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CHLORAL HYDRATE AS A PREANAESTHETIC, NARCOTIC AND ANAESTHETIC FOR DONKEYS

(With 2 Tables & 12 Fig.)

By

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أيدارات الكلورال كحيد قبل التخدير ومنوم ومخدر في الحمير

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أجريت هذه الدراسة على ٢٤ حمرا ، حيث نوقشت النتائج الأكلينيكية والتأثيرات الباثولوجية لأيدرات الكلورال على الأعضاء المختلفه. وبينما مكنت الجرعه ٦ جم لكل ٥٠ كيلو من وزن الحيوان الحى من ترقيد الحيوان لكن دون تأثير معظم الحواس ، فقد أعطت الجرعات ٩ ، ١٧ ، ٣٠ جم لكل ٥٠ كيلو من وزن الحيوان تخدير لفته ٦٠ إلى ٩٠ دقيقه. ولكن بأستعمال الجرعه الأخيره غللت الحيوانات راقده لفته طويله أمتدت من ثلاث إلى أربع ساعات ، وقد كان للجرعات العاليه تأثير تثبيطى واضح على درجة الحراره. هذا وقد كانت الجرعه ١٥ جم لكل ٥٠ كيلو من وزن الحيوان جرعه مميتة للحمير. كذلك أوضحت الدراسة الباثولوجيه أن الجرعات الكبيره من أيدرات الكلورال ممكن تؤدى إلى تغيرات لكن غير مستديمه فى الكبد بالإضافة إلى التغيرات الأنحلاليه فى الطحال والكلى والقلب.

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SUMMARY

The present study was conducted on 24 donkeys to evaluate chloral hydrate as a preanaesthetic, narcotic and anaesthetic drug. The clinical results were discussed and the pathological effects on the parenchymatous organs and brain were also evaluated. Chloral hydrate in a dose rate of 6g/50kg body weight enabled to control donkeys in the recumbent position, but most reflexes were not affected in most animals. The drug in a dose rate of 9 and 12g/50kg body weight gave anaesthesia for a period of 60 to 90 minutes, but with the later dose the animals laid down for a long period (3 to 4 hours). The large doses have also severe hypothermic effect. Chloral hydrate in a dose rate of 15g/50kg body weight was lethal for donkeys. The histopathological results showed that the large doses of chloral hydrate may lead to reversible changes in the liver, haemocidrosis in the spleen and focal degenerative changes in the kidneys and heart.

INTRODUCTION

Despite the advances which have been made in the development of new narcotics for horses, chloral hydrate remains the best one. The drug could be used to induce basal narcosis varying in depth from a light stupor to complete general anaesthesia (HALL, 1971). While chloral hydrate has lost much of its popularity as a general anaesthetic in large animals, yet it is still used quite extensively. Its continued use in horses depends on the simplicity and on the duration of the effect of the induction dose, an interval adequate for many routine procedures (LUMB and JONES, 1973).

The use of chloral hydrate alone or with other drugs as a preanaesthetic, narcotic or anaesthetic for horse, was indicated by many authors (BRANDER and PUGH, 1971; HALL, 1971; LUMB and JONES, 1973; JONES, *et al.*, 1977; GREEN, 1979; TRAIMONGKOLKUL, 1979; WHOELER, *et al.*, 1980; BEUTTLER, 1981; CRISPIN, 1981; DIETZ and WIESNER, 1984; DAHYA, *et al.*, 1985; AKPOKODIE *et al.*, 1986; SCHNEIDER and STIEF, 1987 and SHORT, 1987). The use of chloral hydrate in donkeys was recorded by TANTAWY (1980) and SAMY *et al.* (1986).

The present study was designed for reappraisal of chloral hydrate as a preanaesthetic, narcotic and anaesthetic for

donkeys. The pathological effect on the parenchymatous organs and brain was also discussed.

MATERIALS AND METHODS

The present study was conducted on 24 apparently healthy, 4 to 11 months-old-donkeys. Animals were divided into 4 equal groups; each of 6 animals.

Group I: The animals received 6g/50Kg Bwt. chlotal hydrate.

