end of two years, when the ulcer appeared to be healed, but she prolonged at her own desire the period of jejunal feeding for nine months.

The remembrance of her intense suffering before the operation, and her perfect comfort and satisfaction when she took food only by the tube, weighed with her in making this decision. The jejunostomy opening has been closed for nearly three years, and she remains perfectly well, free from pain, and is now 4 st. heavier than when the original operation was performed.

Symptoms after Unsuccessful Gastro-enterostomy.

The symptoms present in cases of unsuccessful gastro-enterostomy are chiefly four—pain, vomiting, haemorrhage, diarrhoea.

Pain may be due to:

1. A revival of activity in an unhealed ulcer.
2. The presence of a jejunal ulcer.
3. Adhesions crippling the proper action of the stomach or jejunum.
4. The presence of some other disease which has been overlooked at the operation—chronic appendicitis, cholelithiasis, and the other diseases enumerated above.

Haemorrhage may be due to:

1. The separation of a suture.
2. Continuing activity in a duodenal or gastric ulcer.
3. A jejunal ulcer.
4. The presence of some other disease which has been overlooked, such as splenic anaemia, cirrhosis of the liver, etc., as enumerated above.

Vomiting may be due to:

1. Obstruction as a result of any of those technical defects in the operation which I have already named.
2. Functional causes.

Diarrhoea may be due to:

1. Too rapid emptying of the stomach.
2. Some pathological condition of the intestine, or other viscera, which has been overlooked.
3. The offensive character of the escaping contents.

The too rapid emptying of the stomach is sometimes a cause, or is supposed to be the cause, of many discomforts, intestinal uneasiness and turmoil, and diarrhoea. When a patient suffering in this way is examined by the *x* ray the stomach is seen to empty itself very rapidly—perhaps in ten or twenty minutes, the food flowing through the opening into the intestine simply by the act of gravity. In the great majority of such cases that have come within my own knowledge the operation of gastro-enterostomy should not have been performed. The stomach which acts or fails to act, is generally a feeble, flabby atonic organ, capable of exercising little or no control on its contents. The fault here is due to the performance of the operation in an unsuitable case. The jejunum below the opening in such cases appears, by *x*-ray examination and on inspection, to be dilated as a result of the rapid filling; its distension may be the cause of great distress.

As I have said, pathological conditions which have escaped notice may be the cause of a continuing diarrhoea; of such cases are tuberculous disease of the ileum or caecum, or carcinoma of the ascending colon.

Diarrhoea may also occur within two or three weeks of the operation, and last a few days, or even a month or two, rarely more. This is especially noticeable in cases of malignant disease of the stomach causing obstruction, and is probably due to the unchecked escape into the intestine of putrid matter from the stomach, an intense intestinal irritation being the result.

In a few patients upon whom gastro-enterostomy has been performed with the usual striking success an occasional attack of diarrhoea may occur; the stools are liquid and green in colour, and irritating to the rectum. In other patients it may be noted that the stools are often clay-coloured, or almost colourless. In such cases the discomfort is trivial and temporary, and does not detract from the otherwise excellent result of the operation.

CONCLUSION.

The enumeration of all these untoward events in connexion with the operation of gastro-enterostomy appears very formidable. But, compared with the excellent results

of the operation performed in appropriate conditions, and in a skilful manner, the disappointments are nowadays extremely few. Many of the errors to which allusion has been made are probably things of the past. For their avoidance the greatest care in preliminary investigation is necessary. The diagnosis of duodenal ulcer is rarely difficult; the diagnosis of gastric ulcer is never easy. A very large majority of patients supposed to be suffering from "gastric ulcer" are in truth suffering from other diseases. There is only one unequivocal method of diagnosis in cases of gastric ulcer, and that is by *x*-ray examination. The clinical symptoms are very treacherous.

But when the operation is begun no clinical diagnosis, however confidently made, can stand against the conditions revealed then to the hand and eye. If an ulcer is not seen and cannot be demonstrated, it does not exist, and therefore no operation designed for its cure or relief should be performed. A search must then be made elsewhere. And remembrance should always be given to the associated and interrelated actions of such organs as the liver, spleen, pancreas, and the alimentary canal.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, 1910, i, p. 241.

THE INFLUENCE OF DEFICIENCY OF ACCESSORY FOOD FACTORS ON THE INTESTINE.

