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The Bulletin

of the

Vancouver Medical Association

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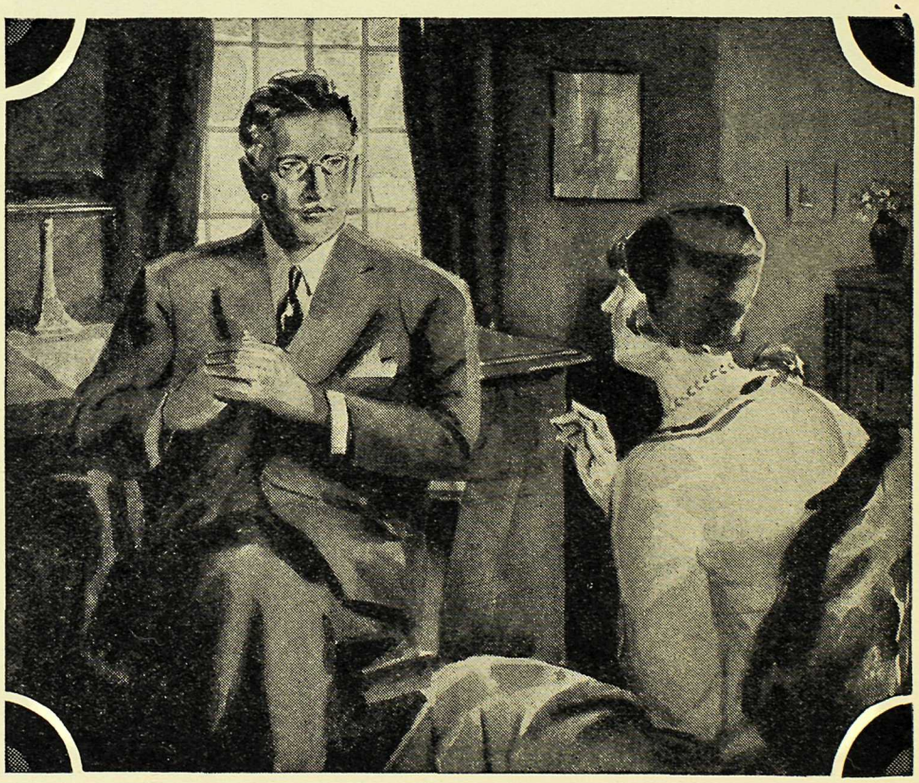

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BULLETIN

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Editorial Board:
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All communications to be addressed to the Editor at the above address.

Vol. V. DECEMBER, 1928 No. 3

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PROGRAMME OF THE 31ST ANNUAL SESSION

GENERAL MEETINGS will be held on the first Tuesday and CLINICAL MEETINGS on the third Tuesday of the month at 8 p.m. from October to April inclusive. Place of meeting will appear on the Agenda.

1928

December 4th—General Meeting:
Papers—Dr. B. D. Gillies; Dr. G. E. Gillies: "Peptic Ulcer, its Medical and Surgical Aspect."

December 18th—Clinical Meeting.

1929

January 8th—General Meeting:
Paper—Dr. Ralph C. Matson, Portland, Oregon: "Surgical Treatment of Pulmonary Tuberculosis."

January 22nd—Clinical Meeting.

February 5th—General Meeting:
Paper—Dr. R. P. Kinsman: "Focal Infections in Infancy and Childhood."
X-ray films to be shown by Dr. H. A. Rawlings.

February 19th—Clinical Meeting.

March 5th—General Meeting.
The OSLER LECTURE—Dr. H. M. Cunningham.

March 19th—Clinical Meeting.

April 2nd—General Meeting.
Paper—Dr. F. P. Patterson: Subject to be announced.

April 16th—Clinical Meeting.

April 23rd—Annual Meeting.

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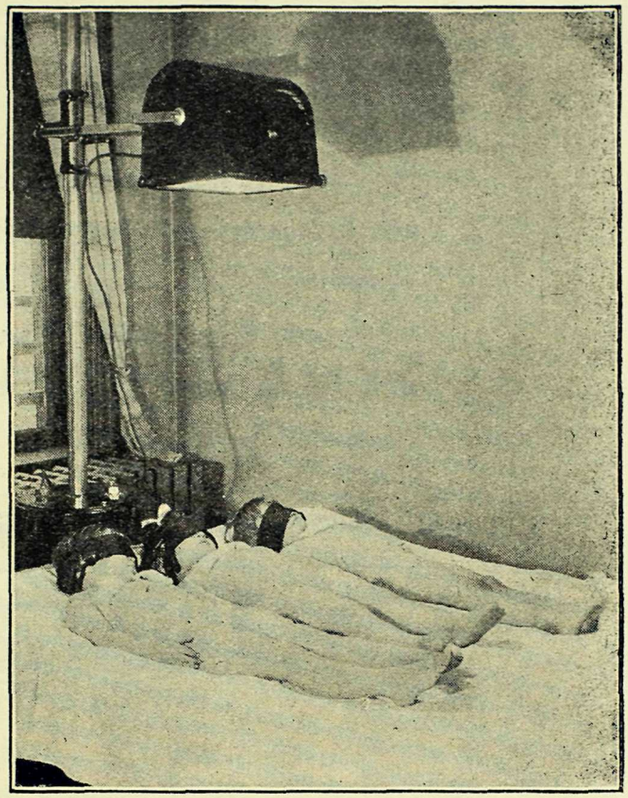

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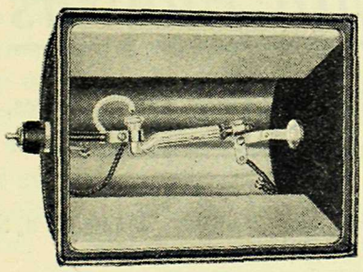

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EDITOR'S PAGE

In this issue we present to our readers a paper of somewhat unusual character. Through the kindness of Dr. C. S. McKee we are enabled to publish some interesting details of a disease in cattle which has been the cause of a great deal of economic loss and which has hitherto baffled all attempts at investigation.

Although the subject of this paper may appear somewhat foreign to the interests of the readers of a medical journal, as a contribution to the study of comparative medicine we feel that it is of considerable importance.

It is possible, nay indeed probable, that much natural experimental work is going on in the lower animals which could be made to yield information of value to the investigation and control of disease in the human family.

