fever and ague as we understand it. It is not malaria, but elephantoid fever for the most part, and if we inquire as to the occurrence of inflammation and cellulitis of limb, scrotum, or acute lymphangitis, we are sure to find that such is the case. The patient is suffering from elephantoid fever, and is or has been the subject of filarial invasion.

The possibility of these various and very different infections should always be present to the diagnostician when he is called on to treat a tropical fever in this country.

Multiple Infections.

I began with a word of warning; I shall conclude with another word of warning, and it is this: Do not infer because you have found in your patient's blood or elsewhere the malaria or some other parasite, that you have the complete and full explanation of the case. In tropical disease malaria is apt to complicate everything, so that multiple infection of patients is rather the rule than the exception.

When you find the malaria parasite the patient has certainly got malaria, but that does not exclude other infections. I have sometimes been "caught" in consequence of ignoring this obvious precaution. Some years ago I was asked to see a patient just returned from Portuguese West Africa. He was said to be suffering from fever and dysentery. He had dysentery sure enough, and his spleen was enormously enlarged. He had taken much quinine; as it seemed to irritate his bowels I stopped it. At my first visit he had no fever. I found nothing in his blood. I left instructions that I was to be sent for should he have an attack of fever.

Some days afterwards I was sent for, his temperature being over 103°. I took a slide of his blood, expecting to find in it the parasite of malaria. Judge, however, of my horror when, instead of the malaria parasite, there was an unquestionable trypanosome staring me in the face! After a few days the fever disappeared, and, with the fever, the trypanosome also.

A fortnight later there was again a return of fever, and I again examined the blood, expecting to find the trypanosome. I found no trypanosome, but I found plenty of tertian malaria parasites. And so the case went on, every now and again a fever spell with trypanosomes in the blood, and every now and again a fever spell with malaria parasites in the blood. By the persistent use of atoxyl and of quinine both infections were finally expelled from the circulation. The patient is now, I believe, quite well.

Last year I had in hospital a patient from an African colony who carried about with him the malaria parasite, the trypanosome, the *Spirochaeta pallida*, the filaria, besides an assortment of intestinal parasites, including *Ascaris lumbricoides*, *Trichiurus trichiurus*, and *Ankylostoma duodenale*—a veritable museum, which, as long as it remained with us, we appreciated very highly at the Tropical School. He could always supply us with a subject for demonstration or for a clinical lecture.

I fear my exposition of the subject has been very sketchy and inadequate; it is necessarily so in consequence of the time limit imposed on me. I trust, however, I have given you the leading points for reliable diagnosis.

COMMON MISTAKES IN OPHTHALMIC PRACTICE.*

By ARTHUR C. ROPER, F.R.C.S.ED., M.R.C.S.ENG.,
SENIOR SURGEON TO THE WEST OF ENGLAND EYE INFIRMARY, EXETER;
SURGEON TO THE ROYAL DEVON AND EXETER HOSPITAL.

IN casting round for a subject on which to write a paper which would prove interesting and useful, the idea came to me that I should formulate my impressions, gathered from twenty-five years of eye work, of the mistakes most frequently made by the medical men who have seen, and usually treated, eye cases before they came under my notice.

I use the word "impressions" because I have no statistics to give you. I do not commit to paper any mistake that I may think another man has made; but

yet, when one sees, as one does see, a considerable number of cases wrongly diagnosed and indifferently treated, they in a way sort themselves and occupy separate pigeon-holes in the recesses of one's brain. I should be sorry if this paper led any one to think that I pass a harsh judgement on a man who makes a wrong diagnosis on an ophthalmic case. I have lived too long and made too many mistakes myself to do this, and, moreover, I know very well the limited amount of experience in eye work that in the ordinary run of work falls to the lot of the general practitioner; and so, if any one finds in this paper a cap that he knows fits him well, let him not think that it is specially designed for his head, but rather consider that his head is fashioned on somewhat cosmopolitan lines, and is therefore capable of being fitted at the first ready-made and reach-me-down hat shop that he chances to enter.

