Lisping.

A CLERGYMAN, with usually an exceptionally distinct utterance, was observed one Sunday morning at the beginning of the service to speak with a pronounced lisp. After a time it wore off, and his speech became as clear as usual. Has it ever occurred to anyone what a very simple thing may cause a lisp? The case in question was owing to a tiny slice of lozenge sticking to the roof of the mouth just to the left of, and close to, the front tooth. This almost imperceptible impediment was sufficient to render the speech so indistinct as to resemble a marked lisp. Of course as the lozenge dissolved the lisp became no longer observable, and the speech assumed its ordinary clearness.

These being the facts, the question that occurs to every thoughtful mind is, If the cause of lisping be so simple, why cannot the remedy be as simple and yet effectual?

The answer I leave to be supplied by some of your scientific readers. A NON-LISPER.

ETIOLOGY OF SCARLET FEVER.[1]

AMONG the infectious or zymotic diseases there are two at any rate (namely, scarlet fever and diphtheria) of which it may be said that their spread is to a lesser extent dependent on defective domestic sanitation than is the case with some of the other zymotic diseases, as, for instance, typhoid fever. Indeed, it is maintained by competent authorities that scarlet fever and diphtheria do not invade the houses of the poor with faulty sanitation with greater frequency or with greater severity than those of the well-to-do, however perfect the sanitary arrangements. This view is based on the important experience gained during the past twenty years, viz. that epidemics of scarlet fever and diphtheria have been brought about by milk. I may here state by way of explanation that a fact well established, and needing no further comment, is that scarlet fever and diphtheria are, like small-pox, measles, whooping-cough, and typhus fever, communicable directly from person to person. This mode of infection, doubtless an important one, and coming into operation in single cases wherever the elementary rules of isolation and disinfection are transgressed, altogether sinks into insignificance when compared with the infection produced on a large scale, if a common article of diet like milk should become in some way or another the vehicle of contagium, as has been proved to be the case in a number of epidemic outbreaks. These epidemics, known as milk scarlatina, milk diphtheria, and I may add also milk typhoid, have this in common, that almost simultaneously, or at any rate within a short time, in a number of houses, having no direct communication by person or otherwise with one another, there occur sometimes singly, sometimes in batches, as it were, cases of illness: scarlet fever, diphtheria, or typhoid fever as the case may be. And it was this peculiar character which pointed to a condition which must have been common to all these households. On closer examination it was indeed found that all these households had this, and only this, in common, that they were all supplied with milk coming from the same source—that is to say, from the same dairyman. Other houses supplied with milk from a different source escaped; and further it was shown that, as soon as the consumption of the suspected milk ceased also, the epidemic, as such, came to an end, except of course the cases due to secondary infection from person to person. The Medical Department of the Local Government Board have had for years past their attention fixed on these milk epidemics, and in the Reports of the Medical Officer many of these are described with great detail; amongst these, Dr. Ballard's Report in 1870 on enteric fever in Islington, Dr. Buchanan's in 1875 on an outbreak of scarlet fever in South Kensington, and Mr. Power's on an outbreak of scarlet fever in St. Giles and St. Pancras in 1882, are specially to be referred to. Mr. Ernest Hart has tabulated all the outbreaks of milk epidemics that have been investigated until 1881, in vol. iv. of the Transactions of the International Medical Congress for 1881. Now, analyzing these outbreaks as far as they refer to scarlet fever, there are several of them where the assumption that the milk acquired the power of infection by contamination from a human source cannot be excluded. This infection if proven would stand on the same footing as if due to contagion from person to person, for it is clear whether the contagium is conveyed from one person to another by air, food, drink, or other articles, it always remains contagion from person to person. Now, in some of the epidemics tabulated by Mr. Hart, and recorded by subsequent observers, *i.e.* after 1881, this mode of milk contamination cannot be excluded, as I said before; but comparing the dates when the milk might be supposed to have become so contaminated with the dates when the milk has actually produced infection, it will be found that a certain discrepancy exists, and as will be shown later another mode of infection, viz. from a person affected with scarlatina to the cow, and through the cow to the milk and then to human beings, cannot be excluded either. There are other epidemics recorded in these tables, in which the mode of infection of the milk is not ascertained; and in a third set, the milk acquired infective power in some way or another, but certainly not from a human source. As an illustration of the first group of epidemics, *i.e.* probable contamination from a human source, I will refer to the table given by Mr. Ernest Hart on page 539:—