While, so far as we are aware, there is nothing in the diseases which afflict mankind immediately comparable to the subject of this paper, our readers will see that the same general problems are present, the same methods of investigation useful. And as is becoming increasingly the case in human disease, workers are turning more and more frequently to the theory that deficiency factors may be of greater importance and that over a wider field than we have hitherto been willing to concede.

This paper and the changing viewpoint which has gradually come over the minds of the authors in the course of their investigation well illustrate the probability that invasion and infection may not contain the whole story of disease. There is a static as well as a dynamic aspect. Moreover the term "deficiency" will have to carry a wider concept. Not only is the food factor including vitamines concerned. Deficiency in oxygen supply or distribution produces effects which have not yet received their due meed of consideration. Deficiency in the digestive juices or in their composition. Deficiency in the secretion of the endocrine glands. At any rate the word will be a useful variant from the word "toxic."

Dr. McKee tells us that the disease under discussion is characterized post mortem by a thickened, ulcerated bladder, from which the bleeding, which lends its name to the disease, arises. The kidneys are not affected and the problem is to account for the condition of the bladder.

Research work was at one time undertaken by the Federal Government, but abandoned at the time of the war.

The disease prevails on bench lands from north of the Fraser river into Northern Oregon. Eastward it is found to a distance of 60 to 65 miles from the coast and is not present on low-lying or prairie farms.

* * *

RED WATER IN CATTLE

A Study of the Pacific Coast Type of Red Water in 63 Head of High Producing Holstein Friesian Cattle on Fernhill Farm, Abbotsford, B. C.

By John R. McKee, B.A., and C. S. McKee, M.D.

Paper read at a meeting of Animal Husbandry and Agronomy experts and Dominion and Provincial Veterinarians on January 7th, 1928.

The writers have jointly suffered severe loss on account of the prevalence of "The Pacific Coast Type of Red Water" which in eight years

has practically wiped out a herd of 63 valuable Holstein Friesians. During this time every precaution was taken to counteract the effect of the disease and naturally, much attention was paid to the affected animals. In the following report we offer some observations, particularly in connection with early symptoms and evidence of pathological changes taking place in the animal long before Red Water itself appears, which to the best of our knowledge have not been recorded before. It may be stated also that animals sold from affected areas to non-affected areas do not develop Red Water, even in cases where they have shown signs of these early symptoms, whereas corresponding animals remaining on the affected areas have invariably succumbed. Once Red Water has appeared, an animal never recovers.

We submit the report, sincerely hoping that our contribution may seem of sufficient importance to cause a reopening of the investigation previously undertaken in connection with this disease, which constitutes a serious economic problem to many dairy farmers in British Columbia.

SITUATION, SOIL AND CROP CONDITIONS

The farm is situated on high land about 3½ miles north and east of the town of Abbotsford, B. C. The soil, a brown loam with a depth of from 1 inch to 7 inches, is fertile and well watered, producing splendid crops of clover, roots and corn. Grain has been grown only as a cover crop for small seeds, on account of difficulties in threshing, and has shown satisfactory growth. The subsoil consists of a blue clay. The land lies in several benches and faces in a general direction of north and west. Mostly cleared from 1912 on, clearing ended in 1924 and it is to be noted that as no new land was brought under the plow the disease progressed more rapidly.

FEED

All roughage produced on the farm has been fed to the animals, but concentrates(including grain) have been purchased. Occasionally small quantities of roughage, some bedding straw, and on one occasion a large quantity of alfalfa hay were obtained from outside.

THE HERD

Seven Holstein grades were purchased in the summer of 1919. A few months later a registered bull calf was secured. Since then, and until the spring of 1924, additions were made to the herd by purchase of springing cows and heifers (mostly high-priced pure-breds), all young animals, none over 5 years of age.

FIRST APPEARANCE OF RED WATER

The first case of Red Water appeared in the fall of 1921—two years and some months after the arrival of the first cows. A second animal showed the red urine a few months later and in 4 years, five out of the original seven cows had become affected (two had died from other causes). Four died from Red Water by the spring of 1923, one lived until May, 1924.

DEVELOPMENT AND SYMPTOMS IN COWS

Since the first appearance of Red Water every animal has become affected sooner or later. In practically all cases the purchased cows re-

mained normal in appearance during their first lactation period and gave satisfactory quantities of milk. Many cows milked sixty pounds on twice-a-day milking and several more than that on three-times-a-day (all on R.O.P.) On second an dthird freshening they never came up to their first showing. In fact, the best never gave more than 40-50 pounds per day with identical feed, care and other conditions. In most cases they rapidly drpoped down to 25-30 pounds per day.

Generally in two or three months after their second calving the cows became unthrifty, their coats rough and dry looking, and they showed a distant tendency to lose flesh. No drop or rise in temperature. They usually went off feed for two or three days, lying down and refusing to eat. A distinct but transitory jaundice could be seen lasting ten or twelve days and then disappearing only to recur later on from time to time. The natural yellow pigmentation of the skin seemed to become unhealthy and unnatural. The change in pigmentation seemed to extend also to the eye. There is, particularly in the more heavily pigmented animals, a natural brown in the iris, even extending into the white. In affected cases this coloring took on a muddy appearance, and the eyes did not look as bright and inquisitive as they do normally.

Our first observation was that a cow would remain about two or three years without showing signs of bloody urine. After that she would live from one to three years. Many of the high-priced animals were kept until they died, in the vain hope that resistant calves might be obtained from them.

Later this conception had to be changed in that "the period of affection" shortened as time advanced. The last "case" on the farm is typical in this respect; the cow in question showed the first indications of trouble eighteen months after coming to the farm; three months later she gave certain signs (red urine); she is already passing small clots of blood, and, unless there is a deviation from other cases, she will die before the first of June this year, 1928.

With one single exception, the period of affection, as indicated before, has become shorter since the disease first appeared. This exception, or respite, refers to the winter of 1924-25, when alfalfa hay and straw, purchased from outside, were fed to the cows. No new cases of Red Water developed that winter. The cows which were already definitely affected continued to retrogress, but the others did better. The four calves which were raised during this period made better gains and progress than calves raised at any other time. But, subsequently they all died from Red Water.

OCCURRENCE AND SYMPTOMS IN CALVES

Most of the first calves born on this farm died from haemorrhagic septicemia. There were some few that lived, however. These few early calves were observed particularly closely during their early period of development. They were, however, rather small for the breed. In these animals red urine appeared at around three years of age, just appearing previous to their second freshening. These animals lived until the fall of 1927, six and five years respectively.

