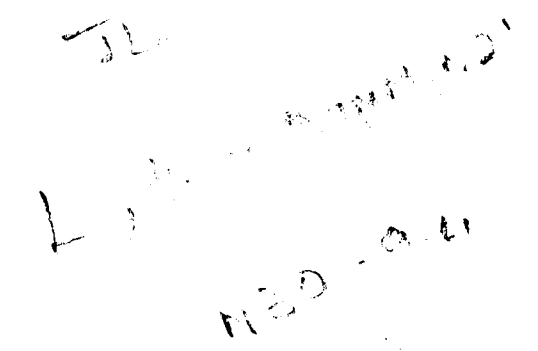


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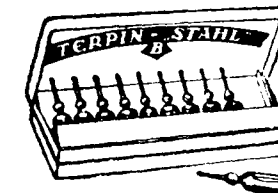
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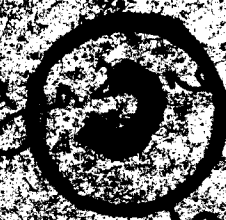
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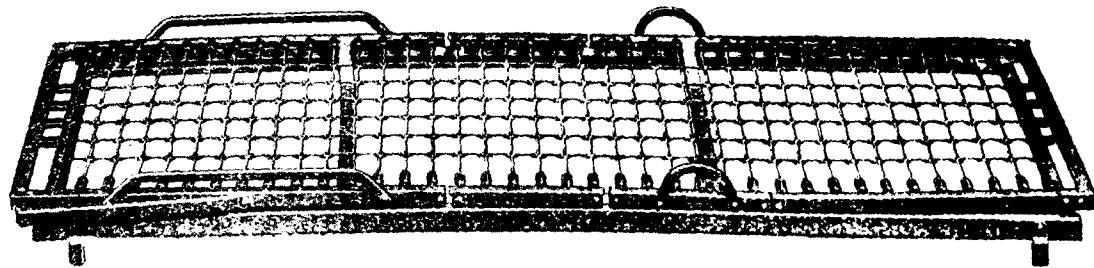
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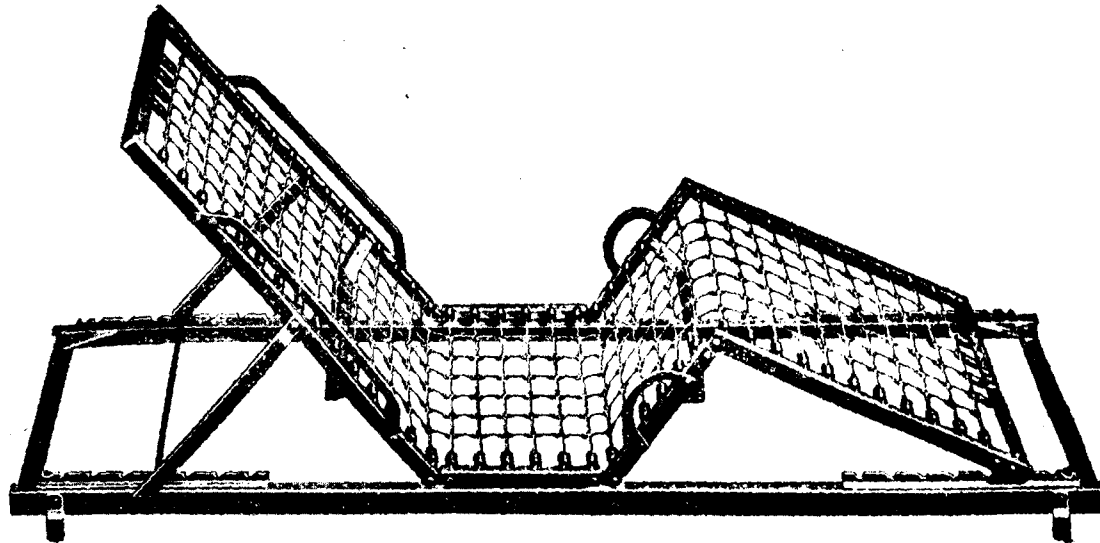
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EDITOR

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April, 1930.

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OLD MEDICOS—WHY DO THEY DIE.

Death is here and death is there
Death is busy every where
Death has set its marks and seal
On old Medicos—Pathetic O I feel.

Will it be that the Gods above
Have a "nursing-home" all fresh and new
And desire hands from down below
For are'nt we clever and a lot we know?

Sebastian—a house-surgeon he was
Up he went and followed alas
Fine chaps Krishnamoorty
Nathaniel and Vishwanathan Chandrasekaran.

Dress and case-sheets students will do to be sure
And a surgeon is there to heal and cure
I forgot on doubtful points of Anatomy
Doctor Swaminathan will be quite ready.

O Gods—art thou now satisfied
And the staff thou needed completed
Or order more of us?—Heavens forbid!
Thou hadst better apply in columns "wanted."

How else then could we account
For deaths of medicos out of count
Students die and doctors die
But the hostel—it never is born why?

B. SANJIVA RAO,
Third year.

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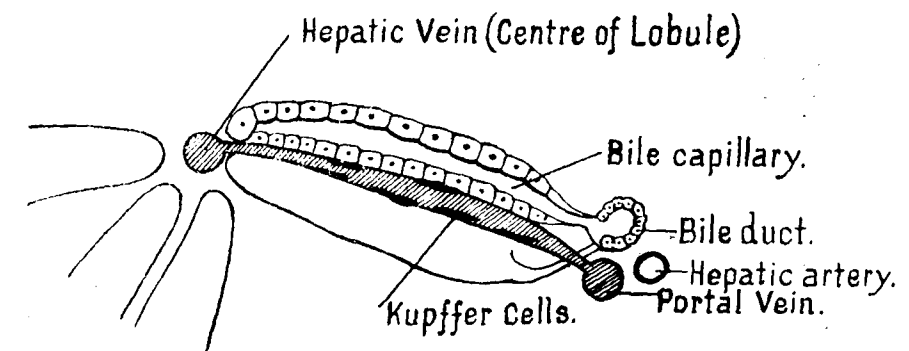
APRIL 1930

No. 4

The Liver with Special Reference to Cirrhosis*

BY LIEUT.-COL. G. E. MALCOMSON, M.D.,
M.R.C.P., I.M.S.

Although the liver functions are numerous, important and complicated, the physiological structure according to the concept of McNee is comparatively simple. This author describes the lobule as consisting a number of tubular glands called bile capillaries with the closed end at the centre radiating from the central hepatic artery and discharging by their open ends at the periphery into the smaller bile ducts. Between these bile capillaries the vascular capillaries course between the portal vein at the periphery and the hepatic vein at the centre. These



* A readership lecture delivered under the auspices of the University of Madras at the Medical College on 30th January 1930.

vascular capillaries are incompletely lined by star-shaped endothelial cells called the Kupffer cells. This is shown diagrammatically thus:—

The functions of the liver are manifold and we have but a very partial knowledge of them in any direction; this organ probably plays a part in every metabolic process known to us.

From its position in the portal circulation it forms a barrier or filter between the alimentary canal and the general circulation and it is to the partial removal of this barrier that no doubt some of the symptoms exhibited in cirrhosis are due. Most of the substances carried to the liver by the portal vein are stored or modified in some way in order to render them non-toxic, or of use in the body economy, or in a suitable form for excretion by the kidneys.

Those stored fall into two groups:—

1. Glucose or other monosaccharids stored as Glycogen for further use.
2. Poisonous substances like alkaloids or the heavy metals, in order to shield the other tissues of the body.

The known functions of the liver may be classified thus:—

1. The hæmopoietic function.
2. The blood destroying function.
3. The biliary function.
4. The glycogenic function.
5. Function as regards protein metabolism.
6. Function as regards fat metabolism.
7. Function as regards iron metabolism.
8. The protective function.
9. Functions in relation to blood coagulation.

I will now say a few words about these seriatim:—

1. *The blood forming function.* 2. *The blood destroying function.*—In these two functions which may be considered

together the liver may be regarded as forming part of a great system concerned with blood breakdown and repair, comprising the spleen, lymph glands and bone marrow. In other words the liver contains a large part of the reticulo-endothelial system of the body.

The hæmopoietic function of the liver only exists during foetal life pending the development of the bone marrow; during this period both red and white cells are manufactured. This is important to remember because in certain diseases of the bone marrow a revision of this function in the liver may take place, in later life. In the process of blood destruction the Kupffer cells of the liver share with the rest of the reticulo-epithelial system the function of converting the hæmoglobin of the broken up red corpuscles into the iron-free pigment bilirubin.

3. *The biliary function.*—The important constituents of bile are three in number—

- (1) Bile pigments.
- (2) Bile salts.
- (3) Cholesterol.

Bile pigments as stated above are formed by the reticulo-endothelial system from the debris of the red blood cells, but this process does not end here. Further changes are undergone by bilirubin after passage through the hepatic cell into the bile capillary; these changes are shown by Van Den Bergh to be manifested by an immediate direct reaction with the diazo reagents, whereas bile which has not passed through the liver cells only gives a delayed direct reaction or possibly only after extraction from the serum with alcohol—*i.e.*, an indirect reaction.

A glance at McNee's liver diagram will show that Jaundice may occur in three ways:—

- (1) Obstruction to the bile duct will cause damming of the bile back in the bile capillary, and re-absorption into the blood stream, so called Obstructive Jaundice.

- (2) Injury to the Hepatic cell, which is thus rendered incapable of passing the bilirubin and other bile constituents through to the bile capillary, so called, Toxic or Infective Jaundice, according to the nature of the injury.
- (3) Excess of blood destruction and therefore of bilirubin may occur, so that the liver cells are unable to deal with the latter and some of it remains in the blood; so called, Hæmolytic Jaundice.

The Van Den Bergh reaction is of use in two directions chiefly:—

1. The detection of latent as opposed to overt jaundice.
2. The discrimination between obstructive and the other forms of jaundice.

As regards the bile salts, nothing definite is known as to their origin. The similarity between the constitution of Cholin and Cholesterol strongly suggests the latter as the precursor but of this there is no direct evidence. Normally the bile salts, after having played a subsidiary part in fat digestion are re-absorbed from the alimentary canal and re-excreted by the liver. On the other hand in cases of biliary fistula where bile salts are lost to the body, there is no diminution of the secretion. There must therefore be a controlling mechanism because if all the bile salts were continually being absorbed and re-excreted and their formation *de novo* kept on at the same time the whole body would ultimately as Whipple remarks, be converted into a pillar of bile salt. In obstruction and toxic, but not in hæmolytic jaundice bile salts are found in the blood and urine. Their presence in the blood is generally assumed to be the cause of bradycardia and less generally is the itching of the skin attributed to the same cause. They are only present in the urine during the early stages of jaundice and then disappear. They are present

however in larger amounts in obstructive than in toxic jaundice.

Hypercholesterolemia also occurs in jaundice under the same conditions as cause bile-salts to appear in the blood. The part played by cholesterol in the bile and the results of its reabsorption into the blood stream are still wrapt in obscurity.

4. *The Glycogenic Function of the Liver.*—It is generally assumed that the cleavage products of Carbohydrates, namely the monosaccharids are taken up by the liver, polymerised to, and stored as glycogen, which is again broken down into glucose by the hepatic amylase. This glucose is passed into the general circulation as required to keep up the percentage of circulating sugar. It would perhaps be more accurate to say that glycogen is an internal secretion of the liver, as Claude Bernard first suggested, because in the absence of available carbohydrates, glycogen is formed by the liver from proteins and possibly from fats.

5. *The Hepatic function in protein metabolism.*—The view is still held by the majority of physiologists that the liver is capable of splitting the amino-acids received from the intestine into a nitrogenous and non-nitrogenous moiety. The nitrogenous moiety is largely converted into ammonium carbonate, which in turn is converted into urea in the liver. The non-nitrogenous moiety can be utilized by the liver for the production of glycogen.

