delivered normally. You will be impressed with the seriousness of the condition when I tell you that the methods practised in three of her deliveries by skilled obstetricians consisted of craniotomy, induction, and, last of all, Caesarean section. In the one confinement in which I attended her I was able, fortunately, to deliver her by more natural means, having diagnosed the state of inertia when first she sought my advice in the last month of gestation.

I have just said that typical uncomplicated primary uterine inertia is due to the insufficiency of pressor substances in the blood. We do not know the biochemical mechanism that precipitates labour, but we do know that the rhythmical uterine contractions are stimulated by pressor bodies; consequently we should be able easily to discover the future possibility of primary uterine inertia before the onset of labour, and without difficulty to prevent this abnormal occurrence.

As a matter of fact this is possible, if the patient be examined during the last two or three weeks of gestation. Normally, at this period the uterus is very sensitive and the muscle contracts readily on simple stimulation, such as that produced by gentle kneading with the hand. If the uterus remain flaccid and tend to sag at the sides without proper central protuberance, there is every likelihood that, unless the condition be remedied, labour will be inert. Confirmation of the condition of primary inertia may be obtained by an estimation of the systolic blood pressure, which should be higher than normal during the last weeks of pregnancy. Usually the pressure at this time is equal to that of 140 mm. Hg. If then the blood pressure be found to register 110 mm. Hg or less, there is almost certainly some insufficiency of pressor substances, and this must be made good.

Normally, the amount of the ionized calcium in the blood tends to rise at the end of pregnancy, owing to the lessened demands of the foetus, and these salts greatly influence the tone of involuntary muscle fibres. It is necessary, therefore, in these cases to supply calcium in an absorbable form. Many persons do not assimilate calcium salts well, but I think pregnant women, on whom the calls in respect of this substance are great, are easily treated. Many years ago I had the preparation known as *mistura calcii lactatis recentis*—pure concentrated lactic acid (200 grains), precipitated calcium carbonate (75 grains), and chloroform water (3 viij)—prepared, and this is now widely and advantageously used. About two ounces of the mixture should be given every night. In addition to this, it is advisable to inject intramuscularly 0.5 c.cm. of infundibulin every night and morning for a fortnight before parturition is due. If proper ante-parturitional investigations be made and this treatment be adopted, I think that one of the commonest and greatest sources of trouble to the practitioner would be banished. I may add that I treated the lady, just mentioned as having suffered so much with primary uterine inertia and from drastic treatment, on the principles enunciated, and she brought forth a living child weighing nearly ten pounds after a labour of a very few hours' duration.

All that I have said to-day is commonplace knowledge. I have discussed a few examples out of many not uncommon local and general conditions in the case both of the child and the mother. But I do not think the facts I have mentioned always occupy an important place in the mind of the practitioner, as, indeed, they should; for I am inclined to think that in practically all cases of forceps delivery—whether rightly or wrongly performed, excepting those of immoral expediency—one of the conditions I have described is present. There are, of course, other factors, such as a rigid muscular floor, occasionally concerned, but these are of less importance. The chief lesson I wish to convey is that all are recognizable before labour commences, and that no one who has had a previous opportunity of examining his patient should be confronted unexpectedly with serious difficulty after labour has commenced. In such circumstances the wrong treatment may be applied—perhaps necessarily applied—with inevitable danger to the life of the child and the possibility of serious maternal laceration and infection, which demand such toll and produce so much physical incapacity in women of the present day.

EXPERIMENTAL RICKETS IN RATS.

BY

V. KORENCHEVSKY, M.D.

(From the Department of Experimental Pathology, Lister Institute.)*

Historical.

THE earliest attempts to produce rickets experimentally in animals were by administering food deficient in calcium (E. Voit, Baginsky, Aron and Sebauner, Götting, Miva and Stoelzner, Dibbelt and others), or in phosphates (Heubner). The view that rickets is an infectious disease receives some support from the experiments of Morpurgo, who isolated a diplococcus from spontaneous outbreaks in rats, and found that rickets ensued after he injected cultures into the animals, provided the inoculations were made at a sufficiently early age. Confinement and limitation of muscular exercise as a possible etiological factor in rickets has been studied by Findlay, Paton, and Watson, and is considered of importance by them.