Group II: The animals received 9g/50Kg Bwt. chloral hydrate.

Group III: The animals received 12g/50Kg Bwt. chloral hydrate.

Group IV: The animals received 15g/50Kg Bwt. chloral hydrate.

Chloral hydrate was prepared as 10% solution and administered intravenously. Heart rate, respiratory rate, and rectal temperature were recorded before and after injection of chloral hydrate and the results were tabulated. Various reflexes (skin reflex, eyelid reflex, coronary hand reflex, conjunctival reflex, corneal reflex anal reflex and tongue withdrawal reflex) were evaluated. The period after which the animals laid down and the duration of narcosis and anaesthesia were also recorded.

Liver biopsy was taken surgically 3 hours after chloral hydrate injection through a midline laparotomy. The line of incision was infiltrated with 2% procaine Hcl solution.

Euthanasia 7 days after drug administration was performed by bleeding after exposure of the common carotid artery under effect of local infiltration analgesia. The postmortem examination was done immediately after euthanasia, and the macroscopical findings were recorded. For the histopathological study, specimens were collected from the liver, kidneys, spleen, heart, adrenals, lung and brain. Paraffin sections were stained with Haematoxylin and Eosin and examined with the light microscope and the results were recorded.

Animals of both groups I and II were divided into two subgroups A&B where the drug was used either as non repeated dose or as repeated doses with 7 days intervals for three times. Animals of group IV were divided into two sub groups (A&B) where the drug was used either with or without atropine premedication. Atropine sulphate 1% solution was used in a dose of 0.05mg/Kg Bwt., intramuscularly, 15 minutes before chloral hydrate injection.

RESULTS

Group I: Animals of This group lied down 2-3 minutes after chloral hydrate (6g/50kg. Bwt.) injection. The skin reflex, eyelid, conjunctival and coronary band reflexes disappeared in some animals 15 minutes after chloral hydrate injection and

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reappeared within 45 minutes. Tongue withdrawal reflex disappeared after 15 minutes and reappeared 60 minutes after drug injection in all animals. The animals stood up 45 to 80 minutes after administration of the drug.

Group II: The animals of this group lied down just after administration of chloral hydrate (9g/50kg Bwt.). The skin reflex, coronary band reflex and eyelid reflex disappeared within 15 minutes and reappeared within 60 to 75 minutes after drug administration. The corneal reflex and anal sphincter contraction only sluggish after 30 minutes and returned completely within 60 to 90 minutes. Within 2 to 3 hours after the drug administration, the animals could stand up.

Group III: The animals of this group lied down during injection of chloral hydrate (12g/50kg Bwt.). The reflexes disappeared within 10 minutes and reappeared within 90 to 120 minutes after injection of chloral hydrate. The animals were able to stand up within 3 to 4 hours after drug administration.

Group IV: The reflexes disappeared within 10 minutes and reappeared within 3 hours after chloral hydrate administration (15g/50kg Bwt.) in 2 animals. The animals began trials to stand up within 4 hours but were able to stand up 6 hours after drug injection. Three animals (two of them were premedicated with atropine) died within 10 minutes after chloral hydrate injection. One animal (premedicated with atropine) died after 105 minutes.

The changes in the body temperature, heart rate and respiratory rate are shown in tables (1&2) and figures (1,2,3,4,5&6).

The postmortem examination revealed only petechial haemorrhages in the spleen of in one animal 7 days after chloral hydrate administration in a dose rate of 9g/50kg Bwt. (Fig. 7). On microscopic examination of the liver of all groups, the pathological alterations were restricted to the centrilobular zones and ranged from parenchymatous degeneration to hydropic and vascular degeneration. The cytoplasm showed coarse acidophilic granules and empty vacuoles (Fig. 8). The hepatic cords were distorted and hepatocytes were swollen (Fig. 9). The heart showed focal degenerative and necrobiotic changes in group III and IV (Fig. 10). The kidneys in one case (group III) showed degenerative and necrobiotic changes (Fig. 11).

The spleen showed haemosidrosis in all group (Fig. 12). The brain and adrenals showed no microscopic detectable changes.

