Onderstepoort Journal of Veterinary Science and Animal Industry, Volume 13, *Number* 2, *October,* 1939.

Printed in the Union of South Africa by the Government Printer, Pretoria.

Alkali Poisoning.

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I. Weak Alkalis (Sodium Bicarbonate and Sodium Carbonate).

Kobert (1906) states that sodium bicarbonate raises the alkalinity of the tissue fluids and enhances the oxidation processes in the body. Ingestion of excessive quantities of sodium bicarbonate causes dilatation of the stomach, anorexia and anaemia.

Fröhner (1919) reports that a cow which had received 800-900 gm. of sodium carbonate aborted, groaned and bellowed, and had to be slaughtered, and that 10-15 gm. of potassium carbonate is fatal for the dog (heart failure and collapse). Dogs fed daily with 15 gm. of sodium bicarbonate for weeks showed vomiting, diarrhoea and loss in condition (Fröhner, 1919). He also states that two horses which ate sodium carbonate from bags took ill with severe colic, profuse diarrhoea, continuous coma, stomatitis and swelling of the lips.

Reid (1921) describes sodium carbonate poisoning in sheep due to the drinking of effluents from dairy factories. The symptoms were staggering gait and hurried respirations. The animals soon lay down and died. The following were the post mortem appearances: Hyperaemia of the subcutaneous tissues, imperfect clotting of the blood, hyperaemia of the small intestines with subserous haemorrhages, slight congestion of the liver, in very acute cases the abomasum was filled with dark, blood-stained, fluid contents, intense hyperaemia of abomasal mucosa with haemorrhages.

Heller and Larwood (1930) conducted experiments upon rats and other small animals with single salts and also mixtures of calcium chloride, calcium sulphate, sodium bicarbonate, and magnesium sulphate. Drinking water containing 20,000 parts of sodium carbonate per million parts (i.e. a 2 per cent. aqueous solution) was found to be decidedly deleterious and reproduction was interfered with at quite low levels. Sodium bicarbonate proved to be less injurious. In subsequent experiments upon rats Heller (1932 and 1933) established that drinking water containing 1 per cent. (10,000 p.p.m.) of sodium carbonate resulted in unsatisfactory growth of the offspring, while a 1·9 per cent. solution caused a rough coat, red eyes, and diarrhoea in mature rats and a very high

mortality in their young. A 1·5 per cent. solution of sodium bicarbonate caused undersized adults and impeded growth in their offspring, while there were unsatisfactory growth and appearance with 2 per cent. solutions. Experimenting with 0·5 and 1·0 per cent. aqueous solutions of caustic soda (NaOH) he found practically normal growth with the former solution, the latter causing retarded growth, "dirty animals", marked nervousness, sore eyes and diarrhoea.

Linton and Wilson (1933) refer to sodium bicarbonate poisoning in pigs due to the feeding of "Flour sweepings" containing 33·7 per cent. of this alkali. The symptoms seen were excessive thirst, animals appeared to be in pain, stood with their heads lowered and oblivious to their surroundings and staggered on their front legs. Only one pig died after the sweepings were removed and milk given. Autopsy revealed inflammation of the stomach. Linton furthermore describes alkali poisoning in pigs through accidental discharging into their milk of the alkali used for cleansing the milk utensils.

Some time ago the author received for analysis a certain proprietary "scouring powder", which was being sold as a cleansing agent for *kitchen utensils*. Pigs had died after their troughs had been scoured with it. Four grams of this strongly alkaline powder caused death in rabbits within a few hours. This is one of the many cases in which a virulent poison is labelled non-poisonous.

Witter (1936) added sodium bicarbonate to the drinking water of chicks and found that: "(1) Sodium bicarbonate given in the drinking water in the usual dosage (¼ lb. to 5 gallons or 0·6 per cent. solution) caused chicks to drink more water than normal and produce moist droppings. Chicks two weeks old developed pale and swollen kidneys from this dosage, but chicks three weeks old and older were not noticeably injured. (2) A double dose (1·2 per cent. solution) of soda caused chicks to drink more water than those fed the 0·6 per cent. solution and produced watery droppings. Chicks two to eight weeks old were seriously injured by this dosage within one to three days and deaths occurred within this time. (3) Two and four-tenths per cent. solution of soda reduced water consumption below normal for chicks under four weeks of age. The injurious effects of this dosage were noted within a day and deaths occurred within three days. (4) Mature cockerels were injured with a 2·4 per cent. solution of soda, but were not affected by a 1·2 per cent. solution. It was apparent throughout the project that the younger the chicks the more susceptible they were to soda injury. (5) Kidneys from chicks affected by feeding soda became pale, swollen and engorged with urates. The kidney tubules showed degenerative and exudative changes indicating severe injury. (6) Chicks affected by feeding soda showed an increase in kidney weight, and an increase of approximately four times in uric acid per gram of kidney and in uric acid in the blood". He found the injury caused by feeding sodium bicarbonate to chicks similar to the pathology and blood chemistry changes present in visceral gout. Two weeks old chicks consumed from 45 to 113 c.c. of "normal water" daily.

