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

MAJOR W. GRANT WAUGH, R.A.M.C.

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

collapse. Not infrequently these cases exhibit premonitory symptoms, consisting of extreme restlessness, blanching, and increase of pulse rate, which are sufficiently definite to lead one to make sure that a tourniquet is readily available. A lesion of a main arterial trunk is almost invariably present in such cases. Experimentally, it was found that manipulation of a large vessel in a wound caused considerable variation in the general blood pressure, even if the vessel were uninjured. These cases lend support to the view that disturbance of the nervous mechanism of the vascular system may be the initial pathological factor in the production of surgical shock.

Amount.

The amount of blood employed was usually 600 c.cm., but amounts of 900 to 1,200 c.cm. have been injected as the condition of the patient demanded. The donor's blood pressure usually fell about 10 mm. Hg, but recovered to normal within an hour.

Accidents.

(a) *Incompatibility* has been met with on three occasions. In two of these transfusion was abandoned, no ill effects persisting; they occurred early in the series, and were probably due to faulty comparison of the bloods. (These cases are not included in the series.) The third case occurred in a patient suffering from pyaemia, and merits a detailed note.

The patient was being transfused for the third time, the same donor being employed as in the second transfusion, performed without mishap three weeks previously. The recipient's condition was normal until about 200 c.cm. of blood had been injected, when acute collapse, with symptoms of haemolysis, occurred, and death took place abruptly. Autopsy revealed no evidence of embolus in lungs or cerebrum, and as a specimen of blood taken from recipient's vein was found to be haemolysed, I have no doubt that death was due to incompatibility.

In addition to emphasizing the necessity for ascertaining the symbiosis of the two bloods before every transfusion, the case seems to indicate that incompatibility may develop as a result of multiple transfusion; unfortunately it was impossible to determine whether the antagonism in this case was peculiar to the blood of the individual donor, or a true incompatibility to an entire blood group. Incompatibility usually declares itself after 10 c.cm. of blood have been injected; in this case injection had proceeded for a considerable time before any symptoms appeared. Accidents of a similar nature have been reported by other observers; a suggested explanation given by Bond is that "a definite alteration has been found to occur in the direction of increased haem-agglutinin content of the blood in the interval between two transfusions," and that this was responsible for the unfortunate result.⁴

(b) *Injury to Donor.*—This occurred once, probably the result of the transfusion tube having been reintroduced to the donor's vein after being employed on the recipient. Infection spread distally in the radial vein, which was removed through an incision at the wrist in the manner of Babcock's operation for varicose veins; complete recovery followed. A transfusion tube should not be filled a second time, and to avoid this it is convenient to employ a tube of 600 c.cm. capacity. In closing the donor's wound I make a point of removing half an inch of the vein opened in case it may have suffered damage during the proceedings.

Conclusions.

The cases are tabulated according to the clinical condition and operation performed. The figures, it should be remembered, represent a considerable proportion of the desperate cases met with in base hospital work, and afford no guide to the general death rate in the various clinical conditions. Transfusion for pyaemia was to some extent experimental, and was in fact an attempt to do something further than merely drain secondary abscesses. Patients who had recovered from some slight septic lesion were employed as donors. I think the number of recoveries in these very severe cases justifies an extension of the method as a therapeutic measure. The results in Class II indicate the usefulness of transfusion as a prophylactic against shock; it seems reasonable to hold that it should come into general use in civil practice as a preliminary to any severe operation.

Clinical Condition and Operation Performed.	Cases Transfused.			Cases not Transfused.		
	No.	Recov-ery.	Death.	No.	Recov-ery.	Death.
CLASS I.						
(a) Secondary haemorrhage; ligation of main artery	52	35	17=32.7%	52	28	24=46.1%
(b) Secondary haemorrhage; amputation of limb	14	10	4=28.5%	14	7	7=50.0%
CLASS II.						
Sepsis or gangrene; amputation of limb	39	35	4=10.2%	29	20	9=31.0%
CLASS III.						
Pyaemia	19	12	7=36.8%	—	—	—
Totals	124	92	32=25.8%	95	55	40=42.2%

A number of sphygmomanometric records were taken of the effects on blood pressure of various substances used in intravenous injections, and will be made the subject of a future communication.

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MASTOIDITIS.

BY

ADAIR DIGHTON, F.R.C.S.,

HONORARY SURGEON, EYE AND EAR INFIRMARY, LIVERPOOL; HONORARY AURAL SURGEON, BIRKENHEAD CHILDREN'S INFIRMARY.

For the last ten years I have been in daily contact with cases of suppurative otitis media, or its complications, in the out-patient department and operating theatres, and some opinions founded on this experience may be of interest.

The term "suppurative otitis media" is a misnomer, as in every case the disease starts in the mastoid antrum, and the suppurative otitis media is only an outward and visible sign or symptom of this invisible disease. I had swabs taken from the bottom of the mastoid antrum in fifty consecutive cases of operation for suppurative otitis media, and in every one pathogenic micro-organisms were found. The cases were not selected, but were taken as they came, and ranged, according to the duration of the discharge, from one day to seventeen years; and, according to the age of the patient, from one year to sixty-nine. It is therefore not a matter of wonder that treatment by drops, syringing, powders, etc., is ineffectual.

During 1915 I had further proof in a school clinic at which the patients could be seen and treated daily. The patients were divided into sets. One set was treated with drops, another set by hydrogen peroxide and syringing, another by massive lavage, etc.; but in no case was there any sign of improvement of any sort, but, on the contrary, the few cases which came to operation later on at the hospitals showed more extensive bone destruction than might have been expected from the duration of the disease. This experience was repeated during the years 1917-18, when, as officer in charge of the aural centre at Blackpool, 4,148 cases passed through my hands; 90 per cent. were cases of suppurative otitis media. They were treated daily by all known methods, with no good result of any kind, and remained in their original category of B 3. A great waste of men who, by means of operation (no beds were available), might, in the space of three months, have been raised to category A 1.

As the mastoid antrum is the real cause of suppurative otitis media, the only feasible treatment is by operation— and early operation. During my period as officer in charge of the aural centre I took particular note of all operated cases, and found that among 53 cases only 9 could justifiably be called healed. Of these 9, 5 underwent a radical operation, and 4 a modified mastoid, or Heath, operation.