The test for rennin is as follows: Ten cubic centimetres of the filtrate of the gastric contents is neutralized with decinormal solution of NaOH. To this ten cubic centimetres of neutral milk is added, and the mixture tested at 38° C. If rennet be present casein will form in ten to fifteen minutes.⁵⁷

The test for the zymogen is as follows in the absence of the ferment: To ten cubic centimetres of the filtrate made slightly alkaline add two cubic centimetres of a one-per-cent. solution of calcium chloride; then ten cubic centimetres of milk and heat at 38° C. If rennet is present casein will form. It is rare to find a condition in which rennet or the rennet zymogen is wholly absent, but a marked diminution of these substances is characteristic of certain morbid conditions.

In some cases, therefore, aid may be derived from a quantitative estimation of these substances.

The quantitative estimations for rennet and the zymogen are as follows: Part of the gastric filtrate is exactly neutralized, and portions of this diluted to different dilutions as one-tenth, one-fifteenth, one-twentieth. Five cubic centimetres of each dilution is placed with five cubic centimetres of neutral milk at 38° C., and the dilution at which the ferment ceases to be active ascertained. By comparing the result with normal results, any diminution and the amount of it can be observed. For the zymogen a portion of the filtrate is made alkaline; different dilutions made, one-tenth to one one-hundred-and-sixtieth; five cubic centimetres of dilution placed with one cubic centimetre CaCl_2 plus five cubic centimetres of milk; and the dilution where action ceases recorded.

Full tables of the normal amounts of the rennin and its zymogen in the normal conditions can be found in Friedenwald's article on the subject.⁵⁸

(To be continued.)

THE TUBERCULOUS DIATHESIS.

AN INQUIRY INTO THE CAUSES THAT LEAD TO PULMONARY CONSUMPTION.¹

BY CHAUNCEY REA BURR, M.D., HARV., PH.D., YALE, SAN JOSE, CAL.

AMONG the morbid conditions associated with pulmonary tuberculosis, one of the most constant is a fatty liver. This lesion may be in the form of fatty degeneration, or fatty infiltration. Morphologically it is often impossible to distinguish between the two. The liver normally contains a certain proportion of fat, particularly after meals.

"This fat," according to Ziegler,² "is partly brought to the liver ready-formed and there deposited, and partly elaborated from albuminoid substances *in situ*." In diseased conditions the hepatic fat may be enormously increased, the increase depending either on increased production of fat or on diminished consumption of fat, or on both. Most authorities when treating of this subject in connection with pulmonary phthisis consider fatty liver a disorder of suboxidation. And so it is, but the reason ordinarily given, namely, embarrassed pulmonary respiration, is inadequate to explain all cases. For, as long ago pointed out by

¹ Read before the Medical Society of Santa Clara County, California, April 13, 1897.

² Path. Anat., 1884, sect. viii, chap. lviii, No. 487.

⁵⁷ Boas: Magenkrankheiten. Loc. cit.

⁵⁸ Friedenwald: Medical News, June 22, 1895.

Rokitansky, there are diseases characterized by embarrassed respiration, more profound than that of pulmonary phthisis, as, for instance, pulmonary emphysema, in which fatty liver occurs with nothing like the frequency it does in phthisis, while in other diseases in which the lungs are sound, as in tuberculosis of the bones, chronic dysentery and anemia, fatty liver occurs with comparative frequency. Some other factor than embarrassed pulmonary respiration must enter into the production of this pathological state.

It has seemed to the writer a promising subject to investigate, and one which might lead to a better understanding of the tuberculous diathesis. Accordingly, the question to be answered is: "Why should the liver of pulmonary phthisis so often be in a state of fatty degeneration?" It has been suggested by Fitz, that the administration of cod-liver oil is largely responsible for this condition. In some cases it may be, but it by no means follows that because oil is taken as a food it appears in consequence as oil in the tissues. It may be entirely oxidized to carbon dioxide. On the other hand, foods which are not fatty, as albumins and sugars, may appear as fat in the tissues. Foster mentions a pig³ in which 472 parts of fat were laid on for every 100 parts of fat taken as such in the food; while of every 100 parts of a mixed diet, including substances other than fat, 21.2 parts were retained in the body as fat. "Clearer proof than this," he says, "could not be afforded that fat is formed in the body out of something which is not fat."