Carbonates, bicarbonates and caustic soda tend to cause alkalinity of the gastrointestinal juices and of the blood and tissue fluids (change in pH), consequently the continued drinking of alkaline waters and the constant taking of alkaline waters and the constant taking of alkaline powders will lead to serious digestive and other disturbances. Carbonates are more harmful than bicarbonates.

Cope (1936) draws attention to the fact that the constant use of alkaline stomach powders by individuals suffering from chronic gastric hyperacidity may have serious consequences. These powders generally contain calcium carbonate, magnesium carbonate, bismuth oxycarbonate, and sodium bicarbonate. He states that the following symptoms may arise in the course of time when alkaline powders are taken over long periods: Loss of appetite; slight vomiting; irritability; thoughtlessness; unreasonableness; depression; vague headaches and muscle pains; red and inflamed conjunctiva (sore eyes); " vision may be slightly blurred, but is as a rule not interfered with "; in some cases the patient himself complains about increasing impairment of mental efficiency; the skeletal muscles are sometimes abnormally excitable to direct percussion; polyuria is usual, but sometimes the urine volume is reduced below normal; the urine is mostly alkaline and always contains albumin; finally there may be coma. " The rapidity with which these successive stages are passed through varies considerably "—from a few days to weeks. Cope states that " very characteristic of all conditions of this type in which the body contains excess alkali is the extremely low concentration of chloride in the urine. This may be very simply demonstrated by the addition of nitric acid and a small quantity of silver nitrate solution." According to Cope the following points are of importance in the diagnosis of alkali poisoning: (1) The symptoms. (2) The presence of albumen in the urine. (3) Low concentration of chloride in the urine. (4) " An alkaline reaction of the urine with effervescence on the addition of acid is of no diagnostic value as it is frequently found in persons taking considerable quantities of alkali in the complete absence of all toxic symptoms." (5) Blood analysis: (*a*) " Estimation of plasma carbonate or alkali reserve is the most certain of interpretation "—a rise above 80 volumes per cent. is practically diagnostic of alkali poisoning. (*b*) " It would seem to be a safe general rule, however, to regard all suspected cases showing a rise in blood urea of about 80 mg. per cent. as cases of alkali poisoning, unless an independent cause for the high value can be definitely found." As treatment he recommends: (1) Removal of the alkaline powders. Alkali sensitive individuals should take di-sodium phosphate or perhaps hydrated magnesium silicate. (2) Administration of sodium chloride *per rectum* facilitates the excretion of bicarbonate. (3) Ammonium chloride can be given as an acidifying agent, but it must be used with care as in all cases of alkali poisoning the kidneys are severely damaged and acidosis may result. It is essential that the urine be examined at frequent intervals.

In experiments upon themselves Joos and Mecke (1934) found that after the administration of sodium bicarbonate there is (1) a marked increase in the sodium content of the blood serum, (2) a

slow fall in the magnesium content, (3) a pronounced and sudden fall in the calcium content, (4) a quick fall in the chloride content, and (5) a slow fall in the phosphorus content of the blood serum.

Little (1937) describes a simple method of estimating the alkaline constituents of washing powders and washing solutions containing mixed alkalies.

The taking of too much alkaline stomach powders has resulted in a serious state of alkalosis (Davies, 1939).

