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THE CLINICAL IMPORTANCE OF ACHLORHYDRIA*

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So much has been written on achlorhydria during the last twelve years that it might be thought that very little more could be said about it. But I think there is still room to discuss it from a somewhat wider point of view than has been possible until recently, by considering how it arises and how it comes to be associated with such a remarkable variety of diseases, and, most important of all, how the latter can be prevented by preventing the development of achlorhydria, or, when this fails, by curing the underlying condition instead of merely treating it symptomatically by the substitution of artificial for natural gastric juice. In order to do this I shall have to begin by referring to some fundamental physiological facts concerning the functions of gastric juice.

Functions of the Gastric Juice

1. *Peptic Digestion*.—The glands of the body of the stomach contain two kinds of cells: chief cells, which secrete pepsin, and parietal or oxyntic cells, which secrete hydrochloric acid. The most obvious function of the hydrochloric acid is to activate the pepsin so that it can digest proteins. This is, however, not an essential function, as tryptic activity in the small intestine is so efficient that digestion of meat is unimpaired in complete achlorhydria and after gastrectomy.

2. *Antiseptic Action*.—The antiseptic action of the acid is more important. It destroys streptococci and other organisms which are swallowed with saliva from the mouth and with mucus from the nose and pharynx. It also helps to protect against infection with bacteria present in food and drink. There is a good deal of evidence to show that bacillary and amoebic dysentery occur much more commonly in people with achlorhydria or hypochlorhydria than in those with a normal secretion of acid (Camps, 1933), and the same probably holds good for typhoid and other parenteric infections and for cholera. I am confident that the Services would save a great deal of invaliding if they took the precaution of giving test-meals to all men before sending them to the Tropics in order to eliminate those with achlorhydria.

Knott (1927) has shown that the increased alkalinity of the contents of the small intestine which results from achlorhydria favours its invasion with *B. coli* from the colon; this organism is consequently found in large numbers in the duodenum, which is normally quite sterile. The spread of *B. coli* occurs with extraordinary rapidity. The stomach and duodenum are infected within a few hours of the perforation of a gastric or duodenal ulcer and the consequent inhibition of the secretion and movements of the stomach (Löhr, 1924). This explains why the peritoneum is sterile if an operation is performed within six hours of perforation but after that becomes progressively more heavily infected.

Infection of the small intestine with streptococci from above and with *B. coli* from below, combined with the mechanical irritation of the mucous membrane by food which is insufficiently softened and broken up owing to the deficient quantity

and solvent activity of the gastric juice, is very apt to lead to chronic enteritis. Moreover, when the gastritis which caused the achlorhydria is a result of the action of some swallowed irritant, the small intestine is liable to be damaged at the same time as the stomach. The functions of the small intestine are difficult to investigate and consequently very little is known about their derangements, but we are gradually accumulating evidence that many symptoms ascribed to the stomach and colon are really due to disorders of the small intestine. Thus chronic diarrhoea is frequently associated with achlorhydria; though these cases, which are sometimes wrongly diagnosed as colitis, are now generally regarded as gastrogenous in origin because of the rapid disappearance of the diarrhoea with acid, true enteritis is often present as well.

Cholecystitis is frequently associated with achlorhydria, and is, I believe, generally due to an ascending *B. coli* infection from the duodenum (Hurst, 1932). Even when free acid is present in the stomach the symptoms often date from an attack of acute gastritis, in which achlorhydria may occur temporarily, as Beaumont (1833) demonstrated in his observations on the effect of a drinking bout on Alexis St. Martin, or from pregnancy, which is also often associated with temporary achlorhydria. On restoration of normal gastric secretion the infection dies out from the duodenum, leaving, however, the seeds for the development of inflammation and stone formation in the gall-bladder. The nausea which is sometimes associated with achlorhydria is probably often due to the accompanying cholecystitis.

