Kerosene Poisoning in Primates

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SUMMARY

Normal lung weight/body weight ratio's were established for vervet monkeys. Three groups of 5 animals were each utilized experimentally. In order to ascertain whether absorption and excretion through the lungs is the underlying physiological process which results in the pulmonary pathology following kerosene ingestion, 2 groups were tracheostomized (groups II and III) and 1 sham operated (group I). Kerosene 45 ml/kg body weight was administered via a nasogastric tube to groups I and II, and 6 - 8 hours later all live animals were sacrificed. Lung weight/body weight ratio and lung wet weight/dry weight ratio's were calculated for each group and the means compared with those of the control group. The differences are statistically significant. Group III had kerosene injected either intravenously or endotracheally. Handled in a similar manner after sacrifice, the means of their lung weight/body weight ratio and lung wet weight/dry weight ratio are statistically heavier than those of the control group and come statistically from the same population as group I. Macroscopic and microscopic investigations confirm the above trend.

All results indicate that the pulmonary effects following kerosene ingestion are not due to absorption and excretion through the lungs, but due to aspiration into the tracheobronchial tree alone.


Experimental Groups

Three groups of 5 monkeys each were utilized. Each animal was anaesthetized, using 10 - 20 mg of phencyclidene intramuscularly. They were then weighed and a femoral artery catheter was inserted. The following procedures were then carried out:

Group I: Monkeys weighing 1 850 - 2 700 g (mean 2 264 gm) received a sham tracheostomy.

Group II: Monkeys weighing 1 640 - 2 750 g (mean 2 202 g) received a tracheostomy and had a plastic endotracheal tube inserted, with the proximal end tied off securely.

Group III: Monkeys weighing 1 900 - 2 550 g (mean 2 162 g) were operated on as for group II.

The animals in groups I and II then received 45 ml of kerosene/kg body weight via a nasogastric tube. After 6 - 8 hours all live animals were sacrificed, using an intravenous barbiturate. The lungs were examined macroscopically, weighed before and after sections of relevant areas had been removed for microscopy, and then dried as for the control group. Lung weight/body weight ratios and lung wet weight/dry weight ratios were then calculated as for the control group.

The animals in group III, had either 1,0 ml kerosene injected intravenously or 0,2 ml kerosene in 5 ml normal saline injected endotracheally. After 6 - 8 hours the group was then handled as above.

RESULTS

Macroscopic and microscopic examination of the lungs in groups I and III showed heavy oedematous lungs with patchy haemorrhagic areas. The lungs from group II could not be distinguished, either macroscopically or microscopically, from those of the control group.

Table I gives the lung weight/body weight and lung wet weight/dry weight ratios for the control group. Tables II, III and IV give the above ratios for the animals in groups I, II and III.

Table V compares the means of lung weight/body weight ratios and lung wet weight/dry weight ratios for each group against those of the control group, and for group I against group III. It can be seen that the established means for groups I and III were significantly larger than those of the control group and came from the same statistical population, whereas group II did not differ statistically from the control group.

MATERIALS AND METHOD

Control Group

Twenty-two normal healthy vervet monkeys, weight 1 812 - 5 436 g (mean 3 243 g) were used as controls. After being weighed, they were sacrificed and the wet weight of their lungs was measured. The lungs were then dried slowly in an oven at 85°C and weighed daily until the dry weights were stable. Lung weight/body weight ratios and lung wet weight/dry weight ratios were then established for the group.

Lung weight/body weight ratios and lung wet weight/dry weight ratios are relatively fixed for any particular animal measured. The first ratio documents increase in lung weight from any cause; the second elucidates the nature of the increase. An increase in both suggests an increase in fluid content of the organ.

The characteristic pulmonary lesion following kerosene poisoning is an acute fulminating, exudative, haemorrhagic bronchopneumonia. This can be objectively demonstrated by microscopy but more precisely delineated by the above ratio's.

Although some authors' claim to have evidence for the excretion of kerosene through the lungs following ingestion, only the work of Ashkenazi and Berman remains valid under close examination. In their group of rats, labelled heneicane was demonstrated in liver, lungs, brain and kidneys 8 - 24 hours following injection of the heneicane into the duodenum. Gerarde and Richardson and Pratt, however, could show no evidence of pulmonary pathology following the intragastric instillation of kerosene into rabbits, chickens, rats and dogs. Rabbits in particular are of interest, as they cannot vomit and thus preclude aspiration as a cause of pulmonary pathology. Lesser et al., in a study using these animals, also failed to demonstrate any lung lesion following gastric instillation of kerosene.