The dietetic theory has recently been revived by Mellanby, whose investigations indicate that those constituents of a diet which are particularly rich in fat-soluble vitamin A possess a profound importance in connexion with the occurrence of rickets in puppies. Mellanby's conclusion is to some extent supported by the experiments of McCollum, Simmons and Parsons, Shipley and Park; but the American observers, who made their observations upon rats, found that changes more closely resembling rickets occurred in these animals when their diet, in addition to being deficient in vitamin A, was also poor in calcium salts or phosphates. Hess and McCann and Pappenheimer, who also experimented on rats, found that a diet deficient in vitamin A did not produce rickets but lack of active osteogenesis.

Lastly, different abnormalities in the function of endocrine glands have been connected with the etiology of rickets (Erdheim, Stoelzner, and others), important experiments having been conducted on this question by Erdheim.

The theories deduced from all these investigations have numerous adherents and numerous opponents.

The Present Investigation.

My own experiments were started at the end of November, 1920—that is, before the works of the American authors were published. A complete description of all the chemical and histological observations, illustrated by microphotographs and accompanied by a complete critical review of the literature on the question, is being prepared for publication. In this preliminary communication I propose to give a brief account of my experiments, and to summarize the main conclusions which I arrive at.

The experiments were conducted on rats born at the Lister Institute from stocks that had been all the time under observation. With few exceptions the rats used for separate experiments belonged to one litter.

The basal diet possessed the following composition:

Purified casein	...	...	...	...	18 grams.
Starch	...	...	...	...	52 "
Fat	...	...	...	...	15 "
Marmite	...	...	...	...	5 "
Orange juice	...	...	...	...	5 c.cm.
Distilled water	...	...	...	...	50 "

This mixture contained about 0.041 per cent. Ca and 0.22 per cent. P. In the food mixture, intended to be rich in vitamin A, a mixture of 12.5 grams of butter *plus* 2.5 grams of cod-liver oil was used as fat. For food deficient in vitamin A cottonseed oil was used.

To this basal diet was added 5 grams of saline mixture. The one used for food rich in calcium had the following composition, as recommended by the American investigators:

(N 1) Sodium chloride	...	...	...	...	5.19 grams
Magnesium sulphate	...	...	...	...	16.4 "
Sodium acid phosphate	...	...	...	...	10.41 "
Potassium phosphate	...	...	...	...	28.62 "
Calcium phosphate	...	...	...	...	16.20 "
Calcium lactate	...	...	...	...	39.0 "
Ferric citrate	...	...	...	...	3.54 "

To the diets intended to be deficient in calcium a saline mixture of the following composition was used:

(N 2) Sodium chloride	...	...	...	...	5.19 grams
Magnesium sulphate	...	...	...	...	16.4 "
Sodium phosphate	...	...	...	...	27.91 "
Potassium phosphate	...	...	...	...	28.62 "
Ferric citrate	...	...	...	...	3.54 "
Sodium bicarbonate	...	...	...	...	21.2 "

* This research was supported by a grant from the Medical Research Council.

Ten to twenty grams of food mixture were supplied to each animal according to age. In the later stages of the experiments with deficient diet this amount was not always wholly consumed as the appetite of the animals diminished.

In the text following we shall denote the diets by the following letters:

N=Normal mixture; N-Ca=normal mixture deficient in calcium; -A=diet deficient in vitamin A; -A-Ca=diet deficient in vitamin A and calcium.

Altogether twenty-six families of rats, comprising 183 animals, were used for the experiments. Histological examination of the ribs, humerus, and radius was carried out in every case, and in 109 animals the calcium content of the bones of the leg was determined. For histological examination the bones were incompletely decalcified in Müller's fluid, or, in important cases, undecalcified bones were cut. In the first case haematoxylin-eosin staining was employed; in the second the material was treated with AgNO_3 and then stained with alum carmine and eosin.

The Significance of Confinement and Lack of Exercise.—Observations were made upon 28 rats which served as controls to the various series of experiments mentioned below. The animals lived two or three together in cages 30 cm. in diameter and 15 cm. high. I have never observed any indication of rickets, nor was there any variation of the calcium content of the bones outside the normal limits for animals of the same age.