3. *Haemopoiesis*. (a) *Absorption of Iron*.—The diet taken by most people contains amply sufficient iron to maintain the normal percentage of haemoglobin in the blood. When, however, the diet is deficient or excessive quantities of blood are lost in menstruation or from other sources, a microcytic anaemia results if an insufficient proportion of the iron in the food is absorbed. This may occur in achlorhydria, but it is uncertain whether it is due to there being no hydrochloric acid to convert the organic iron in the food to a form suitable for absorption, or to a failure of absorption by the small intestine, which is so frequently in an unhealthy condition when achlorhydria is present. The latter is probably the true cause, as the simple administration of hydrochloric acid has no effect on the anaemia in such cases, though when large doses of inorganic iron are given the intestines are still capable of absorbing a sufficient proportion for rapid recovery to take place. (b) *Production of Haemopoietin*.—Castle has proved that the gastric juice contains a substance, the "intrinsic factor," which acts on something present in protein food, the "extrinsic factor," to produce the normal stimulant for the formation of red corpuscles by the bone marrow, its absence leading to Addisonian (pernicious) anaemia. Wilkinson's (1933) investigations indicate that the intrinsic factor, which he calls "haemopoietin," is an enzyme. According to Meulengracht (1934) it is produced by the glands in the pyloric end of the stomach and probably the fundus, which contain none of the chief and parietal cells of the glands of the body of the stomach, which secrete respectively pepsin and hydrochloric acid.

4. *Production of Neuropoietin*.—The gastric juice contains an enzyme which is produced by the same glands and acts

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in the same way as haemopoietin to form a substance which is essential for the normal nutrition of the central nervous system. This enzyme, which may be called "neuropoietin," is not identical with haemopoietin, as either may be absent from the gastric juice whilst the other is present. Its absence leads to degeneration of the posterior and lateral columns of the spinal cord. Though the resulting disease is generally associated sooner or later with Addison's anaemia, this is not invariably the case, and degeneration of the cord was found in only 85 per cent. of fatal cases of Addison's anaemia in the days before the introduction of treatment by liver. Salus and Reimann found haemopoietin in the gastric juice of five patients with subacute combined degeneration of the cord and achlorhydria whose blood picture was normal, though it was absent in seven other patients who had Addison's anaemia in addition to the spinal degeneration.

Pathogenesis of Achlorhydria

Out of every hundred normal people about ten secrete more and ten secrete less hydrochloric acid than the remaining eighty (Bennett and Ryle, 1920). The hyperchlorhydria and hypochlorhydria are expressions of what I have called respectively the hypersthenic and hyposthenic gastric constitutions, and both are often familial. They are compatible with perfect general health and good digestion, but whereas the 80 per cent. of people with the average type of stomach are likely to pass through life without developing any serious gastric disorder, most of those with hyperchlorhydria suffer sooner or later from gastric or duodenal ulcer, and most of those with hypochlorhydria develop achlorhydria with its many associated troubles. In both cases this is generally the result of the chronic gastritis which follows prolonged mechanical or chemical irritation of the gastric mucous membrane. Mechanical irritation may be caused by insufficient mastication when food is bolted or dentures are deficient, and by the consumption of excess of coarse vegetable material, including the roughage which is so widely and unwisely recommended for constipation.

The chief chemical irritants are alcohol, especially when taken undiluted on an empty stomach or mixed with other irritants in the form of cocktails; strong tea and coffee; excess of curry, pickles, pepper, and mustard; drugs such as bromide, iodide, mercury, digitalis, quinine, and salicylates, which are taken continuously for long periods; and the poisons which are swallowed when too many cigarettes are smoked. Chronic gastritis is also a common sequel of the acute gastritis caused by food poisoning and by the excretion of toxins by the gastric mucous membrane in acute infections such as gastric influenza, scarlet fever, and septicaemia. These exciting causes are so universal that very few of those predisposed by the hypersthenic or hyposthenic gastric constitution escape. Consequently 10 per cent. of people dying after the age of 40 are found to have an active gastric or duodenal ulcer or the scar of a healed ulcer (Stewart, 1929), and at least 10 per cent. of people over 40 have achlorhydria (Hartfall, 1932).

