time made manifest. A brother, in excellent health, was found to have an enlarged spleen, a raised fragility, 0.52 to 0.3, and moderate urobilinuria.

I would like to thank Dr. Robert Hutchison, Dr. Otto Leyton, Dr. Theodore Thompson, Dr. Adolphe Abrahams, Dr. Campbell, and Dr. C. S. Atkin for letting me use their cases. One of Dr. Leyton's cases was particularly valuable. I am indebted, also, to Dr. Creed of King's College Hospital for his valuable method of estimating fragility of the blood. I have referred in the text to the reports of Professor Turnbull and Dr. W. D. Newcomb. I gratefully acknowledge the help and criticism of Dr. Mathilde Hunter and Dr. Donald Hunter in the gathering of numerous observations set forth in the text. I have also received valuable aid from Dr. Daniel Davies, and I think it will be admitted that the illustrations by Messrs. Shiells and Ford and Mr. S. A. Sewell are excellent.

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A Lecture

ON

SOME SURGICAL ASPECTS OF FAULTY NUTRITION*

BY

ROBERT McCARRISON, C.I.E., M.D., D.Sc.,
LL.D., F.R.C.P.

COLONEL, INDIAN MEDICAL SERVICE; HONORARY PHYSICIAN TO THE KING; DIRECTOR, NUTRITIONAL RESEARCH, INDIAN RESEARCH FUND ASSOCIATION

The laboratories of the Indian Research Fund Association, devoted to the study of diseases associated with faulty nutrition, are located at Coonoor in the Blue Mountains (Nilgiris) of Southern India, 6,000 feet above sea-level. Their location is fortunate, for the temperature is equable, never very hot or very cold: the mean winter temperature rarely falls below 50° F., the mean summer temperature rarely reaches 80°. A good deal of rain falls in these hills, Coonoor coming within the range of both the south-western and the north-eastern monsoon: the former brings with it only occasional heavy showers, while the latter may involve us in heavy rainfall during November, December, and January. But it is rare that a day passes without some hours of bright sunshine and clear skies. Conditions are such that no elaborate buildings are required for the animals. Indeed, I have been content with corrugated-iron sheds, roofed with the red tiles of the country, and having floors tiled, sloped, and adequately drained. I am fortunate also in having a large staff of animal attendants, who are now highly skilled in their work. In these favourable circumstances it is possible to ensure ideal conditions of hygiene and exposure of the animals to sunshine practically every day the whole year round. A simple but adequate means of sterilizing the animal cages is adopted, and the hose-

pipe and creosol solution are used freely on the tiled floors. The walls are whitewashed frequently. The test of cleanliness is a simple but severe one. In an animal room of some 30 by 15 feet there may be as many as 500 rats. Should the visitor be made aware of their presence by his sense of smell then there is something wrong with the sanitation. I may add that he rarely is.

FREEDOM OF WELL-FED ANIMALS FROM DISEASE

In these conditions there live some 1,000 stock albino rats—the source from which the large numbers of young animals required for my work are obtained. For this work I use on the average 1,100 animals a year. The pairs are kept in roomy, netted-wire cages, screened and straw-filled, in which they find the peace and comfort necessary for fruitful breeding. As a rule, five or six litters are taken from each pair, and the older animals are thinned out at the age of 2 years—a life span said to correspond approximately to the first 40 to 50 years of life in man. The average number in each litter is eight, but litters of twelve are frequent, and those of fifteen are not uncommon. The mothers invariably rear the whole of their young.

This stock is fed on a diet similar to that eaten by certain peoples of Northern India, among whom some of the finest physical specimens of mankind are to be found. The diet consists of whole-wheat flour, unleavened bread (*chapattis*) lightly smeared with fresh butter, sprouted Bengal gram (legume), fresh raw carrots and cabbage *ad libitum*, unboiled whole milk, a small ration of raw meat with bones once a week, and abundance of water, both for drinking and for washing purposes.

The point of these somewhat lengthy, but necessary, details is this: during the past two and a quarter years there has been no case of illness in this "universe" of albino rats, no deaths from natural causes in the adult stock, and, but for a few accidental deaths, no infantile mortality. During this period I have myself performed complete post-mortem examinations on 1,189

* The first of two lectures on experimental research at the Pasteur Institute, Coonoor, S. India, delivered before the Royal College of Surgeons of England.

of these rats, ranging in age from the newborn to 2 years. In none have I encountered any macroscopical evidence of disease, with the exception of an occasional cyst in the liver, containing tapeworm larvae. How the animals became infected with tapeworm I do not know, unless it be from the straw bedding which they nibble. Both clinically and at post-mortem examination this stock has been shown to be remarkably free from disease. It may be that some of them have cryptic disease of one kind or another, but, if so, I have failed to find either clinical or macroscopical evidence of it. Disease and death have been excluded, almost completely, by minute attention to three environmental conditions: cleanliness, comfort, and food.