1881, April.	Keswick.	J. Robertson, M.D., M.O.H.	A dairy closely adjoined a house where scarlet fever had existed for several weeks. The cows were milked, every night and morning, into an open tin can carried across an open yard past the affected house.	The children who first caught scarlet fever in the locality played about the yard whilst in a state of desquamation.	On one particular day a general epidemic of scarlet fever broke out in the town, between thirty and forty families being invaded. All those suffering from the disease received their milk-supply from this particular dairy-farm. Some member of every family supplied had either a scarlatinal sore throat or scarlet fever on this day. Other families supplied from a different source escaped the disease.	A lodger had the milk raw for supper and was attacked. His landlady boiled her milk the same night and escaped.

[1] Lecture delivered by Dr. E. Klein, F.R.S., at the Royal Institution on Friday, May 27, 1887.

Now, mark this, that on one particular day the fever broke out. We will return presently to this point.

As an illustration of the second kind (viz. probably not from a human source), I will refer to the outbreak of scarlet fever in Oxford in the spring of 1882, recorded by Dr. Darbishire in the St. Bartholomew's Hospital Reports, vol. xx.

The substance of Dr. Darbishire's Report is this:—Three cows were kept by those who sold the milk, and nine houses, containing eighty-five persons in all, were supplied morning and evening; the milk was never stored, as there was generally barely enough at each milking for all the customers. In the house to which the cows and paddock belonged, there was a case of diphtheria in a young lady. She was removed to the infirmary on March 1. The cowman had a child ill with scarlet fever in his cottage from February 27 till March 3. On March 3, Dr. Darbishire had this child removed to the hospital and the cowman's cottage thoroughly disinfected; the cowman left his cottage to sleep in lodgings near, the care of the cows having been handed over to another man, engaged for that purpose. Now, if the milk had become infected from either of these two cases (one diphtheria and the other scarlet fever), this must have occurred for the first before March 1, for the other before March 3; and as the period of incubation of scarlet fever is known to be as a rule less than seven days, it follows that March 3, being the last day on which the milk could have received the contagium from a human being, March 10 would be the last day on which scarlet fever could have been produced by that milk, and the majority of cases of scarlet fever must have occurred before that day, as one cannot assume that in all these cases the period of incubation was protracted to such length as seven days. But mark what really did happen. Dr. Darbishire states that no case occurred till March 10, on which day 2 cases of sore throat and 1 case of scarlet fever occurred; on March 11, 1 case of sore throat; March 12, 2 of sore throat and 1 of scarlet fever; March 13, 4 of sore throat and 2 of scarlet fever; March 15, 1 of sore throat and 1 of scarlet fever; March 16, 2 of sore throat and 1 of diphtheria; March 17, 1 of sore throat; March 18, 1 of sore throat.

Now, all these cases were proved by Dr. Darbishire to have been caused by that milk. There occurred subsequently other cases, but these were traced to have been due to secondary infection from person to person.

This is a good illustration of a milk epidemic, in which the milk most probably did not receive the contagium by human agency. And there are other milk epidemics which on analysis of dates lead to the same conclusion. The infection of this milk was probably brought about as I shall show you hereafter in some other way.

As an instance of the third kind, viz. where milk has clearly not been infected from a human source, I will refer to Mr. Power's Report in 1882 on an epidemic outbreak of scarlet fever in St. Giles and St. Pancras. "The disease was distributed with a milk service derived from a Surrey farm. In this case two facts could be affirmed: the one that a cow recently come into milk at this farm had been suffering from some ailment, seemingly from the time of her calving, of which loss of hair in patches was the most conspicuous manifestation; the other that there existed no discoverable means by which the milk which had coincided with scarlatina in its distribution, could have received infective quality from the human subject." (Medical Officer's Report for 1885-86, pages v. and vi.)

