paratively late, the various vomits following a definite sequence of which faecal vomit is the last and comparatively late stage, except in impacted gall stone, whether in the cystic duct or intestine, when it appears very early.

4. The amount vomited is far greater than the amount taken in.

5. In cases of intestinal occlusion—say, of annular stricture of the small intestine—the more the faecal vomiting, the more relieved the patient should feel. On the contrary, the more toxic he appears.

6. In cases that recover there is an intense diarrhoea at first of small quantity of dirty brown material, then of large amount of light liquid, lemon-yellow faeces, showing that the liver cells have not regained their normal powers.

7. If the faecal vomiting occurs sometime subsequent to the insertion of a drainage tube in the peritoneal cases—that is, non-occlusion cases—then the patient will probably recover; whereas if operation is deferred until it has occurred the case is hopeless. An exception must be made in the case of impacted gall stone, as faecal vomiting is so often an early symptom, and followed by temporary relief.

8. A most fatal objection is that the higher up the obstruction the more profuse is the vomiting. The contrary should be the case. My explanation of this phenomenon is that the number of bacilli which should have been spread over the whole length of small intestine now flood the upper reaches of peritoneum, and stress is more rapid and urgent on stomach and liver: the need being great early, the liver early puts forth all its strength.

The reaction and colour of the vomit have, to me, become important indications, not only in the diagnosis, but still more in the prognosis of inflammatory conditions inside the abdomen.

For instance, if the case has been ill for more than three or four days and the vomit is still yellow and acid, I know there is no perforation or acute obstructive condition, but simply inflammatory, and if surgical interference is necessary, the prognosis is good. On the other hand, if by this time the vomit is black or coffee-coloured, the diagnosis is that of acute virulent, thin turbid fluid peritonitis—for example, pneumococcal (rare and fatal cases), or perforative gangrenous lesions of some part of the intestinal tract, and if surgical aid has not already been given the case is absolutely hopeless. A dark, spinach-green vomit before operation makes the prognosis exceedingly grave. A home-made beef-tea-like vomit generally indicates pus and a certain amount of lymph, and one sees it in some cases of gonorrhoeal peritonitis and general purulent peritonitis from appendicitis. Operation, to have any hope of success, should be undertaken instantly. The prognosis is bad. Early faecal vomiting is a certain sign of impacted gall stone, and prognosis, if operated upon, is good. Late alkaline faecal vomiting means acute strangulation, acute gangrenous appendicitis, etc., and operation, if it has not been previously undertaken, is now out of the question. If it comes on after operation, is acid or neutral, with clean tongue, even though there still be absolute constipation and great tympanites, if the pulse be under 100, the prognosis is quite good.

In operating on septic abdominal cases it should be remembered that the liver and stomach, if chloroform is given, have to excrete this drug from the circulation as well. The double duty handicaps them. Ether is better in such cases.

It is not necessary to enter upon a detailed explanation of the pathology, physical signs and symptoms of a mid-way type, such as pneumococcal peritonitis, chronic proliferative peritonitis, etc. The variation in the reaction of the peritoneum is due to the difference in the virulence of the organisms permeating the intestinal wall.

Localized peritonitis—for example, round an appendix—should present no difficulties in the way of explaining both its method of localization and why its symptoms are those of a general peritonitis. The vomiting in these cases is said to be reflex. I claim that my theory is the more reasonable. Does it not also throw light on the symptoms of "appendix dyspepsia"?

A boy's bilious attack is a peritoneal toxæmia; so also are the summer diarrhoea of children and Asiatic cholera—that is, the peritoneal cavity is the seat of the elaboration of the poison by the bacteria.

Some of the cases of "vicious circle" after gastro-jejunostomy are instances of septic peritonitis. Some of the failures of gastro-jejunostomy are due to the fact that the operation has been performed, not on account of pyloric obstruction or gastric or duodenal ulcer, but in cases of chronic infection of the peritoneal cavity, and the seat of this was not found; the poisoning, therefore, continuing,

the liver and stomach continued their necessary work of excretion.