In addition to amino-acids the liver has a function in purin metabolism the details of which are not well understood. Suffice it to say for the present, that the human liver is not effective in oxidising uric acid, as are the livers of lower animals, hence the tendency to gout in the human subject.

6. *The Hepatic functions with regard to Fat Metabolism.*—These are even more obscure than the foregoing, but the liver probably exercises an important function in

converting saturated fatty acids into non-saturated, the latter being more easily oxidised.

7. The functions of the liver with regard to iron metabolism are important and form a very interesting chapter. The liver deals with a large portion of the non-fixed iron in the body whether this be obtained from normal phagocytosis of red cells, or abnormal lysis, or from iron absorbed from the alimentary canal. Muir and Dunn have shown that in acute hæmolytic anæmia $\frac{1}{3}$ of the total iron in the body is contained in the liver, spleen and kidney. Hæmosiderin is found in the liver, in every case of excessive blood destruction in the body, in hæmolytic jaundice, in malaria, in extensive hæmorrhage and in other forms of severe anæmia such as Hodgkin's disease or leukæmia. The same authors show that the iron derived from hæmolysis occupies a different position in the liver from that derived from normal blood destruction, the former being found in the hepatic cells, the latter only in the Kupffer cells.

That hæmosiderin is utilized in blood regeneration is indicated by the remarkable change in the blood picture in pernicious anæmia which may occur from time to time either in the normal remissions of that disease or under treatment with arsenic or liver extract. In the normal process of blood destruction in the body, the liver, that is the reticulo-endothelial system, conserves the iron derived from the hæmoglobin and hands it on to the bone-marrow for disposal. According to the view of Rossle amplified by Eppinger, the disease known as hæmochromatosis is produced by damage to, or defective activity of the reticulo-endothelial system whereby the normal iron metabolism of the body is thrown into disorder.

8. *The protective function of the liver.*—The function of the liver in protecting the body from exogenous poisons is well known. In most cases of rapidly fatal poisoning the bulk of the poisonous substance is found in the liver after

death. Should the amount of poison be excessive the liver tissue may be damaged and toxic jaundice or acute yellow atrophy result as happens frequently in arsenic and atophan poisoning. This untoward result may be to a large extent prevented by reinforcing the liver with glucose and therefore glycogen before administration of any of these dangerous substances, thus calling into play the so-called glucuronic acid synthesis, the available glucose being able to enter into combination with the poisons rendering them harmless. The knowledge of this mechanism has to a large extent done away with the dangers of poisoning from therapeutic administration of these substances.

The protection afforded by the liver against bacterial infection is well known and is exemplified by the removal from the blood and excretion in the bile of typhoid bacilli and other organisms; whether similar protection is afforded against bacterial toxin is uncertain and the experiments of Roger who perfused the liver with diphtheria toxin finding the latter in no way diminished in activity give much cause for thought.

Regarding the action of the liver towards products of intestinal decomposition, little is known. Intestinal toxæmia has been blamed for nearly every ailment to which flesh is heir, and in those cases of cirrhosis where no other cause can be found the process may be tentatively regarded as a reaction of the liver to a prolonged bombardment of this nature.

9. *Function of the liver in regard to blood coagulation.*—That the liver has an important influence on the coagulation of the blood is evinced by the tendency to hæmorrhage in long-standing jaundice, by the delayed coagulation of the blood under the same condition, and by the experimental results of extirpation of the liver causing great delay in, or total inhibition of the formation of blood clot.

The mechanism of this phenomena is not understood and it has been variously suggested that there is a low fibrinogen content in jaundice, and that bile salts over a certain concentration hinder coagulation.

On the other hand it seems equally clear that the liver secretes an anti-coagulant. This substance has been named "heparin" by Howell and the rareness of thrombosis in the hepatic as compared with the portal vein has been attributed to its presence.

Having now briefly mentioned the chief hepatic functions I will proceed to consider some of the more important diseases of the liver.

Now like the kindred subjects of arterial and renal diseases the classification of liver diseases is confusing, due largely perhaps to our want of knowledge of the etiology of these conditions.

I will first give in a slightly modified form Rolleston's and McNee's classification of Hepatitis under which term they include all results of damage to the liver, chemical, physical or microbic, and the various processes of reaction and repair which follow the injury; in this way the close affinity will be shown between various well-known hepatic diseases, whose separation is rather a matter of degree than of essential difference.

Thus

Hepatitis is divided into—

1. Acute Hepatitis—Acute damage of every degree
Catarrhal jaundice.
Atophan, chloroform, &c. poisoning.
Acute yellow atrophy.
2. Subacute Hepatitis—damage prolonged
Subacute yellow (or red atrophy) ending in
nodular Hepatitis (Toxic cirrhosis)
3. Chronic Hepatitis (or cirrhosis)
4. Suppurative Hepatitis

- | | |
|---------------------|--|
| (a) Via portal vein | { Amœbic hepatitis and
abscess. Suppurative
pylephlebitis. |
| (b) Via bile ducts | |
| | { Suppurative cholangiec-
tasis. |

Now I want you to recognize the essential similarity between the various forms of Acute Hepatitis in this table, the difference being one of degree only. I do not mean to say that all cases of Catarrhal Jaundice are identical in etiology, that although it was formerly believed that this condition was due to obstruction of the duodenal orifice of the bile duct, it is now understood that most cases are due to an infection involving both the bile duct and the liver cells, that is, both a cholangitis and a hepatitis. The Van den Bergh reaction in the later stages of the disease at any rate is indicative of the toxic-infective group. In my ward clinics I frequently refer to the scrap heap of Catarrhal Jaundice meaning thereby that the diagnosis is tentative only, that it includes diverse conditions, that although the majority are mild some may take on a more acute condition and develop into a clinical picture of the severer forms or even of acute yellow atrophy. Catarrhal Jaundice may then be an ascending infection, from a gastro enteritis; it may be a manifestation of a more specific infection like, malaria, Weil's disease, Dengue, Relapsing fever, Yellow fever, Pneumonia, Enteric, Bacillary Dysentery, Secondary Syphilis; it may be a manifestation of an exogenous poison like phosphorus or chloroform or one of an endogenous nature as occurs in pregnancy or in the recurrent vomiting of children. Any of the above may develop into the serious condition culminating in acute yellow atrophy.

And now a few words about the second heading, namely, Subacute Hepatitis. This results from a more mild poison acting over a more prolonged period or perhaps it would be more accurate to say that the acute stage has advanced to a certain degree of necrosis not sufficient to cause death,

and repair has taken place. In acute yellow atrophy, necrosis is the dominating change, usually affecting the whole lobule, occasionally the centre, or as in eclampsia, the peripheral part may be attacked. The necrotic cells are removed by autolysis and phagocytosis and if the lobule is not completely destroyed the bile ducts which are more resistant proliferate to re-establish connection with the lobules. The surviving liver cells divide and the lobule is regenerated, such regeneration nodules forming island like structure surrounded by bands of fibrous tissue which have taken the place of the lost liver cells. The term insular cirrhosis is sometimes given to this. With the regeneration of the liver tissue and the linking up of the bile capillaries again jaundice disappears though the liver may exhibit many varying degrees of distortion according to the amount of destruction of hepatic tissue which has taken place. The patient may be enabled to carry on a normal existence for many years, and the condition only discovered post-mortem. Occasionally these cases may terminate with the clinical picture of portal cirrhosis, but more often as the result of an intercurrent infection.

Now, this case which I have here, illustrates, I think the disease known as Toxic Cirrhosis.

He is a young Hindu, 22 years of age, who gives a history of jaundice for the past 5 months.

There is nothing of importance in his family history; both his parents are alive and healthy.

He gives a history of enteric fever some five years ago which was followed by an attack of jaundice; after that his medical history appears to have been uneventful until the onset of the present trouble some 5 months ago when he became jaundiced with some indigestion and vomiting. The jaundice appears to have subsided after a few days to recur again after a month. At the same time his temperature rose in the evening to 101° or so. His urine was high coloured and his motion clay coloured and constipated.

This condition persisted until his admission into the General Hospital some 3 weeks ago and he noticed a tumour forming in his right hypochondrium.

On examination the patient was wasted, aëmic and jaundiced. The liver was tender and reached nearly to the umbilicus and the spleen was about 2" down. In the evening he showed a moderately raised temperature (101°).

Blood picture

Hb	...	60%
Red cells	...	2,900,000 per cm.
White cells	...	12,500 per cm.
Colour index	...	1.

Differential leucocyte count:

Polymorphs	...	78%
Lymphocytes	...	14
Large mononuclear	...	6.2
Eosinophiles	...	1.8

Van den Bergh Reaction

Direct Biphaseic	...	7.04 unit
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Wassermann Reaction...Negative

Coagulation time 4' 45"

Fragility of red cells

Hæmolysis between 0.35 and 0.3 % saline

Blood cholesterol 139 mgms per 100 c.c.

Lævulose tolerance test

Before administration	...	0.129
½ hour after	...	.147
1 hour after	...	.147
1½ hours after	...	.152
2 hours after	...	.134

Urine: Neutral

Sp. Gravity 1012

Albumin trace

Bilirubin present.

Culture: sterile

Inoculation experiments: No evidence of spirochætal infection.

Stools: Clay coloured

Culture: Streptococci grown

Duodenal contents: Sterile.

Since admission he has had 9 injections of sodium salicylate intravenously in addition to the *Mistura Hepatica* of our pharmacopœia.

His condition has improved considerably, the temperature has come down to normal and the liver has decreased somewhat in size. He feels generally better although he is still jaundiced and the urine still contains bile pigment.

Now if we may attempt to reconstruct what has happened in this case, we may imagine that some 5 years ago as a result of an enteric septicæmia there was an inflammation affecting the smaller bile ducts and the liver cells. This infection has persisted in a chronic or latent condition till a few months ago when owing to some temporary reduction of vitality or other cause, it again took on an acute form with destruction of liver cells followed by extensive hyperplasia of liver tissue. That the liver function is being re-established, probably by the joining up of the newly formed liver tissue with the bile capillaries is shown by the fact that the jaundice is gradually decreasing. That the inflammation is settling down is shown by the shrinkage of the liver and the temperature becoming normal. No doubt there is considerable formation of fibrous tissue and it is possible that the case will ultimately develop into the clinical picture of cirrhosis.

Next we have to deal with Rolleston and McNee's third form of Hepatitis, *viz.*, Chronic Hepatitis or Cirrhosis. I have here two tables, one, according to Chauffard, which was constructed as long ago as 1902, and indicates the methods by which Cirrhosis is induced. Rolleston and McNee state that the "Classification is too elaborate,

for the toxic and infective forms overlap and from a clinical standpoint some of these sub-divisions are too minute to be of practical value."

The following is Chauffard's classification:—

I. *Vascular* (a) Toxic—

(i) Poisons taken by the mouth.

(ii) Poisons manufactured in the intestinal tract.

(b) Infective—

(i) The direct action of micro-organisms.

(ii) The action of bacterial toxins, either produced by bacteria in the liver itself or manufactured elsewhere in the body and carried to the liver.

(c) *Dystrophic*

(i) Due to arteriosclerosis, this form of cirrhosis would be homologous to a granular arteriosclerotic kidney, the fibrosis being a replacement fibrosis.

(ii) Due to chronic venous engorgement, the fibrosis of a nutmeg liver.

II. *Biliary*. (a) Secondary to obstruction of the larger bile ducts.

(b) Primarily an inflammation of the smallest bile-ducts, leading to a unilobular cirrhosis—Hanot's hypertrophic cirrhosis with chronic jaundice.

III. *Capsular cirrhosis or perihepatitis*.

(a) Chronic localised

(b) Chronic universal.

The other table by Mallory indicates the relative frequency of the different varieties of cirrhosis from the point of view of etiology, which holds in western countries as determined from a large series of *post mortem* examinations.

Incidence and Classification of Cirrhosis (Mallory).

