Fig. 6, in which the dark bands are collecting tubules. For the sake of clearness the vessels have been blocked out of this and the subsequent figure. These granular cells can be seen emigrating from surrounding capillaries through the walls of the collecting tubules. The renal blood vessels contained a marked excess of these blood constituents. This condition of the collecting tubules is also indicated in Fig. 7, which shows in addition the following features:

(a) The presence of granular leucocytes lying free in the intertubular tissue.

(b) An atrophy of the epithelial lining of the convoluted tubules, doubtless due to pressure of the leucocyte exudation.

(c) Several convoluted tubules which appear perfectly normal.

A transverse section of the ureter in the same section from which Fig. 6 was taken shows that the leucocytes had undergone marked degeneration in the course of their downward journey, very few leucocytes being present as such in the ureter.

The gouty deposit in the tissues, the changes in the synovia, the widespread thromboses, and the areas of necroses in the kidney clearly indicate the nature of the disease. It is a typical case of gout. It is furthermore a case of acute gout which had proceeded to its natural termination. The fact that early indications of pericarditis were present might suggest the opinion that the animal, undoubtedly the subject of gout, succumbed to some terminal infection not immediately related to the gouty disorder. The clinical history and pathological appearances alike fail to substantiate this view. In this connexion reference may be made to the:

1. Absence of any alteration in the joint structures other than in the synovia, the nutritive fluid of the joint. There was no gouty arthritis nor other indication of chronic disease.

2. The absence of any cirrhotic changes in the kidney and fibrous overgrowth in relation to the deposits in the extra-articular tissues such as we would expect in chronic disease.

3. The blood vessels of the animal were singularly healthy, which in all probability would not have been in a case of chronic disease that had succumbed to what is commonly known as a terminal infection.

These negative facts fail to substantiate this hypothetical view as to the existence in this fowl of two or more diseases not etiologically related, and this failure is emphasized by reference to some of the other unusual features of this case, more especially to the condition of the pancreas and collecting tubules of the kidney, both of which present pronounced morbid appearances of a character extremely rare in the history of simple terminal infection. We are therefore driven to the conclusion that we have to deal with a case of pure gout in which the disease had run a natural course, in which an examination of the tissues shows that the *post-mortem* appearances generally are characteristic of an infection by bacteria or their products.

Particular interest centres in the condition of the intestinal tract, pancreas, and kidneys. The condition of the pancreatic duct is a very striking one; it appeared to be an extension from the duodenum. A reference to the figures shows that the lesion would be in its results analogous to the effect of a ligature of the duct. The interesting question is raised as to the existence of any relationship between these appearances and the occurrence of gouty glycosuria in the human subject. The changes in the tubules of the kidney are remarkable. I am not aware that these appearances have been previously described. The alterations in the leucocytes have a special interest in connexion with the changes in the blood in acute polyarticular gout recently described by the author² and confirmed by Bain.³

The main facts learned from this investigation emphasize the necessity of further examination into the comparative pathology of the disease. It is specially important to obtain material sufficiently fresh to allow of an adequate bacteriological investigation. A question that at once presents itself from the foregoing record is, How would the acceptance of the theory of a bacterial origin of the disease harmonize with the known clinical facts of the disorder? The consideration of this point is not relevant to this communication. It may, however, be stated that in my opinion the clinical features of gout—regular or irregular, acute or chronic—are more adequately explained by the light of our present knowledge of infections, relapses, and immunity than by any other theory. The distinctive feature of the infection in gout is that the toxin or toxins have the special property of disturbing nitrogenous metabolism in a manner favourable to the deposit of uric acid in certain tissues.

In conclusion, I would only add that the addition of a bacterial factor to our present conception of the disease would in no way minimize the importance of attention to dietetics in its treatment. It would correct the teaching of some writers whose standard of dietary has special reference

to nuclein-holding foods, and would focus attention on the by-products of deranged digestion of carbohydrates and proteids arising from abnormal bacterial activity in the digestive tract and other tissues.

REFERENCES.

- ¹ *Encyclopaedia Medica*, vol. iv, article Gout. ² *BRITISH MEDICAL JOURNAL*, January 6th, 1900. ³ *Ibid.*, January 31st, 1903.

GLANDULAR FEVER.

By JOHN W. BYERS, M.A., M.D.,

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SINCE the middle of November an epidemic has been prevailing, chiefly among children, in Belfast and in parts of the North of Ireland of a kind which I have not seen before, either in private practice or during the years that I was physician to the Belfast Hospital for Sick Children, and which seems to me to correspond to the condition described in 1889 by Pfeiffer, and termed often by German writers Pfeiffer's *Drüsenfieber*. O. Heubner, Dawson Williams, Comby, Donkin, and J. P. West have also contributed largely to the literature of glandular fever. My knowledge is based on 33 cases, some of which occurred in two public institutions, while the others were seen in consultation.