The calves that came from this time on, however, show some very interesting symptoms. At birth they appeared quite normal, whether their dams were showing Red Water or not—nor did it matter whether their dams had just been purchased and given birth to a calf carried previous to coming to the farm or had carried the calf here for full time. For the first six weeks in all cases and in some cases for five or six months the calves were fed whole milk. After that they were changed to skim milk, with a mixture of ground oats and oil cake meal fed dry. Hay (grown on the place) was before them all the time.

From six weeks of age onwards (with four exceptions—fed on alfalfa hay, purchased during the winter of 1924-25) the calves did not thrive. By the time they were three months of age their coats had become rough and dry. They were small for their age, and continued to be so.

These calves seemed to show distinct signs of "jaundice." The pigment of the skin seemed an unnatural yellow. The eyes were muddy looking and seemed to have an unnatural brownish yellow pigmentation, not very pronounced but noticeable. In those born during 1922-23 Red Water appeared just previous to first calving.

From 1924 on, in those calves bred on the place, Red Water often appeared at six or seven months; sometimes it would clear, and again show just previous to freshening. They never lived to freshen again. Of those calves in utero, when the dam was purchased, the Red Water usually appeared just previous to first freshening. They died, usually in two to three months after freshening.

Of those calves born on this place in late 1925 and the spring of 1926, none lived to freshen. They were very poor and unthrifty. Red Water usually first appeared at six to seven months. They never cleared, and died or were slaughtered (when just about dead) at from 10-20 months.

Of the last calves born on the place and kept, (late 1926-spring of 1927) one died at just over one year of age, the other at 11 months.

In connection with this, it may be of interest to note, that these calves at all times *ate their bedding,* which was straw purchased from a farm on Sumas Prairie. They would leave "good" home grown hay to eat this. This was particularly noticeable at all times. And many cows ate dirt the first thing after leaving the barn for pasture in the morning.

The only calves that seemed to thrive at all, were those fed alfalfa hay during the winter of 1924-25.

SPECIAL DISCUSSION OF BULLS

Three bulls were used on this farm. The first one lived for four years and did not come under observation as to the first occurrence of the disease. Very shortly after the first red urine appeared he was dead.

The next bull used was one born on the place. He was sold as a yearling to a farmer on Matsqui Prairie and was small for his age. He was repurchased as a two-year-old for use here. When repurchased he was in nice shape and fairly well grown. He was kept in a pen by him-

self and never allowed to run out to pasture. He had a small run about 150 feet square to play in. He was fed hay, roots and ensilage grown on the place, and a good ration of ground oats and oil-cake meal which was purchased. About 18 months later the first signs of approaching Red Water appeared. The bloody urine showed, and a few months later he died.

The last bull used was also one born on this place. This bull presents a rather interesting study. The first six months of his life, he was fed as the other calves—on hay, grain and milk—the hay grown here. He was, however, an animal of outstanding type and of excellent record backing—(over 800 pounds fat for his three nearest dams and 23,000 pounds milk). All his life he was never allowed to run out. At first alfalfa hay was purchased and fed to him after he was six months of age. He was fed liberally on grain. He received a good allowance of mangels or ensilage grown here. This was kept up until he was two years old, and he was fat and sleek, full of life and did well. At two years he was transferred to a small shed with a yard 20 x 25 feet attached. The ground here had been previously dug and limed. He was from now on fed on roughages grown on this farm plus his usual grain ration. Six months later the prognosis was Red Water. A year or so later the first Red Water appeared, and in a few months he was dead.

The bulls go quickly after the Red Water first appears.

POSSIBILITIES IN REGARD TO CURE AND MANAGEMENT

The cows on this farm were always well fed and cared for, particularly so from the spring of 1924 until the end. They were brushed daily in the winter. The barn was kept clean and the cows well bedded. Water was before them in automatic fountains. In the cold weather (a few weeks) the fountains were shut off and water fed from pails. This water was warmed. The source of water supply is a spring coming out of the rock about 2,000 feet up the hill back of the farm. It is piped down. The water is soft, cold and pure.

The animals were fed roughage grown on this farm at all times, with the exception of the winter of 1924-25, when alfalfa hay and some stra wwas purchased and fed. During this winter no new cases of Red Water developed and the calves did much better.

During the summer months care was always taken to see that the cows had ample feed when pastures were short. The silos were never allowed to become empty. A summer silo of peas and oats was filled in July and some clover was blown in. This was very good feed and relished by all the stock. Either clover, oats or oats and peas were fed to supplement this silage. In August and September there was always ample after grass to carry the stock into winter in grand shape. Usually a little silage was fed after the middle of September to supplement the pasture. Grain was always fed in ample quantities to both milking and dry stock. In fact to many of the Red Water animals, grain in excess of what normally would be required was fed. This heavy grain feeding seemed to make no difference to the course of the disease. Quite often, in fact, some of the diseased cows would refuse their grain for a day or so.

BACTERIA OR PROTOZOA

The blood and urine were examined many times for the presence of bacteria or protozoa. The urine was particularly well examined, both of apparently normal and diseased cows. No signs of bacteria or of protozoa were at any time in evidence. The possibility of its being a leptospira involving the liver has not been excluded.

MILD CARRIER TYPE OF HAEMORRHAGIC SEPTICEMIA

Since this was started no haemorrhagic septicemia has appeared. The Red Water went on as before. This may be excluded by the fact that after our first losses, all animals were inoculated, the calves ten days after birth.

MINERAL FEEDING

Several mineral mixtures were tried. The first consisted of calcium carbonate, bone-meal, ferrous carbonate and iodized salt. No difference in the course of the disease was noted. Also a mineral mixture put out by the Triangle Chemical Company was tried. The cows refused to eat their grain wth which this was mixed at first. They ate it finally, but the use of the mixture was discontinued as the stuff smelt like bone-meal fertilizer and had an offensve taste. A first-class quality of bone-meal flour was then fed, with a little ground limestone and iodized salt. No difference was ever noted in the course of the disease.