1. Alcoholic cirrhosis		45.6
2. Pigment cirrhosis		7.6
1 and 2 combined	...	20.0
3. Syphilitic cirrhosis	...	6.0
4. Infective cirrhosis		5.2
5. Toxic cirrhosis		4.0
6. Old healed cirrhosis, type not determined.		11.6

We will deal with Chauffard's classification first. The dystrophic variety due to arteriosclerosis or chronic venous engorgement must be of very infrequent occurrence and of but little clinical importance. Although arteriosclerosis may coincide with cirrhosis there is no direct relation between the two conditions. As regards chronic cardiac disease although a certain amount of fibrosis may occur in the nutmeg liver, it is surprising to find so many chronically congested livers over a very prolonged period without any demonstrable fibrotic change whatever.

Next in Chauffard's table we see that Biliary Cirrhosis may be obstructive or non-obstructive. Of the obstructive variety I have nothing more to say than that here we are dealing with a condition whose production is identical with suppurative cholangiectasis as shewn in the classification of hepatitis with the difference that here we are dealing with a slow or mild inflammation so that a fibrous tissue reaction has time to occur. The discussion of Hanot's Biliary Cirrhosis is made much easier by a note by Hurst in the 3rd edition of Price's "System of Medicine."

He says "A condition has been described under the name of Hanot's biliary cirrhosis, which is said to affect children and young adults, often several in a family, and to be characterised by enlargement of the liver and spleen, with chronic jaundice and febrile attacks, but rarely ascites. Though I gave the usual description of the disease in the earlier editions of this book, I have never seen a case which could be accurately described as biliary

cirrhosis, and I am exceedingly doubtful whether such a clinical entity exists. The term has probably been used to include cases of splenic anaemia with cirrhosis (Banti's disease) acholuric jaundice, or ordinary cirrhosis associated with infective cholangitis and very chronic cases of infective cholangitis. I do not, therefore propose to help to perpetuate what I believe to be a clinical myth."

I imagine many careful students of medicine will welcome the disappearance of this disease as a separate clinical entity.

This paper would not be complete without mentioning the infantile biliary cirrhosis which is common in this country especially in Calcutta. The illness starts at about 7 months of age and frequently attacks several children in one family. Early symptoms are fever with enlargement of liver and spleen followed by vomiting and jaundice which may be intense and finally ascitis may end the scene. The cause is unknown but certain etiological factors seem to be established. It is far more common amongst the well to do especially in Brahmin families. There is no relation to syphilis or malaria or (probably) Kala-Azar. It has been thought to be the result of a mother's milk altered in some way by a pampered life and a too rich diet. Forsyth and Frost in a research done some years ago in Madras demonstrated a connection between the disease and superfatted milk. The pathological change is unicellular cirrhosis becoming interlobular, and there is a great destruction of liver cells with much formation of new bile ducts.

And now we have cleared the way for a discussion of common varieties of cirrhosis as we see it in the wards and I would refer you again to Mallory's table. Infectious cirrhosis and toxic cirrhosis have been already dealt with under the heading of subacute hepatitis, which I have already stated may terminate with the clinical picture so familiar to us.

Syphilitic cirrhosis should not cause any difficulty. It occurs in the congenital form as an intercellular fibrosis. In the secondary stage of syphilis we may get an acute hepatitis which may terminate in any of the ways mentioned above. In late congenital and in tertiary acquired syphilis there is gummata formation and according to the size and number of gummata, the subsequent fibrosis will lead to a corresponding deformity, shrinkage and destruction of the organ.

Pigment cirrhosis is either malarial or due to hæmochromatosis. In malaria there are four possible factors:—

1. Necrotic changes (acute hepatitis)
2. Gastro-enteritis.
3. Irritation of the hæmosiderin, which is deposited in the peripheral cells of the lobules.
7. Possibly poisons from the spleen.

As regards hæmachromatosis, a discussion as to its pathogenesis does not enter into the province of this lecture. In my introductory remarks I mentioned that the view perhaps which had most adherents was that of Rossle and Eppinger, namely that owing to defective activity of the reticulo-endothelial system of the body the normal iron metabolism could not be carried out.

About Banti's syndrome I have but little to say; there is however such a close resemblance between malarial cirrhosis, that of kala-azar, that of some forms of syphilis, that one hopes in the future that the still unexplained cases of splenic anæmia will disclose their true etiology. We are now able to draw, however, a sharp distinction between those cases of splenic anæmia which have a thrombocytosis and frequently thrombosis of the splenic vein and which are aggravated by splenectomy, and those which have a thrombocytopenia and which are benefitted by this operation.

The hepato-lenticular degeneration of Kinnear Wilson is a rare disease but of extreme interest. It is occasionally familiar but not hereditary. The very remarkable resem-

blance between this disease and that of chronic manganese poisoning suggests irresistably that it is of toxic origin.

Alcoholic cirrhosis still remains. In this connection I would ask you to compare the remarks made in Taylor's *Medicine* some 30 years ago with those of Rolleston and McNee made last year.

Taylor says:—"But the simple theory of direct irritation by alcohol has not escaped criticism, and the following views have also been advanced: That the real irritant is some toxin produced in the mucous which results from the accompanying gastritis: that the irritant is not alcohol, but some other constituent of the liquid drink. The excessive use of spices is said to be occasionally the cause".

Rolleston says:—"With regard to the role of alcohol in the production of cirrhosis, it may safely be held that alcoholism is frequently an antecedent condition, but that per se alcohol has no specific action on the liver except fatty degeneration. It gives rise to cirrhosis in a secondary manner, either by leading to the production of sclerogenic poisons or by enabling such poisons to have full sway on the liver. The importance of alcoholism has been rather exaggerated, and it is not sufficiently recognised that other factors may cause cirrhosis, and that a congenital or acquired want of resistance on the part of the liver itself is, though hard to estimate, probably of great importance."

No further comment is necessary. In its protective function the liver shields the body from a continuous bombardment of poisonous substances received from the stomach and intestines and some such substances can be found stored in the liver after death. A slow degeneration and necrosis of the liver cells result. These cells disappear and are replaced by fibrous tissue. The connective tissue also shows signs of inflammation so that here we are dealing with a chronic hepatitis in which parenchyma and stroma are alike involved. In the early stages the liver is

enlarged, smooth and tender and moderately hard. The first symptoms are those of dyspepsia. I have one case in my notes whom I saw first in November 1928 with a large and hard liver and in whom to my eternal disgrace I diagnosed neoplasm. He died 9 months after from a typical portal cirrhosis. There was no alcohol and no syphilis and no more addiction to hot things than anyone else.

This lecture would be incomplete without a reference to the third chief heading in Chauffard's table, *i.e.*, capsular cirrhosis or perihepatitis, and I would remind you of Megaw's work in Calcutta where he has found chronic bacillary dysentery to be a potent cause of this condition. It would be interesting to discuss the pathogenesis of polyserositis or Concato's disease which is also occasionally found in association with cirrhosis of liver, but time does not permit me to enter into this discussion here.

There are several points about the treatment of cirrhosis:—

1. In all cases of cirrhosis where medical treatment has failed and no definite poison such as arsenic can be incriminated, the gall bladder should be explored and drained if necessary.

2. In all cases which may be reasonably expected to be early portal cirrhosis and where syphilis can be excluded, put the patient on a non-irritating diet. Give gastric lavage and hydrochloric acid.

3. Splenectomy has been tried in the Mayo clinic for such cases. The results have been disappointing. This indicates a different etiology from at least the thrombocytopenic cases of splenic anæmia.

Ladies and gentlemen, my object in this lecture has been to show you the connections which link together the various types of liver disease which appear to be widely separated in text-book descriptions.

A case of catarrhal jaundice usually ends as such; it may however become suddenly more serious and develop

into acute yellow atrophy. It may on the other hand become subacute and become a case of subacute hepatitis or toxic cirrhosis, or, it may ultimately terminate with the clinical picture of portal cirrhosis.

Therefore remember that in all cases with the clinical picture, it is necessary to go into a careful consideration of the earlier stages of the disease.

And now I will quote from an article written by Dr. Richard Bright on Jaundice in the Guy's Hospital Report as far back as 1836. He says:

“A very large proportion of cases are completely cured: and where the result is otherwise, it generally arises from some complication of diseases; most frequently from previous disorganization of the liver, or from neglect, on the part of the patient, in not applying for medical aid, or in not steadily pursuing the plan laid down: for it is not uncommon to find persons inclined to make light of an attack of jaundice, if the pain or inconvenience attending it is not such as to prevent them altogether from pursuing their usual occupations. Nothing, however, can be more injudicious; and it is the duty of the practitioner to impress this upon his patient: for it can never be a matter of slight importance, that either obstruction or inflammatory action should exist in the intimate structure of a secreting organ, still less in an organ so delicately complex as the liver: and there is no reason to doubt, that although, even without treatment, an attack of this kind may pass off, the liver will be some time before it has completely recovered; and thus a tendency to relapse, or to a renewal of the complaint, may occasionally be observed, showing itself at longer or shorter intervals throughout life, and terminating, at length, in the destruction of all the powers of the stomach, the ulceration of the colon, miserable emaciation, or universal dropsy.”

These words were written nearly 100 years ago, but as often happens they contain a valuable lesson for us to-day.

On the Nature of the Anti-bodies*

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The scientific Phase of experiments on immunity began with Pasteur. He was the first to demonstrate the importance of bacteria in causing infectious disease. It was also he who first conceived the idea of employing pathogenic Bacteria to therapeutic purposes.

It was in 1880 Pasteur was playing with the very tiny microbe that kills chicken with a malady known as chicken cholera. He had to go out for the holidays and on his return resumed his experiments of chicken cholera. He was surprised to observe that the cultures, which had been virulent before his departure had now become a virulent and did not kill the inoculated chickens. The fact that struck him in particular was that the inoculated chickens which survived resisted an inoculation of fresh virulent cultures.

A lesser man than Pasteur might have done the same accidental experiment for this was no test planned by the human brain. A lesser man might have done it and would have spent years trying to explain to himself the mystery of it. But Pasteur stumbling on this chance protection of a couple of miserable chickens saw at once a new way of guarding living beings against virulent germs of saving man from death. His brain jumped to a new way of tricking the hitherto inexorable God who ruled that men must be helpless before the sneaking attacks of his subvisible enemies. So it was that Pasteur ingeniously turned microbes against themselves.

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Thus two discoveries among the most important in biology were made namely the attenuation of the virus and vaccination.

At this time anthrax was ruining the whole of the central Europe depriving prosperous owners of cattle, of their belongings. There was enough superstition among the farmers and horse-doctors of Europe about this disease, strange beliefs in regard to the mysterious powers of this plague that always hung over their animals like an invisible sword. So it is not surprising to find Pasteur trying to discover a way of taming the vicious anthrax microbes and turning them into a vaccine.

As is natural with every great discoverer he had created a number of enemies around him. An eminent horse doctor of that time wanted to lure him into a dangerous public experiment, by inducing the Agricultural Society to invite Pasteur to prepare a vaccine that could protect their sheep and cattle from anthrax. Pasteur, not a bit suspicious, consented to the demonstration. And thus we find him at the farm of Pouilly-le-Fort. On May 5th, 1881 at the farm which is now called 'Clos Pasteur', 24 sheep, a goat and 6 cows received 5 drops of an attenuated anthrax culture by subcutaneous injection. On May 17th, the (24 sheep the goat and 6 cows) received a second subcutaneous injection of an attenuated culture but one that was more virulent than the first. On May 31st (the degree of immunity in the 31 vaccinated animals was determined). As controls Pasteur employed 29 new animals 24 sheep a goat and 4 cows. All the animals were inoculated with a very virulent culture. Two days later that is on June 2nd a number of people met at Pouilly-le-Fort to see the result of the vaccination. The visitors were profoundly moved when they saw the 24 vaccinated sheep, the goat and cows all in good health, while the 21 sheep and the goat not vaccinated already lay dead on the ground. (Two other non-vaccinated sheep died under

their eyes ; while the remaining animal died at the end of the day).

