pupils, low temperature, slow pulse, and appeared to be moribund. Hemiplegia was present, but there were no signs of fracture of the skull; both eyes had extremely choked discs. After trephining over the posterior Krönlein point and opening the dura, the patient obtained relief. The choking of the discs and the papilloedema disappeared, and she recovered health and full vision.

Sir J. H. PARSONS (London), as one largely responsible for the introduction of the term "papilloedema," deprecated the further complication of the nomenclature of these conditions. It was true that papillitis manifested itself first as a slight oedema, to be followed later by cellular infiltration, but the same condition was produced by inflammation in other parts of the body; and the distinction of papilloedema from papillitis or optic neuritis is parallel to the use of the general terms "oedema" and "-itis" (as indicating inflammation). He concluded that Professor Rönne thought that many cases of retrobulbar neuritis were associated with plaques in the optic nerves containing degenerated medullary sheaths with many intact axis cylinders, as in the well known lesions of disseminated sclerosis. This was a very illuminating suggestion; but it must be remembered that the symptoms of retrobulbar neuritis often preceded any pathognomonic signs of disseminated sclerosis by intervals which might extend to several years.

Dr. DE SCHWEINITZ (Philadelphia) referred to Koeppe's method of distinguishing choked disc and optic neuritis by means of the slit-lamp, especially in the early stages of these conditions. Although the test (the absence of "inflammatory cells" in the first instance and their presence in the second) was not universally accepted it deserved to be used more often. Although he fully recognized the difficulty of making a differential diagnosis in certain cases of optic neuritis and choked disc, he believed that a clinical distinction should be maintained; papilloedema (choked disc) indicating a condition in which oedema was the dominant factor, and optic neuritis one in which an inflammatory process was concerned. Clinically, he was guided by Marcus Gunn's description of the six stages of choked disc (papilloedema). The surest method of preventing the end-result of choked disc—namely, atrophy—consisted in a release of the increased intracranial pressure; to use Victor Horsley's expression, by an operative procedure, usually so-called palliative trephining, other things being equal. How long an operation should be deferred must depend upon frequent testing of the acuity of vision and charting the visual field, especially the size of the "blind spot," or, more accurately, that of the increasing size of the pericaecal amblyopic zone. He agreed in deprecating operative intervention with relation to the paranasal sinuses, and was convinced that infected speno-ethmoidal disease was a definite etiological factor in many cases of retrobulbar neuritis, and more frequently the case than the observations in England would seem to indicate. He was in accord with Professor van der Hoeve that in certain cases, after due trial of conservative measures, operation was required, which should be performed by one skilled in this type of operative work. He did not refer to acute retrobulbar neuritis with its well known visual field disturbances and other clinical signs, which depended on various causative factors and was often a self-limited disease.

Dr. W. H. WILMER (Baltimore) agreed that in certain chronic optic nerve inflammations greater emphasis was laid upon the sinuses in America than in other parts of the world. The ordinary x-ray photograph of the sinuses often failed to show pathological conditions in the cells around the optic canals. At the Oxford Congress Dr. Tyson had exhibited a picture of the optic canals of a man who was gradually losing his sight from a post-neuritic optic atrophy, in spite of the facts that all recognizable sources of infection had been excluded or removed and that the sphenoid and ethmoid cavities had been opened four times. The picture showed the complete removal of all diseased cells except those just around the optic nerves. Dr. Wilmer had seen this patient, and doubted whether the remainder of the diseased cells could be removed without injury to the optic nerves themselves. Yet in the absence of aid blind-

ness was inevitable, and further operative procedure seemed to be justifiable. In some cases of chronic retrobulbar neuritis the scotoma disappeared and vision returned after the removal of several possible toxic sources. It was often impossible to say which was the cause of the trouble.

Sir WILLIAM LISTER (London) referred to the form of papilloedema found during the war in cases of injuries to the head or of concussion. He thought that there was nothing characteristic about the appearances. There were the three primary signs: swelling of the disc, blurring of the edges, and fullness of the veins. There was very slight swelling, and it could be recognized very much more easily by the bending of the vessels as they passed off the disc than by measurement with the ophthalmoscope. This pointed to the condition having been due to a very slight intracranial pressure.

