

making an exact diagnosis, an acute psoriasis and a subacute eczema, for instance, often requiring the same local treatment, but in others an accurate diagnosis is essential, as in the inflammation excited by scabies in clean people, which is so often erroneously treated as an eczema, or in not recognising that impetigo contagiosa is secondary to some previous itching lesion such as urticaria, prurigo, animal parasitic irritation, &c. It is, however, often desirable and even necessary to remove the secondary lesions first in order to pave the way for the most effectual means of treating the primary eruption.

#### GENERAL DEDUCTIONS.

The general deductions which may be drawn from the subject we have been considering are:—That a large proportion of inflammatory diseases of the skin are of compound origin. That there is frequently a microbic element and that this too may consist of more than one kind of microbe superimposed on another such as the staphylococcus aureus on the streptococcus pyogenes, the staphylococcus on the seborrhœic micro-bacillus, the seborrhœic micro-bacillus on the bottle bacillus, &c. That not only do these several mixtures produce different forms of dermatitis, but that even the same microbe may produce different forms according to its mode of implantation in the skin. That the microbic element generally requires a suitable soil for its successful implantation and propagation. That this soil varies with the age of the individual and the kind of skin he possesses of which the modifications may be congenital or acquired, certain of the tissue proclivities being probably hereditary. That intestinal and probably other visceral toxins and ptomaines play an important and often unsuspected part in producing many forms of eruption and even that many supposed gouty eruptions are really of toxic origin from the generally present intestinal catarrh. That many of these toxins act through the vaso-motor nerves, central or peripheral, rather than directly on the skin, though they may act directly on that also. That the cerebral nervous system acts chiefly as a controlling influence over the sympathetic system as regards the intensity of the eruption. That with very few exceptions the nervous system, whether vaso-motor or cerebral, exercises but little influence on the character of the eruption though it does on its distribution and intensity. That the character of the eruption is mainly due to individual peculiarities or proclivities of which we can often only chronicle the result without being able to explain it. That while with apparently the same etiology different eruptions may ensue in different individuals, in recurring eruptions in the same individual there is remarkable constancy in the characters of the eruption and in its time, place, and mode of development. That many general eruptions are for a long or short time of local origin, occupying only a small area before generalisation. That other serious affections start from apparently trivial causes, such as seborrhœa, a superficial pustule, &c., and that it is important, therefore, to treat affections of the skin in as early a stage as possible, as most inflammatory eruptions have a much greater tendency to further development than they have to spontaneous involution. That the principles of treatment depend on the due appreciation of the relative importance in any one case of the microbic, the personal, the nervous system, and the toxic elements; and that so far as our knowledge extends the general principles of medicine apply to them, but that inasmuch as our power of estimation is often at fault we have to fall back on certain so-called specifics which experience has shown to be of service in certain conditions. That the most reliable and comprehensive specifics are arsenic, salicin, thyroid extract, quinine in large doses, and iodide of potassium; the first two have a very wide range as compared to any others. That except as regards pustular eruptions and those demonstrably of micro-parasitic origin the character of the local treatment depends comparatively little on the diagnosis of the particular kind of dermatitis, the extent, intensity, and localisation of the inflammation being the most important elements. That in employing microbicide treatment in superficial and widespread eruptions the microbicide should not be irritating, or should at least be capable of being at once neutralised, otherwise the increased inflammation set up defeats the aim of the therapist and affords a favourable soil for further microbic development. It is in comparatively few circumscribed microbic diseases, such as impetigo contagiosa, boils, and carbuncles, that the microbicide is the sole curative agent.

That in all wide-spread forms of dermatitis rest and equability of temperature are the most important and often the essential curative means. Finally, that in proportion as we study diseases of the skin in the same manner as we study diseases of other organs, we shall find that their treatment can be carried out successfully on the sound basis of pathology and that specifics will occupy a diminishing space in our armamentarium.