This experiment marked a turning point in the history not only of anthrax but in medical science as well. This was the beginning of important discoveries which continued to increase from year to year.

Nearly 50 years now separates us from the experiment at Pouilly-le-Fort. Bacteriology has made such rapid strides that we cannot find its equal in any other branch of biology. Bacteriology to-day dominates medicine, surgery and hygiene, to mention only those familiar to us. Workers in all countries have attacked the most varied problems pertaining to bacteria. New chapters have been created, old ones have been modified or begun anew.

What Pasteur did was to get his animals accustomed to tolerate increased doses of a most virulent poison. The process of tolerance was gradual for the tissues of the animals. This we technically call active immunisation.

The process of active immunisation so changes the animal body that it becomes highly resistant against an infection to which it had formerly in many instances been delicately susceptible. In the absence of visible anatomical or histological changes accompanying the acquisition of this new power, investigators in order to account for it were led to examine the physiological properties of the body cells and fluids of immunised subjects. While it was reasonable to suppose that all the cells and tissues were affected by or might have taken part in a physiological change so profoundly influencing the individual, the blood, because of its unquestionably close relationship to inflammatory reactions and because of the ease with which it could be obtained and studied, claimed the first and closest attention.

The study of the blood serum of immunised animals as to simple changes in chemical composition or physical properties has shed little light upon the subject. There is

little or no alteration from the normal in the blood sera of immunised animals, as to index of refraction specific gravity, alkalinity. These observations have helped little in explaining the nature of the process going on in immune sera.

The first actual light was thrown upon the mysterious phenomena of immunity by the investigations of Nuttall 1886 who not only demonstrated the power of normal blood serum to destroy bacteria but also showed that the property became diminished with age and destroyed completely by heating to 56°C.

Soon after Von Behring (1890) made further important advances in the elucidation of the immunising process by showing that the blood sera of animals actively immunised against the toxins of diphtheria and tetanus would protect normal animals against the poisons of these diseases—Antitoxins.

The formation of antitoxins directed against soluble poisons, however, did not explain the immunity acquired by animals against bacteria like B. Anthrax, the cholera vibrio, which produced little or no toxins. It was evident that the antitoxic property of immune blood serum was by no means the sole expression of its protective powers.

In 1894 Pfeiffer did his classical experiment by injecting cholera vibrios into the peritoneal cavity of immunised G. P. where the organisms became granular and underwent complete dissociation. He believed that the destruction of bacteria in the body was due to a substance which circulated in an inactive state in the serum of a vaccinated animal. The reaction was specific in that the destructive process took place to a marked extent only in the case of bacteria employed in the immunisation.

Bordet while confirming Pfeiffer's observation was able to show that the lytic process would take place in vitro. The constituents of the blood serum which gave rise to this destructive phenomenon were spoken of as bacterio-lysins.

Following closely upon the heels of Pfeiffer's observations came the discovery of another specific property of immune serum. Grubber and Durham observed that when certain bacteria were brought into contact with the serum of an animal immunised against them they were clumped together, deprived of motility and finally agglutinated.

The list of antibodies was again enlarged by Kraus who in 1897 showed that precipitates were formed when filtrates of cultures of cholera, typhoid and plague were mixed with their specific immune serum-precipitins.

The treatment of the animal body therefore with bacteria or their products gives rise to a variety of reactions which result in the presence of the antibodies described above.

It was soon evident to early investigators especially in the light of the large amounts of antibodies that are produced in response to the injection of small amount of toxin, that the antibodies were secreted by the body cells and were not due to a conversion of bacterial products.

An ingenious and fruitful attempt to explain the mode of origin and manner of action of antitoxic and bactericidal sera was made by Paul Ehrlich in his widely known side chain or receptor theory. It is well to bear in mind that the much used standard diagrams and pictures are the imagination and not representations of established fact. Whatever be the ultimate fate of the speculations that have been built up around the central hypothesis, there can be no doubt that knowledge of immunity and the immunising processes was greatly increased by the stimulus given to research.

This theory of Ehrlich of the origin and nature of specific anti-bodies beautifully explains and co-ordinates all known immunological facts. Four major hypotheses are implied in the Ehrlich theory, the assumptions (a) specific antibodies are preformed highly specialized chemical substances normally present in the animal body;

(b) that these substances originate in the fixed and wandering cells of the body, and are by them given off into the body fluids; (c) that the humoral elements thus formed and liberated are purposeful, defensive chemical substances for the body as a whole; and (d) that the formation and liberation of these specific chemical defences with their subsequent transmission and absorption by other cells are the only specific adaptive mechanisms in the body.

The theory is characterized by its minor hypotheses based on the specific receptor theory of cell nutrition. These minor hypotheses assume that some of these highly specialized food receptors have accidental affinities for certain antigens: (a) that a receptor having this accidental affinity is permanently injured or denatured by its chemical union with this antigen; (b) that the resulting denatured receptor is eventually cast off by the living cell; and (c) that the cell now regenerates a new receptor of identical chemical structure. This regeneration is over stimulated, more than one receptor being formed to replace the one lost. The superfluous receptors thus formed are in time cast off by the cell and constitute the specific antibodies.

The hypotheses thus formulated explain and co-ordinate all known immunological facts. The high susceptibility of certain animal species for a given antigen is a result of receptor having an assumed unusually high antibody for this antigen. The relative insusceptibility is explained as a result of an assumed paucity or low avidity of the same receptor.

The conviction that there is something wrong in the Ehrlich theory arises from a consideration of failures of therapeutic methods based on the assumptions. According to Ehrlich theory the specific antibody appearing as a result of active immunisation having a specific defensive chemical substance in sufficiently large quantities must have a valuable therapeutic effect. Yet in spite of active researches and commercial exploitation, therapeutic sera

have been lamentably unsuccessful except in a small group of diseases. This conviction gets strengthened by the study of the quantitative characteristics of serum reaction. It is evidently impossible to prove or disprove Ehrlich's theory by data drawn from test-tube experiment. Proof can only come from physiological study of immune tissues.

It is now generally agreed that Ehrlich's qualitative receptor analysis of immunity and immune reaction has out-lived its usefulness. From the presentation which has been given it must be apparent that it is only an ingenious application to immune processes of an hypotheses on cell nutrition and injury that has long since been discorded by students of physiology and pathology. Ehrlich's hypotheses have crumbled under the weight of the special assumptions which were necessitated by each new discovery. Recent investigators have found it more profitable to discord Ehrlich's hypotheses in favour of conceptions borrowed from colloidal chemistry. Though the dynamics of colloid systems are being applied to the study of immunity the general theory on these grounds has not yet been definitely formulated. Hence Ehrlich's pictures are still retained although their values are doubtful.

The studies on immunity outlined above have dealt entirely with the phenomena occurring in the reaction between bacteria or bacterial extracts and the body fluids. These studies have formed the basis of a theoretical conception of immunity formulated by the German School of Bacteriologists. Parallel with these developments, however, investigations on immunity have been carried on which have brought to light many important facts concerning the participation of the cellular elements of the body in its resistance to infection.

The inspiration for this work and the greater part of the theoretical considerations which have been based upon

it have emanated from Metchnikow and his numerous pupils at the Pasteur institute.

In studying the cellular activities which come into play whenever foreign material of any description gains entrance into the animal body a definite reaction on the part of the phagocytic cells has been observed. The presence of a foreign material in the peritoneal cavity has after a primary repellant action upon the phagocytes attracted them in large numbers to the site of the foreign substance. Such repelling or attracting influences upon the leucocytes are spoken of as negative or positive chemotaxis.

We have seen therefore that the invasion of the animal body by foreign material, living or dead, is followed by a prompt response on the part of the phagocytic cells. In the case of bacteria when these are deposited in the subcutaneous areolar tissue the inflammatory reaction which follows brings with it an emigration of microphages (Polymorphs) from the blood vessels, and these are the so called pus cells.

When the injection of bacteria is intraperitoneal, after primary diminution, there is an increase of leucocytes in the peritoneal cavity which soon results in the formation of a copious turbid exudate. If the pus or exudate is examined microscopically it will be seen that many of the microphages have taken bacteria into their cytoplasm. That fully virulent living bacteria can be so taken up has been variously proved. The phagocytosis is therefore not simply a removal of the dead bodies of bacteria previously killed by the body fluids, but represents an actual attack upon living and fully virulent micro-organisms. That the ingested bacteria are often alive after ingestion is proved by the fact of their being able to give rise to infection. It must not be forgotten however, that not all micro-organisms are equally susceptible to phagocytosis. Some may resist ingestion more energetically than others by agencies not fully understood. To a certain extent moreover the variety

of bacteria determines the variety of phagocyte attracted to the point of invasion. It is clear then that the process of phagocytosis is a universal response on the part of the body to the invasion of foreign particles, of dead material, of alien cells, and of living micro-organisms. It remains to be shown upon what basis this process may be regarded as essential feature in protecting the body against infection.

While few bacteriologists of the present day harbour any doubt as to the truth of these contentions the fundamental differences between the conclusions drawn from these phenomena by Metchnikow and by German workers may be clearly stated as follows:—Metchnikoff believes of phagocytosis as the cordinal factor which determines immunity, while Pfeiffer and others maintain that the determining factors upon which recovery depends lie in the fluids of the body, the serous exudate and its contents of immune body and complement, while the phagocytosis occurring coincidently is merely a means of removal of bacteria after the outcome has already been decided.

Although the theories of immunity are generally classified as the humoral and the cellular theories, the separation has never been an absolute one. The serum, according to Metchnikoff, acted not directly upon the bacteria in the nature of bactericidal or lytic substances but rather upon the leucocytes-preparing or arming these for the prey. Others came to the conclusion that the serum aided phagocytosis by its action upon the bacteria than by its influence upon the leucocytes.

It was left to Wright in 1903 and 1904 to determine the direct dependance of phagocytosis upon some substance contained in blood serum. He further proved that this serum component acts upon the bacteria directly and not upon the leucocytes and is bound by bacteria and renders them subject to phagocytosis. This he called Opsonin. This is a rapid sketch of the principal tendency of the problem of immunity as considered at that time.

On the Essential Identity of the Antibodies.

Since Ehrlich's first classical analysis of antibodies, it has been a generally accepted conception of immunity that agglutinins, precipitins, sensitizers, bacteriolysins, hæmolysins, opsonins. Anaphylactic antibodies are separate substances formed in the animal body often in response to treatment with a single antigen. The idea that agglutinins and precipitins might represent one and the same antibody has been more or less prevalent since their first observation. But in regard to the identification of other antibodies with these two, there has been either a complete disregard of such possibility, or when suggested it has been thrown out of consideration because of the frequent and complete lack of quantitative parallelism between the curves of the various antibody functions in one and the same serum.

In 1908 the fundamental idea of such a possibility was expressed quite definitely by Boil and Hoke who made an extensive study of the bacteriolytic precipitating and agglutinating actions of normal serum and immune rabbit serum. They clearly express the opinion that there were no separate bacteriolytic precipitating or agglutinating antibodies in these sera, that the essential fact was the existence, in the sera exerting these effects, of a single antibody which united with bacterial substances and that the various reactions which followed were the results of the conditions under which the different observations were made.

By the unitarian view of antibodies Zinsser formulates that working with a single antigen in a pure state one variety of antibody only would be produced. This would be present in the form of a serum constituent specifically capable of uniting with the antigen. As a result of the union the antigen is altered in its physical and perhaps to some extent in its chemical behaviour. The resulting reaction which may be observed with this sensitized antigen (agglutinins, precipitins, complement fixation, bactericidal, bacteriolytic, opsonization or sensitizing effects in the ana-

phylactic sense) would be determined not by differences in the nature of antibodies with which the antigen has united but rather by the physical state of the antigen itself, the nature of the co-operating substances (alexin, leucocytes tissue cells) and by the environmental conditions under which the observations are made. Thus if the antibody comes in contact with a very finely divided antigen—as in a bacterial extract or in a horse serum, (if electrolytes are present and perhaps other necessary physical factors furnished by the presence of serum) precipitation occurs.