HIGH INCIDENCE OF DISEASE IN ILL-FED ANIMALS

I wish now to turn to my experimentation rooms. Here are the same corrugated-iron huts, the same cages, the same scrupulous cleanliness, if anything more scrupulous than in my stock animal houses; for here there are attendants, armed with forceps and crumb-tray, who pass from one cage to another removing faeces which may occasionally adhere to the half-inch meshes of the screens on which the animals live. There is no coprophagy; it is not allowed. Nor do we meet with the phenomenon of "refection." Here the animals are exposed in the same way to the sun's rays. But they live in separate cages, as do the controls for each experiment—isolated from their fellows; they are thus less contented than their more fortunate kith and kin in the stock animal rooms, and they are more exposed to the effects of chill, since they cannot be given straw bedding, though each evening they are given bedding of clean cotton-waste. The main regard in which they differ from the stock animals is in their food, which is always faulty in some way or another. Further, as my business is to learn, as far as I can, how the foods eaten by the people of India are related to disease, synthetic diets are used comparatively rarely, and then only for special purposes. The materials entering into the various faulty dietaries are usually those in use by the people of India; sometimes actual Indian dietaries are used.

During the last two and a quarter years I have examined 2,243 of these improperly fed rats at post-mortem examination—that is to say, during the same period in which I failed to find disease in the universe of stock rats. The following are the morbid states I have encountered:

Lung diseases: pneumonia, broncho-pneumonia, bronchiectasis, pyothorax, pleurisy, haemothorax.

Diseases of the nose and accessory sinuses: sinusitis.

Diseases of the ear: otitis media or pus in the middle ear.

Diseases of the upper respiratory passages: adenoid growths.

Diseases of the eye: conjunctivitis, corneal ulceration, keratomalacia, panophthalmitis.

Gastro-intestinal diseases: dilated stomach, gastric ulcer, epithelial new growths in the stomach, cancer of the stomach (in two cases only), duodenitis, enteritis, gastro-intestinal dystrophy, stasis.

Diseases of the urinary tract: pyonephrosis, hydronephrosis, pyelitis, renal calculus, ureteral calculus, dilated ureters, vesical calculus, cystitis, incrustated cystitis.

Diseases of the reproductive system: inflammation of the uterus, ovaritis, death of the foetus *in utero*, premature birth, uterine haemorrhage, "hydrops testis."

Diseases of the skin: loss of hair, dermatitis, abscesses, gangrene of the tail, gangrene of the feet, subcutaneous oedema.

Diseases of the blood: anaemia, a "pernicious" type of anaemia, *Bartonella muris* anaemia.

Diseases of lymph and other glands: cysts in the sub-maxillary glands and accessory glands in the base of the tongue, abscesses in the same, and occasionally also in the inguinal glands, enlarged mesenteric, bronchial, and other lymph glands.

Diseases of the endocrine system: lymphadenoid goitre, enlarged adrenal glands, atrophy of the thymus, and, very occasionally, haemorrhage into the pancreas.

Diseases of the nervous system: polyneuritis.

Diseases of the heart: cardiac atrophy, occasionally cardiac hypertrophy, myocarditis, pericarditis, and hydropericardium. Oedema.

Not infrequently animals are found to be suffering from more than one of these diseases.

I say nothing of diseases of the bones and teeth, since the diets were not specially designed for their production, and the practice of exposing the animals to sunlight—a practice followed because the average Indian is daily so exposed—probably compensated for any lack of vitamin D in the diets. But mention might be made of crooked spines and distorted vertebrae, and of imperfect teeth, which are encountered not infrequently. The list I have given includes only those morbid states which I have myself seen (there may have been, and no doubt were, others which I failed to see); it is a list prepared from the post-mortem records of the 2,243 imperfectly fed animals.