Refraction.

The class of cases that provide the most numerous mistakes are undoubtedly refraction cases. One would expect this because refraction work forms such a large proportion of eye work, but I should think that, taking mistakes in this branch alone, they are quite in the proportion of 9 to 1—that is, that in 10 mistakes in eye practice 9 are due to bad refraction work. Nor is it astonishing that so many mistakes are made in this department, for while it has always seemed to me that constant practice at it is essential to its satisfactory performance, the public, and not a few of our profession, seem to consider that any one who sells spectacles is qualified to advise upon refraction troubles. When I occupied—as I did for twenty years—the post of assistant surgeon at the eye infirmary, where the bulk of the work is refraction, I always found that when I returned from my summer holiday my work took me half as long again as it was doing when I went away. Moreover, accuracy comes with speed, and you decide without hesitation such points as whether you shall give full correction in a hypermetrope, or whether it is worth while to give this child cylinders, or wise to give that myope a second pair of spectacles for near vision. These, however, are minor points, though they have oftentimes an important influence upon the reputation of the man whose work is bettered. The gross mistake, and the one which one most often sees, is where ciliary spasm in a hypermetrope is treated with -2 D. spectacles or -1 D.

The type of this is a young dressmaker, or perhaps a tailor's apprentice, who comes complaining that after a few hours' sewing things become misty and her eyes ache. She gets some relief by holding her work nearer to her eyes; but now this manoeuvre fails to relieve, and she is sure she needs spectacles. You test her, and find that she can read perhaps $\frac{2}{3}$ distant vision. You try her with a -1 D., and she says, "Oh, that's lovely; that's what I want"; or "Oh, yes, the print is beautifully clear." You provide her with the -1 D. or -2 D., whichever she sees best with, and she goes away seeing better than she has done for months, while you proceed to your next patient with the satisfactory feeling that "that is a good case." Next day your patient goes to work with a light heart, and finds everything very nice for a few hours; then the eyes again ache and smart. She thinks that they are not yet quite recovered from the bad treatment to which she subjected them before she obtained the glasses from you, and perseveres with them. But she finds that the aching returns at an earlier hour each day, and that her headache, which used to pass off when she left work, now persists all through the evening, and that only sleep, and even that not always, cures it. She then seeks other advice, is put under atropine, found to have hypermetropia, given her proper correction, and is thereafter comfortable, and wonders why you ever gave her those spectacles, which make everything look so tiny. The explanation of course is that you were deceived by the ciliary spasm. She came to you on account of this spasm. Her ciliary muscles, irritated by months of overwork in focussing correctly in a shortened eye had got to a condition of "tonic spasm" and over-accommodation, producing temporary short sight. You had easily corrected this short sight, but by doing this had added to her hypermetropia by precisely the strength of the concave glasses you prescribed. This led to greater efforts of accommodation, and to results which neither the eyes nor the patient could possibly put up with.

*Read before the Devon and Exeter Medico-Chirurgical Society.

Always, then, be exceedingly chary of ordering low concave glasses for anyone under 25 years of age, unless you have been able to ascertain definitely by the use of atropine that it is not a case of hypermetropia with ciliary spasm.

Squint.

Closely allied to this condition is that of internal squint in children. We so often get cases of bad squint brought to us with the history that it came on many months or even years ago; and, when we inquire why the parents did not see about it sooner, the reply usually is that "the doctor said the child would grow out of it." Now I am very far off believing all that people tell me about their doctors and the opinions that they have given. I know full well, from personal experience of the way people have interpreted and distorted my own opinion, how extraordinarily inaccurate the ordinary man, and still more so the ordinary woman, is in these matters; but this is such an oft-told tale that I feel sure the opinion is frequently given.* There is also some truth in the statement that the patient will grow out of it. "May" grow out of it would be quite true as far as it goes, but the whole truth would be "May grow out of it but with grievous loss of sight in the squinting eye and probable loss of binocular or stereoscopic vision." An opinion which causes any delay in the treatment of a squint in a child is hopelessly wrong and should never be given. For all practical purposes squint in growing children is always the result of refraction error. The exceptions are, in my experience, so few as to be negligible for the purpose of this paper, and the exceptions are usually caused by some lesion which is obvious and difficult to miss, such as corneal opacity. The vast majority of these cases of squint are convergent and when they are convergent they are associated with hypermetropia. When divergent they are usually due to myopia, or not infrequently to something which is rendering the diverging eye useless and so depriving it of the normal stimulus to move concomitantly with the other eye.

