The suggestion that the blood serum may be involved in the protection of the host against the parasite could be demonstrated neither in the case of *Cordyllobia* immunity nor, according to Sandground, in the acquired immunity of the dog against *Strongyloides*; while in my own experiments the filariform *Strongyloides* larvae were agglomerated by normal as well as by specific serum. A "local tissue immunity" in Besredka's sense is apparently present in the skin previously infested by *Cordyllobia*; possibly it is also found in the tissue of the intestine protecting it against parasites entering the mucosa (as *Strongyloides* does), and according to Holppli also feeding on its tissue (as hookworms do, or *Ascaris* and related forms). But the experiments of Herrick and Allen Scott, showing that a certain percentage of hookworm larvae in dogs do not even begin to feed on the tissue, but remain undeveloped for a long time in the intestine, are not in favour of this assumption. In addition, the bile secretion may have a "specific" influence, and some observations suggest that parasites can react to volatile substances. These are so highly characteristic for every species that the dog, for example, can distinguish by means of them not only the species, but actually the individual.

On the other hand, the experiments of Steiner and others with plant nematodes, and the behaviour of the various *Strongyloides* strains, tend to show that a parasite can adapt itself to the biochemical conditions of a new host; that such an adaptation precedes morphological changes is evident, the *Ascaris* of the man and the morphologically identical form in the pig being a suggestive example. But in spite of the fact that the biochemical processes of host and parasite seem to influence each other in many ways, there is so far no evidence of the production by the parasite of "antiferments" against the peptic ferments of the host. In any case, the *Ascaris* experiments of Weinland do not constitute such evidence, since, according to F. Einstein, *Ascaris* is protected against digestion by the impermeability of its cuticle.

Finally, some further remarks on hookworm epidemiology. As the "age resistance" to hookworm infection is so pronounced in dogs, one might expect the human child to be more liable than the adult. In nearly all the hookworm countries, however, the percentage of infected children is much lower than that of the adults. But the defecation habits of primitively living people have such a decisive influence in hookworm epidemiology that the relatively low infection of the children may be merely a consequence of the use of defecation places less contaminated with excrement (and therefore with infected larvae) than are the places frequented by the adults. As it is practically impossible to decide to what extent such habits may influence the hookworm statistics in different countries, we must be very careful in drawing general conclusions from any given material. Therefore the following deductions may, perhaps, not be conclusive.

So far as I know, apart from North Corrientes, an infection of young people higher than that of adults has been reported only from Mexico, by Carr, and from the rural population of Alabama, by Smillie and Augustine. In Alabama, however, the hookworm diminution after the fourteenth year of life is explained by the fact that shoes are used only after this age; in North Corrientes and Mexico, however, such differences between the habits of younger people and adults were not noted.

It is quite remarkable that just in North Corrientes and Mexico (where, in contrast to the other hookworm countries, the infection spontaneously decreases in the adults) the infection of the younger individuals reaches so high a degree as reported from nowhere else, a fact which suggests that the reduction of infection in the adults may be caused by an "acquired immunity" in consequence of the high and continuous infection during earlier life. Herrick points out that the epidemiological observations of Sawyer, Hill, and Dochter are also in favour of the assumption that, under the influence of a continuous hookworm infection, a certain degree of immunity against further infections is developed, especially when the former infection has been a high one. That the same seems to hold good for the hookworm-infected dog has already been noted.

The question of the varying susceptibility to hookworm infection in different races is of great economic importance. According to a very important paper by Smillie and Augustine, the negro children in Alabama are much less infected with *Necator* than are the children of the white farmers, and this in spite of the fact that they live practically under the same conditions. The authors suggest that since the epidermis of the negro children is thicker than that of the whites it is not so easily perforated by the hookworm larvae. The same, I think, may hold good perhaps for the skin of other coloured races. That the skin structure can influence the degree of hookworm infection is proved by the experiments of Looss and myself, which show that the tender skin of young individuals is more easily infected than is that of older persons.

Another clinical observation suggests a higher mechanical resistance of the negro epidermis against hookworm larvae than obtains in Europeans. Kirby-Smith, Dove, and White have described from Florida and its vicinity a type of "creeping eruption" clinically resembling the skin affection caused by the migration of a *Gastrophilus* larva. In the Florida cases, instead of a fly larva, the larva of *Ancylostoma brasiliense* of the dog was found in the epidermis. However, this species of hookworm larva may also find difficulties in boring into the negro skin, because the affection, although very common in the skin of whites, is practically absent in negroes.

