are thus produced? However, these questions I leave to you, who are so much more capable of answering these questions.

I am afraid you may think that I have occupied too much time on these subjects; but so much of every-day practice is made up of attending to affections of this kind, such a large amount of distress is dependent on them, that this must be my apology. My concluding lecture will note the other sexual differences of disease.

THE GOULSTONIAN LECTURES ON PHARMACOLOGY AND ITS RELATION TO THERAPEUTICS.

Delivered at the Royal College of Physicians, London.

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LECTURE III.—Concluded.

EXPERIMENT has taught us that the food begins to be digested in the mouth, and its starchy constituents are even then partially converted into sugar. In the stomach it meets with the gastric juice, which converts the albuminous matters partly into syntonin and partly into peptones. In the duodenum it is mixed with the bile, which emulsifies the fats, and with the pancreatic juice, which combines the properties of all the three others, digesting the starch, albumen, and fats. Lastly, the intestinal juice probably finishes the solution of albumen and starch, as well as converts cane into grape-sugar. In each secretion, we find its ferment: ptyalin in the saliva, pepsin in the gastric, and trypsin in the pancreatic juice. But these ferments do not exist ready-formed in the glands. They are stored up in the form of a mother-substance or zymogen in the secreting cells, and, when these are called into activity by the nerves of the gland, the zymogen yields up the free ferment ready for its work. But something more is required than the nervous impulse, or, as Mayow would probably have called it, the vital spirit. Unless the glands have a free supply of blood to yield them oxygen and nutriment, they cannot secrete much, however strong the nervous stimulus may be. The secretory nerves of a gland are, therefore, accompanied by other filaments, which cause the vessels of the gland to dilate, so that a copious stream of blood pours through them, making the veins pulsate and jet like arteries. But, though these nerves may often run together, yet their functions are quite distinct. Stimulation of the secretory nerves alone will only cause the gland to yield a few drops of juice and then cease, if the vaso-dilator nerves do not quicken the blood-stream at the same time; and no action of the vaso-dilator nerves, no rapidity of the circulation, however great, will produce a single drop unless the secreting nerves act also. Usually, they are both excited simultaneously, and in the

part to which it was applied, and the secretion is poured out. Each gland seems to have its own favourite stimulus—for the salivary glands acids and sapid substances, for the stomach alkalies, for the liver acids applied to its duct, for the pancreas food in the stomach and duodenum, and for the intestine the presence of food. When savoury substances or acids are taken into the mouth, the stimulus they apply

Fig. 2.

any other drug which we can use? It is not very long since we would have been obliged to reply in the negative; but pharmacological research, besides explaining the *modus operandi* of opium, has given us another remedy more powerful still. This is atropia, which Heidenhain (*Pflüger's Archiv*, v, p. 40) found to paralyse the ends of the secreting nerves in the submaxillary gland, so that no irritation would excite the slightest flow of saliva. At the same time, it leaves the vascular nerves unaffected, and, on the application of the stimulus, the vessels dilate as usual. The paralysis may be removed by Calabar bean, and secretion again will occur. The bean alone has a curious effect, for it excites the secreting nerves, but at the same time causes contraction of the vessels. The gland, therefore, begins to secrete, but soon stops from want of blood. The profuse flow of saliva occasioned by smoking is known to everyone, and examination of the action of nicotine shows that this is just what we might expect; for the drug has a double action on the gland, and does not merely stimulate the sensory nerves of the mouth as pellitory would do, but stimulates the ends of the secreting nerves within the gland itself. In larger doses it paralyzes these nerves and arrests the secretion; but, as the poison is quickly excreted, the stoppage only continues for a short time. Thus we can understand how moderate smoking may increase the flow of saliva, and excessive indulgence in it may cause temporary dryness of the mouth, especially if the tobacco be strong.