Let me briefly remind you of the physiology of accommodation and the supposed cause of converging squint; but first let me say that the last annual report of the Eye Infirmary shows that we had 97 cases of converging squint during the year and 10 of diverging; and of those 10 it is probable that not more than half were due to myopia.

As a working theory of the etiology or physiology of concomitant converging squint, Donders's is as good as any, and I can give you the outline of it in a few lines.

1. There is hypermetropia, that is, a short eye.
2. The short eye requires more accommodating power to focus objects on the retina than the normal or long (myopic) eye.
3. Convergence of the eyes is always associated with focussing. You will remember that the normal eye at rest is accurately focussed for distant vision. When objects nearer the eyes are looked at the internal recti converge the eyes so that they look at the desired point. Focussing—that is, increasing the convexity of the lens—also becomes necessary for a clear image to be projected upon the retina. Thus, convergence and accommodation are always associated, and so it comes about that convergence stimulates accommodation and increases it.
4. A hypermetrope of 2 dioptries has to be always accommodating to that amount to see distant objects clearly, and usually the ciliary muscle, which does the accommodating, manages it all right; but if the child gets some debilitating disease, or if anything occurs to weaken its ciliary muscles, convergence is brought into play to stimulate accommodation. Suppose, for example, that this hypermetrope wants a clear image of an object 30 ft. away. He converges to a point, say, 20 ft. away, and so excites the required amount of focussing. But if both eyes converge to 20 ft. neither will be directed towards the object at 30 ft.; and so it comes about that one eye is steadied by its own external rectus so that it looks at the desired object, while the other eye does convergence for both and so comes to be looking at only 10 ft. There you have the squint. One eye, the better one, is fixing the object to be seen, and the other is pro-

viding the necessary stimulus for accommodation by excessive convergence. We now come to the serious part of the business. It is obvious that the child will, the first time he performs this manoeuvre, see double, and so it soon comes about that the image of the converging or squinting eye is obliterated, and thus you get in most cases—

5. *Amblyopia* or impaired vision in the squinting eye. I will not even mention the many theories advanced to account for this. It is sufficient to say here that it does occur, and occurs so rapidly that some authorities think that the amblyopia is the predisposing cause of the squint, instead of the squint causing the amblyopia. Amblyopia means loss of sight, and it also means, if it is present to any important extent, loss of binocular or stereoscopic vision; and so I urge it upon you that at the first sign of squint you see that proper treatment by glasses is at once adopted.

All the treatment that is required at an early stage is correction of the hypermetropia by suitable glasses, and an operation for concomitant squint ought never to become necessary.

Ocular Headache.

One does not see nearly as many headaches from this cause overlooked as one did fifteen years ago. Medical men and the public have become thoroughly alive to the fact that this is a frequent cause of headache; but it is worth remembering that slight errors of refraction more frequently cause headache than serious errors. This is probably due to the fact that people with serious errors have them corrected early because they are aware that they cannot see properly.

Foreign Bodies.

Passing now from refraction troubles to actual pathological conditions, I will first just mention that minute foreign bodies in the cornea are a not infrequent cause of severe neuralgia in the forehead and temple, nor are these foreign bodies always easily discovered.