DISCUSSION

When chloral hydrate was used in donkeys, anaesthesia was obtained with a dose rate ranging from 9 to 12g/50kg Bwt. When
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the sub-anaesthetic doses (6g/50kg Bwt.) were used, some of the reflexes remained unaffected, some sluggish and some disappeared for a short time in comparison to the long recumbency time. JONES *et al.* (1977) recorded that this may be attributed to the very weak analgesic action of chloral hydrate. Although a good anaesthesia for 60 to 90 minutes was obtained with the use of chloral hydrate in donkeys, the anaesthetized animals took a long time (3-4 hours) to be able to stand up.

While the body temperature decreased following administration of chloral hydrate for donkeys, the heart was accelerated. The respiratory rate was accelerated, then fluctuated around the original values and lastly decreased.

While it was reported by some authors that chloral hydrate may result in degenerative changes in the liver (HALL, 1971, GREEN, 1979 and AGRAWAL *et al.*, 1983), JONES *et al.* (1977) stated that the drug has little or no detrimental effect upon liver in the absence of hypercapnia and hypoxia. In donkeys the changes in the liver after chloral hydrate administration were reversible.

The constant findings after administration of large doses of chloral hydrate were haemocidrosis in the spleen and focal degenerative changes in the kidneys and heart. These changes may be related to the use of the drug in high doses. On the contrary the petechial haemorrhages which were detected in the spleen in one case may be due to parasitism. The drug seemed to cause no detectable changes in the adrenals or brain.

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Table (1): Showing the mean values of the body temperature, heart rate and respiratory rate after injection of chloral hydrate in donkeys.

Time (minutes)	Group I			Group II			Group III			Group IV		
	T.	HR.	RR.	T.	HR.	RR.	T.	HR.	RR.	T.	HR.	RR.
0	37.2	58	15	36.9	45	18	37.4	46	17	37.9	48	18
15	36.8	54	19	36.7	48	16	37.4	57	17	37.4	44	25
30	36.7	50	21	36.4	48	19	37.2	67	21	37	44	18
45	36.4	49	20	36	54	18	37.1	63	21	36.6	50	21
60	36.3	49	16	35.6	58	17	36.6	61	17	36.5	61	20
75	36.3	50	14	35.4	53	20	36.3	60	17	36	65	22
90	36.1	52	15	35.3	56	17	36	58	18	35.3	68	14
105	36.1	56	14	35.1	51	17	35.8	56	16	35.2	69	20
120	36.2	54	14	*	44	16	35.7	54	15	35	65	22
150	36.7	56	15	*	48	15	35.5	50	15	*	60	18
180	37.2	58	16	*	41	12	*	50	14	*	64	11

T. Body temperature.

HR. Heart rate.

RR. Respiratory rate.

* Lower than 35 degree centigrade.

Table (2): Showing the mean values of the body temperature, heart rate and respiratory rate after injection of chloral hydrate (repeated doses) in dokeys.

Time (minutes)	Group I a			Group I b			Group I c		
	T.	HR.	RR.	T.	HR.	RR.	T.	HR.	RR.
0	37.8	50	18	37.4	46	20	37	42	18
15	37.5	65	24	37.5	69	18	37.2	59	19
30	37.2	63	22	37.1	56	18	36.7	47	16
45	36.9	60	23	36.6	55	19	36.5	43	18
60	36.7	51	19	36.3	54	16	36.4	40	16
75	36.8	54	21	36.2	51	14	36.3	41	15
90	37.3	54	19	36.1	47	14	36	40	16
105	37.5	53	18	36.2	44	14	35.9	40	15
120	37.8	54	17	36.3	45	15	36	41	16
150	38.6	60	16	36.8	43	15	36.1	42	17

T. Body temperature.

HR. Heart rate.

RR. Respiratory rate.