Here, then, is an experiment on a large scale extending over a period of two and a quarter years. On the one side there is, as controls, a community of hygienically housed rats, fed on a well-balanced diet composed of natural foodstuffs—whole-wheat, pulses, raw milk, butter, fresh vegetables, and fresh meat with fat and bones occasionally, so combined as to provide in proper amount and proportion all elements and complexes necessary for normal nutrition. On the other, there is a community of rats, also hygienically housed, but fed on ill-balanced diets composed mainly of cereal grains or cereal products and vegetable fats with little or no milk, butter, and fresh vegetables. The contrast between these two communities is remarkable; and I claim for this experiment that it proves conclusively that one-sided diets which are disproportionately rich in cereals and poor in animal fats, milk, and *fresh* vegetables are capable of inducing in albino rats a large proportion of the diseases included in our calendar of human ailments. I may add that of all the faulty diets I have used that composed of white bread, margarine, tea, sugar, jam, preserved meat, and scanty overcooked vegetables—a diet in common use by many people in this country—proved to be one of the worst, and most likely to be associated in rats with many of the morbid states I have mentioned, especially diseases of the lungs and of the gastro-intestinal tract.

Rats are not the only animals I use; guinea-pigs, rabbits, pigeons, and monkeys are also employed. In them I have found many of the above-mentioned diseases, and some others besides. Of the latter I may mention the following: prescorbutic states, scurvy, and duodenal ulcer in guinea-pigs, hyperplastic goitre in rabbits, epithelioma contagiosum, polyneuritis, and true beri-beri in pigeons, and ulcerative colitis in monkeys. Two conditions I have not, so far, encountered: gall-stones, observed by Fujimaki (1926), de Langen (1929), and others; and cataract, observed by Eckstein and Szily (1924).

In every species—pigeons, rats, guinea-pigs, rabbits, and monkeys—I have found the same thing: properly fed animals remain remarkably free from disease, improperly fed animals are remarkably subject to it. It is reasonable, therefore, to assume that the human species is no exception to this rule, and that many of the ailments to which man is erroneously supposed to be heir are the outcome of his improper feeding. Some may hold that it is not possible directly to translate to human experience the results of an experiment such as that just described. This may be true, if we seek to do so too rigidly and fail to take sufficient count of other environmental factors to which human beings may be exposed. But, in general, the results of an experiment of this kind do, in my opinion, apply to man, and point the way to a solution of many etiological problems connected with his physical infirmities.

DEFICIENCY IN VITAMIN A

In the above list of diseases there are many of interest to the surgeon. Sinusitis, adenoid growths, otitis media, gastro-duodenal ulcer, gynaecological ailments, certain types of goitre, and urinary calculus are some of them. At first sight it would appear that morbid states so dissimilar as adenoids and stone in the bladder had nothing in common, yet etiologically they are not so far apart. All these surgical conditions were found to have this in common: they occurred in animals whose ill-balanced diets contained little or no vitamin A bearing food materials, such as raw milk, butter, cod-liver oil, carrots, and fresh green vegetables. In my hands these food materials, when suitably combined, have protected my animals from these diseases, and I do not doubt that in the hands of the surgeon they will go far to protect his human patients from them and facilitate their cure and non-recurrence after operation. I lay stress on the food materials themselves rather than on any particular ingredient of them, or any particular quality possessed by them, believing, as I do, that in Nature's laboratory all elements and complexes needed for normal nutrition are combined in a way which we cannot wholly achieve, and that the health-giving properties of food are largely dependent on this combination. For my own part I have been able to devise no synthetic diet which can equal in these properties a mixture of natural food materials such as that on which I feed my stock rats and on which some of the finest physical specimens of mankind are reared. Nevertheless, a principal fault of the diets I have used, and one which is in considerable part responsible for the surgical diseases I have mentioned, is insufficiency of the substance or quality which we speak of nowadays as vitamin A. The effects on the organs and tissues of the body of the insufficient use of vitamin A bearing foods have thus a particular interest for the surgeon.

[Here followed a lantern slide demonstration in which the lecturer showed the typical keratinization of epithelia, the concourse of lymph cells, the secondary inflammatory changes, the ulceration of, and epithelial new growths in, the stomach, and the lymphadenoid goitre occurring in rats fed on diets containing an insufficiency of vitamin A bearing ingredients.]