A great part of our knowledge regarding secretion in the stomach has been obtained from the observations made by Dr. Beaumont, through a fistulous opening accidentally made by a charge of duck-shot in the stomach of a Canadian voyageur. But this information would have been very imperfect, had it not been supplemented and extended by observations on the stomachs of animals, through circular openings, made by a much less tedious and painful process. From both classes of observation, we learn that the healthy stomach during fasting has a pale colour and velvety appearance, and is covered with a thin coating of transparent mucus. When a moderate stimulus is applied, such as gentle friction with a glass rod, the introduction of food, or a weak alkaline liquid, it begins to secrete, the gastric juice oozes out from the follicles, and collects in drops on the surface of the mucous membrane. At the same time the circulation is increased, as in the salivary gland, and the colour becomes rosy. The stimulus has been conveyed by efferent nerves to nerve-centres, and then through efferent nerves to the vessels and cells, as indicated by the letters A, B, C, D in the diagram (Fig. 3). But if the stimulus be stronger—if, for example, the stomach be violently rubbed with a rod introduced through a fistulous opening, the rosy colour at once disappears, the vessels contract, the membrane becomes pale, and secretion of gastric juice stops, a flow of mucus is produced, and nausea comes on. The stimulus has now awakened a different nervous mechanism (A, E, F, G, in the diagram), and the results are different. If the irritation be still increased, the muscles of the abdominal wall are called into action, and vomiting occurs (A, H, K, K).

Fig. 1.

following manner. A stimulus applied to the mucous membrane lining a part of the alimentary canal, mouth, stomach, duodenum, or ileum, is conveyed by afferent nerves to a nervous centre (Fig. 1). Thence the stimulus is reflected down efferent nerves, as in the adjoining diagram, both to the cells and vessels of the glands connected with the

Weak alkaline solutions, such as saliva, are amongst the most powerful stimulants to the gastric secretion, while acids diminish it. Thus vinegar will lessen the quantity of juice, and will thus interfere with digestion.

Fig. 3.

should dilute the gastric juice, and should take especial care to avoid alkalies, which would neutralise its normal acidity, and prevent its dissolving the iron. The same reasoning may be, and has been, applied to the ordinary process of digestion; for pepsin will not act except in acid solutions, and one might imagine that fluids at meal times, and especially alkalies, such as soda-water, would prove very injurious. But Bernard's experiments show that, although a weak alkali will saturate a little of the acid contained in the gastric juice, yet it will so stimulate secretion that very quickly it will become completely neutralised, leaving a large excess of acid fluid ready to digest any albuminous food that may be presented to it.

Thus it is that weak alkalies, such as bicarbonate of soda, given before meals, are so useful in the treatment of atonic dyspepsia. When the food is taken into the stomach, the organ only responds slowly to the stimulus; too little gastric juice is secreted; the food lies heavy at the epigastrium; and, decomposition of an abnormal kind occurring, gas is formed and eructations annoy the patient. But, when a small quantity of alkali is given shortly before the meal, it stimulates secretion; a quantity of gastric juice is poured forth before the food is actually swallowed, and is ready to attack it so soon as it reaches the stomach; the food is quickly digested, and the unpleasant symptoms disappear. If given with or immediately after a meal, the alkali may have a similar action, but will not be so effectual, for the stomach will already contain some gastric juice secreted under the stimulus of the food. This will immediately neutralise part of the alkali, so that little or none may remain to stimulate the stomach. It is possible that a pipe of tobacco, smoked after dinner, may act by stimulating a flow of saliva, and thus acting upon the stomach; but it is not impossible that it may have an action upon the gastric follicles similar to that which it exerts upon the salivary glands. That it has some action upon the stomach is rendered probable by its power to allay hunger when no food can be obtained.

The question, What is the cause of hunger? is one which cannot be regarded as definitely answered. It is, no doubt, immediately dependent on some condition of the nervous centres in the cranium, and may be present when the blood circulating in these centres is impoverished, although the stomach be full, as in children suffering from *tabes mesenterica*; yet the stomach is the part in which it is apparently felt, and it may be produced by local conditions of the stomach which do not affect the nutrition. For the stomach has little power to discriminate sensations; and the bitterness which quassia or quinine causes in the mouth, and the heat caused by mustard or cayenne, are both felt by the stomach as appetite; and so is the slight irritation caused by small doses of tartar emetic or arsenic, which on this account are said to act as gastric tonics. The usual appetite felt by the healthy stomach after a short period of abstinence seems to depend upon slight irritation of the nerves in the stomach, caused by some