A man came to me recently with a story of frightful attacks of neuralgia, for which he had been treated by his doctor for a month, with no good result. He considered that the neuralgia was in some way due to his right eye, because bright light was the chief exciter of the pain. The attacks were becoming more frequent and more severe, and quite incapacitated him for his work. He could not remember anything getting into his eye, and the eye looked quiet and normal, save that there was slightly excessive lacrymation and a very contracted pupil. I suspected some corneal trouble and had herpes in my mind when I examined the eye, but, after a careful and minute search with a lens, I found two minute foreign bodies. They were very difficult to find and see, but their removal cured his neuralgia, though not immediately. I saw him a fortnight after, and he told me that he had had a few attacks, all of them shorter and less severe than formerly, but none for four days past, and he considered that they were gone for good. As I have not had another visit from him, I feel sure that he is cured.

There are, somewhat rarely, cases in which a foreign body has got into the upper sulcus of the conjunctiva, where it is reflected from the orbit on to the eyeball; these are easily missed, and it is not sufficient merely to evert the tarsal cartilage and look for the body on its inner surface. With a bent probe or a spatula you should, after everting the lid, lift it off the eyeball and look carefully along the whole length of the upper sulcus. Eyelashes are specially prone to lodge here and cause irritation with mucopurulent secretion.

Corneal Ulcer.

I now come to a more serious group of cases in which eyes are often lost for want of careful and vigorous treatment at an early stage. Ulcer of the cornea, with hypopyon, following an injury which is often very slight. Almost invariably these cases arise among the poor, underfed, and hard-worked labourer, and in my experience from one of two causes. The man has either been stone-breaking and his eye hit by a chip of stone, or hedge-trimming, when it has been flipped by a twig or scratched by a thorn. The apparent injury may be so slight as to elude detection even with a corneal microscope, but the eye is usually painful, angry-looking, and watering. If you do detect anything by careful examination, it is a small scratch, abrasion, or cleanly-incised wound, the latter from a sharp chip of stone.

Many men err from not treating this apparently slight injury with sufficient respect. They give a boracic lotion

*In the discussion following the paper Mr. Stirk, the Medical Inspector of School Children for Exeter, said that he could corroborate this statement, that parents of squinting children had often told him that doctors, consulted about the squint, had said "the child will grow out of it."

and perhaps some weak cocaine drops for the pain, and think that the eye will be all right in a day or so when the cut or abrasion has healed. A great many eyes are thus sacrificed every year. We had 20 such cases at the Eye Infirmary last year, and I doubt if one of them recovered with any useful vision, and I am sure that they provided a proportion of the eighteen eyes which were excised during that time. Such an eye should be treated as though it were inoculated with a virulent septic organism, as indeed it probably has been.

My advice to you is to send such a case to the Eye Infirmary at once. There is no money to be made out of them, because they have no money; there is no credit to be got from them, because if they get well they say that it was such a trifling injury that "of course it got well"; and if it does badly they say, and with some truth, "It was your fault." I always insist, when these cases come to me first-hand, that they shall come into the Infirmary. They require most careful nursing, antiseptic treatment, rest, and good food, and I cannot recall a case that has "gone wrong" when placed under these conditions. On the other hand, they go home with their bottle of boric lotion, which they pour into a dirty saucer, sop up with a not too clean cloth or rag, and squeeze into the eye with thoroughly septic fingers, and the eye does not get better. Slowly the cornea gets hazy with the hosts of leucocytes which muster to limit and destroy the invading and multiplying host of germs. The battle-field becomes a mortuary for the destroyed blood cells and germs, and this mortuary we recognize as a tiny yellow spot in the cornea—a minute abscess, which slowly, and day by day, grows larger. It not only loosens and destroys the surface epithelium, forming a shallow ulcer, but it penetrates inwards and perforates the inner, Descemet's, membrane, and the highly septic pus is then in the anterior chamber, to the bottom of which it gravitates as what we call hypopyon.