II. Caustic Alkalis (Caustic Soda and Caustic Potash).

A. *Onderstepoort Experiments.*

Some time ago a specimen consisting of a fairly dark-brown, semi-coagulated, jelly-like fluid was submitted by the Government Veterinary Officer, Greytown, Natal, to Onderstepoort for analysis. It was stated that it was a specimen of milk which had turned this colour when placed on the stove to boil. The specimen was submitted in an air-tight fruit jar and was about eight days old when examined. It emitted a very pronounced alkaline odour, was slimy and jellylike, and yielded a marked alkaline reaction with litmus paper. It was impossible to ascertain the degree of alkalinity by means of titration as the filtrate was dark-brown in colour and could not be decolorised with charcoal. It was then decided to determine the sodium content of the specimen. This was found to be far in excess of the sodium content of normal milk.

The following rabbits were drenched by means of a stomach tube with the above specimen:—

Rabbit A (2·0 *Kg.*): 100 *c.c. on* 7/7/38.

Within five minutes after drenching pronounced laboured breathing set in, the animal breathing through its mouth. General weakness (paresis) followed soon and the animal was unable to sit up or hold its head up. Muscular twitchings were seen on different parts of the body. The heart-action was very much accelerated and weak in the beginning and became progressively weaker until it was imperceptible a short while before death. The animal died within ten minutes after the development of symptoms. A few minutes before death occurred the animal became completely paralysed, was comatose and lay prostrate with the head drawn backwards. It died with terrific convulsions (jumping about in the cage) of the whole body. These were probably due to asphyxia.

Post mortem appearances: General cyanosis; pronounced hyperaemia of the lungs, liver and kidneys; pronounced dilatation of both heart ventricles; pronounced hyperaemia and sliminess of the gastric mucosa; contents of small intestine very slimy.

Rabbit B (2·0 *Kg.*): 50 *c.c. on* 7/7/38.

Symptoms very similar to those described above set in within ten minutes after drenching. The only difference in the

symptomatology was that there were no muscular twitchings and that the animal died quietly in a state of paralysis and coma. Death occurred approximately twenty minutes after drenching.

Post mortem appearances: Very similar to those described in rabbit A with haemorrhages in the gastric mucosa.

The following three experiments (specimens 1, 2 and 3) were then conducted:—

Specimen (1):

Four grams of granular caustic soda were added to 200 c.c. of fresh cow's milk. A light yellow colour appeared almost immediately. It soon became more intensely yellow and then developed a brownish colour as the temperature of the milk was raised by means of a gas-flame. As the temperature of the milk rose it became dark yellow-brown and when it was boiling it turned a dark reddish-brown colour. Between 50° and 70° the milk jellified and became fluid again before it boiled. It emitted a pronounced alkaline odour.

Specimen (2):

Twenty grams of granular caustic soda were added to 200 c.c. of fresh cow's milk and boiled for two minutes. The milk showed the same changes as those described under (1); the changes in colour took place more rapidly and the colours were more intense. The cooled specimen was then placed in an air-tight fruit jar and was drenched to two rabbits seven weeks later.

Rabbit A (1·9 *Kg.*): 50 *c.c. on* 14/9/38.

The animal developed dyspnoea and apathy within a few minutes after drenching. After having shown loss of appetite for a day it appeared normal again.

Rabbit B (1·95 *Kg.*): 100 *c.c. on* 14/9/38.

Symptoms more pronounced than those described in Rabbit A set in within five minutes after drenching. The animal appeared normal after twenty-four hours.

Specimen (3):

Forty grams of caustic soda were added to 200 c.c. of fresh cow's milk and boiled for two minutes. The results of the addition of caustic soda and boiling were similar but more pronounced than those described under (2). The appearance, colour and odour of this specimen of milk were identical with those of the specimen submitted by the Government Veterinary Officer, Greytown. Two rabbits were drenched with specimen (3) after it had been kept for seven weeks in an air-tight fruit jar.

Rabbit C (1·25 *Kg.*): 50 *c.c. on* 14/9/38.

The only symptoms the animal showed were transitory laboured respiration and loss of appetite.

Rabbit D (1·65 *Kg.*): 100 *c.c. on* 14/9/38.

Laboured respiration set in within a few minutes after drenching. There were violent intestinal movements within thirty minutes. After having shown pronounced apathy, loss of appetite, accelerated heart-action and after having passed dark brown urine for two days the animal appeared normal again.

The slight toxicity of specimens (2) and (3) was thought to be due to the fact that they had stood too long (seven weeks) before being drenched. A further three specimens (4, 5 and 6) of milk were then treated with caustic soda, boiled for two minutes and left standing for eight days in air-tight fruit jars. The changes in the milk were the same as those noted before. On cooling the specimens jellified.