Take, again, some—I do not say all—cases of chronic atonic dilatation of the stomach (without obstruction). I believe the cause of the condition to be due to the following sequences of events:

Abnormal microbes in excessive numbers in the intestine (due to unsuitability of, and over-indulgence in, food and drink), permeation of these into peritoneal cavity; there being constipation, the stomach assists the liver in the work of excretion, and therefore constantly contains toxic secretion; this duty and digestion cannot both be performed properly, the former, being of the most vital necessity, is proceeded with; food therefore ferments, the numbers of the bacteria are thus increased, and a vicious circle set up: the muscle coats are constantly soaked in toxic lymph and become atonic, etc.

I have a desire to believe that gastric ulcer has somewhat of the same causation, and that, in addition to toxins, bacteria themselves are thus excreted. Some of these bacteria, during the time that they find themselves in the submucous plexus, begin the mischief of ulceration, but I merely whisper the suggestion at present.

The symptoms of acute intestinal obstruction, as given in the textbooks, are not symptoms of obstruction at all, but of the supervening acute virulent peritonitis due to the excessive permeation of virulent micro-organisms through the bowel wall. Such a permeation may take place without obstruction, and Hemmeter says that "many cases have died with the symptom of acute intestinal obstruction where none could be found at the *post-mortem* examination."

TUBERCULOSIS IN INFANCY AND CHILDHOOD.

FROM THE STANDPOINT OF PREVENTIVE MEDICINE.

BY

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IN the JOURNAL during 1910 an article by Hamburger appeared, setting forth the results obtained by him in Vienna by the use of the cutaneous tuberculin reaction in a large number of apparently healthy children of all ages from infancy to puberty.¹ These results were remarkable, for they showed that tuberculous infection, entirely, of course, in a latent form, and beyond the reach of the most refined physical examination, began to occur in infancy, and so rapidly and steadily increased during childhood, that by puberty about 90 per cent. of poor children in Vienna reacted positively to the tuberculin test. Von Pirquet's classical investigation, published in 1907, had demonstrated for Vienna the same striking fact—namely, the existence in childhood of a widespread latent tuberculosis, in addition to the mass of active and clinically apparent tuberculosis.²

The following is a series of 371 cases in which cutaneous tuberculin tests were performed on in-patients of the Royal Edinburgh Hospital for Sick Children; they make it possible to institute a comparison between Edinburgh and Vienna. Both von Pirquet's results and my own included cases of active and recognized tuberculous disease, and are therefore strictly comparable; while Hamburger's figures, showing only latent or concealed tuberculosis, must be kept separate.

The Edinburgh cases were not selected in any way, but were taken systematically from all the medical wards of the hospital. The series was not consecutive, being interrupted from time to time. It includes a number of clinically tuberculous cases, but no attempt was made to secure a large number of these, or of any other clinical condition. The series, therefore, forms a representative group of the in-patients of this hospital, drawn from the poorer classes of the population of Edinburgh. The few cases from rural districts will not materially injure the deductions, and such cases are no doubt also included in the Vienna figures. The cases are arranged in six age periods, similar for both places. In the Edinburgh table there are no cases over 12 years.

Age Period.	Edinburgh.		Vienna.	
	Number of Cases.	Percentage of Positive Reactions.	Number of Cases.	Percentage of Positive Reactions.
Under 1 year	64	14.1	156	5.1
1-2 years ...	61	29.9	98	22.4
3-4 years ...	75	46.6	108	37.9
5-6 years ...	52	28.8	85	62.3
7-10 years ...	79	51.9	119	59.6
11-14 years ...	40	55.0	65	73.8
Total ...	371	37.7	631	38.5

These results can be conveniently shown and compared by means of graphic curves, as in the accompanying chart:

Fig. 1.—Cutaneous tuberculin reactions.

The two curves for Edinburgh and Vienna agree in some respects and differ in others. In both towns tuberculous infection begins in the first year of life, and increasing steadily in the successive stages, is found to prevail in a high degree about the age of puberty. But there are some differences. The curve for Edinburgh rises more steeply at first, and almost attains its maximum at the age period 3-4 years; after a drop in the next period (possibly to be accounted owing to the small number of cases), it rises again by smaller increments. The curve for Vienna is more uniform in its progress throughout, being distinctly lower than that of Edinburgh in the first three age periods, but overtaking it and decisively surmounting it in the latter half of the chart. In trying to interpret these curves from the standpoint of the sources of infection, it may be possible to show that this different behaviour of the two curves is significant.