The sites in which these changes have so far been found in experimental animals are the nares, larynx, trachea, bronchi, submaxillary glands, parotid gland, and the accessory salivary glands of the tongue and pharynx; the pancreatic duct, bladder, ureter, and pelvis of the kidney; the uterus and oviduct; the epididymis, prostate, and seminal vesicles; the conjunctiva, Meibomian gland ducts, cornea, lacrymal gland, Harderian gland, and the extra-orbital (lacrymal) gland; the thymus (Wolbeck and Howe, 1925). To this list I must myself add the thyroid gland. "The distinctive or peculiar gross changes are those due to mechanical factors sequential to the formation and retention of desquamated, keratinized epithelial cells" (Wolbeck and Howe, 1925). Thus cysts, bronchiectasis, hydronephrosis, dilatation of the bladder and ureters may be produced by blockage of ducts or other passages.

These changes occur more readily, and within a shorter time, in young than in older animals—a thing to be expected in the growing animal. They are focal in distribution, and may be confined to narrow or to wide areas, to a few tissues or to many. They vary in their location in different species of animals and in different individuals of the same species. I have, for instance, found the adenoid-like growths in the upper respiratory tract in rats to all appearances normal except for sub-normal growth. They are best seen and more widespread in animals fed on synthetic diets devoid of vitamin A ;

but since man's food is rarely or never wholly lacking in this factor it is of importance to realize that they occur also in animals fed on human dietaries which are by no means devoid of it, though containing it in insufficient quantity. It seems probable that these changes are helped in their occurrence by the existence of other food faults in the diet, such, for instance, as deficiency of suitable protein and mineral elements, and by the condition of semi-starvation into which the animals fall. It seems probable also that other environmental conditions, such as climate, altitude, humidity, may favour or hinder their occurrence. Thus it is universally agreed that the changes in the eye and its neighbourhood are, in rats, the most frequent; but they are not so in the climate of Coonoor; indeed, the eye is often not affected when other tissues are. I mention these matters to illustrate the interplay between nutrition and other environmental conditions (climatic and bacterial) in giving rise to the final morbid state. The worn tyre may travel far along the smooth-surfaced road before encountering a flint; its course along the flinty byway is less secure. So it is with the epithelial treads of the body surfaces when deteriorated by faulty nutrition.

The varying occurrence and location of the changes due to vitamin A deficiency are among the most interesting things about them. Thus in monkeys fed on a diet said to be complete in other regards, but lacking a sufficiency of vitamin A, eye changes, sinusitis, and mastoiditis, so common in rats, are, as Tilden and Miller (1930) have lately shown, conspicuous by their rarity. Twelve years ago (1919) I demonstrated that the colon was of all organs in monkeys the most sensitive to faulty nutrition; and the recent work of Tilden and Miller, while confirming this observation, re-emphasizes the importance of vitamin A deficiency in causing the ulcerative colitis which is so conspicuous a feature in these animals. The monkey's colon is also very sensitive to deficiency of other vitamins (McCarrison, 1919), particularly of vitamin C, "which causes a condition varying from local congestion and thickening of the mucous membrane to one indistinguishable from ulcerating and sloughing dysentery" (Mackie and Chitre, 1928).

In some of these respects man resembles the rat more than the monkey. Thus he is very susceptible to the eye changes resulting from insufficiency of vitamin A bearing foodstuffs in his diet, if one may judge by the prevalence of keratomalacia in the Madras Presidency, and by its amenability to treatment by cod-liver oil (Wright). But I suspect that he resembles the monkey in the sensitiveness of his colon to food deficiency; the number of cases of ulcerative colitis of "unknown" cause (Menon, 1930) occurring in India is sufficient to justify this suspicion. But however the changes vary, either as to extent or location, they are always to be found in animals of all species so far examined—rats, guinea-pigs, pigeons, fowls, and monkeys—and in man himself. Reference has already been made to their occurrence in the eye, and its neighbouring tissues, in Madrassis, and to its proven association with the specific deficiency. But there are few post-mortem studies of pure vitamin A deficiency in man. Fortunately there is one: I take it from a paper by Wolbeck and Howe (1925):

"*Vitamin A Deficiency in the Human.*—There has been but one pathological description of a fatal human case (Wilson and du Bois). This post-mortem, done in the pathological service of one of us at the Children's Hospital, is adequately recorded. We have recently re-studied the tissues and can add nothing of importance. Keratinization of the epithelium of the renal pelvis and large size of Hassall's corpuscles in the thymus were overlooked. The keratinization of the pancreatic ducts is more striking than any seen by us in the pancreas of rats. In respect to the keratinizing changes in the lungs, uterus, submaxillary gland ducts, and thymus, the picture was identical with that in our experimental rats.