When we are dealing with whole bacteria or relatively large mass and correspondingly small surface exposure, agglutination is the result. Further quantitative parallelism with the precipitin reaction is not to be expected because of the much greater dispersion of the antigen in the latter test.

When alexin is present complement fixation or haemolysis and bactericidal effect result.

When there are leucocytes present the union makes possible the phagocytosis of the antigen; and when the antibody is absorbed by the cells of the animal, anaphylactic sensitisation occurs.

We must not forget that agglutination and precipitation are actually only secondary phenomena after the union of antigen and antibody has taken place and are dependant upon a great many environmental factors which may not to the same degree influence phenomena in which alexin, the leucocyte or the body cells of animals are involved. In brief the final result of antigen-antibody reaction whether it reveals itself as a precipitation, an agglutination, or a fixation of complement is always a flocculation.

It is not by any means my wish to convey the impression that the unitarian view should be taken as absolutely or rigidly proven. If there is nothing further to be said in favour of this view one might at least wait for further

evidence before one tried to deny an existing view. But there is much to be said in favour of it and evidence in this direction is accumulating.

So far Immunological analyses have, of necessity, dealt almost entirely with the consequences of antibody formation and the mechanism by which these consequences are governed, but have, up to the present time, left us almost entirely in the dark concerning the fundamental cellular physiology upon which antibody formation is based. The fundamental source of all changes induced by antigen injections is the tissue cell. The tissue cells of higher animals are normally prepared only for the metabolic processes concerned in the nutritional and excretory functions essential to the maintenance of life. In the bodies of the higher animals the substances which reach the cells for nutritional purposes—*via* the blood, or lymphatics—come into actual cellular contact only after elaborate preparation by preliminary digestive processes. Proteins-aminoacids; fats in the form of glycerol, fatty-acids and soaps, carbohydrates simple sugars.

Thus the cell is normally attuned only to deal with substances in the chemical and physical conditions into which such preliminary digestion has put them. Only such substances can come into repeated contact with cells without in some way altering the quality or degree of the cellular reactions around them. It seems to be a fairly general biological principle that most materials which are not in this chemical and physical class of pre-digested nutritive matter give rise sooner or later to a specifically altered state of reaction capacity on the part of cells. Even when dealing with substances of relatively low molecular structure as alcohol, quinine, morphine, etc., the mere fact of habituation and occasional idiosyncrasy indicate that such altered conditions have been produced. As we proceed upwards towards the more complex substances with which the bacteriologist and immunologist more particularly deal-

toxins and proteins proper, contact of cells with these substances—incident to processes of infection invariably induces an altered reaction capacity which is recognised either as some form of hypersusceptibility or of tolerance.

If we consider the varied chemical and physical constitution of the many substances which may come into contact with the body cells from the drugs at the bottom of the list, to unchanged proteins it need not astonish us if the manifestations are subject to a wide range of variations. Common to all of them is the fact that the tissue cells are basically changed in their reaction to the particular material concerned.

These phenomena of reaction have been classified into two main sub-divisions:—Those in which definite antigen-antibody reactions are involved and in which passive transfer to normal animal is therefore feasible. And those in which no antibodies seem to be concerned and passive transfer unsuccessful.

The ability of the cell then to express its response in the production of specific antibodies which may become free in the blood stream is the chief point which separates the mechanism of one type of reaction from all others.

Ehrlich conceived the cell as giant molecule—a homogenous chemical system provided with a large number of side chains.

Since his time the conception of cell entity has changed considerably. The cell is no longer regarded as a large molecule of living matter the life of which is maintained by a constant interchange of chemical unions and dissociations, but must be conceived as a heterogenous series of phases separated from each other by semi-permeable surface layers.—Colloidal state.

Investigation upon the so-called colloids has led to a change in our conception of the reactions which take place between proteins and other substances. Colloidal particles carry a definite charge which is dependant upon H-ion

concentration. The particles carry a negative charge on the alkaline side of the so-called iso-electric point and a positive charge on the acid side.

When we are working with bacterial suspensions or suspensions of blood cells and the various normal and immune sera which are the instruments of immunology we are using materials all of which come under the classifications of colloids. All substances capable of provoking in the organism the formation of antibodies are called antigens, and all protein substances and protein substances only can function as antigens. To be antigenic the albumin must be present in a colloidal state and be derived from an animal different from that in which it is to function as antigen. The heterogenous albumin may be of very diverse origin. Sometimes they may be derived from foods. Another more important source is by the elaboration of foreign material during the course of bacterial infection. The reaction of the body to the bacterium as a banal protein substance is fundamentally different to that of the pathogenic bacterium in its character as a living parasitic being.

We have seen that the penetration of the body of an animal by a foreign albumin results in the appearance of antibodies in the blood of the animal. These antibodies possess the property of flocculating an albumin of the same nature as that which led to the formation of the reacting antibodies. The flocculating antibody is most probably not a substance but is rather a property resulting from a state of colloidal equilibrium of the blood plasma. Thus a single phenomenon takes place under the action of antibodies—a flocculation. Can then all the antibodies, which have been described be reduced to a single one? Though this hypothesis has been suggested by several scientists the idea has not yet been generally accepted obviously because it has been all along believed that the phenomena provoked have been multiple. The formation of

antibodies is the expression of a reaction tending to re-establish the colloidal equilibrium of the serum albumins which have been disturbed by the invasion of the body by a heterogenous albuminous colloid. The result is that it is not the foreign cell which undergoes the reactional shock but the body itself.

A first introduction of an antigen into the body causes—at the same time that it leads to the appearance of a principle which provokes the flocculation of this antigen—the sensitisation of the animal by virtue of the formation of an unstable state of equilibrium of the blood colloids. The acquisition of the sensitising property predicates the anaphylactic state and this unstable equilibrium is susceptible to rupture under the effect of second introduction of the same antigen. This rupture constitutes anaphylactic shock.

Anaphylaxis (Contra Protection.)

A guineapig injected subcutaneously with colloidal solution of any albumin. Some days later it gets another injection by the intravenous route of a small quantity of the same albumin. In a few seconds the guineapig presents a violent crisis of short duration which terminates usually in death. The first injection is called the sensitising injection.

We find here the same conditions as those essential to the formation of antibodies. The sensitisation is not produced immediately after the sensitising injection. It is not established until after a period of incubation varying with the dose of the albumin being shorter as the dose is smaller. It never appears before the sixth day, and once established it persists for a very long time. The second injection called the intoxicating injection must be relatively larger than the sensitising injection. The intoxicating injection especially if the amount is small should be given quickly at a single time, preferably by the intravenous route. The sensitising and intoxicating injections must be effected with the same albumin.

Physiologists had known for a long time that protein shock could be prevented by injecting a short time before the massive injection a minimal amount of the harmful substances. That which is peculiar to anaphylaxis is that the first injection creates a special sensitivity, such that a second injection of a minimal quantity suffices to provoke a crisis which, without the preliminary sensitisation, would have required a massive single injection. Nevertheless in the two cases the intimate causes of the crisis are identical; there is a sudden rupture in the colloidal equilibrium of the blood revealed by a sudden variation in the refractometric index of the serum.

In man the symptoms are similar to those presented by animals—dyspnoea, convulsive phenomena, gastrointestinal disturbances, collapse. Sensitisation may be produced naturally by the penetration into the circulation of native albumins, either by passage through the digestive mucosa or by penetration through the mucosa of the respiratory tract. In man alimentary disturbances often observed presenting symptoms which warrant the conclusion that such intolerances are of anaphylatic nature.

The most common symptom of alimentary intolerance is urticaria—such as we see as a characteristic symptom in serum disease. Then come the intestinal disturbances.

Foreign albumin may gain entrance to the circulation not only by way of the digestive tract but also and even more readily, by the pulmonary route. Hay fever and certain types of asthma are certainly of anaphylatic origin.

There is still another cause and a very important one which may lead to anaphylatic accidents. The bodies of bacteria contain a very small amount of albumin. Among the diverse symptoms which characterise a given infectious disease the greater part are specific that is they pertain to this disease solely and they are related to the action of products elaborated by the bacteria. But other symptoms are common to all infectious diseases; they have a common

cause and are due to the protein nature of the bacterial substance. So it is necessary to distinguish in each disease the part of the symptom-complex resulting from the bacteria by virtue of their toxic substances and that part which pertains to the colloidal substances of the bacteria.

Not only in a natural manner in the course of disease do bacterial colloids gain entrance to the body they gain access in a manner still more abrupt in the course of antibacterial vaccination. Since such vaccinations are usually carried out with bacterial bodies which have been heated the danger is not great, and is less, the higher the temperature used to kill bacteria, the colloidal state of the albumins being the more altered the higher the temperature to which they have been subjected. In fact accidents never occur from the injections of heated vaccines, but the danger becomes considerable when a series of injections is given with a colloidal solution of un-heated bacterial protein. In this case the first injection may cause a protein shock and re-injections may lead to a serious even fatal anaphylatic crisis. The reaction to vaccine may assume one of two forms:—It may be characterised by disturbances which do not become severe, such as fever, head-ache, lack of appetite, symptoms due to bacterial toxins. Or it may induce vaccine shock characterised by dyspnoea, tendency to soncope-cyanosis and anuria. The latter is purely a protein or anaphylatic reaction and is observed only following re-injections of bacteria in colloidal solution.

De-sensitisation:—Can be effected by a minute dose; then a slightly larger dose. Inject intravenously with extreme slowness.

Serum disease:—In man shock may follow the re-injection of a therapeutic serum. It may be well here to recall what we have said regarding serum disease as it follows a first injection. This condition appears only after an

incubation period of from 7 to 12 days. But circumstances may require a second injection of serum after a greater or less interval. The first injection may have been diphtheria anti-toxin administered therapeutically. Some months later the same person is affected with dysentery, with cerebrospinal meningitis or suffers an injury which renders tetanus possible. He is then injected with anti-dysenteric, anti-meningococcus or antitetanic serum. All of these sera are derived from the horse. After a second injection the reactions to the serum appear after a period which is the shorter as the interval of time elapsed since the first injection is less and these reactions are apparently the same as those resulting from a first injection—fever, joint-pains urticaria. This is the case when the injection is given by either the subcutaneous or intra-muscular route. But if the second injection is given by the intravenous route a typical often violent anaphylatic shock follows.

Cause of shock:—Why this difference in the length of the incubation period—7 to 12 days in the case of a first injection and a few hours or days after a re-injection? The foreign albumin introduced into the body especially when in a dose of 10 or 20 c.c. or even more in case of therapeutic sera, is eliminated but slowly; 12 to 15 days after the injection it is still possible to reveal its presence in the blood.

From quite a different point of view we have seen that all albuminous colloids if heterologous lead to the formation of antibody. And serum disease occurs at the precise moment when this sensitizer appears in the blood. Anaphylatic shock is a result of the union of this antibody which has been formed with antigen which still persists in the body. So much for the accidents which follow a first injection. If the second injection is given while the sensitizer developed by the first injection is still present in the blood the reaction appears after a few hours only. If the first injection antedates the second by a long time

the reactions are delayed, because the body is required once more to produce an antibody. But during this interval it has conserved a special aptitude to produce it anew under the influence of a second injection. It is clear that it is the antigens and the antigens solely which are capable of sensitizing an animal and of eliciting anaphylatic shock.

A re-injection is not intoxicating except it be made with an antigen of the same type as that which produced the sensitisation. The quantities required to sensitize are precisely those which are sufficient to provoke the formation of antibody. Finally the incubation time necessary for sensitization is exactly the time required for the formation of antibody.