It is well to understand, before proceeding further, to what uses the fat is put after it is formed. Thompson⁴ mentions several such uses, namely, to furnish energy for the development of heat; to spare the tissues from disintegration; to serve for the storage of energy.

Fat is, therefore, a fuel, a producer, if properly oxidized, of heat and power. It has already been stated that the fat found in the liver cell may be brought thither ready-formed or may be elaborated from albuminoid substances *in situ*.

Brunton⁵ gives a table which makes clear this latter change. According to this table, albuminous tissues split up into non-nitrogenous substances, as fat, etc., and nitrogenous substances, as leucine, tyrosine, etc. The fat is normally converted into carbonic acid and excreted by the lungs; while the leucine and tyrosine are converted into urea and pass off by the kidneys.

Smith⁶ says that "fat when entirely deprived of water consists of three elements only, namely, carbon, oxygen and hydrogen ($\text{C}_{77}\text{O}_{11}\text{H}_{12}$). When the fat is decomposed in the body these elements unite, so that the carbon takes a part of the oxygen and becomes carbonic acid, whilst the hydrogen takes another part of the oxygen and becomes water, any deficiency in the quantity of oxygen for this purpose being supplied by the inspired air."

An hepatic cell, therefore, in a state of fatty degeneration, is one in which the fat has been elaborated from albuminoid substances *in situ*. Furthermore, there has been some interference with the normal chemical process, for the change to stop at the fat formation and not pass on to that of carbon dioxide.

Fat is a fuel, burned up or oxidized in the vital processes of the cell. The fat droplets appear in the parenchyma of the cell shortly after the ingestion of a

³ Physiology, 1883, p. 533.

⁴ Practical Dietetics, 1895, p. 173.

⁵ Pharmacology and Therapeut., 1885, p. 380.

⁶ Foods, 1873, p. 45.

meal, and disappear in large part before the next meal. The oxygen necessary for combustion comes from without; in other words, the cell has a respiration: it breathes; and if it breathes there must be a respiratory mechanism.

Quain⁷ speaks of the streaming movements of cellular granules. "This phenomenon," he says, "can be seen to occur both in cells which are stationary and in those which are exhibiting ameboid movements. It consists in the protoplasm of the granules which are suspended in that substance." He refers the production of the phenomenon to contractions in the protoplasmic substance. Here, then, is the mechanism by which the cell breathes. If it contracts, it also expands, and probably draws in oxygen in the process through the cellular walls. As if to put the seal of *imprimature* on this supposition, he further says: "The contractility of protoplasm is dependent upon the supply of oxygen. If this be withheld, the movements will, it is true, proceed for a time as usual, but this is because protoplasm, like other forms of contractile substance, such as muscle, has the power of storing away and using oxygen in some form of combination. For it is found that the active manifestations will not proceed indefinitely in the absence of oxygen, but cease after a time, to be renewed only on the accession of fresh oxygen."

A cell, therefore, in which fat is not consumed, a cell, in other words, in a state of fatty degeneration, is a cell which has ceased to breathe sufficiently to oxidize its fat. This failure of respiration may be due to failure of oxygen in the body at large, or to intrinsic weakness in the cellular respiratory mechanism. Cases of the first class occur in any disease in which the lungs are involved or in which the oxygen-carriers, the red blood corpuscles, are involved. Such are pulmonary complaints of long standing, anemia, infectious diseases and the intoxications, as lead, syphilis, alcohol, etc.

Cases of the second class may develop independently of the first class, or may be the direct sequence of this. The stationary cells of the body are in relation with the nervous system. Stimuli sent through this manifest themselves in signs of cellular activity. One of these signs is cellular contraction, and this, as has been shown, is the probable essential feature of the cell's respiratory mechanism. Failure of contraction involves failure of nerve force, and this in turn involves lesions of the nerve substance. In the case of the liver, the nerve substance, or nervous supply, is derived from the hepatic plexus of the sympathetic system. Filaments from this accompany the hepatic artery in its ramifications in the portal canals. Any lesions of these filaments or of their supplying plexus must perforce affect the physiological functions of the hepatic cells.

It is astonishing that more attention has not been paid to the pathology of the sympathetic system. Most writers ignore it altogether or make but passing allusion to it. What few facts it has been possible to collect for the purposes of this paper are rather in the nature of processes common to nervous matter in general than to the sympathetic system in particular.