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Fig. (1) Effect of Chloral hydrate on body temperature

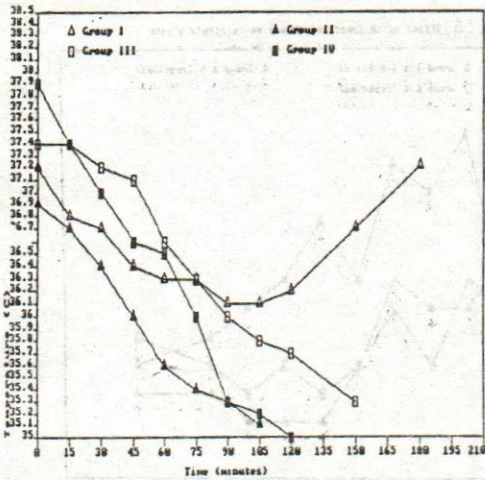


Fig. (2) Effect of Chloral hydrate on the heart rate

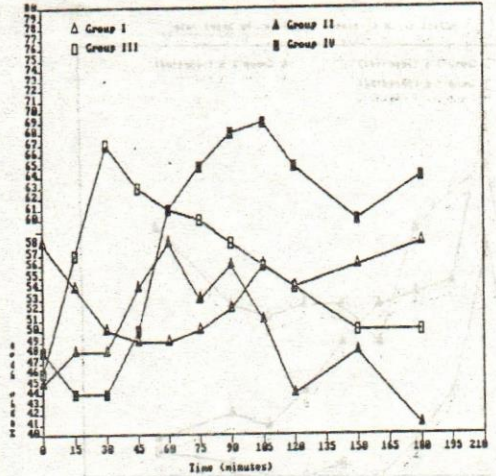


Fig. (3) Effect of Chloral hydrate on the respiratory rate

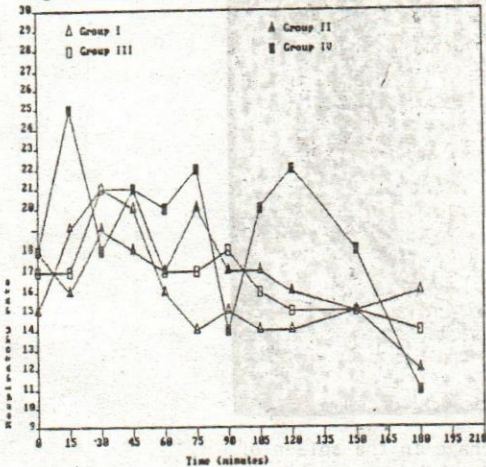


Fig. (4) Effect of CH (repeated doses) on body temperature

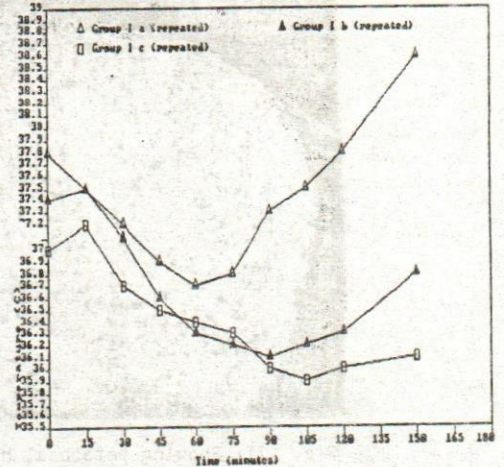


Fig. (5) Effect of Cl (repeated doses) on the heart rate

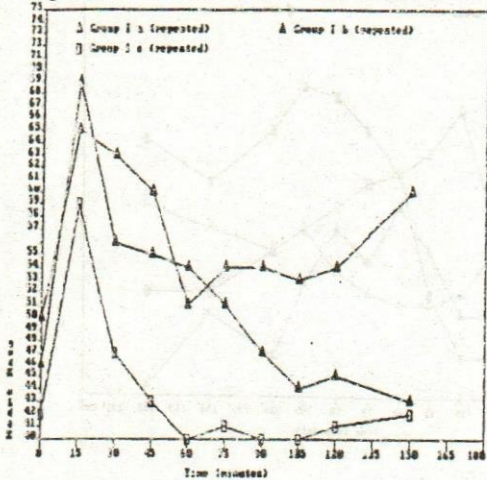