The local treatment which I adopt in the early stage of these cases is a drop of atropine, cocaine, and quinine. The atropine dilates the pupil before iritis sets in—a very good start. The cocaine soothes the pain, and the quinine is an efficient, sufficient, and un-irritating antiseptic. I also use a little yellow oxide of mercury ointment, and these, together with rest, good food, and thorough, frequent irrigation of the eye by a good nurse, are means which I find effective, sometimes even in cases which have reached the stage of abscess, and even slight hypopyon. If the case is a bad one, or threatens to be so, I cauterize the ulcer freely with the electric cautery, let out the hypopyon by an incision in the corneal margin, and thoroughly irrigate the anterior chamber with 2 per cent. boric solution or with normal saline. I am getting to use the cautery earlier and oftener in these cases, because I am convinced that the resulting opacity of the cornea is far less dense than is that which follows the natural cure.

Therefore, in cases of corneal trauma, especially from stone-breaking or hedging, watch with care and treat with vigour and precision. If possible, send them to an eye hospital for skilled nursing, rest, and feeding. If the case does well, a week or ten days in hospital will cure it; if it does badly, it will have been placed under the best conditions, and will be under treatment for many weeks.

Irido-Cyclitis.

Acute iritis is not often overlooked, but the low plastic type of irido-cyclitis is often most inefficiently treated, because its seriousness is not appreciated. I have had several such cases in the last two or three years, which have been mismanaged before they came to me, with vision reduced to less than $\frac{1}{20}$, with pupils not properly dilated, and so forth. Many are misled by the apparent mildness of the trouble. The eye is only very slightly inflamed, scarcely pink. There is absolutely no pain, and usually the only complaint is that sight is failing. On examination you may find that the iris does not react to light, or only very sluggishly. After atropine, you find that it has dilated either irregularly or not at all. Perhaps, if you are early enough, it dilates regularly but not fully, and you see spots of uvea on the lens. If you look at the fundus you find that the view is not clear, because there are cobwebby opacities in the vitreous; and, finally, close examination of the cornea shows you a few or many minute pin-point specks on its lower segment—keratitis punctata.

Such an eye is the seat of a very nasty and intractable disease. It will take months, running very likely into years, to recover, and will almost certainly suffer visual loss as the result of the inflammation which permeates it. The mistake which is usually made is that the iritis is recognized and treated, very likely correctly, with atropine, salicylates, iodide of potassium, or what not, and that the case is given up as cured when active iritic symptoms have passed. Men forget that the iris is, though physiologically a separate organ, anatomically continuous with the ciliary body and choroid, so that inflammation can, and does, spread backwards by continuity of tissue. In a case of simple iritis I am always very diffident about leaving off atropine, and watch the case anxiously for a month or two after omitting it from the treatment.

Do not forget that the iris is only the anterior termination of the uveal tract, and that an inflammation of it may spread back and involve the choroid, retina, and vitreous. Look out for keratitis punctata.

It may impress it upon your minds if I tell you that this condition is practically the same as that which you all know and dread as sympathetic ophthalmia. One sees very few cases of that nowadays, and there is no one here who would not at once recognize it if it arose in his practice after injury to the other eye. That is sympathetic irido-cyclitis, and what I have been speaking of is simple or, if you like, idiopathic irido-cyclitis, and is often as formidable a disease.

Glaucoma.

I feel that you will expect me to say something about the diagnosis between iritis and glaucoma, and at the same time I am conscious of being quite unable to condense the subject into a paragraph. The mistake is still made of using atropine for a glaucomatous eye, and is made in cases in which ignorance is not only the excuse for it, but the reason. A typical case of glaucoma in the acute or subacute stage is like nothing else. The suddenness and severity of the onset, the hardness of the globe, the partial dilatation of the pupil and its immobility, the shallow anterior chamber, and the steamy cornea, together with the rapid loss of sight, form a very perfect clinical picture. In iritis the onset is more gradual, the tension is but little if at all increased, the pupil is contracted and immobile, the anterior chamber is not diminished in depth, the cornea is not hazy, and the loss of vision is neither rapid nor, at first, great. But these are the easy cases to decide upon. One occasionally sees cases which give rise to great difficulty in diagnosis, and I believe that when you are in such a difficulty the best thing you can do is to order cocaine drops for the relief of pain, and send the case to a specialist at once.