BY

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(Abstract.*)

THE pathological changes in the intestine which result from a dietary deficient in accessory food factors have been studied in 152 pigeons fed exclusively on autoclaved milled rice.¹ This dietary contains an excess of carbohydrates, is deficient in proteins and fats, and is almost wholly lacking in all classes of accessory food factors. The experiments were continued to a point at which death of the birds occurred, or was about to occur.

MACROSCOPICAL APPEARANCES.

The chief macroscopical features observed at autopsy in the intestine were atrophy and congestion, both of which might be extreme. All cases presented atrophy in greater or lesser degree, while a proportion, estimated at about 30 per cent., showed no congestion appreciable by the naked eye.

The upper portion of the intestine of healthy pigeons, especially that part embracing the pancreas, is thick and muscular. It is this portion which suffers most in consequence of the deficient dietary. The atrophy is of all grades of severity; it may be comparatively slight, or progress to a point at which the intestinal walls are so thin as to be transparent. As a rule the atrophy is more marked the longer the bird is under the influence of the defective diet.

The congestion is commonly confined to the upper part of the alimentary tract; less often it involves its whole extent. The branches of the mesenteric vessels surrounding the bowel may be greatly engorged, and extravasations of blood are occasionally to be seen under its serous coat. An attempt was made to correlate the degree of congestion with the degree of enlargement of the adrenal glands; no correlation could be established.

On splitting open the bowel, the upper three to six inches are often found to be distended with tapeworms, more rarely with round-worms—the latter usually dead, the former almost invariably alive. Round-worms have occasionally been found in the oesophagus.

On examining the surface of the bowel with the hand lens varying degrees of atrophy and erosion of the mucous membrane are encountered. These are not always uniformly distributed over the whole surface of the bowel; some areas show them in greater degree than others. Ecchymoses are common over the mucous surface; in the more severe cases pin-point haemorrhages into the bowel lumen are to be seen.

*The full text, with illustrations, is published in the *Indian Journal of Medical Research*, July, 1919.

HISTO-PATHOLOGY.*

Apart from some degree of atrophy of all coats of the bowel, which is universal in birds dying in consequence of this dietary, no marked histological changes, with the exception, it may be, of slight congestion, are found in approximately 30 per cent. of cases. In the remainder the histo-pathological appearances are very striking, although widely variable in degree in different birds. They comprised the following pathological changes:

1. *Congestion and Haemorrhage.*

In its mildest form this change consists in engorgement of the subperitoneal vessels, and in distension of the small vessels ramifying between the muscularis mucosae and the base of the crypts of Lieberkuhn, or the glandular structures corresponding to these in birds. The process of congestion is not usually limited to distension of vessels in these areas but may extend to those of the villi. The next stage in the congestive process is the occurrence of actual haemorrhages around the bases of the crypts. In some cases an almost continuous layer of haemorrhage may surround the bowel at this point. Such remnants of villi as remain are often seen to be much engorged or to contain effusions of blood; free corpuscles may be found in the lumen of the bowel or exuding from its congested and frayed mucous membrane.

A further, or simultaneous, stage in the process is the spread of these haemorrhagic areas from the submucous coat into the myenteron; these may involve only the circular layer of muscle fibres or penetrate to the longitudinal layer. It is not unusual to see larger tracts of the circular layer of muscle broken up by extensive haemorrhages into its substance. Such haemorrhages cause separation and rupture of the muscle fibres; the ruptured fibres may present ragged edges enclosing the collection of effused blood; the haemorrhages may be more diffuse, the corpuscles being scattered throughout the whole thickness of the myenteron and penetrating to the serous coat. Added to these appearances is the engorgement—often intense—of the vessels running under the serous coat and ramifying in the muscular layers. Oblique and longitudinal sections of these vessels may at first sight present the appearance of haemorrhagic effusions. Rupture of the vessel walls is, however, rare. Oedematous infiltration of the coats of the bowel has occasionally been encountered.

It thus appears that intense congestion of, and haemorrhages into, the coats of the upper bowel, sometimes leading to actual breaches in their continuity, are amongst the results of a dietary deficient in accessory food factors. It may be assumed that resolution of such haemorrhages would lead to the deposition of much fibrous tissue in the myenteron and serous coat.

2. *Atrophy of the Myenteron.*

The thickness of the circular layer of the muscular coat is not the same at every point of the bowel's circumference; it gradually merges from about 0.35 mm. to about 0.25 mm. In pigeons fed on the deficient diet the degree of atrophy of the circular muscular layer varies considerably. The muscular coat is usually thinnest in birds which survive longest, or in which infection of the bowel walls has occurred; it is atrophied, as a rule, to the extent of one half to one quarter its normal thickness in health, and its efficiency is thus proportionately reduced. Added to this are frequent haemorrhages, the rupture and separation of muscle fibres thereby, and, less commonly, segmentation and fragmentation of these fibres. The extent to which the motor function of the bowel may be impaired by the deficient dietary will be realized from these statements.