The order of changes in the human case was somewhat different, in that the salivary glands showed only slight changes. The peculiar swollen epithelial cells with nuclear inclusion bodies in the ducts of the parotid and submaxillary glands are unlike any changes seen in rats. The lacrymal gland showed only slight changes and no keratinization. The eyes were not removed for study.

"The importance of this human case is that it did show the epithelial changes which we have produced in rats, and, therefore, contributes irrefutable evidence for an important part of our thesis to the effect that vitamin A deficiency affects epithelial tissues in a specific manner."

The testimony of these experienced observers is of the highest value, and leaves no room for doubt that the tissue changes occurring in the lower animals, consequent on the *insufficient* supply of vitamin A bearing foodstuffs in the diet, occur also in man. I again emphasize the word "*insufficient*," since, in my own experience, and in that of Tilden and Miller (1930), in monkeys the diets associated with them are not necessarily devoid of vitamin A, though always they are poor in this factor.

These changes are independent of infection, though infection is often superimposed upon them. Indeed, it is because of them that infection becomes possible, since they occur for the most part in "outside" coverings of the body; the respiratory passages, the urinary tract, the reproductive tract, the conjunctiva, the nasal passages, the ear, and the ducts of organs. They are breaches in the walls of the body's defences, through which the invading microbe may enter. In this sense vitamin A is "anti-infective"; but the "anti-infective" quality is shared with other vitamins—C, B, and D—though exhibited by them in other ways.

Two points of interest to the surgeon remain to be mentioned: (1) If rats, fed on a diet deficient in vitamin A, be artificially infected with a bacterium, such as the Breslau bacillus (paratyphoid), they usually die from the resultant infection. But some may live, and in them the infection persists in the lymph glands "for a very long time after the general infection has been overcome" (Lassen, 1930). It seems to me probable that chronic enlargement of lymph glands in childhood, due usually to infection of one kind or another, may often be due to such persistence of infection as a result of a vitamin A deficient diet. Further, it seems probable that the focal breakdown of the tissue defences induced by this deficiency may be concerned in the causation of those "septic foci" about which so much is heard nowadays. (2) I have spoken above of breaches in the walls of defence against bacterial invasion consequent on the use of vitamin A deficient diets, and I have mentioned that other vitamins may also exercise an anti-infective action, though exhibiting it in a different way. This way may not need the breaching of the walls, but their scaling. I take the following from an unpublished manuscript which Dr. Lloyd Arnold of the Department of Pathology and Bacteriology, Chicago, has recently sent me:

"McCarrison has repeatedly called attention to the early appearance of digestive disturbances in vitamin deficiency studies. The pathological examination of the dead animals following avitaminosis experiments shows evidence of tissue changes due to infections of various kinds. We have found

that one of the first demonstrable changes in avitaminosis is an alteration in the distribution of the bacterial life within the lumen of the intestinal tract. The bacteria ingested by the mouth are not destroyed in the usual manner, but remain viable for many hours within the contents of the whole length of the small intestine. The mixed bacterial flora of the large intestine ascend into the upper levels of the small intestine. There is a gradual and progressive loss of the power of the body surface to control the microbic life existing in contact with it. These findings have been constant, and hold for vitamin A, B, and D deficiencies. By the time the adult animals show a loss in body weight, or the young animals show a stationary weight-curve, there is evidence of bacterial invasion of the mucosa of certain surfaces. These latter observations have been made by numerous investigators. We attribute the early inflammatory changes in the lining membranes of the eye, mouth, throat, alimentary tract, etc., to the loss of power of these body surface coverings to regulate the bacterial flora in contact with their cells. This precedes evidence of invasion of the membranes by bacteria."

This statement, so amply demonstrated by Dr. Lloyd Arnold's investigations, calls to mind the contentions of Sir Arbuthnot Lane and Dr. Nathan Mutch in regard to infection of the upper reaches of the intestine consequent on chronic gastro-intestinal stasis: a condition which, as

my own researches (1921) have shown, may be due to the consumption of faulty food from the "first bottle" onwards through life.

The function of vitamin A, so far as it is known at present, is thus to maintain the functional integrity of the cells covering body surfaces, thereby preventing invasion of the organism by microbic agents.