Broadly speaking, such processes may be divided into inflammatory and degenerative, the one often following or becoming merged in the other. Thus, cellular infiltration is followed by fibrous hyperplasia or

sclerosis and degenerative atrophy of the nerve elements. Among the specific causes which induce inflammation of the nervous structures may be mentioned typhoid, variola, acute rheumatism, pyemia, puerperal fever, ulcerative phthisis. Nor must syphilis, alcohol, lead and other mineral poisons be forgotten. It will be observed that these are the same disorders in which fatty glandular change is so common. Lead in particular gives rise to degenerative change in the intestinal plexuses. Wherever degeneration occurs, the nervous filaments on the distal side atrophy, and consequently cut out of circuit more or less completely the structures to which they are distributed. This is descending degeneration. There is also an ascending degeneration of more limited extent extending upward to the nearest ganglion cell. In such cases, the trophic centre is in the periphery, and hence the course of the degeneration is away from the cut-off base of supply.

Intrinsic weakness in the cellular respiratory mechanism is simply another name for cellular paresis and the anatomical basis of this is inflammatory or degenerative change, or both, in the nerves supplying the cells concerned. The fatty cell is an index of this condition, and the writer is firmly of the opinion that the majority of cases of fatty degeneration wherever situated will be found to be associated with a degenerative neuritis of the nerve supply. This has already been shown to exist in the case of Bright's disease by the researches of Da Costa and Longstreth,⁸ who found an apparently constant degeneration of the renal ganglia, along with the other lesions peculiar to the disease. The associated albuminuria may be due to a similar condition, for the blood-vessels and the renal parenchyma have a similar nerve supply, and if the integrity of the one is attacked, the other must suffer also, and allow a leakage of serum albumin through their damaged walls. For all that is known to the contrary, the amyloid or lardaceous change may be due to a similar condition, for fatty degeneration is a constant concomitant with this disease.

In diabetes, it is estimated that half the cases are associated with a degenerated and fatty pancreas. In fatty degeneration of the heart a careful search would probably disclose degeneration of the cardiac ganglia. Eichhorst has demonstrated that section of the vagi is followed by fatty degeneration of the heart, and branches of the vagi terminate in these ganglia. In peripheral neuritis a dropsical condition of the limbs is often observed. All admit that this is due to paresis of the blood-vessels. If paretic blood-vessels in this situation allow their fluid contents to escape, will they not do the same in other situations? In short, there is good clinical evidence in support of the conclusion that a fatty cell is a paretic cell.

If the liver of pulmonary phthisis is so often in a state of fatty degeneration, it is because it is paretic. The cause of the paresis may be difficult to determine. From what has already been said, it will be understood that the site of the lesion may be either at the termination of the nerves in the hepatic parenchyma, or in the hepatic plexus. The former is probably the more frequent site of the two. Any glandular structure may be worn out by excessive use. Inflammation follows, then degeneration and atrophy. Here, then, is one cause of hepatic paresis—overwork. The liver of beer drinkers is often fatty and cirrhotic.

Anat., 1882, pp. 6 and 7.

⁸ Bartholow: *Materia Medica and Therapeutica*, 1884, p. 597.

They also often develop pulmonary tuberculosis. Cattle fed upon malted grains become, after a time, emaciated and tuberculous. In both cases the liver is overworked. Again, from the position it occupies, the liver is peculiarly liable to septic intoxications. With the exception of fats, all the products of intestinal digestion pass through the liver on their way to the general circulation. Poisonous peptones and many other poisons are here rendered innocuous. But it is conceivable that the task may be too great and the liver itself be poisoned. Over-feeding and its consequent dyspepsia is therefore a frequent cause of a disordered and paretic liver.

Another cause is improper food. Strong meat is not for babes, nor is alcohol or starch for adults. The late Dr. Fothergill, of London, has set forth in his inimitable way the reasons why meat in excess is not a proper food for certain individuals.⁹ According to this authority, lithogenesis is reversion. Reversion to an archaic ancestry, which shared with birds, reptiles and fishes conditions of life which resulted in certain similar metabolic processes. Among these, he places the excretion of uric acid in place of urea. Birds excrete a solid urine rich in uric acid, but deficient in urea, as do also reptiles and fishes. The reverse is the case with mammalia. Here a fluid urine is excreted, rich in urea, but deficient in uric acid. The fetus *in utero* is a reptile with the potentialities of a mammal. Its circulation and heat production are reptilian, and it manifests a tendency to the reptilian uric-acid formation. "When it breathes," writes Dr. Fothergill, "as by the touch of an enchanter's wand, the circulation becomes that of the mammal, and its heat production that of a warm-blooded animal."

