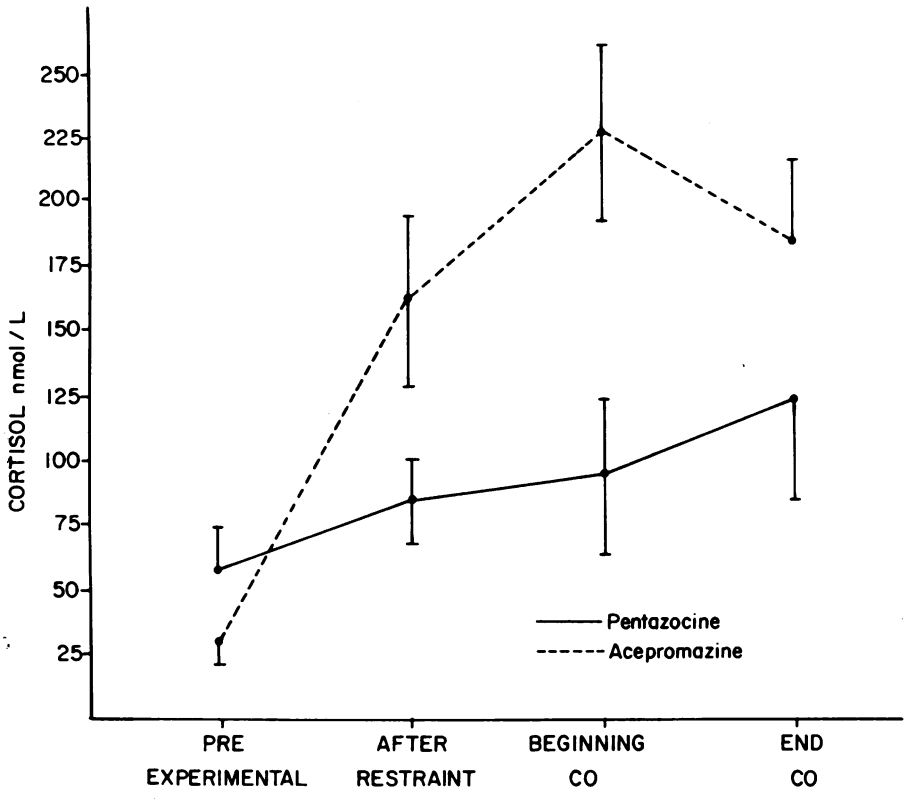

Fig. 5. Variations of the cortisol levels during CO euthanasia of dogs pretreated with acepromazine or pentazocine. With the use of acepromazine the serum cortisol values increased significantly during the preexperimental handling of the dogs. During CO inhalation no increase was noted. Pentazocine inhibited the secretion of serum cortisol before and during CO inhalation, the increases noticed at the different phases of the experiment were not significant.

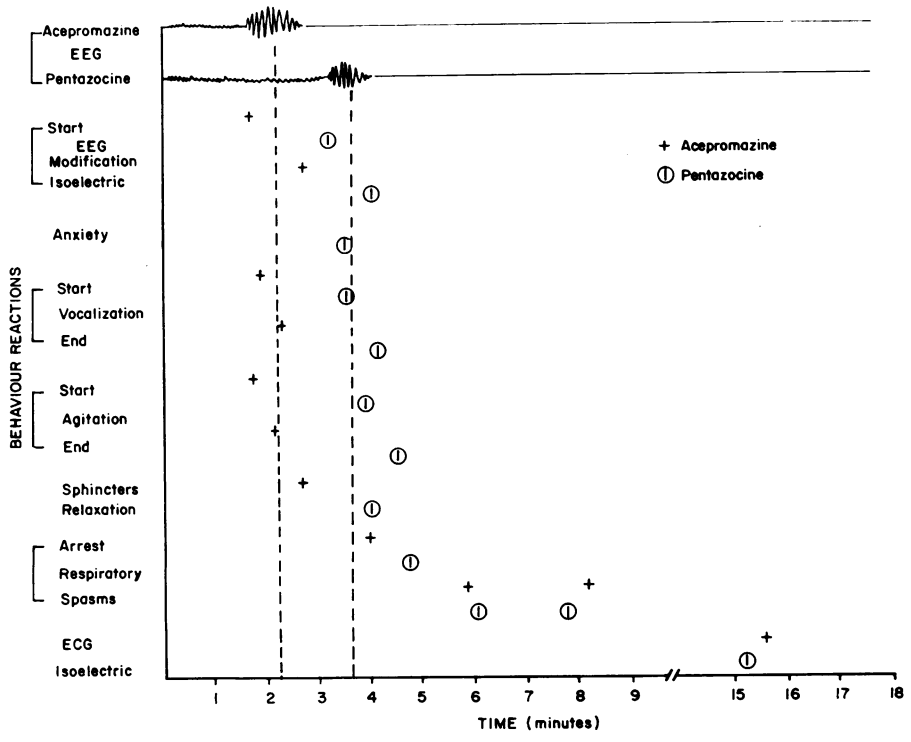

Fig. 6. Behavioral and physiological reactions in relation with EEG modifications during CO euthanasia of dogs pretreated with acepromazine or pentazocine. Signs of stress and anxiety such as agitation and vocalization still started in the conscious phase irrespective of the drug used. Note that these behavioral signs did not occur in all dogs.