The Medical Department of the Local Government Board, have from these facts drawn the conclusion that "distrust must be placed on the universally accepted explanation that milk receives infective properties directly by human agencies," and further that "the question of risk from specific fouling of milk by particular cows, suffering, whether recognized or not, from specific disease, was seen to be arising." This view received striking confirmation and proof by a report of an outbreak of scarlet fever that occurred at the end of 1885, and the beginning of 1886, in the North of London, which was investigated by Mr. Power; his report is published *in extenso* in the Report of the Medical Officer of the Local Government Board for 1886. I will here give you the substance of it. Mr. Wynter Blyth, Medical Officer of Health for Marylebone, "had last December observed a sudden outbreak of scarlatina in his district to be associated with the distribution of milk coming from a farm at Hendon, and had found reason for believing that the disease had prevailed exclusively amongst customers furnished with milk from that source." Mr. Power on a more extended inquiry found that a similar prevalence of scarlatina had occurred about the same time in other parishes in and near the metropolis that were furnished with milk from the same farm. By careful inquiry, Mr. Power could with certainty exclude any contamination of the milk from a human source, or that anything of the kind known as "sanitary" conditions could have had any concern with the infectivity of the milk. Mr. Power showed conclusively that only certain sections of the milk-supplies of this farm, and finally only certain cows from which these sections of milk were derived, had any relation to the observed results. "In the end," says the Medical Officer, "he has demonstrated, beyond reasonable doubt, the dependence of the milk scarlatina of December on a diseased condition of certain milch cows at the farm—a condition first introduced there in the previous month by some animals newly arrived from Derbyshire; and he finds strong circumstantial evidence for believing that the later phenomena of this dependence were brought about through the extension of the diseased condition of one set of animals to another set, after the fashion of an infection."

Now this disease, as it presented itself in some of these Hendon cows, consisted in the presence of sores in different parts of the skin with loss of hair in patches, ulcerations on the udder and teats, and a visceral disease, notably of the lungs, liver, kidney, and spleen, which, although milder in character, very much resembled the visceral lesions occurring in cases of human scarlet fever. By experiment it was shown that the matter of the ulcers of the udder is possessed of infective power, inasmuch as on inoculation into the skin of calves the same ulcers are reproduced; further, it was shown that in the ulcers of the cow there existed in large numbers a species of micrococcus, which, on being planted on artificial nutritive media, such as are used for the study of bacteria, produces in a few days a crop of micrococci, possessed of very distinct characters by which they are distinguishable from other bacteria.

When calves are inoculated from a cultivation of this micrococcus, they become, after an incubation period, affected with a cutaneous and visceral disease the same as the disease of the Hendon cows. From the blood of these animals the same microbe was recovered by cultivation.

To sum up, then, it has been shown that at this Hendon farm there existed certain cows affected with a communicable disease which, in many points of its pathology, bears a great resemblance to human scarlatina; further, that the milk of these cows gave scarlet fever to human beings; and, lastly, that a particular microbe was obtained from these cows, which in calves produced a disease similar to the one from which those cows were suffering. In order to complete the evidence thus far obtained, it was necessary to prove that scarlet fever in man is due to the presence and multiplication in the blood and tissues of the same micrococcus, and that this microbe, if obtained from human scarlet fever, produces in the cow the same disease as is produced by the micrococcus of the Hendon cows. Now, this proof has been satisfactorily given. In the first place, it has been shown that in the blood and