The uric-acid infarcts of infantile kidneys are well known, as well as the deposit of urates on infantile napkins. At puberty the uric-acid formation is largely left behind. Exceptionally it persists, and where before the passage of uric acid and its salts through the kidneys occasioned little or no irritation, it is, later in life a fruitful cause of Bright's disease and many other ailments. Most authorities agree that urea is excreted by the liver cells. Vaughn has shown that uric acid is excreted by leucocytes. The liver cell, therefore, excretes a fluid urine rich in urea, the leucocyte, a solid urine rich in uric acid—an interesting distinction, as showing the one to be mammalian in its development; the other reptilian. In hepatic disorders, a heavy output of urates is a very common occurrence; and as these disorders are generally plethoric in their character, it would seem as if the old contention was true, that urea is simply oxidized uric acid; or, to put it more in the language of this paper, that the liver cell being specialized and of the mammalian type, breathes more deeply and uses more oxygen than the reptilian leucocyte.

Again, Haig has shown¹⁰ that uric acid and the xanthin compounds from which uric acid is derived may be introduced with the food, particularly where a meat diet is used. A feeble liver, therefore, fed with meat peptones containing uric acid and xanthin, is easily upset and hepatic congestion and inflammation follow. Feebleness of hepatic function is often inherited. The diathetic state in which this occurs is called scrofulosis, and the frequent association of this with tuberculosis has long been recognized. Fothergill

says that the children of town dwellers in the second and succeeding generations are very apt to be litho-genetic. How common the type is the out-patient waiting-room of any metropolitan hospital will show.

There is the bulky personage of large frame, clumsy and rheumatic, with features like a blurred photograph, and the delicate, fragile creature of emotional temperament, feeble digestion, light lower jaw and arched palate. The air of cities is notoriously deficient in ozone, and this may have much to do with the persistence of the uric-acid diathesis, particularly as such patients improve when removed to the ozonized air of the sea-shore.

With regard to the use and abuse of alcohol, but little need be said. Its influence is sometimes beneficial, often deleterious. It robs the system of oxygen to satisfy its own needs. In addition, sclerosis develop under its continued use. Whether this is due to the alcohol itself or to the impurities which often accompany it does not appear to have been determined. Fusel oil is generally regarded as one of the offending factors.

Starch, while admitted by all as unsuitable for infantile digestion, is not so generally recognized as unsuitable for adult digestion. And yet it is often a stumbling-block to those of the dyspeptic habit. The secretions of the salivary glands, Brunner's glands, pancreas and liver may all be called on in turn to struggle with starch and convert it into glucose. Sugars, fats and albumin, on the contrary, are readily digested and absorbed. The axiom has already been stated that "any glandular structure may be worn out by excessive use." The constant struggle of a delicate organism to convert the starch into glucose is an illustration in point. If the truth could be known, it would probably appear that the diet of baked beans and doughnuts, common to so many American families, had much to answer for in the production of insufficient livers and pulmonary phthisis. The rice-eating Bengalee escapes gout, but falls a frequent victim to diabetes and consumption.

So far, the inquiry has endeavored to show the causes which lead to fatty degeneration of the liver, and the fact has been pointed out that this condition is often associated with tuberculous deposits in the lungs. It remains to study the one condition with the other and ascertain what relation, if any, exists between the two.

No time need be wasted over the proximate cause of pulmonary consumption. All admit its identity in the tubercle bacillus. This microbe is not an aggressive fellow. He requires time for development and quiet surroundings. The cultures grow slowly. On the other hand, the leucocyte is an aggressive fellow and is the sworn enemy of microbes in general. How happens it then, that the battle is often so languidly waged and that the victory so often rests with the peace-loving invader? For the very reason that there is peace in his immediate neighborhood. He has pitched his camp by the slow-flowing stream of dropsical fluid that exudes from paretic capillaries. He is not rudely jarred by draughts of air sweeping through the passageways of the lungs. On the contrary, he chooses the apices where the respiratory movement is normally slight, or some dependent portion of the lung already infiltrated with the serous exudate. The leucocytes hasten to do battle, and crowd about the seat of invasion in ever-increasing numbers. But

⁹ Keating: *Cyclopedia of Diseases of Children*, 1889, vol. II, pp. 256-319.