The statistics which I gave you in my first lecture show that the task of acquiring the power of diagnosis such as every practitioner should possess is not difficult in the majority of instances, as three-fourths of all cases of dermatitis are comprised under a very small number of diseases, but a considerable practical experience is necessary to grapple successfully with the variations which even these few common diseases present according to the several conditions we have already discussed; but attention to these points will gradually make this easier and will bring success which will add interest to the further study of a class of diseases which is a sealed book to many otherwise well-informed medical men, because they have not started on their investigation in a systematic manner.

I trust that this brief *résumé* that I have given of a wide subject will be of some assistance to a serious study of the inflammations of the skin which will well repay the time spent upon it both by yourselves and him whom I must now call "your late Lettsomian lecturer."

## A CASE OF ACUTE SPLENIC ANÆMIA TERMINATING FATALLY WITH GENERAL BACTERIAL INFECTION.

BY HERBERT P. HAWKINS, M.D. OXON., F.R.C.P. LOND.,  
PHYSICIAN TO, AND LECTURER ON MEDICINE AT, ST. THOMAS'S  
HOSPITAL;

AND

C. G. SELIGMANN, M.B., M.R.C.P. LOND.,  
SUPERINTENDENT OF THE CLINICAL LABORATORY OF ST. THOMAS'S  
HOSPITAL.

AN Italian *chef*, aged 37 years, was admitted into St. Thomas's Hospital on Feb. 7th, 1902, where he died two months later. His previous history threw no light on the nature of his illness. He had had no illness except slight indigestion occasionally, had always been temperate, but had smoked about an ounce of cigarettes a day. There was no history of syphilis or malaria or rheumatism, and he had not been in Italy for 26 years. He presented an odd appearance, for at the age of four years he was run over and his lower jaw was so damaged that some of the bone on the left side necrosed and was removed and at the age of 14 years he lost the sight of the left eye by an accident. His statement was to the effect that in January, 1902, having been previously in good health, he was admitted into the Italian Hospital suffering from pneumonia. The anæmia probably dates from this time, for on his recovery from the pneumonia he was sent to a convalescent home with the advice to return to the hospital later to obtain treatment for the blood state. After a few days at the convalescent home he began to experience difficulty in swallowing solid food, apparently through a dryness of the mouth, and becoming discontented on other grounds he left the home and was admitted into St. Thomas's Hospital.

On admission his chief complaint was a distressing feeling of weakness. But he stated also that he had difficulty in swallowing solid food, had to roll it about in his mouth before he could get it moist enough to swallow, and was frequently aroused from sleep by a choking dryness of his throat. There was some scarring over the lower jaw at the site of the injury which he had received in his infancy and the molars and the bicuspid had gone. But the rest of the teeth were in good order, there was no apparent lack of saliva, and there was no apparent change in the buccal or pharyngeal mucous membrane; and, as a matter of fact, he never showed any difficulty in swallowing after admission, but gave play to a very considerable appetite. He was rather thin and pale, with a slight yellowish tint of the skin and conjunctivæ. The

spleen was considerably enlarged (post mortem it weighed 25 ounces), but its borders were always difficult to determine, owing to general fulness and resistance of the abdomen. The liver extended for two inches below the costal margin, as determined by percussion, but no clear signs could be felt. The heart was healthy but for a systolic murmur in the pulmonary area; the pulse was 102, small, and of low tension, and the temperature was 101° F. The optic fundi were pale but healthy, the pupils were equal and active, and the knee-jerks and plantar reflexes were normal. The shins were slightly cedematous. The urine was strongly acid, brownish-yellow, clear, with a specific gravity of 1018, and contained no albumin or sugar. With the spectroscope it showed the absorption band of urobilin at F.

During the first fortnight of his stay in hospital he improved considerably under the use of iron and arsenic. His temperature became subnormal, his appearance improved, the appetite was good and at times voracious, and he expressed himself as feeling quite well. He gained three pounds in weight. During the second fortnight, however, his temperature began to rise, reaching 103° on two occasions, and coincident with this pyrexia there was profuse diarrhoea, there being on one day nine watery and rather offensive stools. There was no blood or anything to indicate any necrotic process in the bowel and the diarrhoea was, in fact, attributed to the irritant effect of the arsenic. The temperature fell to a subnormal point rather suddenly at the end of this period and during the third fortnight it remained

preserved the characters already mentioned and contained no bile. Bile was always present in the stools. During the whole course of the illness there was no alteration in the physical signs as regards the spleen and liver, but he complained occasionally of pain over the former. There were no ascites, no necrotic process in the mouth, and no cutaneous or visceral hæmorrhage.