DR. MILTON'S MIXTURE

This was a preparation secured from Dr. Milton of Chilliwack. The cows were drenched three times daily before feeding in December, 1925, for 14 days. Eight animals were so treated. In seven cases of young heifers, the Red Water temporarily disappeared (for about two months). All animals treated became very sick and went off their feed. When the disease again appeared similar treatment had no effect on these same animals. Some milking cows were also treated. It had no effect on the disease. The cows quit milking, however. Several other farmers in the neighborhood tried it on mature anmals, but reported that it did not relieve the cows treated.

It may be well to state here that other iron, lead and arsenical salts were tried, with no effect on the course of the disease.

THEORY OF DEFICIENCY

Therefore in view of the facts as herein presented—that as time went on, the land became more intensively cultivated and no new land was brought under cultivation; that the cows purchased lived for shorter and shorter periods; that the calves died earlier and earlier in life; that the severity of the disease increased, we have come to the conclusion that this type of Red Water is a deficiency disease. We believe that the liver is involved some time before the appearance of the red urine, which in our opinion is the terminal point of the disease.

SUGGESTED INVESTIGATIONS

From what has been previously stated it would seem fairly conclusive that deficiency of some kind must exist, and unless this conference reaches other conclusions it is respectfully suggested that investigations should

be carried out along the following main lines in order that this important economical problem may be solved:

SOILS AND CROPS

1. A survey establishing areas involved and losses sustained in at least ten specific areas. (Widely distributed areas).
2. An examination of soils in the districts affected.
3. Chemical analysis of water, soils and feed (with special reference to Ca, K, P, Mg, Mn) grown upon the soils in question.
4. Feeding experiments with a sufficient number of cows so that comparative results may be obtained.

(a) From feed grown on "deficiency" land.
(b) From feed grown on unaffected areas.
(c) From a combination of feed from affected and unaffected areas.
(d) With different kinds of drinking water.

ANIMALS

Blood chemistry—Ca, P, K, Cholesterol, Mg, Sugar, NPN.

A series of animals should be taken to the farm and one killed every four months and sections of the livers taken.

* * *

Case Histories

By John R. McKee, B.A.

I. *Star*—Grade Holstein. Purchased August as a springer. Calved late September, 1919—a bull which was vealed.

(a) Calved October, 1921—heifer.
(b) Calved December, 1922—heifer.

She showed signs of red water shortly after her second calving (November-December, 1921). She gave birth to another calf in December, 1922, and as the Red Water was affecting her production severely, she was shortly after butchered.

(a) Grade—born October, 1921. Was a mother of bulls throughout her life. She showed signs of Red Water shortly before her second calving. It cleared up after this calving. Red Water again appeared just before her next freshening. Never entirely cleared thereafter. Died October, 1927. P.M.—Liver badly adherent to diaphragm—elongated downwards—felt tough and leathery. Bladder, usual Red Water type. Other organs seemed normal.

(b) *Star*—born December, 1922. Freshened four times with the following calves:

*(b1) Heifer—Grade II.—born February, 1925.
*(b2) Heifer—*Little Star*—born December, 1925.

Her next calf, born October, 1926, was a monster and in May, 1927, she aborted.

As a calf she did not thrive well. Was small. Coat always rough. Calved at 23 months. Just previous to this first calving she showed

* These dates are correct. I bred a number of my grade cows when they first came around in order to have them freshen sooner for the purpose of balancing my herd.

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bloody urine. The urine cleared until three months before next freshening. It never cleared thereafter. The rest of her life she kept going down hill until she died from Red Water in November, 1927. P.M.—Liver badly adherent to diaphragm—elongated downwards—felt tough and leathery. Bladder, usual Red Water type. Other organs seemed normal.

(b1)—Grade II.—born February, 1925. Small unthrifty calf. Was bred and gave birth to a bull. Showed Red Water previous to calving. Was bred again. Died of Red Water before next calf was born. No. P.M.

(b2) *Little Star*—born December, 1925. Was a small and unthrifty calf. Passed bloody urine at 11 or 12 months. Died before she was bred ,aged 20 months. P.M.—Liver as usual. Bladder typical. Other organs seemed normal.

II. *Mandy*—Grade Holstein. Purchased August, 1919. Calved November, 1919, a mother of bulls. Gave birth to one daughter. Do not know when she first showed signs of Red Water. Died March, 1925. P.M.—Liver very badly adherent to diaphragm and spleen. Seemed to be general adhesions all around the liver, which was elongated and tough. Bladder very badly ulcerated and hard.

(a) *Little Mandy.* Born April, 1924. Grew fairly well. Was fed purchased hay in the summer of 1924 as our own had run out. Was not allowed out of the barn. Fed purchased alfalfa winter of 1924-25 plus roots and ensilage grown on the place. Thrived well. Was turned out with the herd to pasture in the spring of 1925. Became unthrifty by the summer. Was bred in the fall of 1925. Did not grow much the winter of 1925-26. It was observed that she looked very poorly when put in the barn in the fall of 1925. She showed a muddiness of eye and looked yellow. Told my brother she would develop Red Water before the winter was out. In March, 1925, she passed bloody urine. Calved July, 1926. Died end of August, 1926. P.M.—As usual.

III. *Hejelkje Rooker*—Purebred Holstein. Purchased February, 1920.
Calved June, 1920—bull—(a) *Fernhill Rooker Posch.*
Aborted, 1921 and 1922.
Calved March, 1924—heifer (b) *Fernhill Hejelkje Korndyke.*
Calved March, 1925—heifer (c) not registered.

This cow first showed Red Water in the spring of 1923. Usual development. Died September, 1925. P.M.—As usual.

(a) Bull—*Fernhill Rooker Posch,* born June, 1920. Raised to a yearling. Sold to a lowland farm. Repurchased at 2 years of age for service in our herd. Noticed him rather rough looking in the spring of 1924. Red Water appeared in June, 1924. Died September, 1924. No P.M.

(b) Heifer—*Fernhill Hejelkje Korndyke*, born March, 1924. Never thrived. Was rough looking. Seemed yellow. Was so small she was not bred at the usual time. In April, 1926, showed bloody urine. Met with an accident and was destroyed. P.M.—Liver not adherent. Was tough and leathery to the touch. Bladder showed ulcers.

(c) Heifer calf—not registered, born March, 1925. Very small and weedy looking. Never grew much. Looked sick and jaundiced all the time. Eye not clear and bright. Showed Red Water in August, 1925 at 6 months of age. Cleared. Sold her cheap to farmer on Sumas Prairie. (He know she had Red Water). She died of Red Water on his place sometime in summer of 1926.