It is still uncertain whether achlorhydria is ever congenital and due to an inborn error of secretion. In their investigations on a hundred healthy students Bennett and Ryle found that four of the ten with deficient gastric secretion had complete achlorhydria. Combining their statistics with those of Baird, Campbell, and Hern (1924), Wright (1924), and Apperly and Semmins (1928), it is found that 3.4 per cent. of 497 healthy students and children had achlorhydria. But the achlorhydria may have been acquired as a result of acute gastro-enteritis or some general infection in infancy or early childhood. Achlorhydria is often familial; the associated abnormality of the gastric mucous membrane rather than any constitutional weakness of the bone marrow or spinal cord is the

cause of the frequent occurrence of Addison's anaemia and subacute combined degeneration of the spinal cord in more than one member of a family. Records have been published of about 140 families in which two or more members in one to four generations were affected with Addison's anaemia with or without subacute combined degeneration of the cord, and fifty-nine families in which one of these diseases occurred in one or more members of a family and achlorhydria in others, some of whom were young children (Hurst, 1927, Conner, 1930, Wilkinson and Brockbank, 1931). Ellis and Frank have both observed twins suffering from Addison's anaemia. All these facts point strongly to the achylia being constitutional. It is possible, however, that the constitutional condition was an extreme degree of hypochlorhydria, which would be likely to result in achlorhydria at an early age and in Addison's anaemia or combined degeneration of the cord in later life in several members of a family. That achlorhydria may be a sequel of familial hypochlorhydria was shown by two sisters under my care with achlorhydria; treatment of the gastritis led to restoration of the secretion of acid in one but not in the other. Moreover, Wilkinson and others have found that hypochlorhydria as well as achlorhydria is frequently present in other members of the family of patients with Addison's anaemia.

The strongest evidence in favour of the existence of a true constitutional achylia is the occasional discovery of normal, or almost normal, gastric mucous membrane in patients with Addison's anaemia. Passey (1922) found normal oxyntic cells and no evidence of inflammation in a fragment of gastric mucous membrane removed during an operation for appendicitis in a patient of mine with Addison's anaemia and achlorhydria.

Effect of Gastritis on the Gastric Juice

The gastric mucous membrane attempts to protect itself against mechanical and chemical irritants by the secretion of mucus. This mucus is probably produced by the tall columnar cells which line the whole surface of the stomach; it is independent of the dissolved mucus which is always present in normal gastric juice and is presumably secreted by some of the cells of the gastric glands. It is such a weak alkali that its buffer action is hardly appreciable, but it contains sufficient sodium bicarbonate to neutralize some of the acid of the gastric juice (Bonis, 1930). At the same time some of the tenacious mucus adheres to the surface of the mucous membrane and blocks the mouths of the secreting tubules, thus reducing the quantity of gastric juice which can gain access to the lumen of the stomach. The inflammation of the gastric mucous membrane depresses the functional activity of its glands. The oxyntic cells are the most delicate, so that the hydrochloric acid is the first constituent to be seriously affected. If the healthy stomach produced a gastric juice of average or more than average acidity, the reduction of acidity brought about in these three ways is of no importance. If, however, the healthy stomach secretes a gastric juice of less than average acidity, as occurs in individuals with the hyposthenic gastric constitution, achlorhydria results.

In about 60 per cent. of cases the excess of mucus is sufficient by itself to account for the achlorhydria, as on giving a second test-meal after washing the stomach with plain water so as to dislodge the mucus, free acid is found in some of the fractions. In an additional 20 per cent. of cases removal of the exciting causes of the gastritis and treatment of the inflamed mucous membrane by lavage are followed by restoration of normal secretory activity of the stomach.

In rare cases the initial inflammation is so intense that some or all of the oxyntic cells are at once destroyed.

In severe chronic gastritis with achlorhydria the mucous membrane is invaded by streptococci swallowed from the mouth, and progressive atrophy of the glands takes place. Restoration of secretion is then impossible, and the injection of histamine produces no response, even if all active inflammation is overcome by treatment, the condition being, in fact, a true achylia gastrica and not merely achlorhydria. In true achylia most of the cells secreting pepsin are also destroyed.