The state of the blood can be gathered from Chart 1. It showed the type of secondary anæmia, the colour index being below par (from 0·6 to 0·9) except on one occasion (Feb. 24th), when it was 1·3. There were good rouleaux-formation and little, if any, variation in the size or shape of the red cells, though on one observation (Feb. 24th) a few showed polychromatophilic degeneration. No nucleated red cells were ever seen. There was no leucocytosis at any time and the normal relative proportions of the different kinds of leucocyte were roughly preserved, polymorphonuclear neutrophils varying from 62 to 80 per cent., lymphocytes from 18 to 35 per cent., and eosinophiles from 0·6 to 2·0 per cent. A percentage of 0·6 of mast cells was noted on two occasions. Blood platelets were unusually abundant in one specimen. On three examinations marked leucopenia was found, the counts being 2200, 3000, and 4000 respectively.

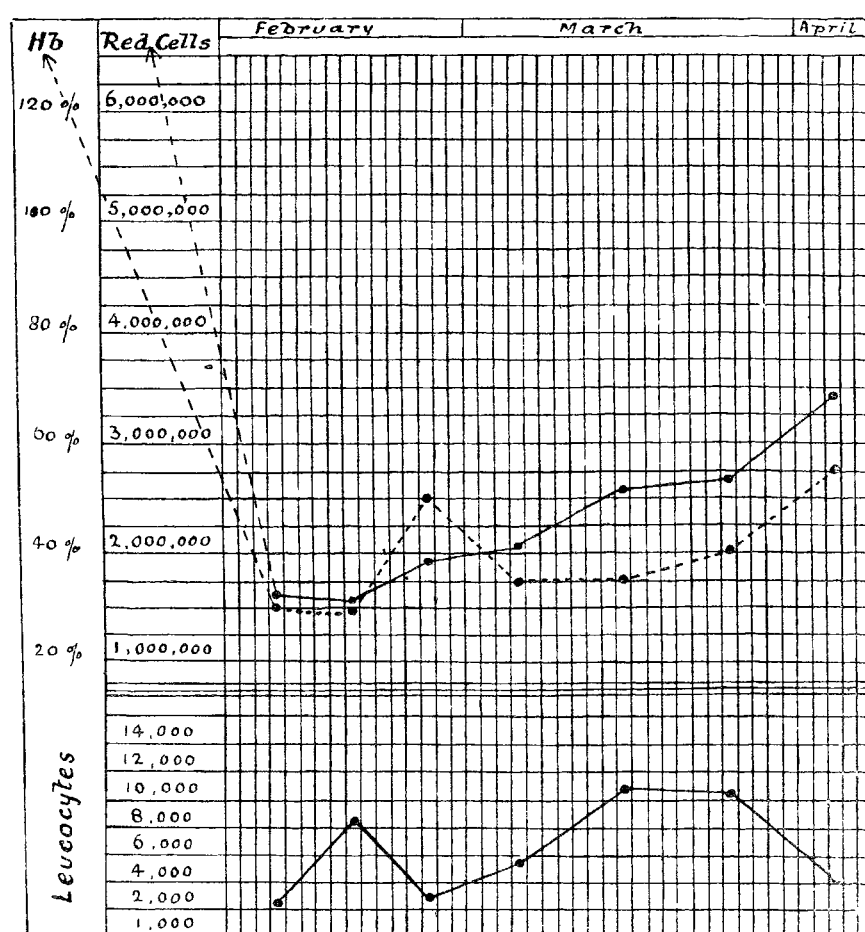
*Morbid anatomy.*—The liver weighed 67 ounces, the patient's weight at death being 102 pounds. It was large, soft, and yellow. To the naked eye there was no appearance of cirrhosis. It was thickly studded throughout with dead-white specks, the largest having the size of a pin's head, and these could be seen to be surrounded by a zone of hyperæmia. There was no iron reaction. Microscopical examination showed no trace of cirrhosis or leucocytal infiltration. The whole organ was degenerate, the cells being cloudy and largely pigmented and the nuclei staining badly or remaining invisible. The white specks consisted of necrosed liver cells, numerous irregularly-shaped nuclei showing up among the granular structureless debris, and they were teeming with bacteria, mostly rods resembling the bacillus coli.