IV. *Countess Canary Mercedes*—Pure-bred Holstein. Purchased September, 1920. Aborted and again aborted. Calved April, 1923—twin heifers.

(a) *Fernhill Canary Segis* (twin), born April, 1923.
(b) *Fernhill Canary Segis Girl* (twin), born April, 1923.

This cow showed Red Water just previous to this calving in April, 1923. She died in August, 1924.
P.M.—Liver badly adherent—bladder as usual.

(a) *Fernhill Canary Segis* (twin), born April, 1923. This animal was a mother of bulls and aborted. She had one daughter, (a) not registered. Born June, 1925. The heifer showed Red Water just before her first calving in June, 1925. From then on she passed bloody urine. She lived until August, 1927, when she aborted. Her Red Water then became so severe that I shot her. P.M.—As usual.

(a1) Heifer calf—unregistered. Born June, 1925. Small, but did not do badly. Coat fairly rough. Was fed purchased alfalfa hay as a calf. Sold her at 23 months to a farmer on Sumas Prairie. She never, to my knowledge, passed bloody urine. She looked to me, however, as if she was going down with Red Water. Later her coat became harsh and dry, unhealthy looking yellow pigmentation appearing and eye becoming muddy looking. I am watching her at her new home. She has been there now for 3 months. Her present owner informed me (February 23, 1928) that she was picking up and looking fine, no signs of Red Water.

(b) *Fernhill Canary Segis Girl* (twin), born April, 1923. She was a mother of bulls. Did not thrive very well as a calf. Did not breed her until nearly three years old. She showed Red Water when just over two years of age. Never cleared. She calved in February, 1926. She did not milk very much. She was killed in May, 1926 for examination. P.M.—The liver was not adherent. Was not of normal color. Felt tough and leathery. Section of the liver showed gross inflammation and destruction. (Round cell infiltration).

V. *Colantha Walker Beauty*—Pure-bred Holstein,purchased August, 1922.

Calved October, 1922—heifer, (a) *Fernhill Colantha Walker.*

Calved February, 1924—heifer, (b) not registered.
Calved February, 1925—heifer, (c) not registered.
Calved February, 1926—bull, stillborn.

This was a big heavy producing cow with a very strong constitution. She had great depth and heart. She milked well during her first lactation.

In the fall of 1924 it was noticed that she seemed unwell. Made a close examination. Cow appeared jaundiced. Was off feed. Eye seemed muddy. She dropped from 40 pounds of milk a day down to 17-18 pounds of milk a day in about 6 weeks. Recovered from her illness and got on feed again. Did not milk well again, 15-16 pounds daily.

She calved again in February, 1925. Did not milk well, only 45 pounds when fresh—dropped in 3 months to 30 pounds. Red Water appeared in the summer of 1925. She seemed to throw it off. Did not milk well, even on good grass.

She calved again February, 1926, and was never bred again. Red Water became worse. She did not milk at all well, only 37 pounds at freshening and did not come up in the spring on good grass. She kept her flesh well, but was fed very heavily on grain. She dropped, in spite of good grass to about 25 pounds daily and gradually dried off. She was kept to see what would happen. Fed heavily on grain, but she kept getting worse and worse, until in fall of 1927 she was sold to the butcher for her hide. No. P.M.

(a) *Fernhill Colantha Walker,* born October, 1922. Did not thrive well as a calf. Was bred late. Calved February, 1925 (a bull which was vealed). Milked very poorly. Was very thin and could barely stagger around. Recovered slightly but was not bred again. Died June, 1926. P.M.—As usual. Very severe liver adhesions.

(b) Heifer calf—unregistered. Born February, 1924. Was small and unthrifty. No Red Water. Sold January, 1926. Is still alive and doing well.

(c) Heifer calf—unregistered. Born February, 1925. Did not grow well. Small and unthrifty. Looked jaundiced and eye seemed muddy. Passed bloody urine in August, 1925. Cleared. Was sold January, 1926. Die dof Red Water later in year. (The owner does not remember the date of her death. She had Red Water, he knew, and he took a chance).

VI. *Nikomekl Butter Girl*—pure-bred Holstein. Purchased August, 1922.

Calved October, 1922, bull. Died of haemorrhagic septicemia.
Calved November, 1923, bull, *Fernhill Douglas Segis.*
Calved February, 1925, twin heifers (sold as calves).
All other calves bulls and vealed.

She milked well during her first lactation. After she again freshened, she soon became poor and rough. Afterwards milked poorly

always. In the fall of 1924, she first passed bloody urine. She became steadily worse and died August, 1927. P.M.—As usual.

(a) Bull—*Fernhill Douglas Segis,* born November, 1923. Did poorly as a calf. The in spring of 1924 I started feeding him purchased alfalfa and lots of grain. Also he was fed roots and ensilage grown on the place. This feeding was continued until he was 2 years old. He was *never* out of the barn till then. He was fat and sleek, full of life, a beautiful animal in every way. In the fall of 1925 I put him in a pen with a small yard 20 ft. by 25 ft which I dug and limed. From then on I fed him home grown feed entirely. In 6 months he lost his sleekness; his coat became rough and dry; his skin tough and his eye muddy. He grew quite a bit, however. He was fed 10 pounds of grain a day, 30 pounds of roots—i nsummer about 25 pounds of ensilage instead of roots. All the hay he could eat was given him. He began to get mean and I had his horns cut off in the spring of 1927. H ebecame dangerously mean. I watched closely, suspecting Red Water some time previously. In September, 1927, he passed bloody urine. He died in November, 1927, just four years of age. No P.M.

VII. *Colony Girl Newman*—Pure-bred Holstein, purchased 1924.
Calved June, 1924—heifer (a) *Fernhill Aggie De Kol.*
Calved April, 1925—heifer (b) *Fernhill Posch Newman.*
Calved April, 1926 and 1927, bulls—vealed.

This cow did extremely well her first lactation. Milked over 90 pounds a day part of the time—tested 3.4 (on R.O.P.) She calved again in April, 1925. She did well at first, but in about 3 months she dropped very materially. Her coat became a little rough. She calved again in April, 1926. She did not do very well—only 55 pounds when fresh. In a few months (on clover pasture up to her belly) she dropped to about 20 pounds daily. She looked unwell. Her eye was not clear. (She was a black cow and it was difficult to be sure of her skin pigmentation). The prognosis was for Red Water. She calved again in April, 1927. She passed bloody urine in May, 1927. She was sold in November, 1927 to a farmer on Sumas Prairie who was gambling on a calf from her. She calved in March, 1928, and is going down hill. Calf is doing well.