3. *Degenerative Changes in the Myenteric Plexus of Auerbach.*

Careful scrutiny is necessary in order to detect, in healthy birds, the ganglionic swellings of this plexus, as they lie between the circular and longitudinal layers of the myenteron. They are compact bodies of oval form varying considerably in size, the variations being, of course, dependent on their point of section. They fit snugly between the layers of muscle and give rise to little or no bulging in the contour of these layers.

*This account of the histo-pathology is based on an examination of serial sections of the intestine in twenty-four pigeons fed to the point of death on autoclaved rice.

In sections of the intestine from birds fed on the deficient diet these ganglia are much more obvious and catch the eye at once. Their readier recognition may be due in part to the fact that the atrophy and recession of the circular and longitudinal muscle fibres renders them more prominent. Their greater prominence is, however, often the result of swelling due probably to oedematous infiltration. In size they vary within wide limits dependent in some measure on their point of section. In general, they are double or treble the size of healthy ganglia.

The swollen ganglia have frequently been noted to cause bulgings of the longitudinal muscular layer towards the serous covering of the bowel or of the circular layer towards its lumen, or both. The cytoplasm of the ganglionic cells may appear shrunken, their nuclei more eccentric than normal, and the nucleoli almost invariably fragmented, the last appearance being the most constant. Occasionally, whole cells or groups of cells appear to have disappeared, leaving in the ganglion lacunae empty of cellular structure.

There can, I think, be little doubt that the plexus myentericus is involved in a degenerative process. This being so, it may be concluded that the nervous control of the bowel is impaired in proportion to the degree of degenerative change.

4. *Atrophic and Inflammatory Changes in the Mucous Membrane.*

These consist in (a) atrophy and partial or complete disappearance of the villi; (b) frequent congestion of such villi as remain; (c) atrophy of the glandular cells of the crypts of Lieberkuhn, and separation of these from their basement membrane; (d) partial or complete disappearance of the lymphoid cells lying between and around the crypts; (e) thinning, or actual disappearance in places, of the muscularis mucosae; (f) a relatively greater proportion of reticular cells, and the scattered deposition of fibroblasts; (g) an increased proportion of leucocytes in the atrophic mucous membrane; and, more rarely, (h) bacterial invasion with intense inflammatory and necrotic changes in the mucous membrane and underlying coats of the bowel.

These appearances are, as a rule, most marked in birds which have survived the dietary for the longest time or in which infections of the bowel wall, are found at autopsy. They are rarely present in the same degree at every area of the bowel's circumference. Sections of even the most severely affected intestines may show some areas in which these changes are much less marked than in others.

5. *Atrophy of Lymphoid Structures.*

In the healthy pigeon the lymphoid cells fill in the spaces between the crypts, and present a continuous layer of varying thickness between the bowel lumen and the muscularis mucosae. In diseased birds this protective layer is atrophied in greater or lesser degree. In some areas it was completely wanting, and the muscularis mucosae also had frequently disappeared.

6. *Fibrosis.*

The evidences of fibrosis are scanty in so far as the mucous membrane itself is concerned. Areas, however, are encountered in which there is a deposition of scattered fibroblasts. Some long-standing cases present areas of fibrotic infiltration in the muscular coats; but this being a reparative process, it is likely to be encountered only rarely in birds not permitted to recover. It may be expected that where such recovery is brought about the resolution of the haemorrhagic effusions will give rise to extensive fibrotic deposits in the muscular and serous coats of the intestine.

7. *Changes favouring Infection from the Diseased Bowel.*

Systemic infection is rendered more easy in the presence of these pathological processes owing (1) to impaired production of digestive juices; (2) to continued congestion of the mucous membrane and consequent malnutrition of its secretory cells; (3) to loss of the protective layer of lymphoid cells; (4) to increased leucocytic invasion of the mucous membrane and to increased leucocytic traffic in micro-organisms between the bowel mucous membrane and the blood stream; (5) to the greater facilities which

the debilitated mucous membrane provides for the growth of bacteria on its surface and in its substance; (6) to the imperfect digestion of food in the upper alimentary tract, which affords a favourable medium for the growth of micro-organisms; and, although more rarely, (7) to actual breaches of continuity in the walls of the bowel itself. Occasional specimens have shown a direct continuity of blood corpuscles from the eroded villus to the blood vessels running external to the serous coat.