The Significance of Certain Intestinal Bacteria.—In human rickets various disturbances of the digestive processes frequently occur, and it has been supposed (Vierordt and others) that these disturbances have some bearing on the etiology of the disease. In the rats kept on -A, -A-Ca, or N-Ca diets digestive disturbances, accompanied by more or less abundant diarrhoea, were frequently observed. In view of Morpurgo's experiments on the infective nature of rickets, it seemed possible that autointoxication with toxins produced by intestinal microbes might be of significance in the mechanism of origin of rickets. Up to the present time I have conducted only one experiment on 10 rats kept on diets N-Ca and -A-Ca. To the food mixture of 5 of these rats was added a meat culture of *B. sporogenes*, *B. bifementans*, and *B. welchii*. Two of these rats received in addition, at intervals, 1 to 2 c.cm. of a live culture of *B. sporogenes* or *B. bifementans* subcutaneously. These experiments, however, gave no indications regarding the influence of these intestinal microbes on the production of rickets.

INFLUENCE OF A DIET DEFICIENT IN EITHER CALCIUM, VITAMIN A, OR BOTH, UPON THE BONES.

General Considerations.

Before passing to the results obtained from N-Ca, diet as well as from -A and -A-Ca, I shall discuss some theoretical considerations which led me to expect certain peculiarities in the rickets developed on the above diets. As is known, animals kept on the diets named cease to grow, and after a variable interval profound cachexia develops.

It is current opinion that in infantile rickets continuous growth and a sufficiently well nourished condition of the child are essential for the development of the typical rachitic changes in the skeleton (Heubner, Fischl, Stoelzner, Pfaunder, Schlossman, Schmorl, and others). Heubner and Fischl, for instance, declare that ill-nourished, slowly growing children never become rachitic; and, according to Schmorl, the characteristic pathologico-anatomical changes in the skeleton are always less developed in weak, ill-nourished children.

In the study of human rickets, attention has naturally been devoted more to the pathological changes in the bones than to the preceding disturbance of metabolism. Rachitis I regard as a disease existing in the child in a cryptic form before the changes in the skeleton evoked by it take place. I believe that the condition may be already present in children who are emaciated and do not grow, although obvious rachitic changes in the bones begin to develop only from the moment when the organism starts growing.

Looser's investigation on "rachitis tarda" in patients whose growth was retarded and nourishment frequently poor is an important contribution to the question of rickets viewed from the above standpoint.

This excellent investigation has shown that in such patients, as well as the usual manifestations of rickets, atrophy of the bones occurs. The occurrence of osteoporosis is, however, not infrequently referred to as an

argument against the diagnosis of rickets (Schmorl). The amount of osteoid tissue and the development of subchondral spongiosa appeared to be small in the cases investigated histologically by Looser. The development of periosteal osteophytes was not always observed, and, when observed, was moderate in amount. On the other hand, Ziegler regards osteoporosis as frequently accompanying rickets.

Returning to my own experiments, and taking into consideration that animals kept on diets -A and -A-Ca were in most cases in a state of retarded or inhibited growth and of more or less profoundly disturbed nutrition, one would on *a priori* grounds expect rachitis, if it is developed, to appear more in the form described by Looser.

In the diagnosis of rickets in rats I have been guided by Erdheim's excellent work on spontaneous rickets in these animals.

Whether obvious bony deformities, bending, swelling of epiphyseal junctions, spontaneous fractures, etc., were present or not, I have considered the following conditions to be essential for the diagnosis of this disease:

1. Diminution of calcium content in bones.
2. Presence of osteoid tissue in amounts distinctly exceeding the normal.
3. Enlargement and disorganization of the zone of proliferating cartilage.
4. Absence or defect of deposition of lime salts in the zone of provisional calcification.
5. Presence of periosteal osteoid tissue.
6. Absence or marked deficiency of calcareous deposition in callus after spontaneous fractures of bones.

The presence of osteoporosis in the conditions named above, as observed in a certain number of our animals, does not prevent the diagnosis of rachitis.

Effect of Feeding on the Basal Diet but without Calcium in the Salt Mixture.