Specimen (4):

Twenty grams of granular caustic soda were added to 200 c.c. of fresh cow's milk. After standing for eight days the specimen was dark brown in colour and had a jellylike appearance and was slimy. It emitted a pronounced alkaline odour and smelt very strongly of ammonia. It was drenched to two rabbits:—

Rabbit E (0·9 *Kg.*): 50 *gm. in a small quantity of water on* 23/9/38.

The animal retched and vomited within a few minutes after drenching, and died within a further two minutes.

Post mortem appearances: Most pronounced hyperaemia of gastric mucosa with partial dissolution (slimy appearance of the mucosa; similar changes in the small intestine; pronounced congestion of the liver; a fair quantity of vomitus in the lungs.

Rabbit F (1·7 *Kg.*): 100 *c.c. with a small quantity of water on* 23/9/38.

Symptoms identical with those described in Rabbit A, which was drenched with the specimen submitted by the Government Veterinary Officer, Greytown, developed within three minutes after drenching. After a further ten minutes the animal retched, vomited and died soon afterwards.

Post mortem appearances: Gastric wall dark reddish-brown in colour and almost completely dissolved (very slimy); mucosa of small intestine dark reddish-brown and slimy (partial dissolution); pronounced hyperaemia of the liver; no vomitus in lungs.

Specimen (5):

Forty grams of caustic soda were added to 200 c.c. of fresh cow's milk. After eight days appearance and odour of this specimen were the same as those of specimen (4). The colour was dark yellowish-brown. The following rabbits were drenched with it:—

Rabbit G (1·25 *Kg.*): 50 *c.c. with a small quantity of water on* 23/9/38.

The symptoms resembled those described in Rabbit F. The animal vomited and died within ten minutes after dosage.

Post mortem appearances: As in Rabbit F, only more pronounced. No vomitus in lungs.

Rabbit H. (1·65 *Kg.*): 100 *c.c. with a small quantity of water on* 23/9/38.

Symptoms as described in Rabbit F appeared within a few minutes and death occurred within ten minutes after drenching. *Post mortem appearances:* As in Rabbit G. The gastric wall was almost completely dissolved and showed a rupture.

Specimen 6:

Sixty grams of caustic soda were added to 200 c.c. of fresh cow's milk. After eight days the colour, appearance and odour (alkaline and ammonia) were identical with those described under specimen (5).

This specimen was not dosed to rabbits as specimens (4) and (5) had yielded positive results.

In considering the toxicity of milk to which caustic alkalis were added it should be realised that there are two different kinds of poisons present, namely, the caustic alkali and decomposition products caused by the action of the alkali on some of the constituents of the milk, especially the protein. That the alkali causes pronounced destruction of the protein in the milk is evident from the pungent ammonia odour which is emitted after a few days. Toxic amines are probably also formed. It appears that the toxicity of caustic alkali milk decreases in the course of time. The ammonia odour which was not pungent after eight days decreased and eventually disappeared.

On an earlier occasion three groups of four rabbits each were drenched with 1·0 (Group I), 2·0 (Group II) and 5 (Group III) per cent. aqueous solutions of caustic soda respectively. The two rabbits (Group I) which received 40 c.c. of a 1·0 per cent. solution developed no symptoms beyond transient loss of appetite. Those two animals of Group I which were drenched with 80 c.c. died within forty hours. In Group II two rabbits received 40 c.c. and the remaining two 80 c.c. of a 2·0 per cent. solution of caustic soda. They died from within twenty to thirty hours after drenching. The two rabbits in Group III which received 40 c.c. of a 5·0 per cent. solution of caustic soda developed symptoms within a few minutes.

One of these rabbits vomited and died within one-and-a-half hours after drenching, whilst the other one lived for approximately thirty hours. The remaining two rabbits of Group III which received 80 c.c. of a 5·0 per cent. caustic soda solution died within three hours.

The symptoms and post mortem appearances closely resembled those described below under II (B). Even with 80 c.c. of a 1·0 per cent. solution of caustic soda there was pronounced dissolution (corrosion) of the gastric mucosa and wall. In one of these cases the gastric wall was almost perforated in two places. The remaining portion of the internal surface of the stomach wall was very slimy and intensely dark (dirty) reddish-brown (alkali haematin). With higher concentrations of caustic soda the lesions were much more pronounced.