The spleen weighed 25 ounces. It was large and rather soft. The capsule was slightly thickened. Section revealed a large white infarct and the cut surface showed numerous white specks similar to those in the liver. Microscopically there were seen to be a slight fibrosis affecting the trabeculæ and sheaths of the vessels and considerable evidence of endothelial proliferation. The white specks proved to be patches of focal necrosis similar to those in the liver. There was no obvious change in the Malpighian bodies. The kidneys were large, soft, and yellow, the cells being cloudy or very granular. The pancreas showed no change and there was no apparent alteration in any lymphatic glands. The marrow was not examined.

The heart was pale, with large subpericardial hæmorrhagic blotches and numerous petechiæ in its muscle. There was an early stage of pericarditis, and both mitral and aortic valves showed distinct vegetations of recent origin. The lungs showed some broncho-pneumonia in the lower lobes, and here also, as in the heart, the serous surface showed early inflammation with deposit of lymph. The stomach and small intestine showed no change and there was no sign of ankylostoma or other worm. The cæcum and adjoining portion

of the colon for a total length of eight inches presented the very peculiar appearance seen in the illustration. The normal folds were greatly swollen in height and in breadth, and being separated only by slight sulci they bore some resemblance to the convolutions of the brain. The thickening is shown by the fact that in places the distance from the peritoneal coat to the summit of a fold in a section through the bowel wall amounted to more than half an inch. The tissue was exceedingly firm, so that felt from the outside it suggested a new growth. As seen from the inside the whole patch was dark-grey or black. In the transverse and descending colon were two patches of croupous inflammation, each being covered by a dirty-white