Dr. WILDER (Chicago) quoted the statement of Dr. Paterson that around the eye there was frequently evidence of the presence of oedema when inflammation was not present, though it might be impending. Such oedema, which began round the orbit, might be equally of inflammatory nature, though the stage of inflammation had not been reached. It seemed that in some of these conditions there were plaque formations such as Professor Rönne had described, which might be the beginning of inflammatory oedema and might ultimately result in severe disturbances. In many cases the inflammatory oedema lasted long enough to produce definite trouble; this also suggested that if it was very transitory recovery would be rapid; if it continued for some time a degenerative condition might ensue. In some operations upon the sinuses the results were so pronounced and startling that there must have been some connexion between the sinus and eye condition. With definite inflammation and clear evidence that there was some infection of the sinuses operation became justifiable.

Sir GEORGE BERRY congratulated the office bearers on the choice of subject for discussion. Optic neuritis was far from being a topic which justified the rigid maintenance of fixed opinions. It was necessary to keep an open mind, and to be ready to accept new ideas when presented.

Dr. J. V. PATERSON, in his reply, stated that he did not take up the position that intranasal disease was never a cause of retrobulbar neuritis. In his opinion, in ordinary cases of retrobulbar neuritis adequate nasal cause was seldom present.

Dr. RÖNNE, referring to a remark made by Dr. Paterson regarding vitreous opacities in optic neuritis, said he had examined a great number of specimens in cases of lesions of the optic nerve, and had never found inflammation present anterior to the lamina cribrosa. Any change occurring in front of the lamina was always an oedema.

VITAMIN A DEFICIENCY AND UROLITHIASIS.*

BY

PROFESSOR E. C. VAN LEERSUM,
Netherlands Institute of Nutrition, Amsterdam.

SOME years ago Osborne and Mendel¹ drew attention to the frequency of calculi in the urinary tract of rats fed on rations deficient in vitamin A. In 857 sections they found calculi 81 times. "In every instance," they wrote, "where calculi developed the animals were without an adequate source of the fat-soluble vitamin." Later, in 1920, on the occasion of a vitamin symposium of the New York Medical Association, Mendel said:

"When it is recalled that phosphatic calculi deposited in a neutral or alkaline urine, which in turn frequently owes its reaction to bacterial decomposition, are found extensively among peoples living, for example, in the tropics and the Far East, on diets quite unlike the mixed regime of most Americans and Europeans, the possible reaction of the calculi to dietary factors is at once prominently suggested."

* A paper read at a joint meeting of the Sections of Pathology and Bacteriology and of Comparative Medicine at the Annual Meeting of the British Medical Association, Edinburgh, 1927.

On this McCollum² wrote:

"Osborne and Mendel observed calculi of calcium phosphate in the urinary tract in 91 [read 81] animals among 857 necropsies; 43 per cent. of these had not had a satisfactory supply of vitamin A. In McCollum and Simmonds's experience calculi have occurred so frequently in animals whose diets contained an abundance of this factor, but were faulty in other respects, that it would seem to be the results of general debility rather than lowered vitality brought about by specific cause."

The contradiction in the observations of the experimenters led me to control the records of the rats of the laboratory of the Netherlands Institute of Nutrition. I have examined 886 rats, piebalds and albinos, of which 405 were males and 481 females. Among them are 241 rats (see

Diet.	Number of Rats.	Cases of Calculosis	Other Lesions.		
			Haemat-uria.	Cylind-ruria.	Xeroph-thalmia.
Adequate vitamin A ...	211 (108 ♂ 133 ♀)	0	0	0	0
Deficient vitamin A ...	645 (297 ♂ 343 ♀)	130 ♂ 67 ♀	46	12	92

table) which certainly never suffered from vitamin A deficiency. Their diet consisted in wholemeal bread, raw milk, butter or cod-liver oil, raw carrots or cabbage, and, now and then, some cheese and spinach or lettuce. In none of these 241 rats (108 males and 133 females) were calculi found. Hence the conclusion does not seem too bold that in healthy, adequately fed rats calculi do not appear, or are at any rate rare.

Among the other 645 animals (297 males and 348 females) 197 cases of calculosis were found—namely, in 130 males and 67 females. These rats had been fed on an A-deficient diet. Among them were some that were fed on a rickets-producing diet. That all these rats suffered from vitamin A deficiency was shown by clinical examination. They grew little, and the mortality among them was great. In a number of those I examined the oestral cycle was disturbed. There were also many cases of xerophthalmia. That this eye disease did not occur still more frequently must be attributed to the short life of many rats. The search for calculi was made with care. In all cases the pelvis, the ureters, and the bladder were examined, and, to make quite sure, the bladders which were apparently without calculi were also investigated microscopically. In this way it was possible on several occasions to find concretions invisible to the naked eye. X-ray photographs were taken of many of the rats.