About two and a half years ago the same affection developed on the forearm of an assistant and myself after some skin-penetration experiments with the larvae of the dog hookworm *Uncinaria stenocephala*. As this species is found also in European dogs, one has to consider the possibility of such skin infection in Europe. On the other hand, according to White and Dove, in contrast to *Ancylostoma brasiliense*, the larva of *Ancylostoma caninum*, so common in most countries, is not involved in "creeping eruptions." This evidently is true also of the larvae of the human species. I have studied the normal course of the affection in my own skin, and although bacterial activity cannot be excluded, of course, I think that the essential cause of the peculiar pathological changes is a toxic secretion of the larva, small as this may be.

TUBERCULOSIS IN CHILDHOOD.*

BY

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THE high incidence of tuberculosis in childhood, its characteristic forms which differentiate it so markedly from the consumption of adults, the inevitable fatal prognosis of some types and, with correct treatment, the favourable outlook of others, make the subject of considerable interest and of the utmost importance. Reference to the Registrar-General's annual report for 1926 gives ample evidence of the frequency of tuberculosis in children. In that year tuberculosis was responsible for 8.3 per cent. of the total deaths in England and Wales, whilst 14.5 per cent. of all the deaths from tuberculosis occurred in children under the age of 15.

The susceptibility to the tubercle bacillus is not spread evenly over all the years of childhood. The disease is uncommon during the first eight months of life; but thereafter the liability to infection rapidly increases, reaching its height at the age of 2 years. From then onwards the body gradually acquires a certain degree of immunity. The lack of resistance to tubercle at an early age accounts for the marked difference of the life-history of the disease in children and in adults; in the young child it rapidly assumes a generalized and fatal form, whereas phthisis, the more characteristic adult type, is rarely seen in childhood. As the child grows older the lymphatic system comes to play a more important part. The lymphatic glands act as a powerful second line of

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defence, and are usually successful in holding up the infection. This leads to the second common form of the disease, glandular tuberculosis, which is much more frequently seen in childhood than in later life, when the function of the lymphatic system tends to be superseded by the whole defensive mechanism of the body.

When the bronchial or abdominal glands become tuberculous the disease may remain latent, and if left undisturbed it may finally heal. Meanwhile, however, these infected glands are a continuous menace, and are easily rendered active by some additional infection or lowering of the resistance. The infection then no longer remains confined to the glands, but spreads to neighbouring structures or bursts into the blood stream. Even in adult life the onset of consumption can often be traced to an original latent infection during childhood.

THE PATH OF INFECTION.

Three varieties of tubercle bacilli are known—the human, the bovine, and the avian; the last of these, however, is so rarely the origin of infection that it need not concern us any further. Much has been written on the relative preponderance of human and bovine infections, but beyond indicating the nature of prophylactic measures, a differentiation is of no great practical value. Certainly there is no evidence that one is more virulent than the other. Whatever the relative proportions of the two varieties may be in tuberculous children, it has long been established that the infection is either acquired through drinking tuberculous cow's milk or through continuous close contact with an adult suffering from the disease—in other words, either by oral ingestion or by inhalation.

The exact route taken by the tubercle bacillus has been the subject of much discussion. Calmette believes that inhalation plays a small part, and that both in thoracic and in abdominal tuberculosis the bacilli enter through the mucous membrane of the throat or of the alimentary tract. This is in direct opposition to Ghon, who, by very careful microscopic examination, claims to be able to demonstrate a minute focus of tuberculosis in the lung in every case where corresponding caseous glands are found at the hilum. This minute lesion in the lung is thought to be the primary seat of invasion through inhalation. It is often so small that without a microscope it would certainly be missed, and in many cases healing has progressed so far that only a small scar remains.

Analogous primary foci of invasion are held to exist in the tonsils and the intestines, but in these situations also the lesion has become healed, or is so minute that its identification is often impossible and caseous enlargement of the glands draining these areas is the only obvious sign of tuberculosis. The disease may, in fact, spread back from such infected glands to the lungs and intestines, or ulcerate through into the general blood stream. In infancy the infection spreads easily to neighbouring organs, and is liable to take on the extremely fatal form of generalized septicaemia. In early childhood the glands become severely affected, and it is only when the barrier they form is broken down that adjacent structures are involved. Even then the disease may remain localized and recovery may follow.