¹⁰ *British Medical Journal*, October 3, 1896.

they have pushed their way into a practically airless region, and one impregnated with toxins. If Dr. Hirschfelder's contention is true that a toxin oxidized becomes an antitoxin,¹¹ then conversely a toxin unoxidized remains a toxin. The leucocyte cannot continue to elaborate germicidal nuclein without a supply of oxygen. If the supply is deficient its ameboid movements cease and therewith its functional activity. The toxins clamor for oxygen, and so likewise do the leucocytes, with the probable results that the leucocytes perish, and the toxins continue toxins.

The evidence of a pulmonary paresis antecedent to the outbreak of tuberculosis is clinical rather than pathological. It rests on the fact of dulness on percussion over the affected area, and diminished respiration. These may precede by a considerable interval the demonstration of the presence of the tubercle bacillus.

The nervous supply of the lungs is derived from the anterior and the posterior pulmonary plexuses, which are formed by branches of the sympathetic and pneumogastric nerves. Section of the pneumogastric nerve, according to Ranney,¹² gives rise to a remarkable set of symptoms: the larynx is impaired in its action; there is dyspnea and hoarseness: the lungs are engorged and consolidated; sometimes there is hemorrhage; the heart is altered in its rhythm, accelerated, often irregular; swallowing is difficult; the liver loses its glycogenic function; the gastric functions are imperfect and there is constipation. These symptoms appear if both nerves are divided below the jugular ganglion. If only one nerve is divided, there is hoarseness, dyspnea, emphysema or pulmonary congestion. A paresis of the pneumogastric, therefore, would manifest similar symptoms, different in degree but not in kind. Such paresis may arise from direct injury to the pulmonary nerves, as is seen in pneumonia and capillary bronchitis, or from injury to some other branch of the vagus, in which case the pulmonary symptoms are reflex.

The pneumogastric, or vagus nerve has its origin in a nucleus near the lower part of the floor of the fourth ventricle, and terminates on the right side in the celiac and splenic plexuses, and on the left side in the hepatic plexus. The left pneumogastric is, therefore, more in touch with the liver than the right pneumogastric. In cases of tubercular deposits in the left lung the liver is more frequently found to be diseased than where the right lung is affected. This is a clinical point of some value, as directing attention to the liver in such cases. The nervous connection between the two explains this.

The pneumogastric communicates through its auricular branch with the glosso-pharyngeal, facial and auricularis magnus nerves, and consequently influences hearing (through the stapedius and laxator tympani muscles), the condition of the tonsils, soft palate and tongue, the muscles of facial expression, and those holding the ear close to the head, as well as the sensation and nutrition of the posterior scalp. Through its junction with the spinal accessory, it further influences the tonicity of the sterno-cleido mastoid and trapezius muscles. The other branches are distributed to the larynx, heart, esophagus and stomach.

A general paresis of the pneumogastric nerve would be evidenced by some such collection of symptoms as

the following: Hoarseness, dyspnea, pulmonary congestion, feeble cardiac action, dyspepsia, coated tongue, constipation, relaxed and edematous soft palate, catarrh, deafness, emaciated face, neck and upper extremities, prominent ears and silky hair on the posterior portion of the scalp. It is also a picture of many cases of pulmonary consumption.

As has been said, pneumogastric paresis may arise from direct injury to some one of its numerous branches. The frequency with which fatty liver occurs in pulmonary consumption points to this as the origin of morbid change in many instances. The interstitial framework of the organ, in which the supplying nerves ramify, becomes inflamed. A neuritis is developed which issues in degeneration, and is thus transmitted to the hepatic plexus and thence into the pneumogastric nerve. This would explain what to Ziegler is a mystery, namely, that the vagus is subject to degenerative change without any compression, inflammation, or other injury.¹³

Inflammation of the hepatic interstitial tissue at once suggests cirrhosis. Osler says,¹⁴ "A remarkable feature (in cirrhosis of the liver) is its association with acute tuberculosis. In seven cases of my series, the patient died with either acute tuberculous peritonitis, or acute tuberculous pleurisy. Pitt states that twenty-two and a half per cent. of the cases of cirrhosis dying in Guy's Hospital during twelve years had acute tuberculosis."