The production of haemopoietin and neuopoietin by the mucous membrane of the pyloric end and fundus of the stomach is very rarely diminished sufficiently to give rise to Addison's anaemia and subacute combined degeneration of the spinal cord, except in the severe cases of gastritis in which true achylia occurs. In exceptional cases of Addison's anaemia the damage to the mucous membrane is not irreparable. I have had three cases in which the secretion of hydrochloric acid returned as a result of treatment of the gastritis; in one of these, recorded by Shaw in 1926, the patient is still after eleven years completely well with normal gastric juice and normal blood, though he has never taken stomach extract or liver. In the other two cases the restoration of gastric acidity was not accompanied by any return of haemopoietin production, and the patients still require liver. Similar cases of restoration of acidity have since been recorded by McPeak and Neighbors, Wilkinson, Heeres, Seyderhelm and Opitz, and Davidson. In Davidson's case haemopoietin is now also secreted, so his patient can be classed with my first case as an example of complete recovery. In those cases in which the secretion of hydrochloric acid, but not of haemopoietin, returns, the glands in the pyloric end and fundus of the stomach, but not those of the body, are presumably degenerated, just as they must be in the 1 per cent. of patients with Addison's anaemia in whom free acid is present throughout. Hydrochloric acid continues to be secreted in subacute combined degeneration of the cord, though no neuopoietin is produced, in about the same proportion of cases.

Post-Operative Achlorhydria

1. Gastro-jejunostomy.—Partial neutralization by the alkaline duodenal secretions which enter the stomach through the stoma after gastro-jejunostomy, together with the increased rapidity of evacuating, reduces the quantity of free acid present. As, however, the operation is generally performed for duodenal ulcer, in which hyperchlorhydria is almost always present, it is rare for the fall to be sufficient to lead to achlorhydria. When achlorhydria is present after gastro-jejunostomy, the operation has generally been needlessly performed on a patient who already had achlorhydria, or on one with a chronic gastric ulcer in whom the original hyperchlorhydria had been replaced by hypochlorhydria as a result of chronic gastritis. In rare instances, however, the hyperchlorhydria of a duodenal ulcer may be followed by achlorhydria after gastro-jejunostomy. This appears to be due to exceptional severity of the chronic gastritis which always occurs when excess of bile enters the stomach, but which fortunately does not often give rise to symptoms.

Though the achlorhydria makes it impossible for an anastomotic ulcer to develop, it is not an unmixed blessing. Chronic enteritis with diarrhoea and chronic cholecystitis are occasional sequels. Simple achlorhydric anaemia may develop under the same conditions as those which lead to its occurrence in ordinary achlorhydria. I have seen one case of the kind in a man (Cosin and Hurst, 1930) and one in a woman, and one male and fifteen female cases have been recorded by Witts, Davies, and Meulen-

gracht, the haemoglobin varying between 24 and 70 per cent. and the interval after the operation between four and twenty-five years.

The gastritis following the operation may be of such severity that the gastric juice is not merely neutralized, but is no longer secreted, and the achylia may then be associated with absence of haemopoietin or neuopoietin. I know of nine cases in which these diseases followed gastro-jejunostomy; in the majority nothing was known about the gastric acidity before the operation, but one patient of mine had a duodenal ulcer with hyperchlorhydria and developed achylia with Addison's anaemia and subacute combined degeneration of the cord eighteen months after gastro-jejunostomy (Glanvill and Hurst, 1930), and Salus (1932) has recorded two fatal cases of subacute combined degeneration of the cord without anaemia which appeared fifteen years after gastro-jejunostomy, test-meals having shown that free acid was present before but absent after the operations.

2. Complete and Partial Gastrectomy.—One would expect the complete achylia which follows removal of the entire stomach to be followed before long by Addison's anaemia and subacute combined degeneration of the cord, owing to the loss of the glands which produce haemopoietin and neuopoietin. This occurred in cases described by Dennig, Hochrein, Poole and Foster, Ungley, and possibly in a patient of Moynihan's. On the other hand, Mr. H. B. Butler of Guildford tells me that a patient on whom he performed complete gastrectomy in 1926 has shown no signs of anaemia or nervous disorder since, although he has had neither stomach extract nor liver, and Walters (1933) described a similar case two years after operation.