Much has been done in England, by proper sterilization of milk and regular inspection of cattle, to eliminate the bovine source of infection, but the human source of infection is far more difficult to eradicate. Unless the utmost precautions are taken, a child brought up and nourished in tuberculous surroundings can hardly fail to become infected through his food, through kissing, or by inhalation of an atmosphere which must necessarily from time to time become charged with the germs.

PROPHYLAXIS.

The surest way of stamping out the disease would be to remove all children of tuberculous parents from their homes to the country during the first year of life, but as in most cases this course is impracticable we are left with less satisfactory methods of prevention, of which the following are the most important. Tuberculous mothers should not breast-feed their infants, and the greatest care must

be taken in the preparation of food so as not to contaminate it with unwashed hands, or by coughing or sneezing. The child's feeding utensils must be washed and kept apart from those of the rest of the household. The infected person must only spit into a special antiseptic receptacle, and must never kiss or breathe on the child. Windows must always be kept open; the child should be in the fresh air as much as possible, and should sleep on a balcony. If possible, the whole family should move to the country. The value of oral administration of attenuated live cultures of tubercle bacilli, as advocated by Calmette, has yet to be confirmed. Grade A tuberculin-tested cow's milk is guaranteed free from tuberculous infection, but if the milk supply is suspected, scalding, boiling, or pasteurization renders it safe.

Various tests have been devised to detect the presence of tuberculosis. The most valuable is the von Pirquet test, performed by the inoculation of Koch's old tuberculin into the scarified skin. This, when a tuberculous lesion is present, produces a circumscribed red wheal in about twenty-four to forty-eight hours. The Mantoux test is more reliable, but is virtually the same as the von Pirquet, except that the solution is injected intradermally. The test depends on the hypersensitiveness of the patient, or otherwise, to tubercle bacilli or their extracts. This allergic state is best seen in the skin, where a local reaction occurs as part of the body's defensive mechanism when these specific bacterial products are injected into people who have previously been sensitized to them. A mere passing tuberculous infection is sufficient to provoke this hypersensitiveness. It follows, therefore, that nearly all adults, and many children by the age of 10, will give positive Mantoux reactions, and the value of the test as an indication of activity is seriously limited. The reaction, furthermore, is positive whether the foci of infection are active, latent, or healed, and the test is therefore of greater diagnostic value in very young children, who are less likely to have old tuberculous lesions present. An additional disadvantage is that the reaction is often negative in the final stages of generalized tuberculosis and in tuberculous meningitis.

A hard-and-fast demarcation of the various forms in which the disease is found in childhood is not always possible; for instance, the association of hilum glands and abdominal tuberculosis is not uncommon, and any form may terminate in tuberculous meningitis.

GENERALIZED TUBERCULOSIS.

Generalized tuberculosis closely resembles ordinary marasmus, and is often difficult to recognize in infants. The temperature remains normal or subnormal, and only shows an irregular pyrexia a few weeks before death. In some cases vomiting is troublesome; in others, with extensive lesions in the lungs, there is persistent cough, rapid breathing, pallor, and generally cyanosis. A mild bronchitis is the only pulmonary sign, and the spleen is generally enlarged. Death results from progressive debility, lung disease, or meningitis.

In early childhood the manifestations are equally obscure and the symptoms generalized and indefinite. Peevishness, indigestion, loss of appetite, wasting, languor, and sleeplessness are the usual complaints; but the irregular pyrexia, marked pallor, and cachectic appearance of the child soon indicate a graver illness than the first mild symptoms suggested. Later, the occurrence of severe symptoms in the lungs, brain, or abdomen often helps to clear up the diagnosis. Generalized tuberculosis can be the cause of persistent high temperature for many weeks, and the diagnosis may present difficulty until meningitis appears as a terminal event.

The presence of an enlarged spleen or the discovery of other foci of tuberculous infection, such as tuberculous epididymitis and tuberculous dactylitis, is a great help in the diagnosis of such an obscure case. The discovery of small shotty glands at the outer border of the pectoralis major muscle on each side is sometimes an indication of tuberculosis when there is no other explanation of their presence. Ophthalmoscopic examination of the fundi should never be omitted, and the discovery of choroidal tubercle may often give the final clue. All these signs are, however,

often absent, and the child may finally die without the condition having been suspected. Once tuberculosis has become generalized no treatment is of any avail, and the patient can only be kept as comfortable as possible.