B. *Mode of Action, Toxicity and Symptoms of Caustic Alkalis.*

Caustic alkalis dissolve the epithelium and form soluble alkali albuminates when coming into contact with proteins (skin and mucous membranes). The mucous membrane and skin becomes oedematous, transparent, and soapy, and the corroded area becomes necrotic, soft and slimy. If blood vessels have been corroded by the alkali the affected surface may bleed and alkali haematin is formed. In such cases the colour of the affected area may be reddish-brown, black brown (rare) or often bright red (excessive haemorrhage). In the course of time the colour changes to greyish-white—in dilute solutions within 1-2 hours and in concentrated solutions after longer periods.

Acute alkali poisoning facilitates the deposition of calcium in the bone system, hence the blood and tissues are deficient in this element (Rentz, 1933; Renvis, 1935).

Histologic changes in the parenchymatous organs in cases of alkali poisoning have been described by Jankovich, Incze and Fazekas (Fazekas, 1937), but unfortunately the publication is not obtainable in South Africa. Fazekas (1937) drenched different groups of rabbits with 1, 2, 3, 5 and 10 per cent. solutions of caustic soda, the animals receiving 0·5, 0·75, 1·25, 1·5, 2·25 and 2·5 gm. NaOH after one day starvation. The following changes were noted in the blood chemistry of the animals:—(1) A disturbance of the glucose metabolism in the form of hyperglycaemia. (2) A pronounced increase in the inorganic phosphorus content of the total blood. (3) A decrease in the serum calcium. (4) A moderate decrease in serum chlorine. (5) A slight increase in serum sodium. (6) Definite acidosis in the form of a decrease in serum alkali reserve.

The degree of corrosion caused by caustic alkali depends upon: (1) Their concentration. (2) The quantity swallowed. (3) Length of period of action. (4) Quantity of food present in the stomach and intestine. (5) The resistance of the tissues concerned. Solutions of caustic alkalis under 1·0 per cent. do not as a rule cause fatal corrosion but mostly only inflammation of the mucous membranes. More concentrated solutions and especially solid pieces of alkali

frequently cause severe corrosion and even perforation of mucous membranes and of the stomach wall. When solutions of caustic alkali are thrown in the face it may be aspirated and fatal pneumonia may result.

Balasz (1934) states that 2,134 cases of alkali poisoning were treated in the Budapest hospital from 1924-1933. Most of these cases were suicidal. He gives a detailed description of the symptoms in alkali poisoning.

The following symptoms are seen in cases where solutions of caustic alkalis of 10 per cent. and higher concentrations are swallowed: —The lips are swollen in almost all cases of alkali poisoning. This is differential-diagnostically of great value in comparison with acid poisoning. Very severe pain in all the organs with which the solution comes into contact. Pain is especially experienced in the sternal region and in the back due to oesophagitis and perioesophagitis. On the first day there are stomach pains in the cardiac and epigastric regions. There are hiccoughs and vomiting. The vomitus may be mucoid, slippery and reddish-brown or almost black with alkali haematin or it may be bright red when excessve bleeding occurs. When necrotic material is present it has a very bad odour. There is difficulty in swallowing which may be due to pain, spasms, an acute, transitory, reflex paralysis, or paralysis due to direct alkali action on the soft palate or oesophageal musculature, swelling of the oesophageal mucous membrane, mechanical blocking of the oesophagus by necrotic mucous membranes, food, or due to fibrosis of the muscles of deglutition. The respiration is accelerated and in bad cases the pulse is slow and small (shock) in the beginning, but eventually it becomes accelerated in most cases. Prognostically a fast pulse is unfavourable as it indicates complications which are described below. The blood pressure is in most cases low. The reflexes are generally increased. The pupils are dilated in bad cases. Patients are mostly quite conscious and convulsions occur very rarely. The skin is mostly pale but in laryngeal lesions it is cyanotic. In the course of time the face turns red when fever appears as a result of complications. In very serious cases death occurs in a few hours with a subnormal temperature. Diarrhoea and bloody faeces are rare and only occurs when there is severe corrosion of the duodenum and jejunum. The urine need not necessarily be alkaline and is sometimes acid in serious fatal cases. An alkaline reaction of the urine often sets in a few hours after poisoning but is transitory. There is protein in the urine in serious cases and often also acetone due to inanition or to decomposition of protein.