Here, however, I must re-emphasize

the position, which I reached more than ten years ago and which all my subsequent work has but strengthened, that, whatever the specific function of any vitamin may be, vitamins are but links in the chain of essential substances requisite for the maintenance and harmonious regulation of the chemical processes in the tissues, and their action must be considered in connexion with metabolism as a whole—that is, in relation to balance of food ingredients in general, to organs of digestion and assimilation, and to endocrine regulation of metabolism. Their deficiency, like that of the other elements and complexes necessary for normal nutrition, leads to depreciation of cellular function; depreciation of cellular function is the foundation upon which disease is built (McCarrison, 1921).

SURGICAL DISEASES WHICH MAY BE ASSOCIATED WITH FAULTY NUTRITION

I find I have left myself little time to discuss specific diseases of surgical interest which may be associated with faulty nutrition. Mention may, however, be made of a few, illustrative of the several departments of surgery, but excluding the ophthalmic surgeon, to whose department some reference has already been made.

Adenoids

In the lantern slide demonstration I showed examples of adenoid growths in the trachea. (See Fig.) In rats these growths were the result of faulty feeding. It is no far cry from the trachea to the nasopharynx, where changes similar to those in the trachea of rats are so

Adenoid growths in upper part of trachea of rat fed on a diet containing an insufficiency of vitamin A bearing ingredients.

commonly found in children. And perhaps I might have found adenoids there also had my serial sections extended up to that situation. In work such as this it is constantly happening that one cannot do everything one wants to do, or that in looking for one thing one overlooks another. So whether or not rats do get "adenoids" as the surgeon understands the term, I do not know, but they certainly get "adenoid-like growths" in a region not far removed from the nasopharynx; and they get them because of faulty nutrition. And if in the trachea of rats, why not in the nasopharynx of children? The more so since Williamson and Pearse, and more recently Wollard (1931), have shown that the pharyngeal entoderm is an anatomical entity which embraces the lymphatic tissue of the nasopharynx and trachea. Evidence provided by the racial distribution of adenoids is not lacking to show that their occurrence may be related to faulty food. Thus Van der Bogert (1927) reports that Labrador Eskimos and those of Baffin Island who have been in contact with Western civilization, and whose food has changed accordingly, develop adenoids, whilst Polar Eskimos, whose diet has remained unchanged for centuries, do not. My own experience in rats would lead me to expect such a sequence of events.

Otitis Media

In rats one of the most common consequences of faulty food deficient in vitamin A is pus in the middle ear; its incidence may reach 60 or even 70 per cent. It is a condition of great frequency among school children in England and Wales, and, although its occurrence, as a sequel of certain diseases of childhood, is well known, the probable nutritional factor in its causation does not appear to me to have received the consideration it deserves. According to Hastings (1925) a high proportion of all cases occur before the fifth year, and many before the first birthday. In 1923 no fewer than 46,000 children had ear trouble; while 90 per cent. of deafness in adults is due to preventable causes in childhood. Ponfick reported that of 100 necropsies in children under 4, who died from various diseases, 91 showed evidence of middle-ear disease, and in only ten of these was the trouble recognized during life. The causes giving rise to middle-ear disease interact with one another: they are squalid poverty—with its accompaniment of malnutrition—neglected catarrh, tonsils and adenoids, mouth-breathing, and the after-effects of measles, scarlet fever, and diphtheria. My rats do not suffer from the three last; but my experience in these animals leads me to believe that the insufficient supply of vitamin A bearing foods may be responsible for middle-ear disease in children to an extent much greater than is generally believed.

Gastro-Duodenal Ulcer

Twelve years ago (1919) I recorded the occurrence of this condition in guinea-pigs fed on a scorbutic diet of crushed oats and autoclaved milk. Recently I have repeated this experiment with the same result; and in this later experience I have found duodenal ulceration at all stages of the process up to perforation (in one case), the resulting abscess being localized. Magee, Anderson, and McCallum (1929) have observed similar conditions in calves fed on food deficient in certain essentials. Gastric and duodenal ulcer are extremely common in Southern India, especially in South Travancore. The suggestion was made to me by Lieut.-Colonel E. W. C. Bradfield, I.M.S., late superintendent of the General Hospital, Madras, and by Dr. T. Somervell of the South Travancore Medical Mission, that the dietaries of the poorer classes, among whom these conditions were most common, might be concerned in their causation; an experiment was accordingly undertaken to put this view to the test.