THORACIC TUBERCULOSIS.

Tuberculous Bronchitis.

Repeated mild attacks of bronchitis may be the earliest signs of tuberculosis, but since, clinically, they differ in no way from ordinary bronchitis, their true significance usually passes unnoticed. It is only when the disease is anticipated by reason of the child's tuberculous surroundings, or when on rare occasions tubercle bacilli are discovered in the sputum, that active treatment can be undertaken at this early stage.

Miliary Tuberculosis of the Lung.

When the infection reaches the blood stream multiple small tuberculous foci are formed in both lungs during the process of general dissemination throughout the body. Miliary tuberculosis is invariably fatal. At first the diagnosis may prove very puzzling, and the condition be taken for influenza or bronchitis, but the increasing pallor, progressive wasting, dyspnoea, and cough eventually give the clue. Irregular pyrexia, generally higher than in bronchitis or influenza, is present, and may remain for several weeks, leading to a suspicion of typhoid fever; enlargement of the spleen further enhances this similarity. An x-ray film of the chest is a great help in the diagnosis, and shows a general mottling of both lungs. Symptoms of tuberculosis elsewhere in the body may supervene, and the illness often terminates in meningitis.

Tuberculous Broncho-pneumonia.

This may arise without any previous pulmonary infection, but more frequently follows a simple broncho-pneumonia, particularly in association with measles or whooping-cough. This stirs up a latent tuberculous infection harmlessly situated in a caseous bronchial gland, and causes it to spread into a neighbouring bronchus. The illness generally terminates fatally in a few weeks, but may sometimes be prolonged for several months.

Tuberculosis is suspected in a case of broncho-pneumonia which fails to clear up after several weeks, the patient meanwhile becoming progressively emaciated, paler, and more languid. Persistent rapid breathing and irregular or continued pyrexia are additional indications. Temporary improvement is sometimes a striking feature, and may give rise to false hopes of recovery. Involvement of the pleura is common, and loculated serous effusions or small empyemata are formed. Examination of such pleural effusions may occasionally reveal the presence of tubercle bacilli, and these must also be sought for in the sputum. Children seldom expectorate, but sputum can often be obtained by touching the back of the throat with a piece of gauze wrapped round the end of a pair of forceps. A reflex cough is excited and the sputum is caught on the swab. Should these two examinations prove fruitless the search may be directed to the stools, or to a stomach wash-out for any tubercle bacilli that may have been swallowed. The discovery of chronic tuberculous lesions elsewhere may prove useful. The presence of opaque fluffy shadows in an x-ray picture of the lungs gives the deciding evidence. At times all methods of examination are inconclusive; the true nature of a persistent broncho-pneumonia with splenic enlargement, continued pyrexia, and progressive emaciation is then left in doubt, since the condition is quite compatible with either tuberculous or pneumococcal infection.

No treatment is likely to meet with success, but, in older children, in the rare event of the lesion being confined to one side, collapse of the affected lung by artificial pneumothorax may occasionally arrest the disease.

Pulmonary Phthisis.

Chronic consumption of the adult type is rare in children, and when it occurs the cavities often do not follow the same distribution as in adults, being principally situated near the hilum or at the base of the lungs. Haemoptysis is a rare event in an advanced stage of the disease, when tubercle bacilli may also be present in the

sputum. A diagnosis of phthisis is undoubtedly too frequently made in children, areas of pulmonary fibrosis and unresolved pneumonia at a lung apex being wrongly interpreted as tuberculous. Such common symptoms of chronic indigestion as wasting, sweating, and evening rise of temperature may also cause confusion. Haemoptysis in childhood should never suggest tuberculosis. It is usually associated with adenoids or is a sign of advanced heart disease.

Tuberculosis of the Bronchial Glands.

The bronchial glands are more often the site of chronic tuberculous disease in childhood than any other region of the body. Children thus affected are not very ill, but are nevertheless potentially tuberculous. Minor maladies or debilitating diseases easily awaken this dormant tuberculous infection. It spreads back into the lungs, giving rise to serious consequences. The caseous contents of the glands may even burst directly into a bronchus or pass into the blood stream.