condition either of the gastric follicles or blood-vessels. Increased irritation causes violent appetite, and such an appetite may be induced by the stimulus of a bitter taken before meals or an irritable condition of the stomach itself. This craving appetite may be observed in some forms of dyspepsia, and not unfrequently precedes a so-called bilious attack with vomiting. When the irritation is increased still further, the appetite, as shown by Bernard's experiments and by Beaumont's observations (*vide* Beaumont, *Physiology of Digestion*, 2nd ed., Burlington, 1847, p. 251), disappears and is succeeded by nausea or even by vomiting. From these observations we learn how to treat anorexia due to different conditions of the stomach. When the stomach is languid and lacks its normal sensibility, there will be want of appetite, because the condition of its walls does not afford sufficient stimulus to its nerves. But, when this is supplemented by the additional stimulus of food, appetite is felt. Thus such persons have no appetite before a repast, but, after they begin to eat, acquire a relish for their food and make an excellent meal. When the appetite is due, on the other hand, to excessive irritability of the stomach, the persons feel beforehand as if they could eat a great deal, but, as soon as they have swallowed a morsel or two, the additional stimulus of the food acts like the excessive friction of the rod in Bernard's experiments, appetite disappears, and nausea or even vomiting succeeds. The treatment is evident from the pathology. If the anorexia be due to deficient sensibility, we must give an extra stimulus, such as bitters before meals, or a little more spice and seasoning with the food. Many a time, however, I have been disappointed with the result, when I have ordered quinine or quassia before meals as an appetiser, without considering the pathology of the particular case I was treating, it being one, not of deficient sensibility, but of excessive irritation. The proper treatment was to soothe the mucous membrane instead of stimulating; and on adopting this, and giving bismuth and magnesia instead of bitters, the appetite returned. Sometimes one meets with persons who complain that, after a full meal, they feel as empty as if they had not had anything to eat. Normally, hunger is appeased by eating, probably because the condition of the gastric walls which causes appetite, whatever it may be, is relieved by secretion; but, in the cases I have just mentioned, the food does not produce its ordinary effect. The reason which suggests itself to explain this is, that the stomach does not react so readily as it ought. We have no experiments, so far as I know, to show what drug will affect the reflex action of the gastric nerves; but we know that strychnia increases other reflex actions, and why not this? Strychnia appeared theoretically to be the most likely remedy for such cases; and, on trying it experimentally, I found it successful. Theoretically, also, it ought to be useful in those cases of atonic dyspepsia in which we give alkaline solutions; and its utility in such cases is practically well known. But, supposing the stomach to be too debilitated, either from old age or from exhaustion by disease, to respond to such stimuli as bitters, alkalies, or alcohol, even when aided by strychnia, what is to be done? The organ is then a passive vessel, and as such we must treat it. It possesses a temperature of 99 deg. Fahr.; and we know that, under such circumstances, weak hydrochloric acid with pepsin will digest meat actively when mixed with it in a glass vessel. By introducing pepsin and acid into the stomach, we shall supply the gastric juice which it cannot secrete; and the products of digestion, by strengthening the body, will gradually enable the stomach to do its own work and dispense with extrinsic acid. Bernard's experiment shows us that great irritation of the stomach will cause vomiting; and we know, from other researches, that it is because the irritation is transmitted by sensory nerves, and especially by the vagus, up to a centre in the encephalon* and probably in the medulla oblongata, whence it is reflected to the muscles of the abdominal walls. It is by irritation of the nerves in the stomach that vomiting is usually occasioned; but it may also be produced by irritation of other nerves, such as those of the intestine† or ovaries.‡ It is a reflex act, and is to be arrested either by removing the irritant or diminishing the sensibility of the nerve-centre through which it acts. If the irritant be undigested food or acrid fluids in the stomach, a little mustard and water will remove it; if it be an inflamed condition of the stomach itself, we must use bismuth and hydrocyanic acid to soothe it and deaden the sensibility of the nerves within it; but if it be an irritant which cannot be removed, like a calculus in the gall-duct or a foetus in the uterus, we must employ opium or bromide of potassium to act on the nervous centre within the cranium and prevent it from transmitting any reflex impulses.