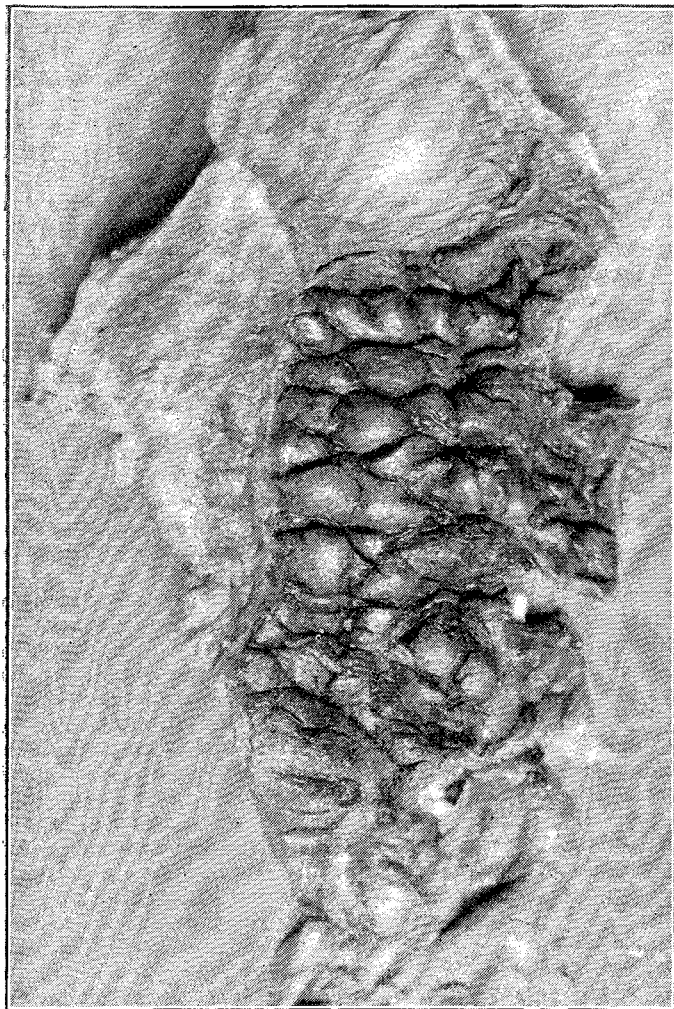
CHART 1



subnormal with nearly normal stools, a good appetite, and considerable general improvement. During the fourth fortnight the pyrexial attack was repeated. The temperature rose slowly at first, but soon reached 104° in the evenings and remained at about that level until death. At the same time the bowels again became very loose. There was a tendency to sweat at night. The nose bled occasionally, the gums began to ooze, and the teeth became foul and clogged with blood. He took very little food. The mind became obscured. The heart failed rapidly and rhonchi were heard over both lungs. During the last four or five days of life he became more and more yellow, eventually reaching the colour commonly met with in catarrhal jaundice. The urine

membrane consisting of necrosed mucosa and coagulated exudation.

Microscopical examination of the affected part of the cæcum showed that the changes comprised hæmorrhage, inflammatory exudation, necrosis, and bacterial invasion. To demonstrate the early stage of the lesion an outlying flat-topped sessile projection of the mucous membrane may be taken. From such a spot the whole of the epithelium has been shed, including the cells lining the glands, though traces of the latter remain. In the underlying submucosa there is some exudation of small round cells and the remains of the mucosa are in a condition of early necrosis. Immediately under the muscularis mucosæ, which is itself altered by the necrotic process to such an extent as not to be recognisable in the central region of the elevation, hæmorrhage has taken place into the submucosa. The muscular



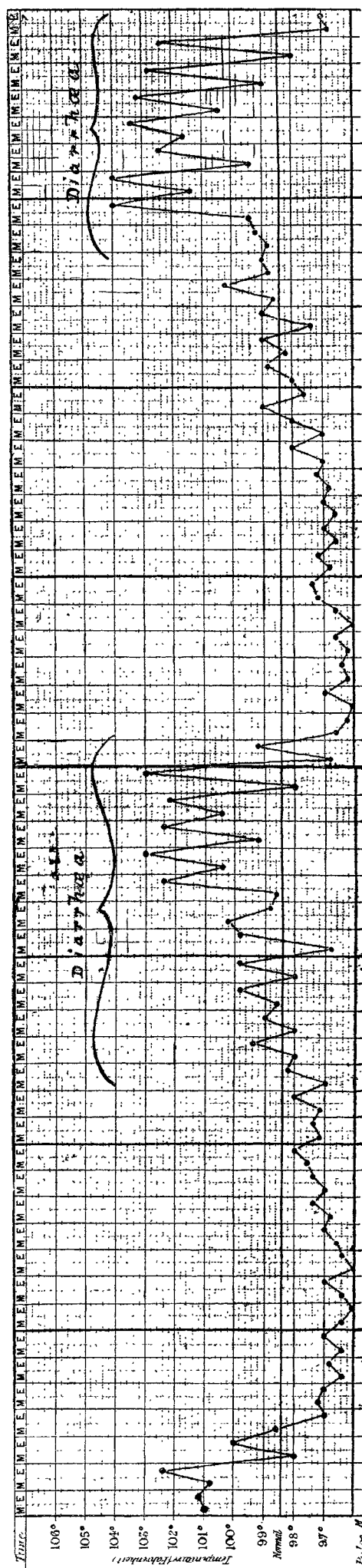
Cæcum in acute splenic anæmia.

tissue, though swollen, is comparatively little affected. One section, which contains the edge of a lymphatic follicle, shows that the follicle has not participated in the change, no necrosis and no infiltration with polymorphonuclear cells having occurred.