Such symptoms as arise from quiescent tuberculous bronchial glands are vague and indefinite. The child has a poor appetite and does not gain weight; his general development suffers, he loses his vigour, and becomes easily tired. The temperature is constantly a few degrees above normal and periodically may be considerably raised. There is a hoarse irritating cough, which is often paroxysmal in character and somewhat reminiscent of whooping-cough. It may, in fact, follow this infection without interruption, and the persistence of a spasmodic cough long after the whoop has disappeared points to the possible complication of enlarged bronchial glands. The glands may become so large in infants that their pressure on the trachea actually embarrasses the breathing, producing snuffles or stridor.

Several physical signs are said to be diagnostic of mediastinal glands, but in my experience they are all untrustworthy. It is true that submanubrial and paravertebral dullness may be present, but the glands need to be very large before either of these signs is elicited. D'Espine's sign and Eustace Smith's sign are of no great value since they can often be elicited in normal children. In every case a radiogram must be made, but even that will not show whether the glandular enlargement is due to tuberculous or to some simple catarrhal infection.

Prognosis and Treatment.

Immediately the condition is suspected and has been confirmed by x-ray examination and a positive Mantoux test, the child must be sent to the country or seaside, where he should remain continuously in the open air. Sun baths or irradiation with ultra-violet light, a nourishing diet, and tonics are also important items in the treatment. This must be continued until all symptoms have disappeared, the normal weight is restored, and the temperature remains normal. Septic tonsils require removal when the general condition has improved. Deep x-ray therapy in the hands of experts is sometimes strikingly successful in cases which are slow in reacting to other lines of treatment.

ABDOMINAL TUBERCULOSIS.

Tuberculosis confined to the abdomen is one of the commonest forms in children and is the most amenable to treatment. The infection is generally bovine through contaminated milk, and reaches the abdomen and mesenteric glands through the intestines. Several clinical varieties exist, and can be enumerated as follows.

1. *Tuberculous Ascites.*—The peritoneum and mesenteric glands are both infected with tubercle, which produces a gradually increased accumulation of fluid in the abdominal cavity. This may lie free in the abdomen or be loculated by the presence of adhesions.

2. *Tabes Mesenterica.*—The infection is confined to the abdominal glands, which become caseous or calcified. They may give rise to symptoms, or remain quiescent and be found unexpectedly at operation or post mortem.

3. *Adhesive Peritonitis.*—In this condition there is little or no free fluid present. Matting of the mesentery and omentum occurs, and adhesions are formed between the coils of intestines.

4. *Tuberculous Ulceration of the Bowels.*—This is generally secondary to pulmonary disease, resulting from the swallowing of infected sputum. In children it can occur as a primary infection, and is then usually associated with severe involvement of the mesenteric glands. In either case diarrhoea is the most striking symptom.

5. *Intestinal Obstruction.*—This may result from ulceration of bowel, or from kinking and narrowing of its lumen through peritoneal adhesions.

6. *Acute Peritonitis.*—This is simulated occasionally when an intestinal ulcer perforates into the surrounding adhesions, or when a tuberculous gland breaks down completely or becomes infected with other organisms.

Clinical Manifestations.

The symptoms depend largely on the part first and most severely affected, but generally they are gradual and insidious. The child's strength is noticed to be failing; he is pale and thin, feverish at night, and the abdomen is progressively enlarging. The appetite is capricious, the stools are often unhealthy, and there may be alternating constipation and diarrhoea. Attacks of colic are not uncommon, and when associated with vomiting may be mistaken for subacute appendicitis. In those cases which simulate acute peritonitis the onset of symptoms is very abrupt, with high temperature, vomiting, acute abdominal pain, and generalized tenderness and rigidity. When the acute inflammation is principally confined to the ileo-caecal glands, this tenderness and rigidity is localized to the right iliac fossa, and the condition closely resembles acute appendicitis. The adhesive type may so constrict the intestines and interfere with their normal movements that partial obstruction results; the intestinal coils stand out in relief under the abdominal wall, and peristalsis may be seen. In certain mild cases there are repeated feverish attacks at intervals of a few months, and little else is noticed at the time except slight abdominal distension. Enlarged glands or tuberculous masses, sometimes mistaken for the liver, can usually be felt in the abdomen at some period of the disease, but these may be masked by the presence of ascites. When suppuration occurs the abscess always tends to point at the umbilicus.

The outlook is certainly favourable in the absence of any severe tuberculosis elsewhere, and patients who have received prompt and energetic treatment recover completely. No case should be despaired of, since the disease rarely proves fatal, and then only in very feeble children. The infection is more dangerous in infants, in whom it is apt to spread beyond the abdomen.