Age.—The youngest child was 13 months, the oldest case was a female servant, aged 25, in a family where the children were attacked; but, with the exception of two cases (the one mentioned and another aged 21), all were in children or young people under 16 years.

Onset.—As a rule this was sudden.

Period of Incubation.—This was from five to seven days.

Symptoms.—In some of the cases the children went to bed in the evening complaining of slight sore throat. The next morning there was sharp fever (102° to 104° F.), pain on one side of the neck, which was tender on pressure and held stiffly; this was succeeded in another twelve hours by a distinct swelling of the glands under and in front of the sterno-mastoid. In the majority of the cases a similar enlargement appeared involving the deep glands of the corresponding and opposite sides of the neck. In the cases seen by me the right side was as a rule first affected. In another group of those attacked the children complained of nothing until the glands of the neck seemed suddenly to enlarge, and such cases when seen at first looked somewhat like aberrant forms of "mumps." In the severer cases headache was an initial symptom, and in a few sickness and vomiting were present and some abdominal pain, but I failed to detect any objective signs of involvement of the mesenteric glands. In none of the cases was the throat in any way affected beyond some slight congestion, and in the majority it was normal and showed no alteration to explain the slight dysphagia. The glands affected were as a rule the deep set under and in front of the sterno-mastoid, but in others those in the posterior triangles of the neck were involved; no suppuration occurred in any of the cases and the swelling of the glands subsided in most of the cases within a week from the onset of the attack, but in one case the enlargement remained for a fortnight, the fever going up and down in an erratic manner most of that time. The majority of the children were convalescent in a week, but in some of the severe cases those attacked remained poorly and looked anaemic for three to four weeks the glands continuing enlarged. When the disease entered a house nearly all the children were attacked, and in one place two of the female servants, both adults. No rash appeared in any of the cases, nor was there any kidney or other complication, and none suffered from depression except the very severe cases. There were no deaths.

Diagnosis.—When the first case appeared in a house, the adenitis naturally suggested some primary throat mischief, but nothing sufficient to cause the glandular swellings could be discovered on an examination of the throat, either on the tonsils or pharynx, and the presence of other cases in the same house showed that one had to deal with an epidemic fever.

Etiology.—In the *BRITISH MEDICAL JOURNAL* of December 5th, 1903, p. 1492, there is described an "epidemic sore throat and suppurative mammitis in cows," but in none of the cows seen by me were there any such conditions observed in the throat, and the cases were in groups, widely separated, and with no common milk supply. The mild type of cases, when

seen for the first time with a well-marked unilateral swelling of the neck, looked like a form of aberrant mumps, but careful examination showed that the parotid gland was not affected, and the children had no pain in closing the jaws or in eating; besides, some of the cases had previously suffered from mumps. It has been suggested that the disease might be a peculiar form of influenza; but as a rule in recent years, when abnormal types of that protean disorder occurred, we were able to diagnose them as due to the influenza poison from the fact that in the same house or locality other ordinary forms of that disease were detected. None of these usual forms of influenza occurred in the houses where the cases I have described were seen. I believe the cases were true examples of that acute epidemic infectious fever in which there is primary inflammation of the lymphatic glands. The poison of glandular fever is probably microbial, and the present extraordinary atmospheric conditions may present precisely those factors necessary for the growth of the infective germ which causes the disease. Some have suggested that infection is through the intestine; in the cases seen by me, it seems more likely that the invasion was by the throat.

Since writing this article I find that in a family where the children suffered from the ailment I have described, two of the adults developed a follicular tonsillitis, but in them although the glands of the neck were tender there was no palpable enlargement.

NOTES ON THE DISTRIBUTION OF CANCER IN THE FREEBRIDGE LYNN RURAL DISTRICT.*

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THE following notes are an epitome of a special report on the subject recently laid before the above district council.

The district in question consists partly of a chalk plateau covered in places by beds of boulder clay, glacial and post-glacial gravels and sand, and partly of the lower greensand; there is also a considerable area of alluvium. Each parish was visited, and the actual house, or its site if it had been pulled down, in which the patient lived, made out. During the forty-one years 308 deaths from cancer were registered; from these were eliminated those which were obviously due to sarcoma, and they were further corrected for immigration and emigration. As thus amended, 241 were found to have lived upon the various geological formations as given below, for a period of not less than three years preceding their death.