3 — Heart Rate

The heart rate doubled within the first 3 min after the onset of CO inhalation and decreased abruptly thereafter to the level found at the beginning of the experiment (time -1 min). This plateau lasted approximately 5 min. The heart rate then decreased gradually to become isoelectric at 15 min 6 s ± 114 s (Figs. 2 and 6). In three dogs, heart beats persisted for more than 20 min.

4 — Respiratory Rate

From the beginning of CO inhalation, a progressive fall in the respiratory rate was observed. Between 3.5 to 4 min the decrease was more pronounced (Fig. 3). Respiration ceased at 4 min 47 s ± 17 s and respiratory spasms were seen at 6 min 7 s ± 23 s and 7 min 50 s ± 38 s (Fig. 6).

5 — Arterial Blood Pressure

The arterial blood pressure increased slightly to reach a peak at 2 min after the onset of CO inhalation. Thereafter, it fell abruptly to 0 mm Hg at 5 min 35 s ± 22 s (Fig. 4).

6 — Serum Cortisol

The serum cortisol levels did not significantly increase before (59.32 ± 14.07 n mol/L), during (94.35 ± 28.42 n mol/L) or at the end (123.33 ± 35.59 n mol/L) of the euthanasia process (Fig. 5).

DISCUSSION

Certain results obtained during this phase of our research project corroborate those obtained in the first part of our work evaluating CO euthanasia without premedication (2). For instance, in dogs premedicated with acepromazine, the preexperimental manipulations were shown, by an increase of the serum cortisol levels, to be more stressful to the animal than the effect of CO itself. This was not the case when pentazocine premedicated dogs were handled before the euthanasia process. Pentazocine would seem to have an inhibitory effect on cortisol release possibly by lowering stress in this group of dogs.

The anxiety period, manifested by anguish, distress, gaze and head movement, seen at the beginning of

TABLE II. Time of Onset of Behavioral Reactions versus EEG Modifications in Dogs Pretreated with Pentazocine

Onset of agitation versus EEG modifications Agitation	Before	After	Same	None
Number of dogs	1	6	1	4
$\bar{X}$ time (s)	13.0	15.0	0	N/A
Onset of vocalization versus EEG modifications **Vocalization**	**Before**	**After**	**Same**	**None**
Number of dogs	1	9	1	1
$\bar{X}$ time (s)	36.0	12.8	0	N/A

CO inhalation without premedication (2) disappeared with the use of acepromazine. This anxiety period occurred in only two pentazocine treated dogs, even though the EEG modifications started at 3 min 17 s ± 14 s. It seems that pentazocine inhibited this period of anxiety in ten dogs.

In acepromazine treated dogs, only the time of onset of the EEG modifications was altered. The onset of EEG modifications in these premedicated dogs occurred very rapidly at 1 min 47 s ± 12 s as compared to 3 min 8 s ± 31 s when CO alone was used. The duration of the EEG modifications was not reduced in dogs treated with acepromazine, being 59.8 ± 13.93 s as compared to 51.39 ± 18.99 s when CO alone was used (2). With the use of pentazocine there was not a reduction of the onset or the duration of EEG modifications.

As previously described, the onset and the duration of the periods of agitation and vocalization varied from dog to dog (2). When CO alone was given, all dogs manifested these behavioral changes which were related to the onset and duration of EEG modifications (2). In this study, two dogs pretreated with acepromazine did not show agitation and four dogs did not vocalize. The average duration of these behavioral manifestations was 28.8 ± 5 s as compared to 47.89 ± 24.18 s when CO was given without premedication (2). Thus, acepromazine pretreatment significantly reduced i) the number of dogs showing (33.3%) and ii) the duration (39.8%) of these behavioral manifestations.

When pentazocine and CO were given, four dogs did not show agitation and one dog did not vocalize. The average duration of these behavioral manifestations was 36.0 s ± 7 s, which is a time reduction of 25% as compared to the results obtained in nontreated dogs (2). Thus, pentazocine pretreatment reduced the number of dogs showing (33.3%) and the duration (24.8%) of these behavioral changes.