tissues of persons affected with scarlet fever there occurs the same micrococcus as was present in the cow, both being identical in microscopical and in cultural characters. In the second place, it was found that the action of this microbe on animals is exactly the same as the micrococcus found in the Hendon cows. Calves and mice, after inoculation or feeding with a trace of the growth of both sets of micrococci, become affected with cutaneous and visceral disease similar to human scarlet fever; in calves, the disease is of the same mild type as in the Hendon cows. I have lately ascertained that milch cows inoculated with the human scarlet fever micrococcus developed readily a disease identical in every respect with the Hendon disease, inclusive of the ulcers on the teats, and the sores and loss of hair in patches in different parts of the skin. Further, it was shown that from the blood and the tissues of these animals infected with one or the other set of cultivations, the same micrococcus was recovered. I will remind you that, in all infectious diseases which have been proved definitely to be associated with a particular species of microbes, this microbe introduced into a susceptible body thrives and multiplies, and thus sets up the diseased condition, differing of course with the different species of microbes. I think I may after this say that this microbe, *Micrococcus scarlatinæ*, is the cause of human scarlet fever; further, that it produces in bovine animals a disease identical with the Hendon disease and human scarlet fever, and that consequently, while the cow is susceptible to infection with human scarlet fever, it can in its turn be the source of contagium for the human species, as was no doubt the case in that milk epidemic from the Hendon farm.

I shall now give a striking piece of evidence well in harmony with what I have mentioned hitherto. In October 1886, Prof. Corfield forwarded to me certain tins of condensed milk, sold under the name of "Rose brand." This milk was under suspicion of having produced scarlet fever in a number of persons that had partaken of it. From one out of three tins of this condensed milk, I have obtained by cultivation a microbe which in every respect, morphologically and in cultures, is the same as the microbe obtained from the Hendon cows and from human scarlet fever. The action of the microbe of the condensed milk was also tested on animals, calves and mice, and it was found that it produced the identical disease that was produced by the microbe of human scarlet fever, and of the Hendon cows. I may add that this Rose brand of condensed milk is, like all condensed milk, obtained from cows' milk. The Rose brand is a cheap article, and meant for the poorer classes; probably it has not been sufficiently heated in the tins before sealing the latter; that this is so can be inferred from the fact that every tin of this brand which I opened contained some organisms. Thus, for instance, I find that one tin contained the scarlet fever microbe and another species of micrococcus; another tin contained a harmless species of micrococcus only; and a third tin opened contained a micrococcus and a species of bacillus.[1]

Another piece of interesting evidence concerning the *Micrococcus scarlatinæ* is this: there occurred during the beginning of this year a severe epidemic of scarlet fever in Wimbledon. This epidemic was also traced to milk coming from a particular farm. In one of the houses supplied with this milk there occurred cases of scarlet fever amongst human beings, and at the same time a pet monkey, who also consumed a good deal of the milk, became ill; it died after five days. I had the opportunity to make a *post-mortem* examination of this animal, and there could be no doubt about its having died of scarlet fever. From the blood of this monkey I obtained by cultivation the same micrococcus as was obtained from human scarlet fever, from the Hendon cows, and from the condensed milk. Experiments made on animals with this micrococcus of the Wimbledon monkey showed that the same disease is produced both by inoculation and by feeding.

It having been proved, then, that the cow is susceptible to infection with scarlet fever from man, the next important question is this, How does the milk of such infected cows assume infective power? Clearly in one of two ways: first, either the milk becomes infected by the milker during the process of milking, particles of contagium being rubbed off the ulcers of the udder or teat; or, the milk *per se* is possessed of infective power—that is, it being a secretion of a constitutionally diseased animal. From previous and from more recent observations, I am inclined to think that both views hold good.

I now come to the question, How is the spread of scarlet fever by milk to be controlled and checked? This question resolves itself into three parts. First, prevention of infection of the cow by man, directly or indirectly; second, prevention of infection of the cow by the cow; and third, destruction of the contagium of the milk of such cows.

As regards the first, all those rules which have been laid down to prevent infection of one human being from another, of milk or any dairy utensil by contact or otherwise with a person suffering from scarlet fever or coming from an infected house, apply also here; and this part of the subject comes under the general aspect of the proper sanitary management of dairies, which is acted upon in all well-managed dairies.

