invaded and replaced by an inflammatory fibrous tissue (Fig. 6, *d*) or new bone may develop in the ankylosed cartilage and thus bring about osseous ankylosis.

Osseous ankylosis takes places by the development of new bone either in ankylosed cartilage or in the fibrous tissue which has already produced fibrous ankylosis.

Ankylosis may occur between the two zones of calcified cartilage, but this form of fusion shows a tendency to become replaced by one of true bone.

Villous outgrowths are found in some joints. These may be due to (*a*) overgrowth of normal villi; (*b*) inflammatory changes in the synovial membrane or in the capsule.

Villous outgrowths in a joint may consist of (*a*) vascular fibrous tissue, (*b*) dense fibrous tissue, (*c*) fibrous tissue enclosing groups of fat cells, (*d*) inflammatory tissue with dilated capillaries and groups of inflammatory cells, (*e*) degenerated or necrotic tissue, (*f*) connective tissue showing vessels deeply congested owing to obstructed circulation.

Villous outgrowths may become detached by rupture of the fine pedicles, and so form a loose body in the joint cavity. The detachment may be preceded by necrosis.

Microscopic villi formed of cartilage are occasionally found on the surface of altered articular cartilage. These may be formed of hyaline or fibro-cartilage.

No constant changes are found after death in the organs or tissues of patients who have suffered from rheumatoid arthritis.

OBSERVATIONS ON THE CAUSE OF RICKETS.

[WITH SPECIAL PLATE.]

BY

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AND

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In the course of an investigation on metabolism in rickets the difficulty of getting satisfactory material from children induced us to produce rickets in a certain number of young dogs, while keeping others free from the disease.

The previous experiences in this laboratory had shown that rickets may almost certainly be induced in the great majority of pups of certain breeds of dog by simply keeping them in the animal house of the department, while the experience of every keeper who breeds dogs is that if pups are to be reared without rickets developing they must have abundant exercise in the open air. One of us (L. F.) has already published an experimental investigation on the etiology of rickets in young dogs,¹ in which he records that puppies freely exercised when fed on bread and water, oatmeal and water, etc., did not develop rickets, but became marasmic and died. On the other hand, puppies fed on oatmeal porridge and milk, if allowed to run about, remained free of the disease, but if confined developed it.

In the large series of experiments made by Mills Renton and Madge Robertson² in this laboratory on the influence of thymusectomy on the production of rickets, all the pups were kept on the same diet of oatmeal porridge and milk with bones for them to gnaw. Some became rickety, some remained free of the disease, their immunity seeming to depend rather upon the breed than upon their mode of life. Thus, all the collies developed rickets; of the retrievers one remained normal while two became rachitic; the two fox terriers showed slight signs of rickets, while the two greyhounds remained normal. Thymusectomy had no effect on the onset of the disease. One collie which had suffered severely from rickets made a complete recovery, and was alive and well except for deformities at the age of six months. The diet had not been altered, but the animal had been kept outside during August and September for most of each fine day.

In 1913 Casimir Funk³ suggested that rickets may be due to absence of some "vitamine" from the food, but his arguments in favour of this view are far from convincing.

As is well known, Bland-Sutton maintained that by the administration of fat the development of rickets in young carnivores kept in confinement might be prevented. But the general experience of zoological gardens at present is that no amount of fat in the food will prevent rickets if the cubs are confined.

Hess and Unger⁴ record observations on negro children in the negro quarter of New York where a very high proportion develop rickets, which tend to show that cod-liver oil has a prophylactic action.

E. Mellanby, on January 26th, 1913, gave a short communication to the Physiological Society reviving Funk's vitamine theory and maintaining that he had demonstrated by experiments on pups that—

"1. Rickets is a condition primarily due to lack of an accessory factor in the diet.

"2. This accessory factor is found in (1) extractives, especially meat; (2) fats, especially butter, but not in linseed oil.

"3. Lack of exercise may play some part, but not a primary part, in the development."

Till his evidence has been published it is of course impossible to estimate the value of the observations. The present vogue of "vitamines" and "accessory factors" seems to have led to a somewhat premature adoption of this theory.

The very extensive study of the "Social and Economic Factors in the Causation of Rickets" by Margaret Ferguson from this laboratory⁵ gives little support to the theory that the disease is due to a dietary deficiency. The tables given on pp. 68 and 69 seem to show conclusively that the difference in diet between rachitic and non-rachitic families is small and that the overlap is large, and indicate pretty clearly that some other factor than feeding is involved in the causation of the condition. The very striking difference in the air space of the houses and in the extent to which the two groups of children had opportunities of free egress to the open air seems to show that in some way these conditions exercise a marked influence upon the immunity to the development of rickets.

Present Observations.

As already stated, the present observations were not undertaken primarily to investigate the etiology of the disease, but to give us a certain number of normal controls, and a certain number of rachitic animals for the study of calcium metabolism.

Since seventeen pups were involved an opportunity was afforded of checking and extending our previous experience.