It is generally agreed that, for practical purposes, there are only two sources of infection in human tuberculosis—the disease pulmonary phthisis in man conveyed by sputum and disease in cattle conveyed in tuberculous milk. It is seldom possible to indicate on a large scale the amount which each of these sources of infection, human and bovine, is contributing to the total of human tuberculous disease; but in the case of Edinburgh and Vienna the figures of tuberculous infection in childhood already given, along with certain other data, can be made to furnish a rough estimate of the respective parts played by the two factors in this widespread infection of the child population with tuberculosis.

The amount of human infectious material in a locality may be fairly gauged by the prevalence of phthisis within it, and so by the phthisis death-rate. The following figures give for Edinburgh (1909) and for Vienna (1906) the number of deaths from phthisis per 100,000 of the population. The figures for Vienna were kindly obtained for me by Dr. J. C. Dunlop, Superintendent of Medical Statistics, Register House, Edinburgh:

Edinburgh, 117

Vienna, 293

In Edinburgh, therefore, the amount of human tuberculous material of an infectious kind is little more than a

third of that in Vienna. Further, Edinburgh has possessed since 1887 in the Royal Victoria Dispensary for Tuberculosis an organization specially directed to control and nullify this mass of infectious material; while Vienna, as far as I know, is without such an organized control. Yet in Edinburgh, with its much smaller phthisis death-rate, and with its special defences against human infection, the poor child-population for the first four years shows a greater degree of tuberculous infection than the corresponding population in Vienna, where the amount of human infectious disease is not only much larger, but is also less strictly controlled. There being only one other source of infection—the bovine—we are driven to conclude that the bovine factor must play in Edinburgh a larger part in child infection than in Vienna—in short, that in Edinburgh the children of the poor are seriously exposed to infection by tuberculous milk.

That conclusion is strongly supported by evidence of another kind. Abdominal tuberculosis, which especially prevails in the early years of childhood, is exceedingly common in Edinburgh; it is very uncommon in Vienna. Thomson and Fordyce³ have prepared figures (from clinical records) showing the incidence of abdominal tuberculosis in various European and American hospitals; they give the following results for Edinburgh and Vienna, which endorse the above statement:

	No. of Cases.	Percentage of Abdominal Tuberculosis.
Edinburgh	15,320	3.6 per cent.
Vienna	11,184	0.46 per cent.

It is not denied that abdominal tuberculosis may be caused by human contagion; but this factor may be expected to operate at least equally, probably more severely, in Vienna. As before, therefore, we are forced to the conclusion that bovine infection is a considerable factor in the tuberculous infection of Edinburgh children, and explains the higher curve in the early years of childhood as compared with that of Vienna.

From the standpoint of preventive medicine, the importance of being able to estimate, however roughly, the varying parts played by human and by bovine infection in this process of tuberculous inoculation is obvious. The statistics collected by Thomson and Fordyce, already quoted, are of great value for this purpose. They show that, while Edinburgh and Glasgow are easily first (from hospital clinical records) in the prevalence of abdominal tuberculosis, all the other British children's hospitals investigated show a much greater frequency of abdominal tuberculosis than do similar institutions in Italy, Austria, Germany, France, and America. The serious position which Great Britain occupies in this respect is graphically illustrated in the following chart, prepared from the figures of Thomson and Fordyce:

Fig. 2.—Showing the relative prevalence of abdominal tuberculosis in different children's hospitals.

I further endeavoured by the simultaneous use of human and bovine tuberculin for the cutaneous reaction to separate

cases of tuberculosis according to the source of infection, human or bovine.

Table showing Results in 330 Children, using alongside one another the Concentrated Tuberculin (Old) Human and Bovine, in each Case.