The direct causes of death in alkali poisoning may be shock, collapse, glottis oedema, pneumonia, exhaustion, perforation, haemorrhage, mediastinitis, pericarditis and (or) peritonitis without perforation. The maximum number of fatalities fall within the first three days and on the eighth to tenth days according to Balász (1934). The shortest period within which death occurred from alkali poisoning was two hours.

The following complications may follow the swallowing of caustic alkalis:—(1) Stenosis of the oesophagus, pylorus and small intestine. (2) Leschke (1932) states that cancer of the stomach may develop in some cases. (3) Inflammatory and purulent phlegmones of the oesophagus and the stomach which may pass over on to the adjoining organs resulting in pleuritis, pericarditis and peritonitis. (4) Extensive internal haemorrhage due to corrosion of large blood vessels. This is rare in comparison with extensive bleeding in cases of parenchymatous haemorrhage. (5) Abortion is rare. (6) Perforation of the oesophagus, stomach wall and (or) intestinal wall resulting in phlegmones, perioesophagitis, pneumonia, pericarditis, pleuritis and peritonitis. (7) Pronounced glottis oedema necessitating tracheotomy. (8) Toxic effects on the heart due to absorption of decomposition products from necrotic tissues. (9) Shock and collapse. (Fröhner, 1919; Lander, 1926; Lewin, 1929; Van Itallie and Bylsma, 1930; Glaister, 1931; Leschke, 1932 and 1934; Rentz, 1933; Fazekas, 1934 and 1937; Balasz, 1934; Schranz, 1934; Smith and Cook, 1934; Renvis, 1935.)

The following table is compiled from information supplied in Balász's (1934) publication and is of great value in differentiating between acid and alkali poisoning:—

Acids.	*Caustic Alkalis.*
1. Coagulate protein of tissues and partly prevent further corrosion.	1. Dissolve tissue protein and enhances further corrosion.
2. Crust is hard.	2. Lesion is soft, transparent and slimy (moist). If corrosion is severe the area may be reddish-brown or bright-red.
3. Very little or no oedema.	3. Pronounced, jellylike oedema.
4. Lips rarely swollen.	4. Lips almost always swollen.
	5. Infections are more frequent in alkali poisoning as the lesions are more favourable for bacterial growth. Hence perioesophagitis and mediastinitis with perforation are more frequent in alkali than in acid poisoning.

The symptoms of alkali poisoning in animals are similar to those described in human beings.

C. *Post Mortem Appearances.*

These depend upon the concentration and quantity of alkali swallowed and are evident from the description of the symptoms.

D. *Detection of Caustic Soda.*

Reaction of specimen is strongly alkaline. Vomit is the best specimen for analysis, and also stomach contents in cases where no chemical antidotes were administered. The specimen, if fluid, should be concentrated by evaporation and then extracted with absolute alcohol and filtered. The filtrate is then evaporated to dryness and tested for the alkali suspected. The dialysis method could also be used. In protracted cases it is difficult and often impossible to detect the alkali which caused the poisoning.

E. *Treatment of Alkali Poisoning.*

Tracheotomy should be performed in cases of glottis oedema. In less severe cases stomach lavage could be executed with small quantities (200 c.c.) of weak acid solutions (diluted vinegar, acetic acid). Milk and eggs should be administered and Belladonna (atropine) to inhibit gastric and intestinal secretion and motility. It is of importance to treat the weak heart. Morphine injections should be given to allay pain, excitement, incessant vomiting and sleeplessness. Infusions are essential in order to replace the loss of fluid. Serious cases of stenosis of the oesophagus, pylorus and small intestine should be operated upon.

III. Summary.

1. Poisoning with weak and caustic alkalis is reviewed and discussed.

2. Analysis of a specimen of milk maliciously poisoned with caustic soda is described.

3. An account is given of (*a*) experiments in which varying quantities of caustic soda were added to fresh cow's milk and also of experiments conducted upon rabbits with this milk. (*b*) The effects of different concentrations of caustic soda on rabbits.

4. The continuous taking of alkaline stomach powders may have serious consequences.

IV. Acknowledgments.

My thanks are due to Dr. J. Louw of the Section of Biochemistry for determining the quantity of sodium in the suspected specimen of milk. To my assistants, Mr. J. G. Malherbe, M.Sc., and Mr. M. G. van Niekerk, I am indebted for assistance rendered in the course of the experiments.

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