This clinical observation of the association with hepatic cirrhosis of pleurisy and peritonitis cannot be explained by any lesion affecting the branches of the pneumogastric nerve. But how about the phrenic nerve? This also supplies the liver, as well as the pleuræ and peritoneum. It further supplies the pericardium and diaphragm. The right phrenic ends in a small ganglion, from whence branches issue to the hepatic plexus, suprarenal capsule and inferior vena cava. The left phrenic ends in the phrenic plexus, a subdivision of the solar plexus which supplies all the viscera in the abdominal cavity. Peritonitis and pleurisy could thus both originate from injury to the branches of the phrenic nerve in the interstitial connective tissue of the liver.

It will be observed that the hepatic plexus receives the termination of the right phrenic and left pneumogastric nerves. A lesion here, originating from hepatic disease, could issue in paresis of the left pneumogastric nerve, the symptoms of which have already been given, while on the right side there might occur pleurisy, pleuro-pneumonia and defective respiration from paresis of the diaphragm. Nor must it be forgotten that the lower lobe of the right lung is packed tightly between the liver and the ribs, and might easily be compressed by enlargement of the liver, thus causing pressure neuritis of the pneumogastric and phrenic filaments and pneumonic consolidation.

Martin has shown that interstitial nephritis often accompanies or antedates the development of pulmonary tubercle. If the latter is the case, the same course of reasoning already followed in the case of the liver would indicate that the pneumogastric or phrenic paresis, on which the pulmonary lesion depends, has its origin in degeneration of the solar plexus, and that this depends on a neuritis in its renal branches. Interstitial nephritis is the nephritis of gout and of the

¹¹ Occidental Medical Times, November, 1896.

¹² Applied Anatomy of Nervous System.

¹³ Pathological Anatomy, 1886, Sect. 687.

¹⁴ Practice of Medicine, 1892, p. 442.

uric-acid diathesis, both of which depend for their production on an insufficient liver. The inquiry therefore begins with the liver and ends with the liver as an etiological factor in the production of pulmonary consumption. The acute and chronic parenchymatous forms of nephritis, so often associated with phthisis, are more likely to be induced by the passage of tuberculous toxins through the renal parenchyma.

Most persons are attacked, at one time or another, with tuberculosis, but in the majority of cases the seed fortunately falls on barren ground. "The frequency with which foci are met with in the lungs and in the bronchial glands," says Osler (*op. cit.*), "is extraordinary, and the statistics of the Paris morgue show that a considerable proportion of all persons dying of accident or by suicide presents evidences of the disease in these parts. The post-mortem statistics of hospitals show the same widespread prevalence of infection through the air-passages. Biggs reports that more than 60 per cent. of his post-mortems showed lesions of pulmonary tuberculosis. In 125 post-mortems at the Foundling Hospital, New York, the bronchial glands were tuberculous in every case. In adults, the bronchial glands may be infected while the individual is in good health."

Bartholow says that incipient phthisis is characterized by indigestion and an abnormal excretion of urea. He thus bears indirect evidence to the frequent hepatic origin of the disease. For urea is the urinary excretion of the liver cell. If this is in excess, the cell must be in a state of increased functional activity. The same condition of increased excretion of urea is found in diabetes mellitus. Indeed the increased production and excretion of urea has probably much to do with the persistence of the polyuria, for urea is nature's diuretic. When the liver has become worn and parietic from over-use, other symptoms are added to the clinical picture. The secretion of bile is deficient; hence fats are but poorly digested or absorbed in many cases and the body wastes. The leucomaines and toxins of intestinal dyspepsia fail to be neutralized on their passage through the liver, and the body at large is poisoned. Again, the glycogen-forming function is impaired.

Pary holds that one of the functions of the liver is to assimilate glucose, that is, that instead of being a sugar-producing it is a sugar-assimilating organ. The change from glucose to glycogen is apparently effected by the subtraction of a molecule of water, the formula of glucose being $C_6H_{12}O_6$, while that of glycogen is $C_6H_{10}O_5$. The failure of the liver cell to accomplish this allows the unchanged glucose to filter through into the general circulation, thus producing diabetes. It is certainly an interesting coincidence, that whereas glucose is not often found in the urine of pulmonary consumption, inosit is found.¹⁵ This substance is simply glucose plus two molecules of water ($C_6H_{12}O_6 + 2H_2O$).