(a) *Fernhill Aggie De Kol, born* June, 1924. Did fairly well as a calf. Was fed purchased alfalfa hay the first winter, and came on well. She was bred. Went to pasture with the herd the summer of 1925 and that winter was fed a home grown ration plus purchased grain. She did not do well. She showed Red Water just previous to calving in July, 1926. She developed a tumor in her udder and was butchered. P.M.—Liver tough—not adherent. Ulcers on bladder.

(b) *Fernhill Posch Newman,* born April, 1925. She never did well as a calf—coat and other symptoms as usual. Red Water appeared in August, 1926. Cleared. Did not breed as she was small. In February, 1927, Red Water again appeared and gradually became more severe. She died in September, 1927. P.M.—Liver adherent to diaphragm. Bladder badly ulcerated.

VIII. *Teussie Victoria Echo 2nd*—Pure-bred Holstein, purchased September, 1924.

Calved October, 1924, heifer (a) *Fernhill Victoria Ormsby.*
Calved September, 1925, bull—vealed.
Calved September, 1926, heifer (b) unregistered.
Calved July, 1927, heifer (sold).

Did fairly well until December, 1926, when she dropped in her milk. She became rough and unthrifty looking. In October, 1927, she looked jaundiced and her eye became muddy. She passed bloody urine in December, 1927. She is still alive but the disease is very severe—clotting is already apparent and she is rapidly losing ground. She eats dirt whenever let out of the barn (February, 1928). She died in August, 1928. P.M.—Liver not adherent, though tough and thickened.

(a) *Fernhill Victoria Ormsby,* born October, 1924. Her history is the same as that of the others. She showed Red Water at about a year. Gradually became worse. Was bred. Calved February, 1927, and died of Red Water in April, 1927. P.M.—As usual.

(b) Heifer calf—unregistered. Born September, 1926. Was very poor and unthrifty. All signs of Red Water from 3 months of age. Passed bloody urine at 6 months. Died of Red Water November, 1927. P.M.—As usual.

IX. *Thorndale Creamo Posch*—purchased October, 1924.

Calved December, 1924—bull, vealed.
Calved December, 1925—heifer (a) unregistered.
Calved November, 1926—heifer (b) unregistered.
Calved September, 1927—heifer (sold).

Usual course. Was sold to a man on Sumas Prairie who is gambling on a calf. Is still alive but passing bloody urine.*

(a) Heifer calf—sold at 2 years. No Red Water but has shown all preliminary signs. Is doing well now and is under observation.

(b) Heifer calf, born November, 1926. Very poor and unthrifty—ill all the time—eye muddy. Red Water appeared at 5 months. Died September, 1927. P.M.—Liver not adherent. Was tough and very hard. Not a good color.

X. *Etta Banostine Canary*—pure-bred Holstein, purchased September, 1924.

(a) Freshened October 15, 1924—heifer.
(b) Freshened October, 1925—bull—vealed.
(c) Freshened November, 1926—bull—vealed.

This cow was never a good milker and was very particular as to her food, though not a good feeder. She wa snot bred after her calving in 1926 and did not show signs of Red Water until spring of 1928. In August she first passed blood and was killed September, 1928 .

* Has died since this report was written.

(a) *Fernhill Banostine McKinley.* Born October 15th, 1924. Grew slowly and never attained full size. When fully matured was smaller than her mother. She was bred and gave birth to a heifer calf (a1) September 7th, 1927. This heifer (a) was a much heavier milker than her dam but was not bred again. She showed early symptoms in the spring of 1928 and passed Red Water first August, 1928, about the same time as her dam and was killed September, 1928.

(a) *Fernhill Banostine Segis.* This calf was fed on purchased hay and grain until sold in March, 1928. She grew rapidly and gives every promise of being a fine animal.

XI. *Ormsby Queen Calantha.* Purchased Holstein. Purchased September, 1924. Half sister of No. X. and No. VIII. Very heavy milker and fine dairy cow. Died of Red Water October 19th, 1927. She was a mother of bulls and is only put in here for purposes of comparison with her half sisters Etta Banostine Canary and Teussie Victoria Echo 2nd, the three animals being purchased and brought to the farm at the same time.

* * *

Pathological Reports

from the Pathological Department, Vancouver General Hospital.

March 28, 1928.

S-27-743, 744—Cow's Spleen and Liver.

(Cow born on farm out of a mother with Red Water).

Macroscopical Examination: . Specimen consists of a portion of liver, on one surface of whch is a rather dense, hyaline-appearing structure which grossly has the appearance of a portion of cyst wall. A small portion of what is evidently spleen, is also present. There is otherwise nothng particularly remarkable about the specimen.

Microscopical Examination: A number of sectons were taken through the spleen and liver and in the former beyond a moderate degree of congestion and some slight increase in the fibrous connective tissue trabeculae, nothing of note is apparent. The liver, however, shows many areas of dense hyaline fibrosis which is not at all of the scirrhous type but is of a diffuse type which completely destroys any growth of liver cells in these particular parts. Many capillary channels are present in these areas and diffuse small round and plasma cell infiltration. Although these plasma cells are not well defined nor typical, it is possible that these may be degenerated hepatic cell nuclei. In these fibrous areas, scattered canalicular structures are seen which are probably bile ducts. Throughout the less fibrotic liver tissue, very extreme congestion is seen, with atrophy of liver cells, leaving vacuolated spaces where they formerly were, giving the appearance somewhat of a fatty infiltration, but it is the former, i.e., cyanotic atrophy due to passive venous congestion. In the portal spaces there is some increase in the fibrous connective tissue but no special inflammatory cell infiltration. There is nothing of a specific nature in any of the sections examined and it is probably a process that in the liver at least, has its port of entry in the portal spaces, but through which component part of the portal spaces—bile, duct, artery or vein—is a question.

S-28-2350—Portions of Cow's Liver.

Shows little of note grossly.