I remember once having to do iridectomy for an acute glaucoma produced by the application of a mydriatic, by one of the best eye surgeons in England, for the purpose of investigating the condition of the lens. He suspected and found cataract, but he did not suspect chronic glaucoma, which was undoubtedly present, and which was exacerbated by the temporary dilatation of the pupil. Men occasionally, and at some risk, use a mydriatic in middle-aged and older people for the purpose of examining the interior of the eye for some unexplained loss of sight. Before doing this they should satisfy themselves that chronic glaucoma is not the cause for which they are seeking, because, if it is, they are running a very great risk of producing an acute attack.

Before using a mydriatic in a person of middle age or over, satisfy yourself that there is neither subacute nor chronic glaucoma present.

Chronic glaucoma is an insidious disease and a dangerous trap for which you should be always on the alert. I have recently seen two very sad cases of this disease in which total blindness was present when they came to me. The cases had passed, by months or years, the period when good could be hoped for from treatment. When a patient past middle age comes to me complaining of failing sight and with a normal looking eye, if correction of the refraction does not produce normal vision I think first of cataract and next of chronic glaucoma. The thing to bear in mind is that the disease is usually far advanced when the vision has become seriously affected, and that there is no time to be wasted in improving the general health in the hope that the sight will improve. The diagnosis in the early stages when treatment may avail to save sight is not easy,

and when the diagnosis is plain and the case a typical one such as textbooks describe, treatment is too often of no avail. It is not very uncommon to find a case of this disease in an advanced stage but able to read $\frac{5}{6}$ and 1 J., but when one examines the field of vision one finds it contracted to very small limits. This is, of course, because the intraocular pressure operates first on the periphery of the retina, where the vascular supply is more easily compressed than it is in the central portion.

It is difficult to give any "tip" for the diagnosis of chronic glaucoma. It always attacks both eyes, I think, but not together, so that if one eye has been lost from it you may expect it in the other. If you can make out that the tension is increased, you are safe in diagnosing it; but you are not safe in saying that a case is not chronic glaucoma because there is no increase of tension, because the tension varies a great deal in some cases, though it is probably increased at times in all. Nothing less than a careful routine examination of the eye and its functional power will suffice for making a diagnosis in an early stage.

Cataract.

There is one silly little mistake which I have made dozens of times, but have not communicated to the patient, in diagnosing cataract by focal (or side) illumination only. In some elderly people the hardened lens reflects the light in such a way as to give the pupil a milky appearance, which is not unlike the pearly reflection of a mature cataract. There is not necessarily cataract present in such a case, and the diagnosis is not good unless you can confirm it with the ophthalmoscope. With a plane mirror at the usual distance for indirect examination throw a light through the pupil on to the fundus. If cataract is present in the pupil you will see the red reflex interrupted by black streaks or dots or spots; or, better still, scrutinize the lens by the direct and close method, and with a +25 D. lens in your ophthalmoscope. That will show you the most minute traces of lens opacity, but requires some expertness. The former method is quite sufficient for all practical purposes, and extremely simple.

CONCLUSION.

I have not exhausted the category of mistakes, and I have not mentioned the question of fundus conditions which cannot well be discussed until they have been demonstrated.

It was not my purpose to deal with the rarer conditions of disease or traumatism, or to exhaust the possibilities of the eye as a field for making mistakes in. I have contented myself, and I expect tired you, with the common mistakes in ophthalmic practice.

THE VALUE OF SOME LACTIC ACID FERMENT PREPARATIONS FOR INTESTINAL THERAPY.

BY

I. WALKER HALL, and

W. A. SMITH,

M.D.,
PROFESSOR OF PATHOLOGY AND
BACTERIOLOGY, UNIVERSITY
COLLEGE, BRISTOL; AND
PATHOLOGIST, BRISTOL
ROYAL INFIRMARY.

M.B.Oxon.,
BRISTOL; LATE SURGEON,
SAFFRON WALDEN
HOSPITAL.