In the intestine we can no longer watch so readily as in the stomach what our drugs are doing, but still experiments are teaching us some-

* Gianuzzi, *Centralblatt für die Med. Wiss.*, 1865, pp. 1 and 129.

† Schiff, *Moleschott's Untersuchungen*, Bd. x, p. 390.

‡ Cyon, *Pflüger's Archiv*, Bd. viii, p. 351.

thing about their action there. It has shown that acids applied to the mouth of the bile-duct stimulate the discharge of bile from the gall-bladder, so that the acid chyme from the stomach causes a flow of bile as it passes along the duodenum; but we do not yet know whether the utility of nitrohydrochloric acid in bilious disorders is due in any measure to its increasing the stimulus afforded by the chyme. Rutherford has shown that podophyllin, rhubarb, aloes, and colchicum increase the secretion of bile; but he, as well as the Edinburgh Committee of which he formed a member, found that calomel does not. The benefit which follows a dose of calomel or blue pill in biliousness is so marked that the discrepancy between clinical observation and the results of experiment led many to believe that the latter was worthless and misleading. But facts never contradict one another, although they may run counter to our opinions. The apparent contradiction in this case may be explained by the farther experiments of Lussana, Schiff, and Heidenhain. These physiologists have found that the liver does not only form new bile; it again secretes old bile which has been absorbed from the intestine. Thus all purgatives which sweep out old bile and food from the intestine (and thus prevent its absorption) lessen the quantity circulating in the blood, whether they increase the activity of the liver or not. The effect of any purgative to clear away bile in this manner will depend on the part of the intestine on which it acts. If it hurry on the contents of the duodenum before they have time to be absorbed, it will clear away the bile; but, if it only act on the large intestine, it will have comparatively little effect. We still want experiments on the part of the intestine affected by different purgatives; but it is probable that it is to an action on the duodenum that calomel owes the cholagogue properties which aloes wants.

The digestion of food in the intestinal canal is, we know, carried on by ferments, which decompose and prepare it for absorption. It would appear that each ferment must do its own work, but must not overdo it; for there seems to be a provision for stopping the action of each after it has acted for a while. The ptyalin will not act in an acid fluid; and the gastric juice lessens, if it do not destroy, its activity. Pepsin is precipitated by bile, and is rendered inert by the pancreatic juice, so that its digestive power is destroyed when the food reaches the duodenum. It has not yet been ascertained whether the action of the pancreatic juice is altered by the intestinal juice, but it will not act except in an alkaline fluid; and thus, if the chyme be very acid, pancreatic digestion will be retarded. The various processes of digestion depend upon one another; and, if one be disturbed, the whole will suffer more or less with it. Hence the satisfaction with which we hail such experiments as those of Bernard and Rutherford on the action of our remedies, and the interest with which we look for farther information regarding the digestive process to Brücke and Kühne, who have already taught us so much regarding it.

But it is not in the intestine only that ferments, or, as they are better called, enzymes, play a part. After the food has been absorbed and actually forms a part of the liver in the form of glycogen, it is still subject to their influence, and they convert it partly into sugar. Here, too, our drugs seem to influence these enzymes, and bicarbonate of soda will lessen their activity and retard the formation of sugar.

Some ferments seem to possess the power of building up compounds, instead of breaking them down; and it appears to be a ferment which causes fibrinogen and fibrinoplastin to unite to form fibrin, while pepsin would decompose fibrin into other substances. Facts all seem to point to ferments or enzymes as the agents by which the tissues are built up and again pulled down in that constant change which continues during life, and the action of drugs upon these enzymes is becoming one of the most interesting questions in pharmacology. Their action can be increased or diminished by the action of alkaloids, such as morphia and veratria, and by various salts, such as nitrates, sulphates, and chlorides. But the results differ with the dose as well as with the ferment and the salt used. The same salt in different quantities may at one time quicken and at another retard the action of the ferment. A muscle cut out of the body becomes acid through the action of a ferment it contains. The same muscle in the body becomes acid in a similar way when it contracts. The process of fermentation and of contraction are thus seen to be closely connected, and the question naturally suggests itself, Does the nerve directly act on the muscular substance, or does it act on it through a ferment which it liberates from zymogen in the muscle in much the same way as it does in the cells of a gland? Is it possible, too, for the nerves to liberate this ferment and decompose the muscles without causing them to contract? These questions are of great practical interest, because one product of muscular decomposition is lactic acid, and this acid is generally regarded as an important agent in the production of rheumatic fever, that disease which so frequently leaves behind it the organic diseases of the heart, the treatment of which we have already considered. It