The amount of calcium in the basal ration was 0.04 per cent., so that, assuming the animals consumed the whole ration, the maximum amount of calcium ingested daily was approximately 8 mg. Two types of experiments were conducted upon 13 animals in all. In the first, consisting of 9 rats, the deficient diet commenced when they were about 30 to 50 grams in weight. The experimental animals remained behind the control ones in weight, but not to such a degree as on -A diet. The rats also manifested nervous excitability and shyness.

The changes in the skeleton agree in general with those described by previous authors, and are characterized by osteoporosis with narrow layers of osteoid tissue. The zone of proliferating cartilage is but slightly changed and nearly always well calcified. Osteoporosis observed in such cases finds explanation in the increased number of osteoclasts. Spontaneous fractures are very rarely observed. The amount of calcium in the dried bones, however, was about 43 per cent. below normal, and the amount of H_2O was 23 per cent. above normal. The picture that resulted cannot be defined as rickets.

The results were quite different when the young rats kept on N-Ca diet originated from a mother who herself had been kept on this diet during the lactation period. Hitherto I have conducted only one experiment on 4 young rats. These all showed the same changes. The bony deformities were pronounced, and to the naked eye closely resembled those of human rickets. The amount of calcium decreased on the average to 64 per cent. below the normal and the amount of H_2O rose to 44-55 per cent. above the normal—that is, the calcium in the skeleton was greatly diminished. Microscopically, the enlargement of the zone of proliferating cartilage was more obvious—in one case to the extent of 20-27 layers of hypertrophied cells. This zone either contained no calcium at all or it appeared in the form of separate patches or bands, usually near the perichondrium. The ingrowth of vessels into the cartilage, when observed, was not deep, and the line of the costochondral junction was not markedly altered, and bent towards the bone marrow.

The spongiosa consisted of numerous trabeculae which formed a network and were thin in some rats and thicker in others. The layers of osteoid tissue were of different thickness, thin ones predominating. The cortical bone was in a state of osteoporosis. The periosteum was in places hypertrophied. The osteoblasts were in many places well defined and numerous, sometimes being arranged in several rows. The number of osteoclasts was also very great. Similar pictures in pups have been described by Dibbelt, and by Schmorl.

When there is much new osseous formation one notices that calcification has occurred over considerable areas also to a different degree, notwithstanding the fact that the calcium content of the bones was 64 per cent. below normal. These facts produce the impression that the incomplete calcification of the bone is due to calcium starvation.

In human rickets in remission calcium is as easily deposited as normally in the osteoid tissue (Schmorl), but during the process of the disease this is prevented by some cause. This difference I consider to be *essential and of great importance*.

Effect of Feeding upon the Basal Diet with the Complete Salt Mixture in which the Butter and Cod-liver Oil were replaced by an Equivalent Amount of Cotton-seed Oil.

The amount of vitamin A in this diet must have been very small.

The experiments arrange themselves naturally into three groups. In the first, the diet commenced about the age of 30 to 50 days; in the second, the mother received the deficient diet during lactation; and in the third, the mother and the father were subjected to the same dietary deficiencies before conception. In both the latter groups the same deficient diet was fed to the offspring as soon as they were capable of feeding themselves. Twenty-three animals were placed on this diet at the age of 30 to 50 days. In most cachexia developed earlier or later, and the rats became very susceptible to casual infections.

The changes occurring in the skeleton of many such cachectic rats can be characterized briefly as osteoporosis with a lack of signs of active osteogenesis and frequently increased activity of osteoclasts. The calcium content of such bones is not greatly decreased, being about 14 per cent. less than normal.

In those rats in which growth occurred, and in which the bones were examined before cachexia ensued, less osteoporosis, more osteoid tissue, and a small increase of the proliferating cartilage, were observed, and sometimes defective deposition of calcium in the zone of provisional calcification. Corresponding to this appearance the calcium content of the bones was 21 per cent. below normal. In a few cases the appearance was suggestive of slight rickets.

In one experiment a rat, after giving birth to six young, was placed on -A diet during the whole period of lactation of the young rats. The young rats, after having been separated from the mother at the expiration of a month, remained on -A diet till the time when they were killed. The chemical composition of the bones of these rats showed further deterioration, the calcium being 36 to 44 per cent. below normal, and the water 17 to 50 per cent. above normal. In accordance with this low calcium content, numerous spontaneous fractures were observed in the ribs of the young rats. However, microscopic examination revealed the picture of osteoporosis typical for -A, and in four cases osteoid tissue was observed. The thickness of the osteoid was equal to, or even somewhat exceeded, that of the calcified bone in three cases.