Partial gastrectomy is frequently followed by simple achlorhydric anaemia. As the fundus probably secretes haemopoietin and neuopoietin, Addison's anaemia and subacute combined degeneration of the cord develop only in the exceptional cases in which severe atrophic chronic gastritis involves the remaining portion of the stomach. In 1931 I collected five cases of this kind, and since then at least seven others have been recorded. We always give a test-meal after operations on the stomach. If achlorhydria is present there is less need for prolonged care in diet than if hyperchlorhydria persists, as an anastomotic ulcer cannot develop, but in such cases we advise the patients to take 30 grains of iron and ammonium citrate three times a day for one week in every four, and some form of liver at least once a week for the rest of their lives, as a safeguard against the development of simple and Addisonian anaemia.

Achlorhydria and Carcinoma of the Stomach

Achlorhydria is present in about 65 per cent. of patients with carcinoma of the stomach. It was formerly believed that the achlorhydria was a result of the carcinoma, but in my Schorstein Lecture of 1929 I brought forward evidence to show that the achlorhydria is really caused by chronic gastritis which is present before the growth develops, the growth being a result of malignant degeneration of the chronically inflamed mucous membrane. All the evidence I have been able to collect since then confirms this theory. I know of no case in which free acid was found when a growth was first recognized and disappeared at a later date. The hyperchlorhydria remained unaltered in three patients with chronic ulcers which subsequently underwent malignant degeneration. In another case of ulcer-cancer, in which operation was refused, the acidity actually rose whilst the carcinoma was growing, presumably owing to the favourable effect on the associated gastritis of the diet and lavage which

kept the patient comparatively free from pain. A similar rise was observed by Pollard and Bloomfield (1931) in two cases of carcinoma after an interval of four and six months respectively. On the other hand, I have had four patients in whom achlorhydria was known to be present before the carcinoma developed, and I have found records of at least nineteen similar cases.

As carcinoma follows chronic achlorhydric gastritis, which may also cause simple anaemia, Addison's anaemia, and subacute combined degeneration of the cord, it is natural that these conditions sometimes occur with or precede the development of a growth. Thus carcinoma of the stomach developed in a patient of mine who had recovered from severe simple achlorhydric anaemia as a result of treatment with iron. The occasional association of carcinoma of the stomach with Addison's anaemia and subacute combined degeneration of the cord has long been recognized; I have myself seen five such cases, and at least twenty-two others have been recorded (Wilkinson). Now that most patients with Addison's anaemia can be kept free from recurrence indefinitely with stomach extract or liver, an increasing number of them will develop carcinoma of the stomach if my theory is correct. I have seen two such cases, in one of which carcinoma developed nine years after the onset and four years after the symptomatic cure of Addison's anaemia as a result of treatment with liver (Plummer, 1931). Dyke and Harvey (1933) found that three out of fifty-two patients with Addison's anaemia under treatment died in a period of five years from carcinoma of the stomach, and from inquiries I have made among physicians who have had many cases of Addison's anaemia under their care I find that a good many cases of this kind have now been observed.

The association of cancer and Addison's anaemia with achlorhydria was strikingly demonstrated in the history of a family described by Borovanská-Felklová, in which a man died of carcinoma of the stomach, his eldest son had Addison's anaemia and subsequently developed carcinoma of the stomach, two other sons had Addison's anaemia, and the fourth had achlorhydria alone.

In almost every undoubted case of ulcer-cancer in which a test-meal has been given free acid was present in spite of the fact that the history was often very long, whereas when the evidence pointed against the growth being secondary to a simple ulcer, achlorhydria was generally present, even if the test-meal was given at a very early stage of the disease (Orator, 1925, Stewart, 1931). Free hydrochloric acid was found in all but one of eleven cases of carcinoma of the stomach with more than eighteen months' history, whereas seventeen out of nineteen with a history of less than six months had achlorhydria; these statistics of my own (1931) are confirmed by those of Pantan and Tidy (1910), Orator, and Stewart. The chance of long survival after a successful gastrectomy is greater in patients with achlorhydria than in those with free acid (Hartman, 1921), although the reverse would be the case if achlorhydria was the result of the presence of a growth in a stomach which had hitherto produced free acid.