As regards the second, viz. prevention of infection of the cow from the cow, this is obviously more important and more difficult to be carried out. I say obviously, because one cow affected with the disease is capable of communicating it to others in the same farm, and when moved to another farm also to the cows there.

The disease in the cow being of a mild character is easily overlooked. The disease in the skin of the cow may be present and slight, or may be absent in its more conspicuous manifestation, whereas the visceral disease is of so mild a character that it requires an expert to diagnose it. When a cow shows the disease of the skin and on the udder well pronounced, such an animal will have to be carefully examined for visceral disease. I need hardly say that amongst the many cutaneous disorders of the cow, known and unknown, there may be one or the other which bears a resemblance to the cutaneous disorder occurring in scarlatina; such cutaneous disease must be carefully excluded before an animal is condemned; but, if visceral disease should be diagnosed as well, the animal should be carefully isolated and its milk should not be used. And it must be clear from this that every dairy should be permanently under the supervision of an expert, and in this the veterinary profession should be as eager for the work as the medical sanitary officers are, and for some time past have been. But judging from the attitude assumed by the veterinary authorities I am afraid the veterinary profession has not yet grasped the full responsibility that rests on them, both towards the general public and the dairy farmers. Instances are on record, when, on the milk from a particular farm having been proved or even suspected to bear any relation to a scarlet fever epidemic, the business of such farm became temporarily or even permanently suspended, and the pecuniary loss of the owner of such farm irrevocable. That the disease in the cow which I have described to you as scarlet fever is as yet unknown to the veterinary profession does not do away with the existence of such disease, and I venture to say that the fact of its being as yet unknown to and unrecognized by them should stimulate them to try to recognize it.

Now the third question, as to the destruction of the

[1] It is well known that no species of micrococci hitherto known are capable of surviving a temperature of 212° F., *i.e.* of boiling water; many of them are killed by an exposure to 180°–190° F.

contagium in the milk. This, I am glad to say, is very easily carried out. Heating milk up to 85° C. or 185° F., that is, considerably under the boiling-point, is perfectly sufficient to completely destroy the vitality of the microbe of scarlet fever. In harmony with these experiments on the influence of heat on the microbe of scarlet fever, I can quote, besides the observation given above by Dr. Robertson, also the following observations recorded by Dr. Jacob, Medical Officer of Health of High Ashurst and Headley, and reported in 1878, to this effect. Between June 1 and 7, there were fifteen cases of scarlet fever in three distant houses, the inmates of which had had no communication with infected persons, but had all been supplied with milk from a farm where a certain cowman worked. This cowman had in his family several children ill with scarlet fever. The cowman continued milking the cows during the illness of his children, though he did not himself have the fever, and the milk was not taken into his cottage; but the point which I wish to bring out is this, that other houses besides those in which scarlet fever had broken out had been supplied with the same milk, but no scarlet fever occurred in them, and why? because all these had consumed only the scalded milk.

I should therefore strongly urge that all milk should be boiled, or at any rate heated to at least 85° C. (that is 185° F.) before being consumed. Judging by the large number of cases of scarlet fever recorded in these milk epidemics, one is justified in saying that a considerable percentage of the total number of cases of scarlet fever would have been avoided thereby. Not all, because unfortunately the rules of isolation of patients suffering from scarlet fever are not always rigorously carried out, and therefore infection from person to person will occur. Nor would prevention of scarlatina by milk exclude scarlatina by cream,—cream cannot be easily subjected to heat; and in the epidemic of scarlet fever that occurred in South Kensington in 1875, and that was investigated by Dr. Buchanan, cream was the vehicle of the contagium. But considering the prominent position that milk occupies in every household with children, the possibility of infection with scarlet fever by raw milk deserves careful attention.

THE SECOHMMETER.