That calculi are more frequent in males than in females has its natural explanation, already given in the Hippocratic book *De Aere Locis et Aquis*—namely, that the urethra of females is short and wide.

Most of the calculi consisted of calcium phosphate or of a mixture of calcium phosphate and calcium oxalate. Calcium oxalate concretions occurred in rats on McCollum's rickets-promoting diet. A number of rats suffered from haematuria. However, this did not occur so often as might have been expected, considering the frequency of the calculosis. Though probably caused by the lack of vitamin A, it is yet of less frequent occurrence than calculosis, and is not directly connected with it, for in many cases of haematuria there were no calculi, while in many cases of calculi in the bladder there was no blood in the urine.

Contrary to all expectations, obvious cystitis was rare. As a rule the urine was clear and its reaction to litmus was acid. In most cases of calculosis, on microscopic investigation the mucous membrane proved to be normal and the surface layer of the epithelium intact.

Calculi may soon appear in case of vitamin A deficiency; the production of calculi in three or four weeks proved to be very common. Several times I found formations in the urine, in shape resembling casts; they were straight or bent crystalline cylinders of calcium phosphate or oxalate. This discovery led me to examine the kidneys, and I found that, in vitamin A deficiency, lime deposits in the tubules are frequent, not to say constant. These lime deposits are found throughout the kidney, especially

in the tubules, but never in the glomeruli. According to the shape of the tubules, they are straight, bent, or sinuous.

Altogether I examined 254 rats with regard to their kidneys; 76 had had a satisfactory supply of vitamin A, and 178 had suffered from vitamin A deficiency. Of the 76 rats, 10 had lime deposits in their kidneys, but these deposits were very small in number, one, or at most two, to a section. Hence I believe that with healthy, adequately fed rats lime deposits of any size in the kidneys may be said to be exceptional. On the other hand, calcium deposits in the tubules were almost constant in the rats suffering from deficiency of vitamin A; of 178 rats deposits were found in 158 (87 per cent.), whereas there was a frequency of calculi in the bladder of only 35.4 per cent.

As a control I first extirpated one of the kidneys from a number of rats on an ordinary diet, and then withheld vitamin A from their food. I then, after some time, killed those which had not already died from the consequences of the operation or some other cause. In 14 rats which died within three to fifteen days after the operation there were no lime deposits in the extirpated kidney nor in the other one; 9 other rats fed in the same way also had no lime deposits in the extirpated kidney, but in 7 of them, after more than twenty days of vitamin A deficiency, I found deposits in the remaining kidney. These lime deposits form quickly; in one case of deficiency in vitamin A they had been formed in thirteen days.

From what has been said above it may be concluded that in rats there is without doubt a connexion between deficiency in vitamin A and the formation of phosphatic calculi.

It is known that absence of the fat-soluble vitamin from the food causes typical changes in the epithelium of the mucous membranes—for example, keratinization; witness the xerophthalmia and the lengthening of that stage of the oestral cycle characterized by the horny cells. Probably such a morbid change of epithelial cells of the tubules gives the impulse to the deposition of lime, which deposition, once liberated, grows to larger concretions in the bladder.

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EFFECT OF EARLY INDUSTRIALISM ON THE HEALTH OF THE COMMUNITY.*

BY

MABEL C. BUER, D.Sc.(Econ.),
Lecturer in Economics, University of Reading.

THE popular view is that the effect of industrialism on the health of the community was necessarily bad, but the fact is that the population of Great Britain grew at an unprecedented rate during the period 1760-1860. To account for this, an increase in the birth rate was postulated; there is, however, no evidence for this, and definite evidence for a decrease of the death rate. An estimate of the death rate for England and Wales in the middle of the eighteenth century shows that, on a rough average, it was 35 per 1,000. In the year 1815 it had been reduced to about 20 per 1,000. In the middle of the eighteenth century the death rate in London and other large towns was 50 per 1,000, while in 1815 the death rate in London was about 30 per 1,000. These figures give very little support for the contention that economic advance was at the expense of health. The main part of the population in the middle of the eighteenth century were living in comparatively isolated rural communities, in which the death rate is usually very high. It is not primarily a hot climate which is responsible for diseases like plague, but the lack of hygiene, along with the uncertainty of food supply. Plagues were frequent in mediaeval Europe, and scurvy certainly and rickets probably were present. The towns were without the most elementary sanitation, overcrowding was rife, and the water

* Read in the Section of the History of Medicine at the Annual Meeting of the British Medical Association, Edinburgh, 1927, the President, Dr. J. D. Comrie, in the chair.