Treatment.

The patients must live in the open air day and night, and remain flat in bed so long as ascites continues, the temperature is raised, or there are any other symptoms present. The general resistance is increased by giving as much nourishment as possible without upsetting the digestion. Milk, cream, eggs, butter, plasmon, raw meat juice, and raw meat sandwiches should all be included in the diet; but if they are not tolerated, and cause flatulence and curdled stools, fats must be restricted and milk may require peptonizing. Starchy foods are contraindicated, and if the appetite is poor or the digestion weak, alcohol in the form of brandy or brandy egg-flip should be given. Cod-liver oil is valuable, but cannot be added to the diet unless the stools are normal and there is no diarrhoea.

A belladonna and glycerin liniment (1 to 4) applied to the abdomen on lint is helpful in easing pain. Various applications are also said to be useful in the treatment of the actual disease, but, with the possible exception of iodoform ointment (10 per cent.) and mercury ointment (B.P. strength), they are of little value.

Irradiation of the abdomen by natural sunlight or ultra-violet rays is undoubtedly beneficial. If the ascites continues to increase, its removal may become necessary by means of a small trocar and cannula or Southey's tubes; but the best method of drainage is through a fairly long incision under general anaesthesia. Surgical removal of tuberculous abdominal glands is an unsuccessful procedure when there are any considerable number, and in adhesive

tuberculous peritonitis surgery is positively dangerous and liable to liberate the contents of the bowel into the abdominal cavity. It is equally unwise to interfere with a tuberculous abscess until it is actually on the point of bursting through the umbilicus.

TUBERCULOSIS OF THE EAR.

Chronic discharge from the ear in children is more commonly due to tuberculous infections than is usually supposed. Any aural discharge in a suspected case of tuberculosis should be examined, since the chance discovery of tubercle bacilli may just give the required confirmatory evidence. There are few subjective symptoms, and the ear continues to discharge profusely, despite ordinary methods of treatment. The lymphatic glands in the vicinity of the mastoid soon become infected and finally break down. In addition, there may be facial palsy as a result of involvement of the facial nerve as it passes through the middle ear. In the treatment efforts are made to eradicate the disease completely by surgical means, but this is often unsuccessful and the outlook is generally unfavourable.

TUBERCULOUS MENINGITIS.

No disease in the whole of paediatrics can appear in so many unexpected ways as tuberculous meningitis and lead to such confusion. The illness has no classical picture, and such physical signs as do appear often arise so late that they give no help towards an early diagnosis. Little reliance should be placed on Koenig's sign, as it is very inconstant and its absence in no way precludes the presence of tuberculous meningitis. Severe headache, persistent vomiting, prolonged pyrexia, gradual change of temperament, drowsiness, or a sudden fit followed by unconsciousness are all possible modes of onset. Should any of these occur after infancy and have no other explanation, tuberculous meningitis should always be borne in mind. Two physical signs that I have found most useful in a doubtful case are enlargement of the spleen and a unilateral extensor response. The ultimate diagnosis will, however, depend upon examination of the cerebro-spinal fluid, and, once established, the prognosis is invariably fatal.

CONCLUSION.

Let me emphasize once again the importance of prophylaxis. The prevention of contact with human sources of infection, the proper sterilization of milk, the maintenance of a high standard of general bodily health, and the provision of adequate country or seaside convalescence after any severe infection are the best safeguards we can give a child against tuberculosis.

CERTAIN ASPECTS OF THE DIAGNOSIS AND TREATMENT OF DIABETES.*

BY

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In the past the identification of a reducing substance in the urine sufficed for the diagnosis of diabetes. Varying degrees of severity were recognized, but anyone presenting a urine which reduced Fehling's solution was regarded as suffering from diabetes of greater or lesser severity. That parturient women and nursing mothers frequently showed sugar in the urine was early recognized, but it was some time before it was realized that this sugar was lactose and that its presence lacked any particular significance. Later it was discovered that the urine of certain persons otherwise in normal health contained pentose, which reduces Fehling's solution in the same manner as glucose. For the diagnosis of diabetes it was then deemed only necessary to demonstrate that the reducing substance in the urine was neither lactose nor pentose, the former being readily excluded by the yeast fermentation test with which it gives a negative result, the latter by Bial's reaction.

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