	Deaths.
Glacial and post-glacial gravels and sands	88
Lower greensand	75
Boulder clay	26
Chalk	25
Chalk marl	16
Post-glacial clay	6
Alluvium	5

Of 265 deaths traced to their altitudes, one-half were found to have resided below 50 ft., and less than one-tenth between 100 and 300 ft. above sea level. The disease was more frequent in the western than in the eastern half of the district in the proportion of 3.1 to 1.8 per cent.

Some of the villages have suffered much more than others, and, furthermore, certain parts of some produced more cases than other parts. Especially was this the case in water-logged places where the water supply is derived from surface wells. There was evidence of the disease being more frequent amongst those who used certain wells. For example, in one village in which there were 10 deaths, 4 of them occurred in a small cluster of cottages around one shallow well from which they all drew their water supply; there were nineteen other wells in the parish. In another village there were 3 deaths all using the same surface well; in this parish there were twelve other wells. In another there were 3 cases amongst the users of one well out of a total of 5 cases; there were twenty-three other wells in this parish.

When the yearly deaths of each parish were "charted," in many cases a tendency to fall into groups was observable, a point quite hidden when the whole series of deaths in the

district was treated in a similar way. This falling into little groups or miniature epidemics, when it occurred, was very striking. There were 5 instances of husbands and wives dying from cancer and 2 instances of the first and second wives of the same husband. In 13 instances 2 cancer cases occurred in the same house, in 10 of which both cases died in the house.

Woodland seems to have had no effect on increasing the number of cases, for the six parishes with the most woodland (2,635 acres) averaged 1.0 case per cent., while six other parishes without any woodland averaged 2.4 per cent. Neither had beer drinking any observable effect.

Respecting the geological formations most liable to cancer, it is interesting to note that they are the same in West Norfolk as those which produce most calculus,¹ namely, the gravels and sands, where the percolation of water is rapid, not upon the chalk, the deep wells of which yield water of great organic purity.

REFERENCE.

¹ On the Distribution of Calculous Disease in Norfolk, by C. B. Plowright, BRITISH MEDICAL JOURNAL, November 7th, 1885.

A NOTE ON DR. PRIMROSE'S PAPER ON FILARIASIS.

By SIR PATRICK MANSON, K.C.M.G.,
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IN his interesting article on filariasis,¹ Dr. Primrose, of Toronto University, gives the details of a case of lymph scrotum associated with embryo filariae in the blood. Two months after removal of the scrotum, in which an adult filaria and several fragments were found, the filarial embryos had disappeared from the circulation. Dr. Primrose concludes that the subsequent absence of embryos from the blood was attributable to the removal of the parent worm.

On January 29th, that is twenty-four days after the operation, embryos were still in great abundance in the blood, as many as 1,764 having been found in 1 c.cm. at 9 p.m. Dr. Primrose's assumption, therefore, postulates that the life of an embryo filaria is at least twenty-four days. I take the liberty of dissenting from Dr. Primrose in his interpretation of the facts.

Dr. Primrose states that his patient, forty-six days after the operation, suffered from an attack of elephantoid fever, accompanied by much inflammatory swelling at the root of the neck; the swelling was incised, and a mass of sloughy broken-down tissue was removed. I attribute the disappearance of the filariae from the circulation, not to the operation on January 5th, but to the attack of lymphangitis on February 19th.

The infection in Dr. Primrose's case was so severe that it could not have been produced by two or three parental filariae. We do not know how many adult filariae it would take to produce a stock of embryos such as he describes, but I think if we regard a minimum infection (that is, the product of one pair of parental filariae) to be five to ten embryos in a drop of blood (and this I consider an over-estimate), such an infection as Dr. Primrose describes would require ten to twenty, or even more, female adult filariae to produce. The scrotum must therefore have contained more parent filariae than were found in it, or there must have been parent filariae in some other part or parts of the body. The latter is my view, and I conclude that the main body of parental worms was destroyed during the very severe attack of lymphangitis on February 19th.

In 1892 and for several years subsequently I frequently saw a gentleman the subject of filarial varicose glands in the groin. His blood contained large numbers of embryo filariae—300 to 400 per drop at 10 p.m. About this time I was endeavouring to find out what became of the embryo filariae during their daily absence from the blood, and what might be the duration of the life of the individual embryo filaria. To settle these points, or at all events to throw some light upon them, I had arranged with the gentleman I refer to to have some of his blood transfused into two monkeys, my idea being that the embryos would continue to live in the monkey pretty much as they lived in their human host, and that by periodical examination of the blood I could watch their disappearance, and so get a key to their life-span. I also hoped from this experiment, should the embryos exhibit their usual periodicity of appearing and disappearing in the general circulation, that by killing one of the monkeys during

* Read at the Autumn Meeting of the West Norfolk Division of the East Anglian Branch of the British Medical Association.