Microscopical Examination: A number of sections were taken through the different areas and these show the central veins to be filled with a pinkish-staining material, homogeneous for the most part. There seems to be increased fibrosis in the portal spaces, with a moderate small round cell infiltration, and moderate congestion throughout the parenchyma. Subcapsularly, layers of liver cells are seen which have a rather vacuolated appearance somewhat resembling a fatty degeneration, but one cannot say definitely that it is such. Otherwise, little of note is apparent.

H. H. PITTS, M.D.

* * *

GUINEA PIG EXPERIMENTS

As we had no more cattle to kill we turned to guinea pigs as a possible experimental animal to use in feeding tests. If they responded in any like way in the nature of similar liver changes, a less expensive and more rapid method of investigation was hoped for.

In May, 1928, half our breeding stock of guinea pigs was taken from Vancouver to the farm. It may be said here that those left in Vancouver did as well as usual.

In July it was noticed that the animals at the farm were not thriving, although given all the fresh clover and carrots they could eat. All the young born up to this time lived, but after the middle of July all young were killed and eaten by the older animals. From this date until October, a guinea pig from the farm was brought down every couple of weeks, killed and sections made of the liver, with the general picture given in the autopsy findings appended. About the middle of October the animals began to die off and the remainder were brought back to Vancouver. Of those so dying, the ones showing most degeneration of the liver were born on the farm.

Some of the guinea pigs brought back were put in a cage in which there were some newly born animals from those never out of the city. These young were promptly killed and eaten by the farm animals, but after a few days on the hay, etc., used for our normal feeding, no further deaths occurred and no more eating of the young. In fact some young have been born amongst the animals brought in from the farm and they are growing normally.

Further feeding experiments as well as blood and tissue chemistry work are in course of preparation now.

Pathological Reports: Numerous examinations have been made by Dr. H. H. Pitts, of the Pathological Department of the Vancouver General Hospital, of the livers of these guinea pigs. A typical reports says "the liver cells stand out rather clearly as far as their borders are concerned as a rather reticulated network in the centre of

which well formed nuclei are seen. The protoplasm is, however, not so well preserved and shows quite extensive vacuolation, probably a fatty change. The central veins are not particularly congested and there is no evidence of any inflammatory infiltration in the portal spaces or elsewhere." (S-28-2309).

On one guinea pig's liver (a guinea pig born on the farm) the report says "there seems to be more of a definite fatty-appearing infiltration so vacuolated as to give the cells a crescentric appearance or so called "signet ring" morphology. Extensive passive venous congestion is also apparent."

NOTE:—The writers wish to thank Prof. P. A. Boving for his constant aid in the many changes they made in their feeding methods.

* * *

CONGRESS OF RADIOLOGY

REPORT ON THE "SECOND INTERNATIONAL CONGRESS OF RADIOLOGY" STOCKHOLM, 1928.

DR. C. W. PROWD

The Second International Congress of Radiology was held in Stockholm, Sweden, in July, 1928. The first held in London three years previously paved the way and, in a measure, set the pace for the Stockholm meeting. Thes recent congress was largely attended and gathered together over twelve hundred delegates from all parts of the world. The papers were read in one of three official languages—German, French or English—the Swedes, with characteristic generosity, relegating their own language to the background. The German group was the largest, Russia second with France, British Isles and Italy well represented. More than one hundred registered from the United States of America. One cannot speak too highly of the splendid organization and complete arrangements. The President, Dr. Gosta Forssell of Stockholm, and his committee, reflected the Swedish efficiency and hospitality everywhere apparent.

One must note particularly the thoroughness and care with which the Information Bureau handled what at times approached a miniature "Tower of Babel," presided over by a score of Swedish women and girls, equally at ease in any language. Charming in manner and appearance they won the favor and plaudits of all present—not excepting your two representatives from Vancouver.

Every phase of Radiology—technical, diagnostic and therapeutic received its share of papers and discussion. The many problems of diagnostic procedure and interpretation were crystallized in world opinion. International standardization in radium and x-ray therapeutics were brought definitely nearer. The papers, proceedings and resolutions of the congress will be published in two special volumes of "Acta Radiologica" soon off the press.

Two resolutions of outstanding importance were adopted by the Stockholm Congress. One, standardized protective measures for x-ray and radium workers, the other defined an international unit of measurement for radiation dosage.

The necessity of increased protection and improved working conditions for x-ray and radium operators in all countries is recognized. The Continent is in advance of America in these measures. The hospital space and room arrangement, also the measure of protection in x-ray equipment is in many cases in striking contrast to our limited space and conditions of work.

Standardization of x-ray dosage and an international measuring unit will make duplication of treatment and comparison of results more accurate and simple.

Continental hospitals and clinics, as a rule, devote much space, equipment and personnel to their x-ray and radium departments and there is a keen appreciation of the growing importance and necessity of their diagnostic and therapeutic measures.

The comprehensive report and demonstration by Dr. Gosta Forssell and his associates on "Therapeutic methods and results at Radiumhemmet" was one of the best offered. Over one hundred cases were presented. The history, method of treatment and result was given in each case. The cancer groups considered most favorable are cutaneous cancer, cancer of the lip, cancer of the oral cavity, cancer of the uterus and sarcoma.

The following summary is of interest:

"Of a total of applicants of 207 cases of cancer of the skin of the face from the years 1910-1915, inclusive, 142 have remained healed, which means a percentage of *absolute cure* of 68 per cent. If, however, we count only technically operable cases, the figure for *relative cure* is 78 per cent.

In *lip cancer* we have obtained a permanent healing in 45 cases of a total number of applicants of 66, that is in 68 per cent. of the cases. For the operable cases the percentage of *relative cure* was 86.

In *cancer of the mouth* we have got a five years' healing in 71 cases of 113, that is a figure of relative cure of 62 per cent. of all cases radiologically treated.

In *cancer of the uterine cervix* our absolute healing figure is 22.4 per cent. by a total number of applicants of 500.

Of 145 *operable and border-line cases* 67 have obtained a five years' healing, corresponding to a relative cure of 46.2 per cent.

In 234 *inoperable cases of cervix cancer* five years' healing is reached in 16.7 per cent. of the cases treated.

In *cancer of the uterine body* the figure of absolute healing is 43.5 per cent. (20 out of 46 cases), and the relative performance 60%, or a 5 years' healing in 15 of 25 operable cases.

Of the total of 543 cases of *sarcoma* treated at the "Radiumhemmet" during the period 1910-1922, 181, or one-third were free from symptoms when reexamined in 1925. Of the 238 primary tumors treated by radiotherapy only 58, 24 per cent., have remained free from symptoms.