As is known, it is a rule that rats kept on -A diet have no offspring, and therefore the males are usually left together with the females. As an exception, however, they do bear young. This was recently observed amongst my rats in one case and amongst the rats of Dr. Zilva at the Lister Institute in three cases. I availed myself of this material in order to determine whether the feeding of parents on -A diet during the periods of conception, pregnancy, and lactation has any deteriorating effect on the condition of the skeleton in their offspring. After separation from the mother the offspring continued to be kept on -A diet. Of these, numbering 18 altogether, I have examined up to the present 12, the offspring of three mothers; 12 were examined histologically, and the skeleton of 7 of these was examined chemically. The investigation was conducted sixty-four to seventy-nine days after birth. In 7 of these 12 rats great proliferation of the cartilage cells and growing-in of blood vessels, and a picture more or less suggestive of rickets, was observed, and in 2 cases the histology was typical of rickets. The average calcium content in the whole series was 25 per cent. below normal and the water 30 per cent. above normal.

To sum up: As the result of feeding the mother during lactation both on the N -Ca diet (4 rats) and on the -A diet (6 rats) the composition of the skeleton in the offspring deteriorated sharply, but changes closely resembling rickets were obtained in an appreciable number of cases only when feeding on -A diet had been started during conception and pregnancy. In other words, if these experiments were confirmed repeatedly on a large number of animals, they would point to the significance of the

condition of the parents, especially the mother, in the origin of the profound changes in the skeleton, in certain particular cases on -A diet even amounting to typical rickets. The supposition frequently advanced regarding the importance of normal nutrition of the parents at the period of conception, and of the mother during pregnancy and lactation, find experimental confirmation in our investigations as well. From this point of view the commencement of rachitic changes in the child could be sought for, not only in the conditions of its own life but also in the state of health—that is, nutrition—of its mother, and maybe of its father as well.

Vitamin A is apparently closely related to the metabolism of calcium in the organism, and particularly in the bone. The experiments of Schabad, Mellanby, Shipley, Park, McCollum, Simmonds, and Parsons, and those related above, support this view. Its action may not inappropriately be compared with that of an amboceptor, provided the analogy be not pressed too far.

Effect of Feeding upon the Basal Diet and Salt Mixture from which both Calcium and Vitamin A have been largely Removed.

This was investigated upon 27 rats. With the exception of eight, the deficient dietary commenced when the animals were three weeks to one month old. In eight cases (two families) deficient diet was given to the mother during lactation also.

The results confirm those of McCollum and his collaborators upon this point. Combined deficiency in calcium and vitamin A in the food has, in my experience, proved the most certain method to produce rickets experimentally in rats.

Rats kept on this diet manifested deformities of the skeleton which are both macroscopically and microscopically typical of rickets, provided that feeding commenced at a sufficiently early age. The amount of calcium in the bones decreased to about 46 per cent. below normal, and the amount of water increased to about 32 to 46 per cent. above normal, whilst the majority presented all the pathologic manifestations of rickets in a pronounced form; in some rats, kept on -A -Ca diet, the amount of osteoid tissue, although abnormally increased, was comparatively small. In these cases osteoporosis was especially marked, and calcium was not infrequently deposited in the zone of provisional calcification.

In animals fed on the basal diet without the saline mixture the resulting changes did not differ from those produced by deprivation of calcium alone.

Effect of Exclusion of Phosphates from the Salt Mixture.

Deprivation from phosphates in conjunction with fat-soluble deficiency was found by Shipley and Park and McCollum and Simmonds to be followed by changes indistinguishable from those of rickets. In three experiments, comprising 14 rats kept upon N and -A diets respectively, I have removed the phosphates from the salt mixture, at the same time compensating for the loss of sodium and potassium and calcium. As compared with the corresponding control animals, no special changes were observed in the bones. This apparent discrepancy is no doubt attributable to difference in phosphorus content in the basal diets used.

Effect of Castration.