Cramer (1934) has shown that the total incidence of cancer is the same in all nations, both sexes, and in all classes, although there are wide differences in the relative frequency in which different organs are involved. This indicates that a constitutional tendency for cancer to develop exists in a certain proportion of human beings, and that the organ involved must depend upon local conditions. I have long believed that cancer never develops in a healthy stomach. In about 25 per cent. of cases malignant degeneration occurs in a simple ulcer and in the remainder it occurs in chronically inflamed mucous membrane, the former being always associated with the presence of free hydrochloric acid and the

latter generally with achlorhydria, though the free acid and the achlorhydria have, *per se*, nothing to do with it. It is the association of the constitutional tendency to cancer with one form of the hypersthenic gastric constitution on the one hand and with the hyposthenic gastric constitution on the other which results in cancer of the stomach. At present we have no knowledge of how the cancer constitution can be controlled, but it should be possible to prevent the development of cancer of the stomach by preventing chronic gastric ulcer and chronic gastritis, or, when this fails, recognizing and overcoming them at as early a stage as possible. Prophylaxis consists in preventing the various exciting causes of ulcer and gastritis which have already been described, and which, trivial as they may appear to be, are none the less of supreme importance.

Treatment

Until recently it was assumed that the discovery of achlorhydria was an indication for treatment with hydrochloric acid. It is true that many of the associated symptoms can be overcome by this means, but the underlying gastritis remains unaltered and may become progressively worse. It may finally affect the production of haemopoietin and neuropoietin, and the chronically inflamed mucous membrane may undergo malignant degeneration. It is therefore clear that the gastritis itself should always be treated. This has resulted in the return of secretion of acid in no less than 82 per cent. of our patients (Hartfall, 1932), who thus have the advantage of secreting their own gastric juice instead of having to buy it from their chemist, and the treatment may also prevent the development of Addison's anaemia, subacute combined degeneration of the cord, and carcinoma of the stomach.

Pyorrhoea must be completely overcome by conservative measures when possible, but otherwise by extraction; infected tonsils must be enucleated, and sinus infection must receive appropriate treatment. It is a good plan to give the patient nothing but milk or milky feeds for the first few days of treatment. In the severe cases in which blood is present in some of the fractions of a test-meal, or, as occurs more frequently, occult blood is found only in the stools, the strict diet should be continued till the blood has disappeared. As a rule, two or three weeks suffice, but in exceptional cases of hypertrophic or polypoid gastritis, in which the danger of malignant degeneration is probably considerable, a longer period may be required. The patient is subsequently given an un-irritating diet for six months, or, if the achlorhydria persists, for the rest of his life. He should be teetotal at first and later should confine his drinks to lunch and dinner or immediately after. He should never indulge in spirits or cocktails on an empty stomach. Smoking should be prohibited during the period of strict treatment; after that it may be allowed in moderation, preferably after meals, and the swallowing of irritating juice should be prevented by the use of clean pipes and of cigarettes with a wool plug in the mouthpiece.

The stomach should be washed out every morning with dilute hydrogen peroxide (1 drachm to the pint) until the washings are clear. The treatment should be continued until no mucus is brought away. It is then given on alternate days, later twice a week, and finally once a week for two or three months. In severe haemorrhagic cases 1/2 to 1 part per thousand silver nitrate solution should be used instead of hydrogen peroxide in the early stages. When the washings no longer contain mucus a second test-meal must be given, as it is only necessary to prescribe hydrochloric acid for the 20 per cent. of patients in whom achlorhydria is found to be still present.

From 1 to 2 drachms of the dilute acid (*British Pharmacopoeia*) are given in 5 to 10 ounces of water, to which the juice and pulp of an orange have been added. As in these residual cases the secretion of pepsin is generally reduced, a saltspoonful should be added to the mixture. The first dose should be taken fasting before breakfast, when its antiseptic action is not impaired by partial neutralization with alkaline food. The second is given as a beverage with lunch, and the third with dinner. Patients rapidly get accustomed to it, and should rarely have any difficulty in taking it regularly for the rest of their lives.