To recapitulate, the seat of the tuberculous diathesis is in the pneumogastric or phrenic nerves and their connections. The causes which induce neuritis or multiple neuritis elsewhere are also operative here. Such are alcohol, syphilis, the mineral poisons, trauma, the toxins of infectious diseases and uric acid. In addition the glandular structures, and the liver in particular, in communication with these nerves, may degenerate from overwork and communicate their degen-

eration to the main sources of their nerve supply. These factors are the predisposing causes of the disease, the tillers of the soil in which the *tubercle bacillus* later takes root and grows luxuriantly.

According to this view of pulmonary consumption, treatment, to be effective, should be begun early. A diagnosis should be made of the underlying dyscrasia, and this properly treated. Success at this stage will depend much on the skill of the therapist. The diet should be most carefully regulated. It is folly to overfeed a phthisical patient. His digestion is feeble at best. Hence none but the blandest foods should be allowed. Milk, eggs, cheese, fruits, green vegetables, toasted bread, an occasional chicken or other bird, game, oysters and soft water, should compose the dietary. Substances rich in xanthin compounds, as meats, meat soups and extracts had better be avoided, as they result in the liberation of uric acid, which acts as so much poison. The ingestion of oils has long been regarded as a *sine qua non* of phthisical treatment. And if properly introduced into the system, they undoubtedly have a reconstructive action. Long experience has shown that cod-liver oil is more effective than the others. The reason for this has been much debated. If the conclusions arrived at in this inquiry are sound, most phthisical patients have a deficiency of bile. Hence their absorption of fats from the intestinal canal is attended with difficulty. Cod-liver oil, on the contrary, furnishes its own bile and is therefore more easy of assimilation. Oils may also be introduced through the skin by inunction, and as they nourish the nerves and so many of these terminate in the skin, much good can be accomplished in this way. The common habit of greasing the throats and chests of persons afflicted with inflammatory disorders of these parts is an illustration of this principle. The workers in woollen mills, particularly in the "feeding" room, enjoy a singular immunity from consumption and scrofula, and as their clothing and persons became saturated with oil the greater part of the time, it is supposed this has much to do with their health. Vaseline or cocoanut oil are the most elegant substances to use for inunction purposes.

Closely connected with the choice of foods is the use of nascent oxygen. The writer has seen striking results from its use in all stages of the disease and has witnessed several cures. It is well known that phthisical patients often derive astonishing benefit from living out of doors and especially from sleeping out of doors. Neither fever, sweating nor debility contraindicate it. Hirschfelder's contention has already been alluded to, namely, that antitoxins developed within the body are simply oxidized toxins. If this is so, and there is clinical evidence in support of the view, it is easy to understand why the fresh air treatment of phthisis is often curative. The atmospheric oxygen combines with the toxins of tuberculosis to form antitoxins, and these in turn kill the bacilli. Hirschfelder oxidizes the toxins outside the body and then injects the antitoxine hypodermically. Good results are reported of this method of treatment. Nature, however, appears to oxidize the toxins within the body.

In giving oxygen by inhalation, the writer uses a strong solution of hydrogen peroxide, mixed with glycerine in a specially constructed atomizer, and under a pressure of twenty pounds. The patient spends five or ten minutes four times a day in breathing this compound. Where there are cavities in the

¹⁵ Tyson: Exam. of Urine, 1891, p. 103.

lungs the peroxide undoubtedly exercises locally, its well-known cleansing and antiseptic properties. Again, where oxygen is given through its normal channel, the lungs, such of it as is not used in oxidizing the toxins, is directly available for systemic purposes, thus burning up within the body, the waste products of metabolism.

Finally, the suggestion of Tidey¹⁰ to strap the chest walls in phthisis, is an excellent one, and is a great relief to the patient.

Clinical Department.

A CASE OF NEPHRECTOMY FOR RENAL ABSCESS, CALCULOUS PYELITIS, AND CHOLECYSTOTOMY IN THE SAME PATIENT: RECOVERY.

BY JOHN HOMANS, M.D., BOSTON.

Mrs. M., age forty-two, was brought to me in May, 1897, by Dr. Dibble of St. Stephens, N. B. She was a pale, spare woman, who had suffered from pain and dyspeptic symptoms for sixteen years. At times she had vomited; had never been jaundiced. A tumor in the right iliac region had been discovered thirteen years before I saw her. Her most pronounced and prominent symptoms were a tumor in the right lumbar region, much pus in the urine, pain and debility. The abdomen was enlarged between the umbilicus, right ribs and anterior iliac spine. This enlargement was formed by a fluctuating, lobulated, cystic tumor about the size of a man's head, which was freely movable. It was flat on percussion and seemed to be the right kidney enlarged. The left kidney, of normal shape and size, was felt in the left lumbar region. The urine contained much pus, an excess of epithelial cells, a few hyaline and coarsely granular casts with occasional oil drops adherent.