Cases were encountered with comparative frequency in which the effects of intense bacterial invasion of the bowel walls have been added to the atrophic and congestive processes. In such cases bacteria of various types were to be found in large numbers throughout the necrotic mucous membrane and lying also in the submucous and muscular coats. By special staining methods bacteria (chiefly cocci) have been detected in the vessels of the submucosa, and bacteria-laden leucocytes are common objects in the microscopic field. In these circumstances bacterial organisms may pass directly into the blood stream from the infected bowel walls. In four such cases I cultivated from the heart's blood at autopsy, by aerobic methods, a small coccus in two, a coliform organism in a third, and bipolar organism in a fourth. Infection of the blood from the bowel is therefore no uncommon consequence of "vitaminic" deficiency.

In cases of intense infection of the bowel walls the atrophic congestion and necrotic processes may be so pronounced as to render the recovery of the mucous membrane impossible. One is occasionally surprised to find that, while in one case the administration of "vitaminic" extracts will cure "polyneuritis" with surprising rapidity, another, to all appearances identical with it, will succumb. Amongst the reasons for this are the varying degrees of pathological change and of bacterial invasion of the bowel walls as well as systemic infection therefrom. It is evident that the ingestion of specific pathogenic organisms (such as *B. dysentericus*, *B. typhosus*, *Entamoeba histolytica*, etc.) in the presence of "vitaminic" deficiency is likely to be followed by their implantation on the debilitated bowel mucous membrane.

In connexion with these lesions in the upper alimentary tract of pigeons fed exclusively on autoclaved milled rice it is to be remembered that Hamilton Wright³ was so impressed with the constancy of pathological changes in this situation in human beri-beri that he attributed this malady to a specific gastro-duodenitis. As is now well known, the pathological changes he described are not the cause of beri-beri; they are, however, important evidences of deficiency of certain accessory food factors in the diet. The fact that Wright found a bacillus of constant morphological characters in this situation is important. Although the organism is not the specific cause of beri-beri, its presence is an illustration of the fact that the wall of the upper intestine may become infected in the human subject in consequence of such a dietary.

During the course of my studies of the pathogenesis of deficiency disease⁴ I have had occasion to feed a number of pigeons (forty-two) on a dietary consisting of autoclaved milled rice, fresh butter, and onions—that is to say, on a dietary containing an excess of carbohydrates and deficient in accessory food factors, chiefly of the water-soluble "B" class. Naked-eye evidences of congestion and other pathological processes are much less frequently encountered in the intestine in birds so fed. The atrophy of the bowel, although often considerable, is usually less marked than in cases dying in consequence of complete "vitaminic" starvation. Histologically the same degree of congestion is not encountered, actual haemorrhages are rare, and the degree of atrophy of the myenteron and of the elements of the mucous membrane, although occasionally considerable, is often comparatively slight. A dietary of autoclaved rice which is enriched with butter and onions thus provides some at least of those factors on which the functional perfection of the alimentary tract is dependent.

COMMENTARY.

Interpreted in terms of bowel function, the derangements to which such pathological changes as have here been described may ultimately lead can be grouped as follows:

1. Impairment of the neuro-muscular control of the bowel; impaired transport of the intestinal contents along the alimentary canal.

2. Impairment of assimilative power.

3. Impairment of secretory function.

4. Impaired protective resources leading to infection of the mucous membrane of the bowel by pathogenic saprophytes, or ingested bacteria, and to systemic infection therefrom.

Unwilling as I am to apply too directly to the human subject the experimental results of intensive "vitaminic" starvation observed in birds, we may, I think, regard the changes I have described as applicable in some degree to man.

It is my experience that, while complete deprivation of accessory food factors will cause the relatively rapid appearance of so-called "polyneuritis"* in birds, sub-minimal provision of these substances, protracted over prolonged periods of time, will, especially in young and growing birds, eventually lead to a like morbid state. It is, I think, with sustained subminimal supply of accessory food factors that the physician is mainly concerned in practice. Complete "vitaminic" deprivation is, as I have pointed out in another place,¹ relatively rare; incomplete "vitaminic" deprivation, or subminimal supply of accessory food factors, is relatively frequent. If, in the light of the histo-pathological findings here recorded, we consider the case of children fed, it may be from birth, on foods deficient in these essential substances, and the frequency with which in later life their dietary continues to be ill-balanced and defective, we are, I venture to think, in a position to realize the ultimate consequences of such a dietary and to anticipate the sequence of events leading up to grave derangements of bowel function. Food deficiency also prepares the soil for bacterial growth, and the resultant morbid states will vary with the nature of the organisms which may become implanted upon it. This knowledge, if it does not enable us to account fully for such gastro-intestinal disorders as mucous disease, coeliac disease, and chronic intestinal stasis, will at least help us to a better understanding of their genesis.