is possible that rheumatic fever is not caused by acid in the blood and secretions, but that the acid and the disease are consequences of one common cause, which may have its seat in the nervous system. But, however this may be, can we arrest the decomposition in the muscles, can we stop the formation of acid by any drug? It would appear that, to some extent, we can; for salicylic acid or its soda-salt not only reduces the temperature in rheumatic fever, but lessens the urea, showing that lessened decomposition of albuminous tissues is taking place. This useful drug we owe entirely to pharmacological research, and, although it is not all we desire, yet the beneficial power it does possess encourages us to hope that we shall ere long discover some other remedy which will satisfy our wants.

We now know that decomposition of the tissues composing the body, whether it be effected by the agency of ferments or not, goes on independently of oxidation, and that it may be increased while oxidation is diminished. According to Voit, albuminous substances, such as those which compose the muscles, liver, and kidneys, do not undergo combustion by direct union with the oxygen supplied to them by the blood, but split up so as to yield urea and fat. The fat should usually undergo farther combustion into carbonic acid and water; but, if the supply of oxygen be lessened or the decomposition be quickened, combustion becomes insufficient and the fat accumulates in the tissues as fatty infiltration and fatty degeneration. Thus it is that, when a limb is disused and the supply of arterial blood to it consequently lessened, fatty degeneration of the muscles takes place. When the coronary arteries can no longer meet the increased calls of a hypertrophied heart for arterial blood, or when they become abnormally contracted, fatty degeneration of the cardiac wall sets in. When the power of the blood to convey oxygen is lessened by diminution of its red corpuscles, the consequent want of oxidation leads to an accumulation of fat frequently noticed in anæmia, and a tendency to obesity frequently appears after severe hæmorrhage.

The pathology of fatty degeneration indicates its treatment. The supply of arterial blood to the organ must be increased, if possible, and its power to carry oxygen is to be augmented by the use of iron, so as to favour combustion. But, at the same time, we must try to restrain the decomposition of the tissues within proper limits. Alkalies have the power to check decomposition of one sort, as in diabetes, but we do not yet know their action upon other changes in the tissues, nor are we able to say that their utility in obesity is due to a power to diminish tissue-metamorphosis. But there is one way of influencing tissue-change which I must not forget. By simply keeping an animal in a hot chamber for a little while, the tissues decompose more rapidly, and the evidence of their waste is to be found in the urine as increased urea. Quicker decomposition is followed by increased combustion, and the temperature of the body rises. Now begins a vicious circle; for the higher temperature itself quickens tissue-change and the fire supplies itself with fuel. The heat, too, stimulates the cardiac ganglia, quickens the heart, dilates the vessels, and accelerates the circulation. Acting on the respiratory centre, it quickens the respiration, increases the supply of oxygen to the body, and thus fans the flame. But, quick as the respiration may be, rapid as is the circulation of arterial blood, it is, as Wickham Legg has shown, insufficient in many instances to keep pace with the decomposition of the albuminous tissues, the products of waste accumulate, and we find them in the fatty heart of fever-patients and in the livers of Strasburg geese.

Now, we clearly see that, whatever may have led to increased tissue-decomposition and combustion in the first instance, the high temperature itself is a cause of mischief, and must be reduced. We no longer regard it as a conflict of vital spirits with which we cannot interfere, nor as an angry struggle of the vital powers with some foe from without, to be encouraged by keeping up a roaring fire in the sick-room, carefully closing every cranny through which fresh air might enter, swathing the patient in folds of flannel, and giving him nothing but warm drink, in spite of his earnest entreaties for a drop of cold water to cool his parched tongue. Instead of this, we regard it as a chemical process going on too quickly, and we try to retard it by securing cool pure air through careful ventilation, while we refresh the patient with washes to his fevered skin and cool drinks to quench his raging thirst. We endeavour to lessen the onward fire which is consuming him by quinine, eucalyptus, or salicylic acid, and to render his feverish pulse slow by aconite. But, when these means fail, and the persistent rise of temperature will assuredly cause death, we know that we can bring it down by the free use of cold water.