Albino rats were used; and the experiment was continued for 675 days. Two diets were employed: one, resembling that eaten by the poorer class Madrassi, and containing rice, red pepper (chillies), tamarind, a little fish, and *conjee* (rice water); the other, resembling that of the poorer class Travancorian, and consisting largely of tapioca root, with rice, a little fish, red pepper, and rice water. Both these diets were very deficient, not only in vitamins of the A, B, and C classes, but in other regards also. The diets were prepared and cooked in the same way as by the people using them. It so happened that about the time this experiment matured I had occasion to examine at post-mortem some hundreds of well-fed stock rats who had lived through the same period of 675 days. In none of these was gastric or duodenal ulcer found. Among the rats fed on the Madrassi diet the incidence of gastric ulcer was 11.1 per cent., while the duodenum showed no signs of ulceration; among those fed on the Travancore diet, 27.7 per cent. had gastric ulcer, and in 11.1 per cent. of these the ulcers occurred both in the proximal (squamous) and in the distal (mucous) portion of the organ. No duodenal ulceration was found in animals fed on either diet, although in 11.1 per cent. of those fed on the Travancore diet a marked duodenitis was present. The limitation of the ulceration to the stomach in rats was the more remarkable because the Travancorian diet is especially associated in man with duodenal ulcer. A further point of difference between the two groups was that while in animals fed on the Madrassi diet the ulcers were limited to the squamous portion of the organ, in those fed on the Travancorian diet the ulcers occurred for the most part in the mucous portion.

Here, then, is unequivocal proof that two diets in use by certain people in India are capable of causing peptic ulcer in albino rats. The high incidence of this condition among the human users of these diets may therefore be definitely attributed to this cause. As to the mechanism of ulcer production—that is another matter; the one that concerns me, as an investigator in the service of the Indian people, is that two of the diets used by them do cause peptic ulcer, while a third, on which my stock rats are fed, does not. For them the moral is unmistakable.

The excellent results reported by Harris (1928) in the treatment of gastric and duodenal ulcer by vitamin-rich food are in conformity with the findings in this experiment.

Gynaecological Ailments

The health of the womb and its appendages is dependent in various specific ways on the quality of the food. It is one of the sites in which faulty nutrition implants its seal, as has been shown, not only in the lower animals, but in the human being. The ovary, also, and the oviducts, may be affected in a similar way; while changes brought about in the bony framework of the pelvis may, as is well known (Stoney, 1930), be a fruitful cause of stillbirth and maternal mortality in childbirth. Fertility, the course of pregnancy, labour, resistance to infection following labour, and lactation may all be affected adversely by defects in quality of the food. It is said that "nothing so predisposes to maternal death at confinement as an unhealthy womb" (Pearse and Williamson, 1931), and while imperfectly terminated pregnancies, aborted pregnancies, and other conditions may, and often are, the causes of this unhealth, faulty nutrition may also be concerned in it. Often I have seen deficiently fed rats that die in pregnancy or at term from causes dependent on their faulty nutrition, and often also rats that come to term only to die with five or six fetuses in their wombs. Some of the anaemias of pregnancy so common in India are dietetic in origin—as the recent work of Dr. Lucy Wills has so conclusively shown. I make no claim that in human beings faulty nutrition is invariably a factor in the production of the tragedy of maternal mortality in childbirth, however frequently it may be so in rats. But here, as in other departments of medicine, experience in rats has pointed

surely to means for the relief of suffering in mankind ; and it might be well were we to accept this guidance more readily in regard to our women, and urge insistently that the mothers of our race be fed with knowledge and understanding.

Lymphadenoid Goitre and Graves's Disease

A lecture might, by itself, be devoted to this subject, so much is the health of the thyroid gland and its departures from health associated with perfect nutrition on the one hand, and with faulty nutrition on the other. Lymphadenoid goitre, originally described in man by Williamson and Pearse (1925), is "the hypertrophic reaction of a physiologically inefficient organ with which there occurs a preponderance of lymphocytic aggregates, fibrosis, and a peculiar atrophy of the parenchyma." I have produced it under experimental conditions in rats (McCarrison, 1927). In these animals the physiological inefficiency of the gland can be induced by faulty nutrition, but the exciting cause of the goitre is some positive factor which has so far escaped me. One cannot be sure when this type of goitre is going to occur ; all that one can be certain of is that it surely will occur, in its own good time, provided the food is of a certain character. It is unrelated in its origin to iodine deficiency. I understand that it is now possible to diagnose this condition clinically in the human subject, and that thyroidectomy has been performed for its relief in not a few cases within recent years in London. Mention of its association with the use of foods deficient in vitamin A recalls the observation of Rabinowitch (1929) that a combination of relatively minute quantities of organic iodine, with a concentrated preparation of fat-soluble vitamins,

causes as marked a lowering of the metabolic rate in Graves's disease as larger doses of iodine when not combined with these vitamins. Fraser and Cameron (1929) have made a similar observation, using small amounts of sodium iodide instead of organic iodine. More recently Abelin (1930) has demonstrated the great value of vitamin treatment in Graves's disease.