H = Human Tuberculin : B = Bovine.	
H and B equal (both positive or both negative) ...	258
H and B both positive, but H greater than B ...	52
H and B both positive, but B greater than H ...	9
H positive, B negative ...	11
H negative, B positive ...	0

The results of this test defeated any hopes of discriminating in this way between a tuberculosis of human and one of bovine origin. They show that in the vast majority of cases, whatever the strain of bacillus present in the body, the reaction of the human tissues is the same, whether it is human or bovine tuberculin that is used to provoke it. It seems, in fact, another piece of evidence in favour of the view that human and bovine bacilli are closely kindred organisms. It is, of course, certain that some, and possibly many, of these 258 cases reacting equally to human and bovine tuberculin must have been bovine infections; but in the present state of knowledge this indifference of reaction cannot be used for or against the question of transmutation of the bovine into the human type of bacillus by residence in the human tissues. All that can be done is to state the results of this test and to leave them meantime unexplained.

So far, therefore, the conclusions are: (1) Tuberculosis, both as active tuberculous disease and as latent infection, is widespread among the poorer children of the Edinburgh population, and that by far the greater part of this is effected in the first few years of life.

(2) When one endeavours to fix the sources of this infection, whether derived from tuberculous disease in man or from tuberculous milk, by a comparison with conditions in Vienna, both as to tuberculosis in childhood and as to adult tuberculosis (phthisis), the conclusion is reached that bovine infection must have a considerable share in the tuberculous infection of Edinburgh children. This infection by tuberculous milk is chiefly operative in the first four years of life. It is specially marked in Edinburgh and Glasgow, but prevails in the large towns of Great Britain to a far greater extent than in most large centres in Europe and America.

(3) These facts of tuberculous infection in infancy and childhood, both as to its extent and its nature, must receive attention in any thorough scheme of administrative control of tuberculosis. They show clearly that the child is very susceptible to tuberculous infection, and that for Edinburgh at least this greater part of infection is effected before school age. This age incidence of the infection, if it can be shown to prevail generally, must make more difficult the problem of control. Some more effective measures for dealing with milk infection, which is a marked and special feature of tuberculosis in childhood in this country, are also called for.

It is very difficult to do more than guess at the relative parts played by human and bovine infection in Edinburgh. It is not meant to suggest that the factor of human infection is negligible. It is precisely during the first few years of life that the child is most dependent on the adult, and most closely in contact with the most susceptible to human infection, and most difficult to guard from such infection. But these conditions are common to all countries and places, and they do not explain the greater incidence of tuberculosis of young children in Edinburgh as compared with Vienna.

Animal experiments, notably those of Findel⁴ and Cobbett,⁵ and those carried out by the British Royal Commission on Tuberculosis, have shown for various animals that it is more difficult to infect by swallowing tubercle bacilli in food than by breathing them in air. The flaw of all such experiments, used as applicable to human conditions, is that while this may be true of the healthy intestine, the same mucous surface in unhealthy conditions of catarrh, etc., may be easily penetrable by tubercle bacilli. In the frequent digestive disturbances of young children the intestinal ports of entrance, normally closed, may be thrown freely open.

The investigation by the British Royal Commission⁶ of the characters of different viruses obtained from tuberculous disease in man is relevant to this point. In 29 cases

of primary abdominal disease, by certain cultural and inoculation tests, 14 bovine strains of bacilli were isolated, and 2 of mixed human and bovine strains; the remainder were human strains. But in 11 of these 29 cases where the mesenteric glands alone were subjected to the tests, the results were 7 bovine and 4 human strains. Here again the possibility of the metamorphosis of types, and especially of the bovine to the human type, comes to the front. The Commission, after rigorous animal experiments, failed to obtain evidence that this transmutation of types takes place, but nevertheless concluded that "they are not prepared to deny that the transmutation of types does not occur in nature." In face of that conclusion, the separation of strains of tubercle bacilli according to the above cultural and biological tests does not give a decisive verdict on this point.

In any case this means of separation is not available clinically, nor is it practicable on a large scale from *post-mortem* material. For practical purposes a rough method of calculating the amount of human and bovine factors in the tuberculosis of childhood is to take, on the one side, the phthisis death-rate for the district, and allowing for any organized control of phthisical patients that exists, to regard its amount as measuring the human factor of infection; and, on the other side, to take the figures for abdominal tuberculosis as roughly indicative of the extent of the bovine factor of infection.

The Relation of Latent Tuberculosis in Childhood to Adult Tuberculosis.