A CIRCUIT containing self-induction acts as if it had a larger resistance than its true one when a current is started in it, and a smaller resistance when the current is stopped. Hence, if balance be obtained with a Wheatstone's bridge in the ordinary way, the fact of any of the arms possessing self-induction, or of any one of the arms having a condenser attached to it, will produce no effect on the balance if the battery circuit be rapidly made and broken, provided that the rapidity of make and break be not too great for the currents in the arms of the bridge to reach their steady values each time that the battery circuit is made, and to die away each time that it is broken. If the currents have not time to reach their steady value when the battery circuit is closed, and to die away when it is broken, then self-induction in any one of the arms will produce a disturbance in the balance; but such a method of measuring a coefficient of self-induction would lead to very complicated formulæ, and is not worth developing with the view of obtaining a simple method of measuring self-induction.

It therefore occurred to us to consider whether, without employing such rapid makes and breaks as would prevent the currents reaching their steady values, the self-induction of a circuit might not be made to act as an apparent steady definite increase of the resistance of that circuit which could be measured in the ordinary way with a Wheatstone's bridge or differential galvanometer; and by this means the measurement of a coefficient of self-induction would simply resolve itself into the measurement of a resistance. And this problem we solved in the following way, in the spring of 1886:—

The coil, the coefficient of self-induction of which it is desired to measure, is placed in one of the arms of a Wheatstone's bridge, the three other arms consisting of ordinary doubly-wound resistance coils possessing no appreciable self-induction, and not only is the battery circuit rapidly made and broken, but, in addition, after each closing of the battery circuit the galvanometer circuit of the bridge is either short-circuited or broken, so as to cut out the galvanometer, and after each breaking of the battery circuit the galvanometer circuit is either unshort-circuited or closed again, so that the galvanometer is now operative again. In this way all the successive impulses of the galvanometer needle that are produced on starting the current in the coil with self-induction produce their *cumulative* effects, but the successive impulses of the needle that, under ordinary circumstances, would be produced on the needle in the opposite direction are cut out. Hence the self-induction possessed by one of the arms causes that arm to apparently increase in resistance by a definite amount depending on the coefficient of self-induction and the number of operations performed per minute. This apparent increase of resistance produces a deflection of the galvanometer which can be noted, and its value ascertained by comparing it with the deflection produced with steady currents when one or more of the arms of the bridge is altered by a known amount, as in making the Rayleigh test. But since the necessity of having to read the deflection limits the speed of performing the double make and break operation, in order that the spot of light may not be sent off the scale, we soon replaced this comparative deflection *cumulative* method by a much more sensitive *zero cumulative* method; and instead of reading the galvanometer deflection we re-establish the balance, and bring the needle back to zero, by altering one or more of the arms of the bridge, as in making an ordinary resistance test with a Wheatstone's bridge.

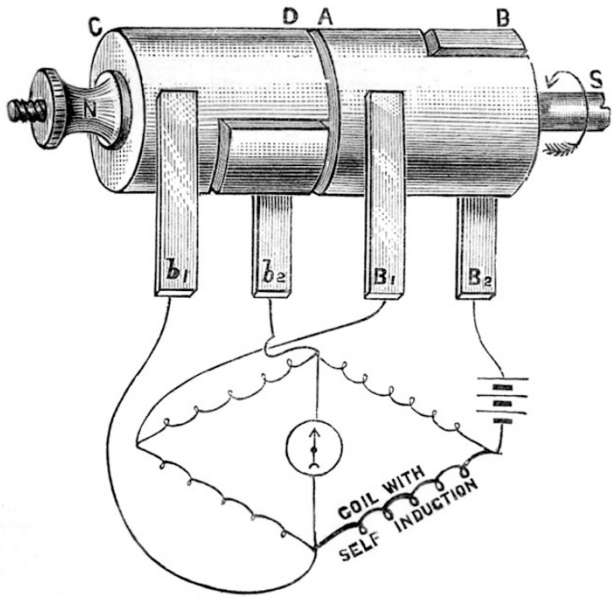

FIG. 1.—Preliminary Apparatus.

The first apparatus for enabling measurements of self-induction to be made in this way was constructed in the spring of 1886, under the superintendence of one of our assistants, Mr. Mather. It consisted of a double commutator, shown in Fig. 1, the spindle, S, to which the