Two litters were used, both collies, a breed peculiarly liable to rickets. The first litter of eight was received on March 30th, 1918, just after they had been weaned at seven weeks old. They were of a small black collie strain. When they arrived they were in poor condition and verminous. The vermin were destroyed, and they improved in condition. They were fed on oatmeal porridge and milk, and were killed at 13 weeks old.

The second litter of nine, of a rough working collie strain, were received with their mother on March 22nd, 1918, at 14 days old. They were all in good condition, and were suckled till about March 28th, and then put on oatmeal porridge and milk.

They were killed when about 11 weeks old.

A. Pups Sent to the Country.

Of the first litter a dog and a bitch were sent to the country on April 12th, 1918, when 9 weeks old. They were in poor condition when they left the laboratory. In the country they were kept in a large loose-box in a stable, and they had the run of a stable yard and garden under the personal observation of one of us (D. N. P.). They were sent back to the laboratory on May 12th and killed at the age of 13 weeks. They were strong and healthy and showed no signs of rickets. (Special plate, Fig. A.)

Of the remaining six of the first litter one was sent to the country on April 19th, 1918, in charge of another laboratory worker, but it did not get the same amount of exercise as the other four, and was returned on May 2nd, looking thin, after which it was allowed to run about in the laboratory. When killed at 13 weeks old the body was well nourished. X-ray and post-mortem examination failed to show definite signs of rickets at the ends of the bones, although the femur was soft and split easily with a knife.

Of the second litter a dog and a bitch were sent to the country on April 18th, 1918, at 5½ weeks old, and were kept under the same conditions as the first litters till June 3rd, when they were returned to the laboratory free of rickets and killed at the age of 11 weeks. Both sets of checks were fed upon oatmeal porridge and skimmed milk, the average amount consumed by each pup per day being: Oatmeal 150 grams, milk (skimmed) 280 c.cm. Taking the fat content of skim milk at even 1 per cent.—it is generally given at 0.3 per cent.—this gives 2.8 grams of fat per day.

B. Pups Kept in the Laboratory.

The remaining five of the first litter were kept in the laboratory in a large airy room. Four were as far as possible kept confined in a stall about 3 ft. by 3 ft.

One was allowed the run of the room, but being alone it took little exercise.

The food consisted of oatmeal porridge and whole milk—oatmeal 175 grams, milk (whole) 100 c.cm.—till May 21st, when.

as the pups were larger, 200 c.cm. were given. Taking 3 per cent. as the average amount of fat in milk, this is equivalent to 5 grams per day, and later to 6 grams.

During life the wrist-joints were examined and photographed by x rays, and careful necropsies were made after killing with chloroform and bleeding. All showed marked rachitic changes, and the femur split easily with a knife. (Special plate, Fig. B.)

The remaining seven of the second litter were allowed to run about in the laboratory till May 21st. One died on May 6th. After May 21st three were confined in a small stall, while the other three had the run of the room. Those in the stall were constantly trying to get out and took almost as much exercise as the others.

Ten grams of butter were given daily, from May 9th, to one of those confined and to two of those running about. One of the latter died on June 3rd, and showed signs of rickets. The others had the same food less the butter. The food was the same as that of the first litter.

Those without butter were getting about 3 grams of fat at first, then 6 grams of fat, while those with butter during the latter period were getting, say, 6 grams plus 8.5 = 14.5 milk fat per day, equivalent to about 450 c.cm. of milk. As was to be expected, they showed a somewhat more rapid growth than the others. All were killed, along with the checks, on May 15th at the age of 11 weeks. They were examined and photographed by x rays, and careful *post-mortem* examinations were made.

All those kept in the laboratory showed signs of rickets to a greater or less degree. One which had been confined and had had butter (Dog 2) was most markedly affected. It was unable to walk. Another of the confined animals which had had no butter was least affected. No difference could be detected between the others. All showed marked splaying of the fore feet, distinct rachitic changes of the wrist-joints, and softening of the femur, so that the bone cut easily with a knife.

LITTER I.
Weight in kilos.

In Country.				In Laboratory.			
				Confined.			Running Free.
Date.	D2.	B1.	B2.	D3.	B5.	B3.	B4.
1918.							
April 1 ...	2.19	1.75	1.65	1.77	1.90	1.65	2.53
" 8 ...	2.15	1.85	1.77	1.80	2.00	1.77	2.45
" 15 ...	—	—	1.75	2.32	2.15	2.00	2.72
" 22 ...	1.90	1.90	—	2.25	2.30	2.00	2.95
" 29 ...	2.78	2.90	—	2.46	2.27	2.20	3.00
May 6 ...	—	—	2.30	—	—	—	—
" 13 ...	4.90	3.95	2.40	2.80	2.60	2.40	3.15
Gain: Total ...	1.90	2.20	0.75	1.03	0.70	0.75	0.62
" Per week (average)	0.31	0.36	0.12	0.17	0.12	0.12	0.10

D2 and B1 were sent to the country on April 12th, 1918, and were returned to the laboratory on May 12th. B2 was sent to the country on April 19th, and was returned on May 2nd.