Forty-five rats were castrated, seven of which were deprived both of sexual glands and spleen. When subsequently placed upon the four diets (N, N -Ca, -A, and -A -Ca) no marked influence on the chemical and histological composition of the skeleton as compared with controls was observed. My experiments did not reveal that beneficial effect which is sometimes clinically observed in man in developed osteomalacia. In some of the castrated rats even greater changes were observed in the skeleton, as compared with the corresponding control animals—for example, on -A diet. Therefore one can only state that castration performed before subjecting the animals to the special diet did not prevent the development of rachitic changes in the skeleton.

Conclusions.

1. The results obtained by me agree in general with the results of the experiments of Mellanby, McCollum, Simmonds, Parsons, Shipley, and Park.
2. Confinement in small cages does not evoke rickets in rats.
3. The introduction of live cultures of *B. perfringens*, *B. sporogenes*, and *B. bifermentans* with the food, and

of *B. sporogenes* and *B. bifementans* subcutaneously, produced no visible effect on the development of rickets in rats.

4. The deficiency of the diet in calcium alone can produce changes in the skeleton of rats which present some resemblances with rickets, especially when the young rats have originated from a mother kept on the same diet during lactation.

5. Usually deficiency of food in vitamin A produces in rats impoverishment of the bones in calcium, enrichment in water, and osteoporosis with deficient osteogenesis, and in some cases a picture resembling slight rickets. Changes in the skeleton more similar to rickets, and in some cases typical of rickets, were observed in young rats on —A diet, provided their parents had been fed on —A diet during conception, pregnancy, and lactation.

6. Vitamin A has a relation to the metabolism of calcium in the organism and particularly in the bones, and therefore to the development of rickets.

7. My few experiments on the feeding of parents on food deficient in vitamin A or calcium during conception, pregnancy, and lactation suggest that this may start disturbances of metabolism in the child which, if the deficient dietary be continued after birth, result in serious disorders of the skeleton.

8. The changes typical of rickets occur most readily and most frequently in rats kept on a diet deficient both in vitamin A and calcium.

9. Castration performed before the commencement of feeding has no marked influence on the chemical and histological changes in the skeleton of rats fed on a normal diet, on diets deficient in calcium or vitamin A, or deficient in both.

In conclusion, it is my pleasant duty to record my gratitude to the Medical Research Council for affording me a grant which has enabled me to carry out this investigation, and to the Lister Institute for the hospitality of its laboratories. I am also deeply grateful to Professor C. J. Martin for his continuous sympathy and support. To Dr. R. Robison I express my thanks for advice connected with the chemical investigations; to Miss E. Luce for assistance in operations; to Miss S. Rutherford for help in the work of feeding the animals.

BIBLIOGRAPHY.

- Aron und Sebaauer: *Bioch. Zeit.*, 1908, viii, S. 1.
Baginsky: *Virchow's Archiv*, 1882, 87, S. 301.
Dibbelt, cited by Schmori.
Erdheim: *Frankf. Zeit. f. Path.*, 1911, 7, SS. 175, 238, und 295.
Rachitis und Epithelkörperchen, Wien, 1914.
Findlay, Paton, and Watson: *BRITISH MEDICAL JOURNAL*, 1908, ii, p. 13; 1918, ii, p. 625; 1921, i, p. 594.
Götting: *Virchow's Archiv*, 1909, 197, S. 1.
Heubner: *Lehrbuch der Kinderheilkunde*, 1906.
Hess, McCann, and Pappenheimer: *Journ. Biol. Chem.*, 1921, 47, p. 395.
Looser: *Deutsch. Zeit. f. Chir.*, 1920, 152, S. 210.
McCollum and Co-workers: *Journ. Biol. Chem.*, 1921, 45, pp. 333 and 343.
Johns Hopkins Hosp. Bull., 1921, 32, p. 160.
Mellanby: *Lancet*, 1919, i, p. 407; 1920, i, p. 604.
Miwa and Stoelzner: *Zeitler's Beitr.*, 1898, 24, S. 578.
Morpurgo: *Zeitler's Beitr.*, 1900, 28, S. 620. *Centralbl. f. Allg. Path.*, 1902, 13, p. 113.
Pfaundler: *Lehrbuch der Kinderheilkunde*, Jena, 1914.
Pommer: *Untersuch. über Rachitis und Osteomal.*, Leipzig, 1885.
Schabady: *Zeit. f. klin. Med.*, 1909, 67, S. 454; 1910, 69, S. 435.
Schmori: *Deut. Arch. klin. Med.*, 1906, 85, S. 170; *Ergebn. d. inner Med. und Kinderheilk.*, 1909, 5, S. 403.
Stoelzner: *Pathologie und Therapie der Rachitis*, Berlin, 1904.
Tozer: *Journ. of Path. and Bact.*, 1921, 25, p. 306.
Vierordt: *Rachitis und Osteomalacia*, Nothnagel's Spec. Path. u. Ther., Wien, 1896.
Voit: *Zeit. f. Biol.*, 1880, 16, S. 55.