I recommended immediate nephrectomy. It seemed needless to watch the openings of the ureters into the bladder with the endoscope, because the tumor must be removed to cure the patient, and it was more than probable that the greater part of the pus came from the right kidney.

Prof. J. C. Warren saw the patient also, and concurred in my recommendation for an operation. On June 1st I operated at St. Margaret's Home. Professor Warren, Dr. Dibble and Dr. Pease were present.

An incision was made over the tumor, near the right linea semilunaris, about six inches long. On opening the abdomen the tumor could be seen under the posterior layer of the peritoneum, moving with the respiration. The posterior peritoneal fold was nicked and then torn with the fingers. The capsule was peeled away without difficulty till the pedicle, that is, the blood-vessels and ureter, were reached. There was very little bleeding. The veins, about the size of the thumb, and the artery, about the size of a slate pencil, were clearly seen, and were tied separately with silk. The ureter below was tied with the artery and vein, *en masse*, in a Staffordshire knot, and the tumor was cut away. No pus was seen in the ureter.

Great care was taken not to tear the renal veins and not to open the kidney, which was evidently full of fluid. The kidney had been removed intact; not a

drop of pus had been spilled. No shock was noticed during the removal of the kidney.

Having the dyspeptic symptoms and epigastric pain in mind, I now looked at the gall-bladder, and found it much distended and filled with gall-stones. Several ounces of straw-colored fluid were withdrawn by aspiration, and later the gall-bladder was incised. A dozen stones, of different sizes and faceted, were removed. Two were very large: one, an inch and a half long and nearly an inch thick, was firmly impacted in the common duct, and could not be dislodged until it was squeezed upwards towards the fundus of the gall-bladder with considerable force by means of the fingers and thumb in the abdomen on the outside of the duct, and at the same time the fingers within the gall-bladder skinned the walls of the duct back and off from the stone. The gall-bladder was united to the peritoneum by six fine silk sutures; the ends of these sutures were left very long, so that the knots could be cut later and the stitches entirely removed.

This proceeding of removing the sutures which have united the gall-bladder to the peritoneum, or fascia, is very important. In a recent case gall-stones formed again around sutures, and were removed, of quite a large size, twenty months after a successful cholecystotomy.¹ Some operators use catgut which is probably absorbed, but I never have confidence in the aseptic qualities of catgut or animal tendon and always use fine silk in all my aseptic work.

The renal tumor was about ten inches long; the kidney was dilated, full of pus and friable stones which had been united in a coral shape before the tumor was opened. There was more than a pint of foul-smelling pus and many stones.

The kidney was given to Dr. J. H. Wright, as were also culture-tubes taken when the kidney was opened. The bacteriological report of Dr. J. H. Wright of the Massachusetts General Hospital Laboratory is as follows: "June 3d. Tubes marked 'kidney': one sterile, the other shows a few unknown cocci. Tube marked 'from cavity after opening' a few bacilli. Two unmarked tubes sterile. A guinea-pig has been inoculated with a piece of the kidney to test for tuberculosis."

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Kidney removed by Dr. Homans June 1st, and sent to this laboratory June 2d for examination as to whether lesions of tuberculosis were present. Microscopic examination shows no evidence of tuberculosis; the kidney tissue is largely replaced by connective tissue. A guinea-pig inoculated with a piece of the specimen was killed to-day. The autopsy showed no lesions of tuberculosis.

J. H. WRIGHT.

Recovery was uninterrupted. In the first twenty-four hours thirty ounces of urine were passed, showing that the left kidney was doing its work well. The bladder was washed out once. In the second twenty-four hours thirty-six ounces of urine, perfectly clear, were passed, showing that the pus had come wholly from the right kidney. Her diet was almost unrestricted. The gall-bladder stitches were all taken out on the 6th of June. On the 9th of June the amount of urine in twenty-four hours was sixty-two ounces, and after that from forty-five to fifty. On June 22d, three weeks after operation, she went home, perfectly well, free from pain and having gained in weight.

¹⁰ British Medical Journal, March 21, 1896.

¹ See Annals of Surgery for July, 1897.