Fig. (6) Effect of Cl (repeated doses) on respiratory rate

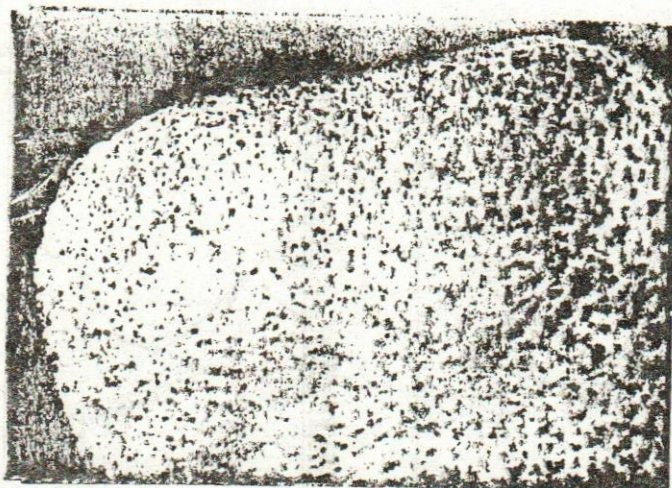
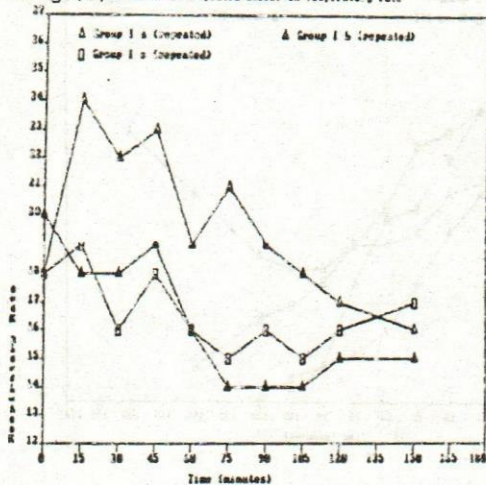


Fig. (7): Showing petechial haemorrhage in the spleen of a donkey, 7 days after chloral hydrate administration in a dose rate of 0g/50kg body weight.

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Fig. (8): Centrilobular parenchymatous degeneration and vacuolation in the liver, H&E (X 400).

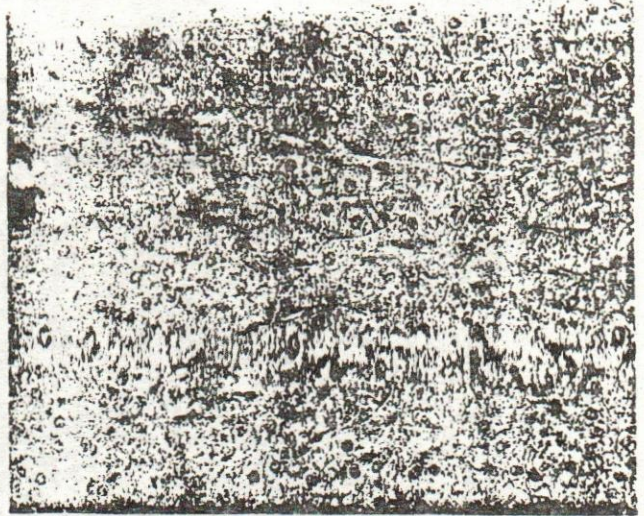


Fig. (9): The liver showing distortion of the hepatic cords and swelling of the hepatocytes, H&E (X 400).