MUCOUS DISEASE.

I have referred to the fact that pigeons fed on an exclusive dietary of autoclaved rice frequently suffer from diarrhoea,¹ and have pointed out that this symptom of "vitaminic" deficiency can in a proportion of cases be made to disappear, often with surprising rapidity, by the administration of the alcoholic extracts of the yolks of eggs. Others have drawn attention to the almost constant occurrence of diarrhoea or of colitis as precursors or concomitants of the condition known as "war oedema," a malady proved to be due to food deficiency.⁴ Clinical evidence of bowel derangements due to this cause are, therefore, not lacking. If, then, we attempt, from the results of our histo-pathological observations, to provide an explanation of the origin of mucous disease, we may, I think, surmise that continued subminimal "vitaminic" supply will lead to that state of chronic gastro-intestinal catarrh which characterizes this disorder.

Mucous disease is very common amongst European children in India who are fed largely on sterilized milk (milk which, in my own experience, is often poor in "vitaminic" content), artificial foods, white bread, polished rice, poor butter, over-cooked vegetables and excessive quantities of sugar. Therapeutic experience of this malady shows how readily it yields to limitation of carbohydrates and to a rationally-balanced dietary of good "vitaminic" quality. The mucous stools, the diarrhoea, the peevishness, the unhealthy appetite rapidly disappear on such a regimen, as also do the "night terrors" from which the subjects of mucous disease so frequently suffer.

I do not wish to dogmatize with regard to the genesis of this malady. I would but draw attention to the fact that an ill-balanced diet, deficient in accessory food factors, is capable of giving rise in pigeons to a state of chronic congestion of the intestinal mucosa, often accompanied with hypertrophy of the adrenal glands; the relief afforded to children suffering from mucous disease by the correction

*The term "polyneuritis," as applied to the morbid state resulting in pigeons from an exclusive dietary of autoclaved milled rice, is a misnomer. It suggests that the nervous tissues are exclusively or mainly involved in the pathological process, whereas in reality no organ or tissue escapes the consequences of "vitaminic" deficiency; some, indeed, such as the reproductive organs, the spleen, the thymus, and the digestive organs, are more gravely affected than is the nervous system. (See BRITISH MEDICAL JOURNAL, February 15th, 1919.)

of their ill-balanced dietaries may find an explanation in this fact.

COELIAC DISEASE.

One cannot read the illuminating account of coeliac disease given by Dr. G. F. Still⁵ in his recent Lumeleian lectures without considering the possibility that it also may owe its origin to similar dietetic deficiencies. Its absence in breast-fed children, its onset between the age of nine months and two years, the diarrhoea which so frequently precedes it, the cessation of growth, the ill-formed, pale "oatmeal" stools, the frequent association of scorbutic symptoms, the abdominal distension, the afebrile nature of the malady, the diminished size of the liver, the blood changes, the occurrence of oedema, the thin bones, the muscular feebleness—all these find their counterpart in pigeons fed on an exclusive dietary of autoclaved milled rice.¹ The pathological findings recorded in this paper indicate with what readiness pathogenic organisms may invade the atrophying intestinal mucous membrane and lead to inflammatory states and fibrotic thickenings therein. The enlargement of the adrenals, which is so constant a result of "vitaminic" starvation, and so frequently associated with oedema in birds, may not be wanting in coeliac disease; I can find no records with regard to the adrenals in this malady.

I venture to suggest that those who have the opportunity should examine cases of coeliac disease, and more especially all available *post-mortem* material, from the point of view of food deficiency. Whether or not this malady is found to be due primarily to such deficiency, it would appear probable that a number of its characteristics are secondary to the imperfect assimilation of these essential ingredients of the food.

If, now, we bear in mind the possibility of subminimal "vitaminic" supply protracted over prolonged periods from childhood onwards, we have, I think, a new viewpoint from which to consider the genesis of that common disorder.

CHRONIC INTESTINAL STASIS.