In the 371 cutaneous tests performed on Edinburgh children, 297 were in cases that clinically were non-tuberculous. Of these, 28 per cent. reacted positively to tuberculin, indicating this proportion of latent as against active tuberculous infection. It is the presence of this large amount of latent tuberculosis that destroys the value of the von Pirquet reaction as a test of clinical tuberculosis. It is necessary to point out that the undiluted old tuberculin must be used if latent infections are being sought for. Mills,⁷ in a recent analysis of the cutaneous test in 253 children, where he used a 20 per cent. dilution of old tuberculin, found negative results in 48 healthy children, whose ages ranged from 9 months to 12 years, and concluded that "a negative reaction may be confidently expected in children apparently non-tuberculous." I myself⁸ in a previous series published in the JOURNAL, and using a 25 per cent. dilution, obtained negative results in about 50 children suffering from diseases clinically non-tuberculous. But if undiluted tuberculin is used for the test, this statement is no longer true. Many apparently healthy children and many with diseases apparently non-tuberculous will then give a positive skin reaction.

The curves (Fig. 3) prepared from Hamburger's tables⁹ give a picture of the frequency of latent tuberculosis at the various age periods of childhood. They represent

Fig. 3.—Latent tuberculosis (Hamburger), cutaneous reactions in 509 children clinically non-tuberculous.

tuberculin reactions given by 509 children, all of whom were by ordinary clinical tests free from tuberculous disease. The lower curve is of cutaneous reactions; the higher curve is of subcutaneous reactions (*Stiehrefaktion*), and represent positive results obtained by this more

delicate method in children who had failed to react to the cutaneous application of tuberculin.

This double curve of latent tuberculosis in childhood is interesting, for it serves to separate what may be called a latent infection of low potential from one of high potential. For the first four years of life the two curves are almost merged into one—that is to say, at this period all latent infections are of high potential; in other words, a quiescent focus may readily become an acute or general infection. But after this period the two curves widely diverge, the lower curve indicating the number of cases of latent infections of low potential at the later period of childhood, cases where the focus of tuberculosis is on the way to becoming obsolete and extinct. The upper curve, however, may represent cases where the focus of tuberculosis is quiescent but not obsolete. It is these cases of latent quiescent tuberculosis in the later years of childhood which may be the forerunners of adolescent and adult phthisis.

Von Behring¹⁰ in 1903 endeavoured to link up tuberculosis in the child with adult tuberculosis, explaining the latter as a late fruit of seed that had been sown in infancy, and regarding the intestinal mucous membrane as the port of entrance, and tubercle bacilli in milk as the source of infection. In all the criticism that has been poured upon this theory, there has been neglect of one point which Behring emphasized—that phthisis is a disease occurring in a person already infected with latent tuberculosis, and that the peculiar character of phthisis is due to this previous infection, which without good grounds he dated back to the earliest period of childhood.

But Hamburger and von Pirquet have now established the fact of latent tuberculosis in childhood, and have traced its progress and increasing frequency through all the periods of childhood. Hamburger¹¹ therefore strongly supports Behring's conception of phthisis as a late sequel of a process begun in childhood. He compares tuberculosis with syphilis, and believes that primary, secondary, and tertiary stages can be marked in the one as in the other. In tuberculosis the primary stage is the first inoculation at some period of childhood—the penetration of a mucous membrane, the settlement in a gland. The secondary stage is represented by the terribly acute manifestations of tuberculous disease in childhood, abdominal tuberculosis, acute phthisis, tuberculous meningitis, general tuberculosis. But this secondary stage may never appear, and yet the tertiary phase may develop many years after in the chronic pulmonary tuberculosis of the grown man. That, so far, is an elaboration of von Behring's view. In childhood the seed is sown as latent tuberculosis; in manhood, the harvest is reaped as phthisis.

Hamburger brings *post-mortem* evidence in support of this view. In Vienna, of 175 children dying of tuberculosis, tuberculous disease of the bronchial glands was found in 97 per cent. The primary focus could in most cases be made out in the lung itself. This disease of the bronchial glands is, according to him, the starting-point of a tuberculous invasion of the lung, such invasion being delayed till adolescence or adult life.