DURING the course of an investigation upon faecal organisms it was found necessary to determine the inhibitory action of certain types of lactic acid bacilli. Some of the bacilli were isolated from ten commercial lactic acid ferments, and it was suggested to us that a statement of our observations might be of interest to those who use these ferments in everyday practice.

The majority of these ferments are sold in the form of dry tablets, obtained by inoculating the milk with an organism, evaporating, and then compressing the residue, or by mixing a dried culture of the organism with a suitable excipient. These tablets are directed to be added to milk which has been boiled for a short time and then allowed to cool; the milk is then to be kept at blood heat for six to eight hours. After this period of incubation the milk is said to be sufficiently "soured" for therapeutic uses. In a few cases it is advised to dissolve the tablets

in sweetened water before consumption. The object of these procedures is the introduction of lactic acid bacilli into the ileum and colon in order to inhibit the action of putrefactive bacteria.

The results of the simple experiments we have carried out tend to show that the ferments at present on the market do not achieve the end in view, and they suggest that until there is a decided improvement in the processes of manufacture the dangers attendant upon the use of these tablets outweigh their benefits. Whenever the treatment is specifically indicated it would be well to obtain young, active, and specially adapted (aërobic and anaërobic) cultures in a fluid medium from local public health or other laboratories, and to determine the efficacy of the organism by bacteriological examinations of the faeces. With suitable media and precautions, such a proceeding need not involve more than the outlay of a few shillings, and the ultimate expense will be considerably less than that occasioned by the longer haphazard medication with inert or inefficacious material.

Development of Acidity.

Since the ferments are used in connexion with milk, we first determined the acidity developed in raw, pasteurized, and sterilized milk during incubation at blood heat. It will be seen from Table I that in pasteurized and sterilized milk a considerable amount of acid is formed, and that it is necessary to sterilize the milk on two successive days in order entirely to free the milk from micro-organisms.

TABLE I.—The Acidity Developed in 25 c.cm. of Milk during Incubation in Sterile Flasks at 37° C. (98.5° F.).

	Acidity when received. Deci-normal Alkali.	24 hours. Deci-normal Alkali.	72 hours. Deci-normal Alkali.	96 hours. Deci-normal Alkali.
Raw milk	12	25.0	96.0	—
Pasteurized milk	6	11.5	19.5	21.0
Pasteurized milk, the acid produced each 24 hours being neutralized	6	11.4	30.9	63.4
Pasteurized milk heated to 100° C. for 1 hour	6	13.0	29.0	36.5
Pasteurized milk heated to 100° C. for 1 hour on two successive days	6	—	—	—

These figures emphasize the observations of Robertson and Muir,¹ who found that:

Milk pasteurized at 70° C. for 30 minutes killed off cocci, lactic acid *coli*, *proteus*, and ordinary pathogenic bacilli.
Milk heated to 100° C. for 1 to 1½ hours killed off anaërobic spore-bearing bacilli, including *Bacillus enteritidis*.
Milk heated to 100° C. for 2 hours killed off *subtilis* group and aërobic spore-bearing bacilli responsible for summer diarrhoea.

As it rarely happens that the domestic heating of milk ensures complete sterilization, it is obvious that the addition of "ferments" to milk partly sterilized induces considerable antagonism between the added bacilli and the unkilld organisms, which, freed from the restraint of the ordinary milk bacteria, are multiplying more rapidly than usual. In summer time some of these resistant organisms possess pathogenic properties, and their free growth is not without danger. If this form of treatment demands the "souring" of milk, it is obvious that the process should be carried out with new milk collected under cleanly conditions. The slight bactericidal properties of fresh milk may then act upon undesirable organisms and the lactic acid bacilli outgrow other bacteria. As this, however, requires several days for completion, we are forced to the conclusion that the conditions of the collection and sale of milk in this country make it an untrustworthy vehicle for the administration of this medicament. It is better, apparently, to discard milk and to administer the cultures in a liquid sugar medium.

I.—The Acid Contents of the Tablets.

It appeared to be worth while to determine the acidity of each tablet in order to ascertain the activity of the medium from which the tablet was prepared. The figures