In the recent edition of Sir Arbuthnot Lane's work on this subject⁶ Professor Arthur Keith draws attention to two anatomical factors which he regards as of primary importance in the causation of chronic intestinal stasis: (1) Defective action on the part of the abdominal musculature, and (2) a lesion of the neuro-muscular system of the intestine.

The histo-pathological findings recorded in this paper indicate one means by which both the abdominal musculature and the neuro-muscular system of the intestine may be simultaneously impaired in functional capacity. Deficiency of certain accessory food factors leads to atrophy of all muscular tissue as well as to disordered function or actual degeneration of motor nerve cells throughout the entire body. The abdominal musculature and the nerve elements controlling it must of necessity suffer along with other muscular and nervous tissues. It may be concluded, then, that defective action of the abdominal musculature will ultimately result in consequence of deficiency of accessory food factors.

In addition to this functional defect of the abdominal wall, we find also in pigeons starved of "vitamines" unquestionable evidence in the wall of the intestine itself of neuro-muscular lesions of great significance. Whether or not we are justified in applying these findings to the genesis of stasis in the human subject, we may at least regard with suspicion human food which does not conform to standard in respect to perfect "vitaminic" balance.

There can be no doubt that the dietaries of children amongst the poorer, and often amongst the richer, classes are frequently deficient in these important ingredients. Subsistence on such ill-balanced dietaries from childhood onwards may be regarded as calculated to lead to defect of the abdominal musculature, to neuro-muscular lesions of the intestine, and to changes in the glandular elements of its mucous membrane. Indeed, the pathological changes in the large bowel in stasis, as described by Professor Keith, are strikingly similar to those I have enumerated as occurring in pigeons fed on a "vitamine"-free food. He describes the morbid changes as affecting two systems—the glandular and the neuro-muscular:

The chief change in the muscular coats of the diseased great bowels removed from cases of enterostasis is a fibrosis—one which mainly affects the tissue between the outer and inner

coats of the bowel, the intermediate stratum which insheaths the myenteric plexus. Degeneration in areas of the musculature, particularly of the outer longitudinal coat, also occurs. In every case there are inflammatory changes varying in degree. There are many marked changes in the lining glandular epithelium and in the submucous coat. One notes also that there is engorgement of the subperitoneal vessels and that the subperitoneal tissue is much thicker and denser than is normal.

Having regard to the fact that the pathological processes I have described are acute in nature, and induced by intensive "vitaminic" deprivation, whereas those recorded by Professor Keith are chronic, the similarity between them suggests that subminimal "vitaminic" supply protracted from an early age has a fundamental influence in the origin of the lesions found in stasis. However this may be, we must regard with suspicion an influence which is capable of providing at once the anatomical conditions necessary for the development of stasis and the facilities for systemic infections which are known to result from it.

CONCLUSIONS.

1. Deprivation of accessory food factors leads in pigeons to congestive and atrophic changes in all coats of the bowel, to lesions of its neuro-muscular mechanism, to impairment of digestive and assimilative processes, and to failure of its protective resources against infection.

2. Certain gastro-intestinal disorders in the human subject—of which three examples are referred to—may owe their origin to subminimal supply of accessory food factors protracted over long periods of time.

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AN INVESTIGATION OF THE END RESULT IN ONE HUNDRED AND TWENTY-FOUR CASES OF BLOOD TRANSFUSION.

BY

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The following is a record of the results obtained in a series of blood transfusions carried out in a base hospital in France between November, 1916, and October, 1918; with a comparative table of the results in cases of as nearly as possible identical clinical nature, treated before blood transfusion was available.

Method.

Indirect transfusion by Vincent's paraffin coated tube was employed; thus unaltered blood was used. The use of citrated blood is open to the objection that citrate is a depressor of blood pressure.¹ Experience has shown this objection to be mainly theoretical, and as transfusion of citrated blood is easily performed and carries least risk to donor or recipient, I believe it to be the best method. It was, however, not available in the period dealt with.

Compatibility.

This was established at first by direct comparison of the bloods under the microscope, later by obtaining Group IV donors by the convenient macroscopic method introduced by Lee.²

Indications.

The main indications for blood transfusion in a base hospital are (1) severe secondary haemorrhage, (2) preliminary to high amputation of the thigh or arm, (3) certain cases of pyaemia. The primary indication for transfusion, surgical shock, is much less frequent at the base than at a clearing station, but I cannot subscribe to the view expressed that secondary haemorrhage is rarely accompanied by shock.³ There is a class of cases in which typical symptoms of shock are present, and the amount of blood lost is so small as to preclude the possibility of the clinical condition of the patient being due to anaemic