This is no new remedy, for by its use Musa saved the life of Augustus. But it is not one to be ignorantly or rashly used; for, while it cured the emperor, it killed his nephew. Even now, in spite of the numerous instances in which life has been undoubtedly preserved by it, many scruple to use it, on account of the dangers which they appre-

hend from it. Experimental pathology and pharmacology have not given us the remedy, but they have taught us how to use it, what are its dangers, and how they are to be avoided. They are death from failure of the heart and death from failure of the respiration. The researches of Panum, Cyon, and others, have shown us that the heart is stimulated to increased action by warmth, but that a sudden fall in the temperature to which it is exposed may act upon its inhibitory ganglia and suddenly arrest its pulsations. The respiratory centre also is stimulated by warmth to increased action. But, while a high temperature quickens the heart and makes it beat more powerfully, it also exhausts it more rapidly. Supposing, then, that both the heart and the respiratory centre should be weakened by continuance of fever, we can readily see that the withdrawal of the stimulus supplied to them by the high temperature may cause them to act so feebly as to endanger life as well as that a sudden fall in the temperature may cause instant death by stopping the heart through its inhibitory ganglia. A knowledge of the dangers teaches us how to avoid them, and the lessons of experiment coincide with those of clinical observation. The temperature in such cases must not be brought down too quickly; it must not be brought down too low, and stimulants must be administered to prevent the action of the heart and respiratory centre from flagging.*

While experimental pathology and pharmacology have done so much for us already, will they not do more? They have taught us how to use such remedies as cold affusion, which were known before, and they have put new ones, like salicylic acid, into our hands. But collapse still sometimes occurs after a cold bath, and salicylic acid does not always prevent the temperature from rising. Will this always be so? I think we may confidently answer, No. We shall yet discover remedies to prevent the collapse, and to keep the temperature within its proper limits. Every day is enriching medical science with some new discovery, diseases are being traced more precisely to their origin, and the action of remedies is being more exactly defined and localised. Order is beginning to appear amongst the crowd of new acquisitions to our knowledge, and isolated facts begin to range themselves under general laws. Pharmacology is allying itself to chemistry, and the rigid laws of the latter are beginning to extend to the former. We no longer attribute the power of drugs to an inherent energy, and say, with Molière, that opium causes sleep because it possesses a *vis dormitiva*. We are beginning to look upon the sleep as only one link in a chain, the beginning of which is a chemical affinity between opium and certain molecules in the nervous system. We observe that drugs having different chemical affinities differ in their effects on the body, while those belonging to the same chemical groups are nearly allied in their action. By altering the chemical composition of our drugs, we can alter the place of their action and change the nature of their effect. We can counteract the effects of one drug by another, and prevent death from a poisonous dose. We are learning the conditions which cause our drugs to affect one part of the organism rather than another, so that we hope are long to foretell from the chemical constitution of any substance the organ it will attack and the functional changes it will effect. At the same time, pathology is teaching us the parts on which we must act, and the nature of the influence we must exert, in order to prevent or arrest the processes of disease. Thus we may hope to predict that a body having a certain chemical constitution will prove an efficient remedy in a particular disease. Hitherto we have been obliged to trust to the substances we procure from plants for our most powerful agents; and, even if we were to know the constitution of the particular remedy we required, it might be impossible to procure it. But now one alkaloid has been made, another and another have rapidly followed it. We can change the constitution and action of these in the same way as we alter those derived from the vegetable kingdom. It is, therefore, highly probable that we shall be able to produce artificially the substances which will act on the body in any way we desire.