Children who develop pulmonary complications following kerosene ingestion generally do so with remarkable rapidity, radiological evidence of lung problems being present within 15 - 45 minutes. The younger the child the more frequent the pulmonary complications, which appear to be dependent to some extent on the presence of spontaneous or induced vomiting. Furthermore, a higher rate of CNS involvement has been noted in those patients who have vomited. The association of vomiting, and pulmonary and CNS signs previously reported, strongly suggests that aspiration is the prime culprit in the production of the pulmonary lesions following kerosene ingestion, with the CNS signs being secondary to the concomitant anoxia that occurs. The study reported here supports this contention.

The amount of kerosene instilled into the stomachs of the monkeys used in this study was far greater than would normally be imbibed by children. In the group of animals (group II) where no possibility of aspiration could exist, no lung lesions were seen, and statistically they could not be distinguished from the normal controls. If absorption and excretion of kerosene through the lungs had been the underlying mechanism in the production of the pul-

**DISCUSSION**

Lung weight/body weight ratios and lung wet weight/dry weight ratios are relatively fixed for any particular animal measured. The first ratio documents increase in lung weight from any cause; the second elucidates the nature of the increase. An increase in both suggests an increase in fluid content of the organ.

The characteristic pulmonary lesion following kerosene poisoning is an acute fulminating, exudative, haemorrhagic bronchopneumonia. This can be objectively demonstrated by microscopy but more precisely delineated by the above ratio's.
monary changes, these animals would perforce have had to be abnormal. In group I, 4 out of 5 animals had lung lesions. Neither all animals nor all children aspirate when kerosene is in their stomachs. In general, however, this group could not be separated from the group of animals where haemorrhagic pneumonia had been produced by endotracheal or intravenous injection of the kerosene (group III).

Finally, the lungs of groups I and III were different from the control group and those of group II, where aspiration had been prevented by the presence of a tracheostomy.

If one can extrapolate from primate studies to man, then this study strongly suggests that excretion of kerosene through the lungs, following ingestion, does not occur, and that the prime problem associated with kerosene poisoning is an aspiration pneumonia.

REFERENCES

History of Medicine

Thomas Hodgkin - The Man and His Times *

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SUMMARY

Thomas Hodgkin was an enlightened Morbid Anatomist. Although he was foremost among those who advocated the correlation of bedside clinical features with the post-mortem findings, his early description of lymphadenomatosis would have remained unknown but for the unsselfish acknowledgement of his colleague Samuel Wilks. He was a self-effacing man with strong Quaker religious beliefs, and after failing to be appointed to a clinical post at Guy's Hospital he gradually withdrew from medicine and turned his attentions to the welfare of the poor, and to the social difficulties of civilization in the mid-nineteenth century. He was particularly interested in the underprivileged, including the Jewish and African peoples. This is a short account of his life and achievements, set against the social climate of his generation.


Thomas Hodgkin was Morbid Anatomist at Guy's Hospital for many years. This hospital has been the alma mater to many South Africans, who have in their turn been in a majority in the hospital's victorious rugby football teams. The fact that few South Africans now graduate overseas is due to our improved medical schools rather than a reflection on our times. The following account of the life of Thomas Hodgkin, however, is a reflection of the times in which he lived.

Thomas Hodgkin was born of middle-class Quaker parents in North London on 17 August 1798. He died from dysentery in Jaffa, which adjoins the new city of Tel Aviv, on 5 April 1866. He and his younger brother, John, who later became a successful lawyer, were educated by their father, who practised as a private tutor. Hodgkin became a student at Guy's Hospital in 1819. The fee was 21 guineas, and this entitled him to perpetual studentship as a Physician's Pupil. He completed his medical training by studying under Laennec in Paris. In 1823 he graduated as a Doctor of Medicine at Edinburgh and in 1825 he received the licentiateship of the Royal College of Physicians.

During this time his interest and skill in morbid anatomy attracted the attention of Astley Cooper and he was appointed as Demonstrator in Morbid Anatomy and Curator of the Museum to Guy's Hospital. He held these posts for 12 years, during which time he also practised as a physician, but it was because of his failure to be * Paper presented at the Congress of the Radiological Association of South Africa (M.A.S.A.), held in Bellville, CP, on 4 - 6 September 1971.