"The hyperthyroid condition," he writes in his recent book, "is one of the abnormal raising of the metabolic rate, together with a lowering of anabolic processes. This disorder of lowered activity of cells can be successfully combated through nutrition. Among other things, for this purpose, the co-operation of vitamins is necessary. The organ destruction in thyrotoxicosis is caused by a vitamin-poverty in the body. The need for vitamin supply is, therefore, increased in hyperthyroidism. Through plentiful use of vitamin-rich substances—cod-liver oil, egg-yolk, yeast, tomatoes, spinach, etc.—the great destruction of sugars has been reduced or almost completely overcome successfully in animal experiments."

Again, it has lately been shown, in my own laboratory, how important is the balance between fat-soluble vitamins and iodine in the diet. The administration of iodine to rats fed on diets deficient in these vitamins may actually cause goitre (McCarrison, 1930). We have found, also, that the maintenance of an adequate iodine content in the thyroid gland is largely influenced by the content of vitamin C in the food (Sankaran, 1931). Few organs of the body, indeed none with the exception of the suprarenals and thymus, are more profoundly affected by faulty nutrition than the thyroid gland, and few are more sensitive to this influence.

My second lecture will deal with the causation of urinary calculus.

THE TREATMENT OF DYSMENORRHOEA *

BY

B. VENN DUNN, M.D., F.R.C.S.ED.

Recent investigations have facilitated the treatment of dysmenorrhoea on a more rational basis than has been possible in the past. The old classification of dysmenorrhoea into spasmodic and congestive varieties may be discarded for several reasons. In the first place the terms are not relative ; "spasmodic" denotes a disorder of function, whereas "congestive" implies a morbid anatomy. Secondly, the two forms cannot be easily differentiated ; cases frequently occur each with a different morbid anatomy, all with similar symptoms—those of spasmodic dysmenorrhoea. Thirdly, there is a danger of cases being labelled "spasmodic," without sufficient examination having been made. The importance of a complete investigation of every case of dysmenorrhoea cannot be too greatly stressed.

CLASSIFICATION

The following classification has been found of practical value in the investigation of cases :

1. Psychological.
2. Endocrine.
3. Mechanical.
4. Gross pathological.

The treatment of the patient will vary according to the category to which the case belongs.

THE PSYCHOLOGICAL TYPE

This applies to those patients, often in their teens, but sometimes much older, who have had an unhealthy psychological upbringing. They may, from their earliest years, have had their simplest physiological acts asso-

ciated with feelings of shame and repugnance. Such an association is bound to make a child magnify any slight discomfort experienced at a time when she is undergoing rapid mental development and in a region she has been told is so important as to be unmentionable. Perhaps, also, too much may have been made of any slight disability the child may have felt in the early months of puberty. The overanxious parent insists on the child lying up when it is more than ever necessary that the circulation in the pelvic area should be encouraged. Gradually a habit of pain becomes formed, the nerve paths for pain become especially sensitive from much use, and, unless a new hygiene is quickly enforced, dysmenorrhoea may continue for the rest of the sexual life.

The treatment of this type of case varies with the age of the patient. The great hope is to catch her young, and before the habit of pain is too well formed. Then we must be ready to spend a good deal of time, first with the patient's parents (either or both) and then with the patient herself. The matter must be talked out. These are the cases which do well under the regime recommended by the Medical Women's Federation, whose leaflet, "Advice regarding menstruation," consists of an exhortation to "business as usual" during the menstrual period. It insists on the necessity of the daily warm bath throughout ; it advocates an adequate amount of exercise, while it deprecates "long expeditions and cycle rides or matches or competitions in which there is a strong incentive to exhaust the reserves of strength." The use of a system of physical exercises such as that published a few years ago by Dr. Harold Dearden, under the title of "Exercise and the will," is extremely valuable for these girls. The older patients with this type of dysmenorrhoea are a trouble to their medical advisers as well as a nuisance to themselves. The only people who profit by them are the vendors of patent medicines and the retailers of gin.

* Abstract of a paper read before the Torquay Medical Society on March 19th.