THE SITE OF OPERATION FOR EMPYEMA.

BY

DUNCAN C. L. FITZWILLIAMS, C.M.G., M.D., F.R.C.S.,
SURGEON IN CHARGE OF OUT-PATIENTS AND LECTURER ON CLINICAL
SURGERY, ST. MARY'S HOSPITAL.

THE best method of treating empyemata has always been, and probably always will be, discussed from time to time in the medical press. As a surgeon attached to a general, a children's, and a consumption hospital, consulting surgeon to a second consumption hospital, as well as having seen over five years of war, perhaps I might be allowed to express my views. In the first place, there is a sharp distinction to be drawn between the cases seen as the result of warfare and those which develop as the result of disease. There is nothing in civil practice which can reproduce what was seen during the war. Even stabs or bullet wounds in civil life are treated so soon and so

thoroughly that the sepsis we saw during the war is not seen in these cases. The methods of dealing, therefore, with war cases do, and should, differ from the methods we use in civil life, and anyone who draws on his war experience too largely will be apt to err if he applies his ideas to ordinary cases. I shall only deal here with ordinary cases. It has been my fortune twice in my life to see the condition known to our fathers as empyema necessitatis—that is to say, an empyema which had not been opened but which was bursting by itself. It was interesting to note that in both cases it was bursting between the second and third ribs in front. Both cases were in children.

In dealing with a large abscess cavity with only one movable wall—namely, the lung—it is obvious that the drainage must be so arranged as to allow the lung to come up to the other walls gradually and evenly, and so obliterate the cavity. Opening at "the most dependent attainable part of the cavity" is the best way to defeat this object. The lowest and the highest points will be those where the lung on expanding will first touch the chest wall, and when that happens drainage from these points must be embarrassed. The same thing applies when the opening is either too far back or too far forward. The opening should be opposite, roughly, the deepest part of the cavity, and at that place which will be closed last. Where this place is, is not always easy to say, especially in purely local collections. The diagrams perhaps illustrate my meaning best.

One always teaches that the seventh rib, in front of the posterior axillary line, is the best rib to resect, but there should be no objection to coming forward to the mid-axillary, or even to the anterior axillary line, if the same level were maintained—that is to say, resecting the sixth, fifth, or even the fourth rib, as one came further and further forward.

It is the ninth rib we resect in cases of abscess or hydatid of the liver on the right side and subphrenic abscess on the left side. The ninth rib is wholly below

the normal lung level in front of the posterior axillary line, and the eighth rib is below that level in front of the mid-axillary line. Only right up behind, near the erector spinae, would it be of any use resecting this rib, and then only in such a position

that the expanding lung would soon render the opening useless. In spite of the authority lent to this teaching, the resection of the ninth rib for empyemata has had a very limited vogue.

In many cases the pouch of pleura between the chest wall and the diaphragm becomes obliterated below the lung level, and after resection of the ninth rib the diaphragm may be opened under the impression that it is the thickened pleura. I once saw Mr. Keetley do this, and the medical registrar, to whom he had not been very polite over the case, acidly remarked that he had wished the thoracic and not the peritoneal cavity explored.

Small operations for necrosed pieces of ribs will always be needed. The maintenance of big unhealed cavities inside the chest wall is due to certain very well known

FIG. 1.—Longitudinal sections of the chest, showing lower part of cavity obliterated by expanding lung.

FIG. 2.—Cross-sections of the chest, showing posterior part of cavity obliterated by expanding lung.