Moreover passive anaphylaxis is conclusive in this regard. Incite in a rabbit the appearance of sensitizer by a series of injections of a given antigen-sheep serum. About 12 days after the last injection remove 1 to 2 c.c. of blood from the rabbit and inject it into the vein of a normal G. P. Test the G. P. a few hours later by an intravenous injection of sheep serum. It will show the anaphylatic crisis and perhaps die. Such sensitization is called passive. The anaphylatic crisis is due therefore to the union within the body of an antigen and its specific antibody. Upon this everybody is in accord. But the unanimity of opinion disappears when the results of this union are considered.

All the phenomena which take place in shock are from the effects of liberation of H-ions in the blood. What is the mechanism of this liberation of H-ions which takes place at the moment when sensitizer unites with the antigen? This is a problem of colloidal chemistry.

The anaphylatic reaction is necessarily provoked by a similar cause. The cause is always the disturbance of the colloidal equilibrium by a liberation of positive ions such as is always produced in the antigen-antibody reactions. But the blood exhibits the phenomenon of automatic

regulation tending to maintain fixed the quantity of H-ions thus controlling the ionic equilibrium of the blood colloids. This instantaneous auto regulation is assured by the buffers. The cause of shock is the excess of ions which remain free after the play of the buffers.

In a word if the conditions in the blood at the moment of the antigen-antibody combination are such that liberated H-ions are immediately fixed no variation in P. H. occurs and shock does not take place. It is clear then that shock may be avoided if care is taken to introduce intoxicating injection slowly. Further it is possible to understand that if the shock is not fatal within a short time all the disturbances may be dissipated without leaving trace.

Quite apart from anaphylatic shock of which the immediate cause is antigen-antibody reaction, each time that there occurs in the blood a sudden liberation of positive ions through some colloidal reaction there will be a crisis of the same order. Such is the case in simple protein shock.

One who has carefully followed the evolution of the science of immunology must feel certain that in many of the protective processes there is concerned some third, and as yet not clearly understood, factor in addition to the mobile polymorphonuclear phagocytes of the blood stream and the antibodies. Natural immunity may in general be ascribed to the activity of the polymorphs of the circulating blood. Acquired immunity whether active or passive involves certain new and enhanced physiological activities. These activities are expressed in the reaction products of serum-antibodies. These antagonistic substances are characterised by their ability to unite in a specific manner with the antigen substance that has given rise to them.

But all these facts, important as they are, do not suffice to explain the numerous phenomena of immunity. Small-pox immunity and the immunity which follows recovery from typhoid fever are not to be accounted for

either by the activity of the polymorph leucocytes or yet by action of antibodies. In spite of the importance of antibodies as indicative of a protective action a very marked immunity may exist without their presence and their presence alone does not ensure protection. It is evident therefore by a process of exclusion we are forced to consider the more or less fixed tissues of the body to find an explanation to certain phases of immunity.

In fact no one from the beginning has seriously disputed that the cells of the body form the back-ground of immunity. In the process of phagocytosis the more mobile cells—the polymorphs—are actively engaged in body defence although in the case of virulent infections their action, to be efficient, must be helped by a sensitizing effect on the invading bacteria by the action of the reaction antibodies of the body fluids. And these antibodies in turn must originate in cells. Too little attention has been paid to the cells of the body; the antibodies of the plasma important as they are in diagnosis are at but only a reflection of more fundamental processes that have gone on in the cells.

From the trend of investigation bearing on antibody formation the outstanding problems of immunity that require further elucidation will be the understanding of the method of antibody formation and an appreciation of the importance of fixed tissue cells in direct defence against micro-organisms through phagocytes.

Wide spread interest in the phenomena of local immunity has been awakened through the recent work of Besredka. He explains the mechanism of active immunity with an assumption that it cannot be accounted for entirely on the basis of antibody. This assumption is based on the lack of parallelism between the antibody content of serum and protection; on the fact that in some instances (anthrax chicken cholera) the known antibodies may be removed by specific absorption and the protective power of the serum remain. The known antibodies should be stripped of their

importance, their function in immunity is entirely secondary.

Active immunity is in essence a cellular phenomenon and depends on rendering insensitive the cells that are sensitive to a particular virus or micro-organisms.

Besredka explains the mechanism of acquired immunity that follows an attack of infectious disease thus:—During the course of the disease the receptive cells are continually brought in contact with the pathogenic bacterium and its derivatives. The reactions between the cell and the virus lead to a condition of adaptability of the one for the other. The initial sensitiveness of the respective cells become diminished so that they become indifferent to a new invasion of bacteria. This reaction is so complete that the cells which previously were so prompt to act now become accustomed or vaccinated. The white cell now not being hindered by a negative chemotaxis attacks the newly arrived bacteria. After capturing the vibrios the leucocytes break them up and then digest the stroma of the bacterium. The breaking up of the bacerial bodies results in the liberation of the anti virus; The digestion of the protein stroma is followed by the liberation of substances called antibodies. It is the free phagocytes in the blood that render the vaccine assimilable from these cells. Active immunisation is therefore brought about by the active co-operation of both these factors.

Thus Besredka believes that the free phagocytes in their process of phagocytosis liberate from bacteria an anti-virus (the precise nature of which is not clear) but which is essentially a non-antigenic toxic extract of the bacterial cell. This anti-virus turns at once to the receptive cells in the body which rapidly become saturated with it and thereby de-sensitise from the further toxic action of the corresponding virus. This process would be essentially similar to de-sensitisation by a small amount of foreign serum in animals that have been rendered suscep-

tible to anaphylactic shock by previous injection of the same protein, in other words it is analogous to the condition of "anti anaphylaxis."

This interesting and important new concept of immunity has been evolved by Besredka from a series of animal experiments and their clinical application extending over a number of years. The clinical results which are both preventive and curative in nature are in themselves increasingly impressive. So far the experimental findings that Besredka has furnished, are concerned particularly with bacteria whose pathogenic localisation (as he considers them) is either in the skin or in the intestinal canal.

The prominence given to the work of Besredka in the field of local immunisation is justified by the stimulus it has given to the more profound study of the possibilities of local immunisation rather than from a belief in the correctness of his explanation of the mechanism of local immunity. We believe that there is now over-whelming evidence of the existence of a truly localised form of immunity confined to certain infectious agents, to localized areas and representing not only the natural resistance enjoyed by many cells but also the exaggerated and important conditions of true active acquired immunity, though we see no evidence for believing that certain forms of active immunisation "close the specific portal of entry."

It may well be that we shall eventually trace all the fundamental immunity processes to one or another of that group of differentiated cells which we collect loosely under the designation of reticulo-endothelial system. Antibody formation relatively great resistance, ultimate disposal, not only of bacteria but of polymorphs that have already injected bacteria, we tentatively refer to them. Without attention directed in the right direction progress in immunity should take a new step forward.

Brachial Pressure Neuritis of the Lower Cord

BY THE CERVICAL RIB OR THE FIRST THORACIC RIB

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The Brachial Plexus may be of three types. The normal type is found only in about 30% and is made of 5th, 6th, 7th and 8th cervical nerves with 1st dorsal nerve. The cephalic type is usually found in about 62% and is made of 4th, 5th, 6th, 7th and 8th cervical nerves with a twig from 1st dorsal nerve. The caudal type is found in about 8% and is made of a twig from 5th cervical and 6th, 7th, 8th cervical nerves, and 1st dorsal with a twig from 2nd dorsal nerve. Therefore the presence of a cervical rib may not give rise to any symptoms in a cephalic type of brachial plexus. In a caudal type of brachial plexus the symptoms may be produced by a normal rib, especially if it has a sharp edge to the groove for the lower cord. Similarly if the Scalenus Anticus has a broad attachment and a stoutish tendon, these symptoms may appear, owing to the pressure of the tendon on the artery and nerve against an abnormal or a normal first rib.

Cervical Rib.—A cervical rib is a supernumerary rib springing from usually the seventh cervical vertebra. The formation of ribs depends on the nerve cords to the arm-buds in the embryo (Jones). The growth of these ribs is prevented by the existence of large nerves and in the embryo the nerves are relatively large. Todd believes that vessels have a similar influence. Anthropoid apes have 13 pairs of ribs and lower animals have a greater number of ribs. According to Todd the anomalies of growth of ribs may be:—(1) Anomalies of lower end of thorax with increase or decrease in the number of ribs; (2) Anomalies of upper end of thorax with increase or decrease

in the number of ribs; (3) Anomalies of both upper and lower end of thorax with increase or decrease in the number of ribs.

A cervical rib may be a true one when it has an articulation with the cervical vertebra or a false one when it is only an elongation of the anterior tubercle of transverse process of seventh cervical vertebra. Cervical rib may be bilateral (more common) or unilateral or two ribs may be present on the same side (rare).

Gruber of Petrograd classifies cervical ribs into following types:—(1) Slight degree of cervical rib that reaches a little beyond the transverse process of the vertebra. (2) Second degree where it reaches further out and may be attached to the first thoracic rib by a strand of fibrous tissue which may also contain muscle fibres. (3) Third degree where it reaches as far as the first rib being usually attached to it by Scalene Tubercle by cartilage or a capsule. (4) Fourth degree where the cervical rib is complete usually attached to the cartilage of first rib or occasionally to the sternum.

Anatomy.—A complete cervical rib extends from the seventh cervical vertebra forwards and downwards passing between the Scalenus Anticus and Medius to meet the costal cartilage of first thoracic rib. As the rib passes downwards the brachial plexus passes over it with Scalenus Medius in front, and the subclavian artery passes backwards over it behind the Scalenus Anticus. In a complete rib the Scalenus Anticus is inserted into the cervical rib; but in an incomplete one the muscle is inserted into the first thoracic rib being interposed between the vein and the accessory rib. Superficial to the plexus are the transverse scapular and transverse cervical vessels with the posterior belly of Omohyoid just above them. The Phrenic nerve passes in front of the Scalenus Anticus muscle. The long thoracic nerve of Bell passes along the outer border of Scalenus Medius deeper to the plexus. The Sternal-mastoid muscle protects and separates the Carotid sheath from the field. The External Jugular vein with its branches lies just along the lateral border of Sternal-mastoid muscle. If the cervical rib is

longer than 2 inches the subclavian artery will pass over it, otherwise it will pass over the first thoracic rib. The dome of pleura lies directly under the cervical rib, covered by Sibson's Fascia, the pleural cavity being increased by one space. If the rib is long enough intercostal muscles, nerves and vessels will also be present (Murphy). If the cervical rib is incomplete but is attached to first rib by a band the course of structures would be the same, but the Scalenus Anticus would be inserted into the first thoracic rib. Shorter accessory ribs are likely to run more laterally.

Etiology.—A cervical rib is a sign of stigma of reversion and a feature of underlying neuropathic diathesis. Other congenital deformities are associated with it. It may accompany a condition of Syringomyelia (Sherren & Bassoe). There is also a familial tendency for it. 80% of these cases are bilateral but only 30% give rise to bilateral symptoms. Complete bilateral ribs are rare. Cervical ribs are found more often on the left side, but the symptoms are more common on the right side. This is due to the facts that the right side is more often used for carrying weights, etc., and there is a greater droop of right shoulder in right handed people; also the right brachial plexus is in closer relation with its rib than on the left side (Adson).

Symptoms.—It is five to seven times more common in women than in men. This is probably due to the fact that there is more movement of thorax in women, the development of breasts which drag the shoulders down, and greater drooping of shoulders in women. Its onset may be brought on by rapid loss of weight, chronic nervous exhaustion, after child-birth, or by general ptosis. Onset of symptoms is usually during adolescence or after child-birth; but may come on earlier or later.

The chief symptoms are pain, disturbances of sensation, muscular atrophy, and circulatory disturbances.