Conclusion

Thirty years ago Professor Knud Faber of Copenhagen came to the conclusion that achlorhydria is always secondary to gastritis, and he has maintained it ever since. When shortly after the war I first became interested in the subject I was so impressed with the constitutional factor that I was inclined to doubt whether gastritis was of any importance at all. I now realize that gastritis and constitutional factors are of equal importance. Without gastritis there is no achlorhydria, but gastritis does not cause achlorhydria unless the patient is predisposed by having the hyposthenic gastric constitution. Gastritis in the presence of the hypersthenic gastric constitution may lead to duodenal ulcer and gastric ulcer, and a gastric ulcer may become malignant, but achlorhydria does not develop. It is the conjunction of the apparently trivial causes of gastritis with the hyposthenic gastric constitution which leads to achlorhydria, and the conjunction of these with the constitutional predisposition to cancer which leads to carcinoma of the stomach.

I expect that this is the last time there will be a discussion on achlorhydria, for the problem has shifted, and we must in future discuss gastritis, its causation, early diagnosis, treatment, and prophylaxis, just as we discuss nephritis rather than albuminuria. It is gastritis which causes achlorhydria, and gastritis, not achlorhydria, which causes Addison's anaemia and subacute combined degeneration of the cord and predisposes to carcinoma of the stomach. The prophylaxis of gastritis is the prophylaxis of these diseases. We may therefore look forward to the time when the prevention of gastritis, and, when prevention fails, its early recognition and adequate treatment, will lead to the disappearance of cancer of the stomach, which every day of the year claims over forty victims in Great Britain.

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ACUTE AND CHRONIC SPRAINS*

BY

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I think the Section is wise to have selected for discussion "Acute and Chronic Sprains," so common in occurrence, but so little discussed, that it is well for us to review our ideas and be sure that they are based on sound principles. These conditions and their treatment did not attract us in our student days; they were not big enough. Who ever heard of an out-patient dresser, or even a house-surgeon, who was really interested in a sprained ankle? And yet when the doctor starts in practice it is the minor injury, far more than major surgery, which forms the bulk of the surgical side of his practice. His reputation as a good doctor will be made not by his knowledge and management of cerebral tumours, but by his handling of the sprained wrist and the Colles fracture. I would focus your attention on what passes as minor surgery, and would remind you that it is only "minor" within the shelter of the hospital walls, and assumes very different proportions when we are faced with the same problem in after-life.

We will consider the acute sprain and its management, and then pass on to the consideration of those sprains which fail to clear up and become chronic. But, before entering into a discussion on the diagnosis and treatment, certain points in the anatomy and physiology of joints must be recalled in order to be sure that our treatment is based on a clear appreciation of the underlying principles.

Physiology of Joint Movements

The essential factor to recognize is the importance of the muscles. Many complex structures go to form a joint—bones, cartilage, ligaments, and so on—but for any real understanding of the pathology of joints the role of the muscles must be emphasized.

Muscle is the first line of defence of a joint against injury, and protects it all the time against the repeated though small injuries which are met with in everyday life. If a false step is taken in walking and the balance momentarily lost, it is muscle action which prevents a strain or sprain of the ankle- or knee-joint if we are to avoid injury. If the force is too great, or is applied for too long a time, the muscle gives way; it can no longer protect the joint, and the force then falls on the second line of defence—namely, the ligaments. Here once again consequences depend on the degree of force. If the latter is slight, the ligament may withstand strain, and no ill effect will follow. If it is moderate, some fibres of the ligament will tear, generally from one or other attachment, and a sprained joint results. If the force be severe, a ligament may tear in its entirety, perhaps taking with it a small flake of bone from the point of its insertion or tearing away from its periosteal attachment, and a sprained joint results. Finally, if the force be very severe or prolonged, first the muscle, then the ligament, and finally the bone itself yields, resulting in a fracture.

This conception must be remembered, and we must guard against thinking of a fracture as a broken bone, or of a sprain as a torn ligament; we must not forget the all-important fact that it is on muscle that the first strain falls. Indeed, the fracture is often the least important part of the injury—that is, when considered from the point of view of solution of continuity in the bone. For example, a sprained back with fracture of the transverse

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