But this pulmonary invasion may not be delayed. It may occur in childhood, producing an acute phthisis, which is rapidly fatal.

Sluka in Vienna¹² has published a series of cases of this kind, in which by the use of *x* rays he demonstrated in life enlargement of the bronchial glands, and later a spread of disease into the lungs. In adult life, however, it is a slow infiltration of the lung, in the first instance by way of the lymphatic tracts, and later gives the familiar picture of a chronic phthisis.

Shennan¹³ has made a valuable analysis of *post-mortem* examinations in the Sick Children's Hospital, Edinburgh, and his table of glandular distribution is exactly comparable with a table given by Hamburger. In both tables the children died of tuberculous disease; the cases are thus not of latent tuberculosis.

This comparison brings Edinburgh into line with Vienna with regard to *post-mortem* material, showing for both places the great frequency of bronchial gland tuberculosis, but emphasizing also the peculiar position of Edinburgh with regard to abdominal tuberculosis.

The evidence in favour of a connexion between tuberculosis in the child and in the man thus rests on several sets of facts: On the great frequency of latent tuberculosis

Table showing Glandular Distribution of Tuberculosis.

Glands Affected.	Edinburgh (Shennan). Total Cases, 243.	Vienna (v. Ghon). Total Cases, 175.
Mediastinal alone	93 (or 39 %)	113 (or 67 %)
Mediastinal and mesenteric together	101 (or 42 %)	169 (or 100 %)
Mediastinal to al	194 (or 81 %)	169 (or 100 %)
Mesenteric alone	45 (or 19 %)	—
Mesenteric total	150 (or 63 %)	56 (or 33 %)

in childhood, especially in the later periods; on the localization of this latent disease in a great majority of cases in the bronchial glands that trap the lymphatic currents through the lungs, and on the special age incidence of phthisis at the periods of life nearest to childhood.

Whether this invasion of the lungs is entirely an auto-infection from the disease in the bronchial gland, or is partly also assisted by a fresh infection, there are no means of judging. The point, in any case, is not important. The fundamental fact, both for Vienna and for Edinburgh, is that among the poor children latent tuberculosis is extremely common, and in the majority of cases is located in the glands at the roots of the lungs.

A question here arises whether these bovine infections, that have been shown to be such a special and important feature of child tuberculosis in Edinburgh, can end in adult or adolescent phthisis. The British Royal Commission only isolated 2 bovine strains from 42 cases of phthisis. The answer to the question thus depends on whether metamorphosis of bovine into human types of bacilli occurs in human tissues; and metamorphosis, though the balance of experimental evidence is against it, has not received a final scientific negative. But even if bovine infections are practically confined to child tuberculosis, there is probably a sufficient number of human infections in childhood to support the hypothesis of adult phthisis as a sequel to a focus of pre-existing local tuberculosis.

If adult phthisis be in most cases a sequel of latent tuberculosis acquired during childhood—and certain facts, both clinical and pathological, seem to support it—any administrative measures that might reduce or prevent the infection of children would have decisive effect in the campaign against tuberculosis.

I wish to express my indebtedness to Dr. Melville Dunlop, Dr. John Thomson, and Dr. J. S. Fowler, who kindly allowed me to perform the cutaneous tests on patients in their wards; and also to the Carnegie Trustees for a grant which has defrayed the expenses of the investigation.

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THE *Boston Medical and Surgical Journal* quotes a report from Albany as stating that on August 26th Governor Dix announced the appointment of Dr. Charles H. Andrews of Buffalo, Dr. Lemon Thompson of Glens Falls, and Dr. Charles C. Duryea of Schenectady, as members of the board, established last year by the Bush Bill, to examine the feeble-minded, criminals, and other defectives, with a view to determining upon the legitimacy of their surgical sterilization. "The examiners are empowered to examine into the mental and physical condition, the record and family history of the feeble-minded, epileptic, criminal and other defective inmates in the State hospitals for the insane, State prisons, reformatories, and penal institutions, and one of its members is authorized 'to perform such operation for the prevention of procreation as shall be decided by the board to be most effective.' Only criminals convicted of such offences against the criminal law as convince the board that they are subject to 'confirmed criminal tendencies' come within the operation of the law."