Pain.—is felt on the inner side of arm and forearm. It may be sharp and lancinating, and may be brought on by

sudden rotation of the head or a forcible pull downwards of the shoulder. Sometimes the pain is dull and aching, burning or boring in character, and occurring during the latter part of the day after work as sweeping, washing, dusting, carrying of buckets etc. Hyperaesthesia, parasthesia, or anaesthesia as shown by tingling burning, or numbness along the inner side of arm, forearm, and inner two fingers may be complained of, depending on the degree of involvement chiefly of the lower and middle trunks.

Muscular Atrophy.—Sets in later and is progressive but rarely complete. It may be of two types: (a) partial Thenar type due to involvement of the cervical nerve, in which paralysis of abductor and opponens pollicis occurs and the thumb remains in adducted position; (b) the Ulnar type which is more common due to involvement of lowest trunk resulting in the paralysis of intrinsic muscles of the hand (except that of abductor and opponens pollicis) and the flexors of the inner two fingers, resulting in a Claw-hand.

Circulatory symptoms: are rarely severe. Dusky hue of the arm and hand, mild trophic changes in the finger tips, changes in the nails, or gangrene of one or more fingers may occur. Diminution of radial pulse is common. Diminution or complete obliteration of radial pulse may be produced by elevating the chin or by rotating the head to the affected side while inspiring deeply. The symptoms vary according to the amount of pressure over the subclavian artery. Sometimes the pulsations of the artery may be seen or felt above the clavicle. Horner's syndrome due to involvement of the sympathetic may be present owing to pressure and traction on the inferior cervical ganglion. Relief of these symptoms is obtained by keeping the shoulder well elevated by a sling to the elbow or by raising the arm up by the side of the head.

Where sensory symptoms are present protopathic sensibility is affected to a greater extent than epicritic (Stopford).

Causes of symptoms.—Todd says that the symptoms are due to the diminished obliquity of thoracic operculum, the distance

of the inner border of the first rib from the middle line, with relative widening of the shoulder girdle. He says that the vascular symptoms are due to trophic changes, the result of paralysis of the sympathetic fibres passing to the vessels. Hence the trophic changes occur in the same areas, either in the vessels, muscles or nerves supplied by the same nerve. But the compression of artery as seen in operation, the diminution of pulse-volume in the vessels of the arm, cyanosis of the skin of the affected arm, variability of the pulse-volume and cyanosis, when the Scalmi are put into tension by suitable movements, the pathological changes in the subclavian artery where it crosses over the cervical rib, gangrene of the fingers, all these are difficult to explain on Todd's theory. Adson is of opinion that all these symptoms are produced by compression of the artery and cords by the Scalmus Anterior against the cervical rib or the first thoracic rib. In adult life when the muscle is most developed the symptoms either appear or are increased, and this is also the period of greatest physical exertion. It is more frequent on the right side owing to greater use of right arm which causes drooping of right shoulder. In cases of cervical rib without any symptoms this muscle is found to be very poorly developed. In cases with symptoms this muscle is found well developed and has a wide attachment to the first rib, the muscle being reflected laterally beyond the operculum on to the cervical rib, narrowing the space between the lateral border of that muscle and the cervical rib. In the absence of symptoms the insertion is small and more medial leaving a wider space. Also if the cervical rib takes a wide lateral curve the space is wider and pressure is unlikely (Adson & Coffey). The symptoms also will depend on the type of brachial plexus as mentioned above.

Pressure on the Brachial Plexus by a normal thoracic rib.
—The same type of symptoms may be produced by a normal thoracic rib especially in a caudal type of brachial plexus, especially when such a rib has a prominent ridge to the groove that accommodates the lowest cord. Edwin Bramwell of

Edinburgh was the first to suggest the relationship between the symptoms, the first rib and the first thoracic nerve root. The symptoms are the same as in the previous type, both in onset and their progress. They are aggravated by carrying weights, etc., and relieved by raising the shoulder. Symptoms are very liable to follow a fracture of clavicle that has not united in good position. This is due to drooping of the shoulder on that side. Majority of cases occur in adult life or adolescence, when the use of right arm for carrying weights, etc., becomes necessary. Even in cases of cervical rib some may be due to compression of lowest cord against the first rib. The tendon of Scalenus Anterior is also a factor to be counted in these cases.

Diagnosis:—The gradual onset of symptoms sensory, motor and vascular during adolescence or early adult life and their relief by raising of shoulder by a sling to elbow is very suggestive. X-ray examination may show an accessory cervical rib. The fact that the radial pulse is affected by the condition, and relieved by raising the shoulder is quite a distinctive feature. Protopathic sensibility is lost to a greater extent than epicritic due to nerve compression (Stopford). The condition has to be diagnosed from Syringomyelia, Progressive muscular atrophy, Klumpke's paralysis and ulnar neuritis. The vascular symptoms have to be distinguished from Anglo-neurotic oedema, Raynaud's disease, etc.

Indications for surgical intervention.—(1) When patient complains of pronounced pain and the circulatory and sensory disturbances are sufficient to incapacitate her. (2) If patient shows evidence of muscular paresis or atrophy in the hand or arm. (3) If there is evidence of circulatory disturbance especially if the radial pulse can be obliterated or reduced at the wrist by movements of neck. (4) If any of the above group of symptoms are present in a case of anomalies in the supraclavicular triangle. (5) In neurotics if paræsthesia only is present the operation may be advised but option is left to the patient as results may not be promising.

Surgical treatment is not advised in mild cases, slinging of the elbow to raise the shoulder being enough. But in those that have to depend on work for livelihood operation is advisable.

Treatment:—Adson and Coffey strongly advise a simple tenotomy of Scalenus Anterior just above its insertion by a 4 inch collar incision just above the clavicle. This gives relief in a large number of cases. Failing to improve the condition or on recurrence removal of the guilty rib may be done by a posterior approach.

Recurrence after removal of the rib may be due to:—(1) Insufficient removal of rib. (2) Compression by adhesions or callus formed from the cut ends of the rib. (3) If periosteum is left behind bone may be formed again with recurrence of symptoms. (4) Intra-neural fibrosis especially in long standing cases. In some pain and objective symptoms disappear or improve after operation, but there is little or no improvement in the atrophy of muscles.

Operation.

Stiles used an angular incision raising a triangular flap upwards and inwards, Stopford raises a triangular flap upwards and outwards by an angular incision. These are very useful in short and thick necks. But in women with thin long necks, a low collar-incision 12cms. long just above the clavicle gives enough exposure and the resulting scar can be hidden by a necklace. After cutting through skin, platysma (supraclavicular nerves being cut in this) the external jugular vein is exposed and cut between two ligatures. The omo-hyoid is divided and retracted. The transverse scapular and cervical vessels are exposed and resected between proximal and distal ligatures. The lateral border of Scalenus Anterior is identified and exposed. Scalenus Medius is now identified and its insertion into first rib defined. The phrenic nerve passing in front of scalenus anterior and the nerve of Bell passing down on the lateral part of scalenus medius are protected. Next the layer of fatty tissue lying in front of brachial plexus is carefully dissected up as a layer. The brachial plexus and artery are

now exposed and protected in the subsequent procedure. The scalenus anterior is divided through three-fourths of its insertion if the tendon is a broad and thick one. Next the plexus artery are lifted forwards and the cervical rib is exposed. This is removed subperiosteally for safety and then the periosteum is sniped away carefully. The Sibson's fascia is the only structure lying between the rib and dome of pleura. When the first rib has to be removed the tendon of scalenus medius is cut through the front three-fourths of it. Now with a curved raspatory hugging close to the rib Sibson's fascia is gently separated and the rib is nibbled by ronguer forceps about an inch from the tip of transverse process of the vertebra and anteriorly it is nibbled just proximal to scalene tubercle. The scalenus anterior tendon is divided through three-fourths of its thickness 1.5 c.m. above the tubercle. Care should be taken of phrenic nerve. The posterior fibrous portion of the muscle should be cut to prevent pressure on the artery. The divided anterior part of scalenus medius is sutured to the remaining posterior part to prevent too much retraction. The space is drained by a rubber dam for 24-48 hours and wound closed in layers.

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Coronary Thrombosis

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Historical.—The syndrome constituting coronary thrombosis was not recognised as a distinct clinical entity until as recently as 1925 by British physicians, who with their habitual but not altogether unjustifiable scepticism are wont to take in all new ideas and new inventions with the proverbial grain of salt and would never accept them until they have passed unscathed through the fire of unrelenting adverse criticism. That is why even the late Sir James Mackenzie who is considered on all hands as the father of modern cardiology failed to differentiate the symptom—complex of cardiac infarction from that of angina pectoris. Even the exhaustive text-book of *Practice of Medicine* by Price has thought it fit to give the condition the dignity of being treated in at least one single page only after the book has run on to its third and latest edition while in the previous editions the subject is barely mentioned. A full clinical description of coronary thrombosis was first given in 1910 by Obrastzow and Strachek and later on in 1912 by J. B. Herrick, subsequent to which it has been exhaustively treated on various occasions in American Medical Journals duly supported by reports both clinical and post mortem. But when once the condition comes to be unquestionably recognised, the mute inglorious Miltons are pooh-poohed by the merciless autocrat as those who "labouring for invention bear amiss the second burthen of a former child". With that view researches have been conducted into the antiquated manuscripts which had been ere long relegated to the museum of archaic ideas, the result being that Harvey's description of Sir Robert Darcy's case in the second Desquisation to J. Riolo in which the wall of the left ventricle was ruptured apparently as the result of "an impediment to the passing of the blood

from the left ventricle into the arteries" has now been recognised as an early example of coronary thrombosis. In 1928 Parkinson and Bedford published 100 clinical cases and 83 post-mortem cases. A most exhaustive description of the syndrome has been given in 1929 September issue of "Medicine" by Levine, who gives in addition, detailed notes about more than 100 cases both clinical and post-mortem along with electro-cardiographic changes in each case. The previous annual reports of the General Hospital, Madras, do not contain any record of cases of cardiac infarction and hence it is a matter for congratulation that the first case to be clinically recognised in the General Hospital ever since its inception came under the meticulous care of the First Physician at the beginning of the current year.

Etiology.—Curiously enough, neither any of the specific diseases nor any of the infectious diseases either acute or chronic have any bearing upon the production of this condition. Nor even the much maligned spirocheta pallida seem to have a direct hand excepting in a few cases where specific arteritis is noticed in the coronary arteries. Chronic primary hypertension and general arteriosclerosis are the most common etiological factors even though some cases have been reported where the blood pressure was not high before the onset. In 34% of Levine's cases glycosuria has been noticed and hence he opined that diabetes has an important bearing to the development of this disease. The fact that the presence of diabetes does not alter prognosis leads one to feel that it has no curative influence in the disease of the coronary arteries, but only indicates the type of persons who have a vulnerable cardio-vascular system. Rheumatism which is perhaps the most common cause of cardiac troubles has nothing to do with the production of cardiac infarction. Heredity is important because many give histories suggesting degenerative diseases of the circulatory system. The patient is generally in the later years of life (average according to Levine being 57) a well set person, rather over weight, good physical strength, with a previous history of having enjoyed an