In a section through the most advanced part of the disease it is seen that the mucosa is swollen and necrotic over large areas, the submucosa is distended with inflammatory exudate now undergoing necrotic change, while hæmorrhages and distended vessels are present in the subjacent parts of the inner circular layer of muscle. Throughout the thickness of the necrotic mucosa, but most abundant in its deeper part, are numerous circular or oval areas of finely granular material, which appear to be the remains of the fundi of glands crammed with disorganised or dead epithelial lining. Bacilli and cocci are abundant in the necrotic mucosa and they extend also into the deeper layers, though here they seem for the most part to be limited to vessels, definite connective-tissue spaces, or lymphatics. The masses of hæmorrhage and exudate in the deeper submucosa and inner muscular layer do not seem to be infected.

*Remarks.*—A splenic anæmia—i.e., an anæmia of secondary type associated with enlargement of the spleen—stands out

CHART 2.



as a distinct symptom-group. It is as definite as pernicious anæmia and leukæmia, and though less common than these it is probably far more common than the scarcity of recorded instances would suggest. It consists of a primary splenomegaly, with more or less profound anæmia of a secondary type, without the characteristic blood changes of pernicious anæmia, without any material alteration, quantitative or qualitative, of the leucocytes (though leucopenia is generally noted), without enlargement of glands, generally showing pyrexia at some period, and in a late stage associated with some fibrosis both of the spleen and of the liver. In many cases coming under this definition the duration is measured in years, and it is to these cases that Osler<sup>1</sup> has particularly directed attention. A duration of 15 years has been noted. It is in such long-standing cases that hæmatemesis and ascites are apt to occur, leading perhaps to an erroneous diagnosis of cirrhosis of the liver. In a smaller number of cases, however, death occurs in from three to twelve months. Thus two forms—an acute and a chronic—may be recognised. It is possible that when the actual cause is discovered these two forms will be found to be distinct. But the symptoms and state in the two forms are identical, except in the occurrence of repeated hæmatemesis in the chronic form. And at present there seems to be little reason for arbitrarily separating them and for considering them as different diseases merely on the score of a difference in duration.

In the example of the acute form here recorded, the size of the spleen and the character of the blood exclude a diagnosis of pernicious anæmia. The blood state points rather to defective formation than to increased hæmolysis. At the same time the latter process is indicated, in spite of the absence of iron reaction in the liver, by the character of the urine and the yellow tint of the skin. The chief points of interest are (1) the apparent onset of the disease after pneumonia; (2) the curious periodicity of the fever as shown in the temperature chart (Chart 2), which might almost be taken as a copy of the chart given by West<sup>2</sup>; (3) the steady improvement under treatment as shown in the blood chart; and (4) the cutting short of this improvement by the supervention of a general infective state, as shown by necrosis of the bowel wall, focal necrosis of the liver and spleen, pericarditis, pleurisy, and acute endocarditis.

As regards the nature of splenic anæmia a string of facts points strongly to the localisation of the cause in the spleen. 1. In the first place it is highly probable that the enlargement of the spleen is practically the first sign of disease. In the case of a man, aged 64 years, recently in St. Thomas's Hospital, who had never had any illness except influenza and had led a perfectly healthy life, the onset of an acute splenic anæmia was very clearly described. In December, 1900, he began to notice a little unusual tiredness after his day's work and some disinclination for exertion. But he was little incommoded by this and did not think it worth while to consult a medical man until the end of January, 1901—i.e., not more than two months later. The spleen was then found to be already greatly enlarged, the lower end being at the level of the umbilicus. As regards the state of his blood (anæmia of secondary type) and his other symptoms he departed in no important particular from the case above described and he died six months after the beginning of symptoms with a spleen weighing 30 ounces. When he went into hospital he was very ill and the time for splenectomy had gone by. 2. In the second place it is certain that removal of the spleen will effect a cure and that it is the right course to pursue if it is estimated that the patient can survive the operation itself. Sippy<sup>3</sup> and Harris and Herzog<sup>4</sup> give a list of 19 cases of primary splenomegaly in which splenectomy was performed. Of these 14 patients recovered and four died; of one the result is not stated. 3. Further there is evidence to show that in long-standing cases at any rate the changes in the spleen have a somewhat distinctive character in the marked endothelial proliferation.