Anal and urinary sphincter relaxation was markedly reduced when acepromazine was used. Sphincter relaxation was seen in only 50% of the dogs and four out of six dogs only showed micturition. With the use of pentazocine, sphincter relaxation was seen in 75% of the dogs. In eight out of nine dogs micturition was noticed, while defecation was seen in six out of nine dogs. This reaction occurred just before cerebral death, whether or not premedication was used.

The heart rate increased significantly 3 min after CO inhalation began in pentazocine treated dogs. This compares favorably with the results found when CO alone was used (2). In dogs pretreated with acepromazine, this peak was not noticed before cerebral death occurred. The antiarrhythmic and antiventricular fibrillation effects of acepromazine could explain the absence of cardiac rate increase (9). In dogs pretreated with acepromazine the heart rate gradually decreased at the time of cerebral death and was stable for approximately 5 min. Thus, the rapid drop in the heart rate, occurring when CO alone was used (2), was not observed after acepromazine pretreatment. With or without pentazocine pretreatment, a similar heart rate reaction was observed. Because of the extensive reserves of the heart, the tracings became isoelectric a long time after cerebral death, whether premedication was given or not. The increased activity found at approximately 9 min could be explained by the presence of ventricular fibrillation in certain dogs.

The arterial blood pressure maintained approximately the same level until the EEG became isoelectric. Thereafter, it gradually decreased until it reached 0 mm Hg at 8 min 10 s ± 57 s when acepromazine was used and at 5 min 35 s ± 22 s with pentazocine. As reported elsewhere, a low arterial blood pressure was also noticed at the beginning of the experiment when acepromazine was used (10,11). In a previous study of euthanasia using CO without acepromazine or pentazocine pretreatment, a rapid drop in the arterial blood pressure began approximately 60 s before cerebral death (2). The 0 mm Hg level was reached more rapidly in animals not receiving acepromazine. This sharp drop could be attributed to a vasculogenic shock due to the important metabolic changes and resulting vasodilatation (12). Due to its tranquilizing and cardiac effects (9), acepromazine probably prevented the occurrence of this phenomenon. In pentazocine treated dogs, the blood pressure increased slightly before cerebral death, then did not drop as quickly.

Neither drug appears to alter the respiratory rate after the onset of CO inhalation. However, respiration became isoelectric more rapidly in acepromazine pretreated dogs when compared with results of a previous study (2). It appears that acepromazine is a more potent depressor of the respiratory rate (11).

Acepromazine did not modify the secretion rate of cortisol. Similar serum cortisol levels were obtained when CO alone was used (2). This part of the experiment confirms again the marked influence of the preexperimental handling of the dogs on serum cortisol levels. Pentazocine markedly controlled the production of serum cortisol during the preexperimental handling of the dogs.

In general, acepromazine reduced the heart and respiratory rates, and the arterial blood pressure before the onset of CO inhalation. It also decreased the number of dogs showing vocalization and agitation during the administration of CO. Most importantly, the duration of these behavioral manifestations was reduced by approximately 40% in the dogs pretreated with acepromazine. When agitation and vocalization were analyzed

separately, a reduction of more than 50% was found. Furthermore, the number of dogs showing sphincter relaxation was markedly decreased (50%). These results indicate that acepromazine diminishes behavioral and physiological reactions of dogs faced with stressful situations.

In pentazocine pretreated dogs we noticed a decrease in the number of dogs that showed vocalization and agitation. The duration of these behavioral manifestations was also reduced by approximately 11 s. The frequency of sphincter relaxation was also reduced by 33%. The arterial blood pressure and the heart rate showed a pattern similar to that found in non-treated dogs.

After pretreatment with either drugs followed by CO inhalation, the respiratory rate gradually decreased showing no peak or sharp drops. When CO alone was used, significant variations (increases and decreases) were found in many behavioral manifestations and physiological parameters (2). These findings could indicate an acute stress or a functional exhaustion of organs.

Cerebral death, as expressed by a flat EEG, was more rapidly attained in dogs pretreated with acepromazine, than in those pretreated with pentazocine or given CO alone. The analysis of these results and the study of such behavioral reactions as vocalization, agitation and sphincter relaxation indicate a reduced sensibility to stress, in dogs pretreated with acepromazine, as compared with those for which CO inhalation without premedication was used. Furthermore, the arterial blood pressure and the heart rate gradually decreased after cerebral death in the acepromazine pretreated dogs. This decrease in stress was not as evident with the use of pentazocine. Although pentazocine reduced some behavioral manifestations, many physiological parameters paralleled those of non-treated animals. A comparative evaluation of behavioral reactions and physiological parameters is found in Table III.