Nor may the time be so very far distant. When the oldest licentiates of this College now living were studying their profession, the discoveries of Lavoisier were novelties; digestion was still regarded as a process of solution; the functions of sensory and motor nerves were unknown, and the doctrine of reflex action was still far in the future. The distinction between the tissues had just been made, and the importance of the cell had not been recognised. Pathology had hardly begun, and scientific pharmacology did not exist. When we think of all this and compare it with our present knowledge, we are astonished. If we compare our practice, as at the beginning of these lectures, with that of the Egyptians and Greeks, we are discouraged; but, if we glance

at the advances made during a single lifetime, we cry, "Slow has been the advance of medicine, because she went astray; now the path she follows is right, swift has been her progress and glorious will be her future."

THE DIAGNOSIS AND TREATMENT OF AUDITORY-NERVE VERTIGO.

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[Continued from p. 289 of number for March 10th.]

A GOOD illustration of the obscurity which gastric symptoms may confer on these cases was afforded by a woman, 50 years of age, who came under my care last spring at University College Hospital. She gave a long history of the symptoms of chronic ulcer of the stomach, for which she had been previously in the hospital. On examination, the stomach was found to be tender and evidently dilated. Vomiting was still frequent, occurring chiefly in the evening, when much of the food taken during the day was rejected. Giddiness was complained of as associated with the vomiting. When she was first seen, the giddiness was thought to be due solely to the gastric disturbance. Subsequent questions as to the character of the vertigo and the time of its occurrence elicited a very definite history of another kind. It appeared that the giddiness very frequently came on when there was no sickness; that it occurred in paroxysms; and that it was produced especially by sudden movements, as lying down, or sitting up, or turning round in any posture. In the attacks, the sensation was always of a definite character, being as if "something went over the top of the head from the right side to the left". If standing, she often fell, and, even if sitting, she would fall from the chair. She always fell to the right. Whether she fell or not, she always had, during these attacks, the sense of falling to the right and backwards, never forwards or to the left. Many attacks were witnessed. In some, she fell to the right; in others, merely backwards, although she felt as if falling to the right. In some, she almost lost consciousness; could be roused with difficulty. She said that she had many times actually lost consciousness for a few minutes. During the attacks there was slight flushing of the face. The eyelids were closed; on opening them, there was no dilatation of the pupils. The eyes were all the time in the median position, never turned either to the right or the left, even when the sense of falling to the right was most intense. The duration of the attacks varied from a quarter of a minute to several minutes. The vertigo commenced suddenly, but ceased gradually.

The definite character of the vertigo led to a careful examination of the state of hearing. It was found that a watch was heard less acutely with the right ear than with the left. (Right at one-third, left at two-thirds the normal distance.) She could hear all the notes of a piano; but, after listening for a little time, the upper and middle notes suddenly ceased to be heard. After a little time, she could hear them again. The upper notes, she said, constantly sounded a "long way off and faint, like the strings of a guitar". She had noticed lately that her daughter's voice, in singing, sounded distant and "squeaky", quite unlike its ordinary character.

Audition through the bones of the skull was tested with the tuning-fork. Placed in contact with the top of the head, she said she could hear it in the left ear only. Placed on the left parietal eminence, she heard it invariably louder when the meatus was closed than when it was open. Placed on the right parietal eminence, she could hear it only faintly, and, when the meatus was closed, she could not hear it at all. Examination with a watch yielded the same result. The meatus was free from obstruction. She had been troubled with occasional singing and whistling noises in the right ear.

Sight was unimpaired, except by presbyopia. The ocular muscles were unaffected. Smell and taste were both diminished. Neither peppermint, assafoetida, nor camphor could be recognised. Neither citric acid, salt, nor sugar could be tasted on either side. Quinine was tasted a little. No pain was referred to the ear, but neuralgic pain and tenderness over the right side of the head were complained of, and, on examination, there was distinctly defective tactile sensibility in the parts supplied by the right fifth nerve. There was a good deal of general weakness and prostration, and she was liable to occasional attacks of great prostration, sense of dyspnoea, surface pallor, without marked failure of pulse. She was disturbed at night by starting in her sleep, accompanied with a sensation of falling: she would wake in a

* A remedy which pharmacology indicates as likely to be successful in cases of impending death from the effects of a cold bath in fever is atropia, which paralyses the inhibitory nerves of the heart and stimulates the respiratory centre.