I will also mention that the results of our comparison between the final achievements of surgery and radiotherapy as regards the treatment of malignant tumors is as follows:

In those types of tumour that offer a fair chance of obtaining a primary healing by radiological treatment, the duration of the healing obtained by radiotherapy is, both as regards the relative and the absolute performance, in every way comparable to the results obtained by surgery.

When primary healing—immediate freedom from symptoms—has once been secured by radiotherapy, the percentage of recurrences in most of those forms of tumour is lower than after primary surgical healing, and in none of the groups comprised in this inquiry is it higher than the percentage of recurrences following radical operation.

The duration of the *period of latency* is very much the same for recurrences after radiological treatment as for recurrences after radical operation. Most of the recurrences occur during the first and second years after primary healing, the majority of them during the first year. After the third year there are only scattered instances of recurrence.

After the fifth year they are rare; still, cases have occurred as late as in the eight and ninth years. After the ninth year our material does not show any recurrence.

In radiotherapy of tumours the *palliative effect* plays at least as important a part as the *curative effect*.

Our system of after-observation gets its strongest support from the fact that the Government, in conformity with a decision of the Swedish Parliament, defrays the travelling expenses of all poor patients to and from the hospital. This makes the maintenance of a personal observation and control possible in most instances. With a few exceptions we have been able to keep constant track of all our cases for the last fifteen years."

The Third International Congress of Radiology meets in Paris in 1931. Those who contemplate the Paris Congress would do well to include Northern Europe and especially Scandinavian countries in the itinerary, both from a scientific and recreational point of view.

VANCOUVER HEALTH DEPARTMENT

STATISTICS, OCTOBER, 1928

Total Population (Estimated)		142,150
Asiatic Population (Estimated)		10,940
	Rate per 1000 of Population	
Total Deaths	128	10.63
Asiatic Deaths	6	6.47
Deaths—Residents only	101	8.39
TOTAL BIRTHS	361	29.98
Male 180		
Female 181		
Stillbirths—not included in above	11	
INFANTILE MORTALITY—		
Deaths under one year of age	8	
Death rate per 1,000 Births	22.16	

CASES OF INFECTIOUS DISEASES REPORTED IN CITY

	September, 1928		October, 1928		November 1st to 15th, 1928	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Smallpox	11	0	29	0	26	0
Scarlet Fever	9	0	11	0	6	0
Diphtheria	37	2	76	3	52	1
Chicken-pox	9	0	24	0	67	0
Measles	0	0	0	0	0	0
Mumps	4	0	5	0	10	0
Whooping-cough	8	0	17	0	14	0
Tuberculosis	9	14	16	7	5	–
Erysipelas	5	1	8	1	3	0
Poliomyelitis	5	3	1	0	0	0
Cases from Outside City—Included in Above.						
Diphtheria	11	0	23	2	20	0
Scarlet Fever	2	0	2	0	3	0
Smallpox	3	0	2	0	0	0
Typhoid Fever	0	0	3	0	2	0
Poliomyelitis	2	2	–	–	–	–

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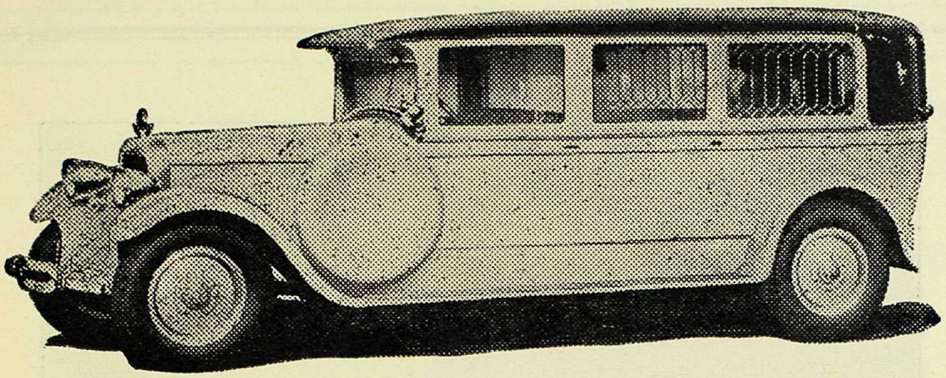

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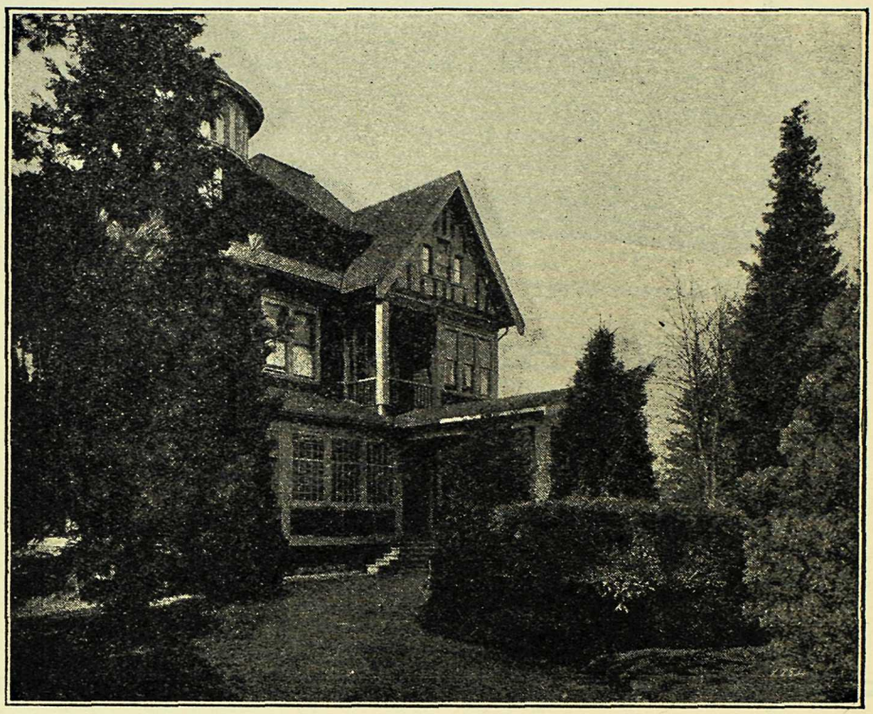

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