It is possible that this endothelial change is itself responsible for the blood state, as has been suggested, by the production of a hæmolysing enzyme, wholly abnormal or an exaggeration of a normal product. But such a theory, however plausible, is incomplete, as it still leaves us without

a cause for endothelial change and the tendency must be to fall back on the hypothesis of the existence in the spleen of a long-lived bacterial or parasitic agent. At any rate, on any view it is difficult to get away from the idea that the actual cause of the disease resides in the spleen. The established disease bears a close resemblance to the state of chronic malarial cachexia with permanent enlargement of the spleen.

As regards the case here described, the question presents itself as to whether the infective termination can be allowed to have any bearing on the causation of the disease. On finding the very remarkable condition in the cæcum above described one was tempted at first from the hardness and blackness of the part and the presumable chronicity of the change to imagine the possibility that this might prove to be a primary lesion, from which the blood state had arisen. But the microscopical examination shows that it was not of long standing and it must be interpreted as a recent necrosis of the bowel wall, consequent on, or followed by, hæmorrhage into its tissue, with secondary bacterial invasion. Though it is a much rarer change, it must be regarded as being essentially of the same nature as the patches of croupous inflammation which occurred in another part of the colon, both of them meaning loss of tissue vitality and secondary bacterial infection. Neither change can be allowed to have direct bearing on the question of the bacterial or non-bacterial origin of splenic anæmia. At the same time it may be noted that a terminal croupous inflammation of the bowel, though it does occur in non-bacterial diseases such as cirrhosis and renal disease, is far more common in bacterial conditions such as pyæmia, septicæmia, and pneumonia.

As regards the state of general infection which was the immediate cause of death, its bearing on the question of the bacterial origin of splenic anæmia is vitiated by the occurrence of the intestinal necrosis. The whole series of changes, the focal necrosis of the liver and spleen, the pleurisy and pericarditis, and the acute endocarditis, may well have been the result of the intestinal infection. But their occurrence seemed to be worthy of record in connexion with a disease of which so little is known. As to the focal necrosis of the liver and spleen we know nothing of such a state except as arising from the action of bacterial toxins. In man it is confined to typhoid fever and dysentery. In the former disease an instance is described and depicted by Glynn.<sup>5</sup> The finding of recent vegetations on the valves after death from any acute febrile disease is a strong indication that that disease has been of an infective nature. Examples may be drawn from scarlet fever and, perhaps, other specific fevers, from pyæmia, erysipelas, and septic diseases generally, and from pneumonia. Considering the presence of the intestinal lesion, however, there seems to be no justification for laying stress on the endocarditis in this case as having a bearing on the origin of splenic anæmia. But, at the same time, it may be noted that a similar acute terminal endocarditis has been recorded in three cases of splenic anæmia by West,<sup>6</sup> Williamson, and Strümpell, and out of six cases of febrile (infectious) purpura occurring under the care of one of us similar early vegetations were found in three cases on the valves of the heart.

## THE HEIGHT OF THE DIAPHRAGM IN RELATION TO THE POSITION OF CERTAIN ABDOMINAL VISCERA.

BY CHARLES T. ANDREW, M.B., CH.B., B.Sc. ABERD.,  
JUNIOR DEMONSTRATOR OF ANATOMY, ABERDEEN UNIVERSITY.

WITHIN the last few years attention has often been drawn to abnormalities in the shape and position of certain of the abdominal viscera, especially with reference to the part these play in the causation of disease. Some short time ago a pamphlet was published by Dr. Arthur Keith on the "Anatomy of Glénard's Disease," and lately his Hunterian Lectures on this subject,<sup>1</sup> in which he laid stress

<sup>1</sup> American Journal of the Medical Sciences, January, 1900, and November, 1902.

<sup>2</sup> Allbutt's System of Medicine, vol. v., p. 544.

<sup>3</sup> American Journal of the Medical Sciences, vol. xcvi., 1899.

<sup>4</sup> Annals of Surgery, July, 1901.

<sup>5</sup> Thompson-Yates Laboratory Report, vol. iv., part 2, 1902.

<sup>6</sup> Loc. cit.

<sup>1</sup> THE LANCET, March 7th (p. 631) and 14th (p. 709), 1903.