In view of the present study, it is recommended that a two step method be used for dogs submitted to euthanasia by CO inhalation. The method favored would be i) pretreatment of dogs with acepromazine and ii) inhalation of CO. Acepromazine reduced the number of dogs showing vocalization and agitation, and shortened the duration of these signs. Acepromazine also prevented the precipitous fall in heart rate and arterial blood pressure. A secondary advantage of acepromazine is its disponibility.

ACKNOWLEDGMENTS

The authors thank Dr. Carlos Laurie and Mr. André Therrien for their assistance. This study was supported by a grant from the Canadian Veterinary Research Trust Fund.

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TABLE III. Comparative Evaluation of Behavioral Reactions and Physiological Parameters in Dogs Submitted to Euthanasia Using CO Inhalation Alone or the Two-step Method, Premedicating Dogs with Acepromazine or Pentazocine Before CO Inhalation

Behavioral reactions — Presence of	CO alone (2) N 18	Pretreatment: Acepromazine N 12	Pretreatment: Pentazocine N 12
Anxiety reaction	15 dogs (83%)	No dogs (0%)	2 dogs (17%)
Vocalization (V)	18 dogs (100%)	8 dogs (67%)	11 dogs (92%)
duration	—	21.0 ± 0.93 s	36.5 ± 6.3 s
Agitation (A)	18 dogs (100%)	10 dogs (83%)	8 dogs (67%)
duration	—	23.5 ± 4.90 s	34.3 ± 8 s
A/V total duration	47.9 ± 24.18 s	28.8 ± 5 s	36.0 ± 7 s
EEG modifications	All dogs	All dogs	All dogs
start	3 min 08 s ± 31 s	1 min 47 s ± 12 s	3 min 17 s ± 14 s
duration	51.4 ± 18.99 s	59.8 ± 13.93 s	48.6 ± 6 s
Sphincter relaxation	18 dogs (100%)	6 dogs (50%)	9 dogs (75%)
micturition	—	6 dogs (50%)	8 dogs (67%)
defecation	—	2 dogs (17%)	6 dogs (50%)
Physiological Parameters			
Respiratory rate			
A[a]	Slight increase	No significant variations	Gradual decrease
B[b]	Gradual decrease	Gradual decrease	Gradual decrease
Respiratory spasms			
B	6 min 41 s ± 68 s	5 min 54 s ± 16 s	6 min 7 s ± 23 s
B		8 min 13 s ± 21 s	7 min 50 s ± 38 s
Respiration ceased			
B	5 min 13 s ± 26 s	4 min 02 s ± 14 s	4 min 47 s ± 17 s
Arterial blood pressure			
A	No significant variations	No significant variations	Slight increase
B	Sharp decrease	Gradual decrease	Sharp decrease
0 mm Hg	6 min	8 min 10 s ± 57 s	5 min 35 s ± 22 s
Heart rate			
A	Significant increase	No significant increase	Significant increase
B	Sharp decrease	Gradual decrease	Sharp decrease
Heart isoelectric	18 min 33 s ± 227 s	15 min 32 s ± 269 s	15 min 16 s ± 114 s
Serum cortisol			
Preexperimental	Significant increase	Significant increase	No significant variation
During CO inhalation	Slight increase	Slight decrease	No significant variation

[a]A : Before
[b]B : After cerebral death

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Book Review

MECHANISMS OF DISEASES. *D.O. Slauson and B.J. Cooper. Published by Williams and Wilkins, Baltimore, Maryland. 1982. 420 pages. Price $37.50.*

This book is about general pathological principles and was written, according to the preface, for students of all kinds. The emphasis is on the application of "understanding the how and why disease states evolve".

It is difficult not to compare this book with Thomson's General Veterinary Pathology published in 1978 by W.B. Saunders. Both books essentially cover the same types of material, are about the same length and are divided into similar chapters. One's first impression that they are the same, soon gives way to the realization that they are different and complement each other. Thomson's book has relatively more illustrations and less text but this book uses the text to advantage and includes some scanning electron photomicrographs along with the transmission electron and light photomicrographs and gross lesion photographs. This book separates inflammation and immunopathology, as well as healing and repair. The chapter on immunopathology is an excellent one for all "students" be they in training, in practice or a laboratory. This chapter makes this book an even more valuable addition to a library. Both of these books have a place for those studying or reviewing general pathology, but this book is recommended for those who wish a bit more detail. Numerous line drawings illustrate the ideas discussed in the text. Recommended further readings are provided at the end of each chapter. The index is adequate and provides the option of finding illustrations by the use of italics but the difference in type is difficult to see.

This book is recommended reading for everyone interested in general pathology and in understanding better the mechanisms of disease.

T.W. Dukes