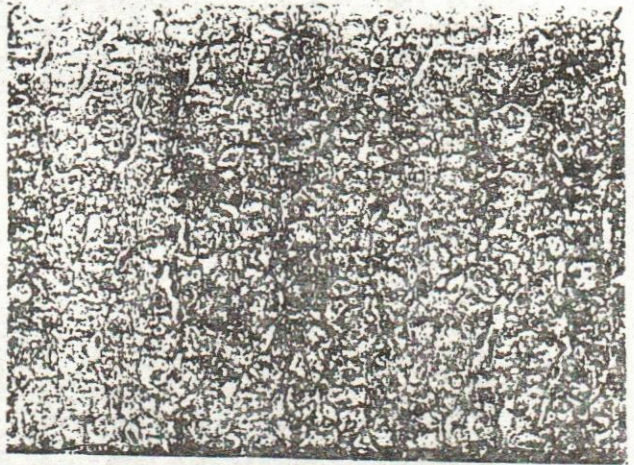
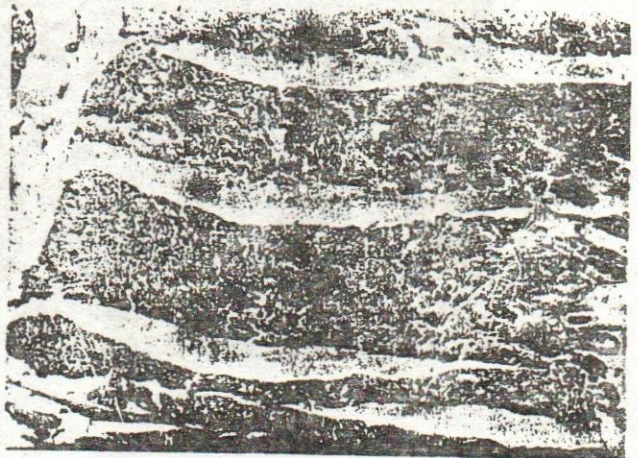


Fig. (10): Showing myocardial degenerative changes, H&E (X 400).



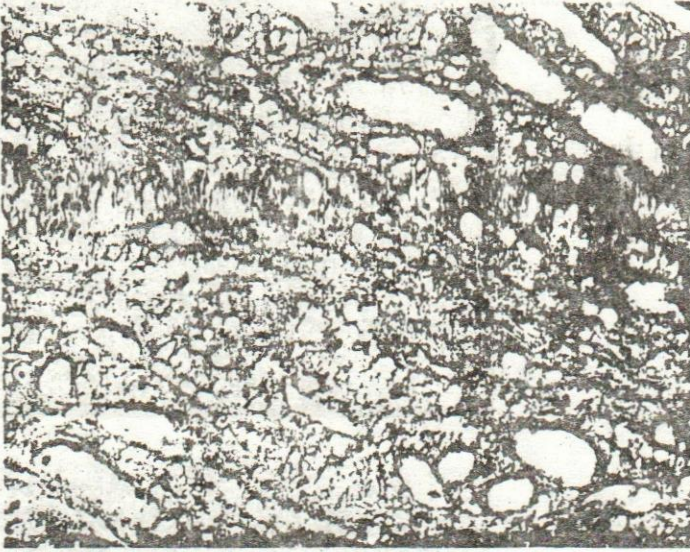


Fig. (11): Tubulonephrosis in the kidney, H&E (X 400).

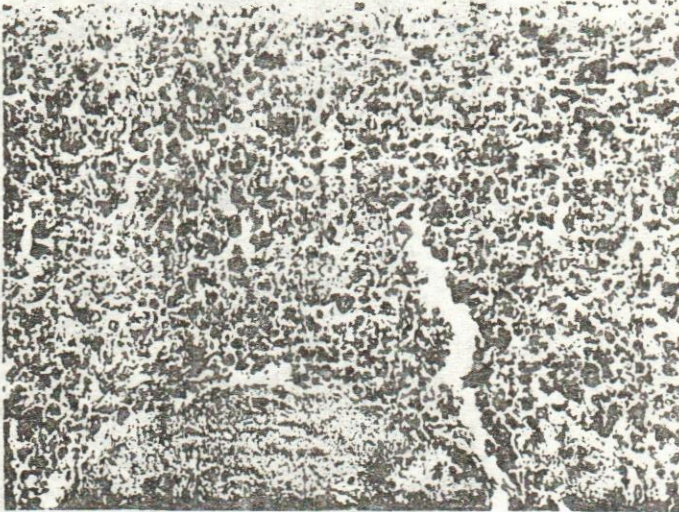


Fig. (12): Haemosiderosis in the spleen, H&E (X 400).