LITTER II.
Weight in kilos.

In Country.			In Laboratory.						
			Confined.				Running Free.		
Date.	D1.	B2.	B1.	B4.	B5.	D2.*	B3.*	B6.*	B7.
1918.									
April 1 ...	0.88	1.17	0.68	0.93	0.70	0.87	0.80	0.66	0.77
" 8 ...	1.00	1.30	0.72	1.04	0.71	0.97	0.84	1.00	0.90
" 22 ...	—	—	1.07	1.42	1.00	1.45	1.18	1.45	1.42
" 29 ...	—	—	1.23	1.62	1.07	1.56	1.34	1.6	1.55
May 13 ...	2.55	2.85	1.37	1.82	Died	1.85	1.25	1.70	1.70
" 21 ...	—	—	1.60	2.20	—	1.85	1.40	2.20	1.92
" 27 ...	—	—	1.75	2.20	—	2.10	1.30	2.15	1.95
June 5 ...	4.00	4.05	1.95	2.35	—	2.65	Died	2.50	2.15
Gain: Total ...	3.12	2.88	1.27	1.42	—	1.78	—	1.84	1.38
" Per week (average)	0.35	0.32	0.14	0.16	—	0.20	—	0.20	0.15

* These three animals were given 10 grams of butter daily from May 9th.

D1 and B2 were sent to the country on April 18th, and were returned to the laboratory on June 3rd.

The pups, both those sent to the country and those retained in the laboratory, were weighed at regular intervals. (See accompanying tables.) It will be seen that the onset of rickets is not associated with periods of active growth.

Conclusions.

Pups kept in the country and freely exercised in the open air, although they had actually a smaller amount of milk fat than those kept in the laboratory, remained free of rickets, while the animals kept in the laboratory all became rickety.

The observations seem to show that some other factor than diet is the prime cause of rickets in dogs, and afford no evidence that milk fat (butter) contains any accessory factor protecting against the development of rickets.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, July 4th, 1908, p. 13. ² *Journ. of Path. and Bact.*, 1916, xxi, p. 1. ³ *Ergebnisse der Physiologie*, Dreizehnter Jahrgang, 1913, p. 197. ⁴ *Journ. Amer. Med. Assoc.*, 1917, vol. xxix, p. 1533. ⁵ Medical Research Committee Special Report, Series No. 20, 1918.

VANGHETTI'S OPERATION.

[WITH SPECIAL PLATE.]

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In the recent article dealing with the utilization of stump muscles as advocated by Vanghetti¹ the writer rightly finds fault with Sauerbruch's method of providing the loop motor, for this is all tunnelization amounts to, on the score of "difficulties in maintaining the nutrition of the long flap (in which the lines of blood and nerve supply are largely cut across) and in bringing together the edges of the gap caused by its removal without dangerous tension and inconvenient traction upon neighbouring parts."

During the last eight to nine months I have, when occasion arose, been making use of Sauerbruch's method with more or less success, getting over the above difficulties by—

1. Cutting the flap in the long axis of the limb of ample width (that is, not less than one-third of its length) and with a good lining of subcutaneous tissue.

2. Avoidance of pressure on the skin tube by leaving in it at the time of operation nothing larger than a probe, and by making the muscle canal of ample capacity.

3. Leaving a layer of subcutaneous tissue on the raw surfaces from which the flap is lifted, and covering in with a Thiersch's graft instead of drawing the edges together.

Even so, however, the range of movement is poor owing to another and fundamental defect which underlies the whole method. On referring to Fig. 8 in the article mentioned and to the majority of the illustrations in Sauerbruch's monograph it will be seen that the tissues tunnelized are not only muscle and skin, but fascial sheaths of muscle as well.

Diagram showing tunnelization of biceps in face of stump during amputation; biceps tunnelled at C, H, Humerus; A, anterior flap split at E and punctured at D; P, posterior flap; S, skin tube fashioned; S', ditto *in situ*.

The continued traction of the working muscle will very soon mobilize skin if free from scar tissue, but on the other hand, fixation of the skin tube to fascial sheath will more or less effectively anchor it and largely defeat the object of the operation. Still, to give the Devil, and even the Hun, his due, the following brief note shows that tunnelization of muscles with a skin tube is likely at times to prove useful in Vanghetti's operation.

I recently had occasion to amputate above the elbow, and was fortunately able to save the whole of the biceps muscle and an ample anterior flap of skin. The musculotendinous stump of the biceps was isolated and tunnelled half an inch from its end (see diagram). The anterior flap

Bent Back of Five Months' Duration. A, Before treatment. B, Under treatment with "back-board." C, After treatment.

MAJOR W. F. BROOK: VANGHETTI'S OPERATION.

FIG. 1.

FIG. 2.

FIG. 3.

D. NOËL PATON, L. FINDLAY, A. WATSON:
THE CAUSE OF RICKETS.

A

B

X-ray photographs of wrist-joints of A, pup kept in the country and free of rickets, and B, kept in laboratory and markedly rachitic.