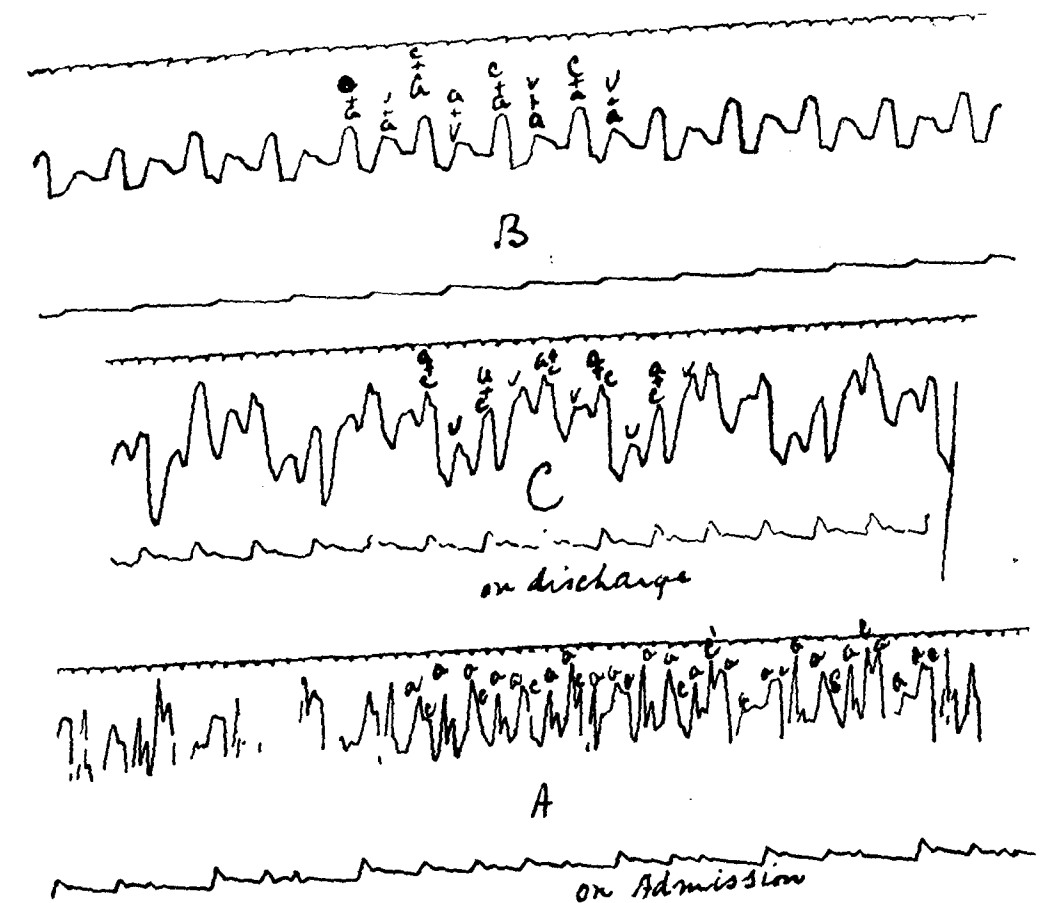
unusually good health. Angina pectoris is related to coronary thrombosis in that many of the persons affected by this syndrome give a history of previous attacks of angina of effort which finally culminated in the terrible disastrous attack. It may be stated in passing that this relationship affords an important argument in favour of one of the theories for the production of angina namely that it is due to a temporary spasm of the coronary arteries causing a transient occlusion of its lumen.

Pathology.—Thrombus formation in the coronary artery causes localised damage to the heart muscle, namely infarction the situation and extent of which depend on the artery affected. The right coronary artery supplies the whole of the right ventricle with the exception of the left one-third of its anterior wall, while the left supplies the whole of the left ventricle and the small left anterior portion of the interventricular septum. So much so, the anterior descending branch of the left coronary being the most commonly affected, the infarct is often found involving the lower and anterior part of the left ventricle and the adjacent part of the septum and sometimes also part of the wall of the right ventricle. Occlusion of the artery causes necrosis with extravasation of blood into the muscle tissue and subsequent early repair by connective tissue proliferation if the patient lives long enough.

Symptoms and signs.—A typical clinical picture of the syndrome of coronary thrombosis is afforded by the recent case in the General Hospital, which I propose to discuss in this connection. This patient had led an extraordinarily healthy life, subjected himself to undue physical exertion and had contributed his quota to the service of humanity by responding to the call of the bugle in the Great War. We have adequate reasons to suppose that his blood pressure before the attack had been unduly high but had not caused any untoward symptoms. On August 6th, 1929, he returned home after a hard day's overwork and went to sleep early after his usual meal. At 11-30 that night he suddenly woke up from his sleep being seized with an excessively severe pain over the precordium shooting up to

the left shoulder and neck. He felt as if an one ton weight had been placed over his chest and experienced a sense of impending dissolution. He was very restless, breathless and sleepless throughout the remainder of the night. Pulse was feeble and rapid, extremities became cold, being covered with a clammy sweat. There was a considerable fall in the blood pressure which used to be as high as 200 mm. Hg systolic before the attack. This condition continued on for several days and even though there was gradual abatement in the severity of the pain, the patient was still in a condition of orthopnœa, complaining of persistent insomnia and anorexia. Subsequently he was admitted in M-1 wards of the General Hospital. After subjecting him to the usual routine investigations and examination we found that he had an enlarged and sagging heart which was confirmed by radiography, sounds typically tictac unaccompanied by any endocardial murmurs, rhythm irregular polygraph tracing showing auricular flutter with one in one heart block with occasional extra-systole (see tracing A), congestion at the bases of the lungs, enlarged and tender liver and a flatulant abdomen. There was no noticeable œdema in any part of the body. His systolic blood pressure was 150 mm. Hg Wassermann negative, urine normal and renal efficiency normal. He gradually became better under the influence of digitalis, physical and mental rest and dietetic restrictions. But unfortunately he had to experience an equally terrible disaster of pulmonary apoplexy. One morning he was suddenly seized with a severe pain over the right side of the chest and difficulty in breathing and began to spit blood-tinged sputum. On physical examination there was a well-marked area of dullness over the right lower lobe with bronchial breathing and pleuritic rub. He was lucky in surviving this superadded catastrophe and was discharged from the hospital relieved of all his prominent symptoms. At the time of discharge his pulse was quite regular though the polygraphic tracing showed distinct nodal rhythm (tracing C.)

Now going from the particular to the general, it is found that the patient often gives a history of previous attacks of



angina and of high blood pressure. It generally occurs as in this case during sleep which is disturbed by the painful seizure, the pain being situated over the chest of upper abdomen and characterised by a sense of constriction or of unusual heaviness. Sometimes the patient may collapse and may fall unconscious. Frequently it is accompanied by vomiting in which case it may be mistaken for one of acute abdomen. Shortly after the onset the patient presents a picture of profound shock, with a feeble and rapid pulse, cold and clammy sweats, ashen gray skin and fall in blood pressure. Oedema of lungs may develop with moist rales heard at the bases, and liver may become enlarged and tender. Very often there is gallop rhythm or pulsus alternans or any other form of cardiac arrhythmia as extra systole, auricular flutter or fibrillation, or ventricular tachycardia. Depending on the degree of infarction there may be a rise in temperature and a distinct leucocytosis. More frequently examination of urine reveals glycosuria and less frequently albuminuria. Many of the patients who survive the attack improve steadily, but some develop signs of congestive cardiac failure and die as a result. If the infarct reaches the pericardium, there will be a pericardial rub heard; if the septum is involved, there will be heart block, and if it spreads to the endocardium mural thrombus formation with subsequent embolism.

Atypical features.—If coronary thrombosis occurs in a case of myocarditis the picture is less striking; also when the occlusion occurs slowly which is often found in cardiorenal diseases where they show a picture of congestive heart failure following previous attacks of angina. There are some cases which do not give a history of angina, some others who had only normal or even low blood pressure. Cases are on record where patients were unfortunately subjected to unnecessary abdominal operations, they being diagnosed as cases of acute abdomen, while in reality were cases of coronary thrombosis with acute abdominal symptoms.

Termination.—In fifty per cent. of cases death occurs instantaneously possibly as result of ventricular fibrillation, while in

some others death may occur in a few days due to rupture of aneurismal dilation of the heart resulting from damage to the heart muscle. Those that survive the primary onslaught may yet die latter of congestive heart failure. There are three types of recovery. The first and the best form of recovery is where the patient after repeated attacks of angina is seized with coronary thrombosis after which the anginal attacks do not return, blood pressure keeps to the normal level and the patient may survive for 3 to 5 years. In the second type of recovery the attacks of angina persist even after coronary thrombosis possibly due to the blood pressure going up again after having fallen during the attack. In the third type, the patient after a period of comparative well being develops gradual cardiac insufficiency and may subsequently die of heart failure.

If at all anything will help in clinching the diagnosis of coronary thrombosis in doubtful cases, it is the electrocardiographic findings, which unfortunately, by some curious dispensation of providence are not made available to us in Madras where the electrocardiograph ever since its installation has perpetually been out of order, the failure being attributed to the idiotic idiosyncrasy of a quartz fibre which has been condemned for misbehaviour in a tropical climate. The absence of this instrument is really a great handicap and we are in fact 20 years behind the times, since no hospital can have any pretensions to be up-to-date so long as it is not provided with a working electrocardiograph. Now that we gain knowledge of the workings of this wonderful instrument only through books I shall but briefly mention the electrocardiographic changes, which are characteristic of coronary thrombosis. These are (1) A diminution in the amplitude of all the waves. (2) Disappearance of the isoelectric interval between the Q. R. S. complex and the T wave which is the terminal ventricular deflection. (3) Complexes change their form from day to day. This may be appreciated by a reference to the case above mentioned wherein even the polygraphic tracings showed changes as the case progressed.

Differential Diagnosis.—Cases of sudden death can only be diagnosed post-mortem. In those that survive for the time being diagnosis is mainly made by the typical clinical history rather than by physical signs and can be confirmed by electrocardiographic changes. From angina it can be differentiated by the long duration and extreme severity of the pain unrelieved by nitrites, fall in blood pressure, increase in pulse rate, rise in temperature, high leucocytic count, irregularity in the cardiac rhythm, occasional presence of a pericardial rub, not uncommon embolic phenomenon, evidence of shock, development of dyspnoea and gastric symptoms all of which are absent in true angina pectoris. It has also to be differentiated from acute abdominal conditions, and in all cases above 40, with acute upper abdominal symptoms, coronary thrombosis must be thought of at least from the point of view of differential diagnosis. It may simulate an abdominal catastrophe because the patient attacked with coronary thrombosis may complain of pain in the epigastrium with vomiting, rise in pulse rate, and temperature, associated with leucocytosis. Points which help in the differentiation are previous history of angina and high blood pressure, cardiac enlargement and irregularity and occasional presence of pericardial rub. It has also to be diagnosed from diabetic acidosis pneumonia and pleurisy.

Prognosis is the most difficult part of the whole affair. A patient who survives the attack may suddenly die in a few days. On the other hand he might pass unseathed through an extremely severe attack and survive for a long time. Immediate mortality is 53% according to Levine's report while the average duration of life after recovery is two years. The younger the patient the more the chances of recovery. Some of the cardiac irregularities as auricular flutter, fibrillation and extrasystole do not alter the prognosis whereas the complications such as heart block and ventricular tachycardia are unfavourable. Prolonged unconsciousness is of very bad omen. On the whole prognosis is gloomy in the extreme.

Treatment of coronary thrombosis, as in many other diseased conditions, is the least interesting though the most import-

ant part of the whole discussion. We are absolutely helpless when the catastrophe occurs. Nothing can be done either for its prevention or for its cure. It takes you unawares when you are in the best of health freed from all the troubles and tribulations of life, and strikes you with the force and cunning of a midnight apathe when you are sound asleep. The only treatment which we can possibly adopt is symptomatic wherein our trusted friend morphia comes to our unfailing aid to alleviate the excruciating pain. As far as possible stimulation is to be avoided even if there is shock. When pulse has become imperceptible adrenalin or digitalin may be given. In the first few hours when there is no danger of either rupture of aneurysm or embolism, stimulation by caffeine or strophanthin is justified if the shock is profound. Digitalis is only of use when congestive heart failure has set in, after the patient has tided over the immediate catastrophe. Perhaps the most important point as regards treatment in this condition more so in this than in any other disease is absolute rest, physical, mental and physiological. The whole treatment consists in alleviation of pain which is terrible, combating shock which is invariable, curing insomnia which is persistent and improving digestion which is impaired and finally preventing the onset of congestive heart failure which is occasional.

I thank Colonel Malcomson, First Physician, General Hospital for allowing me to discuss the case which was under his care, and for confirming the polygraphic findings, and also for his very valuable suggestions.

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The Pathology of Chronic Colitis in the Tropics*

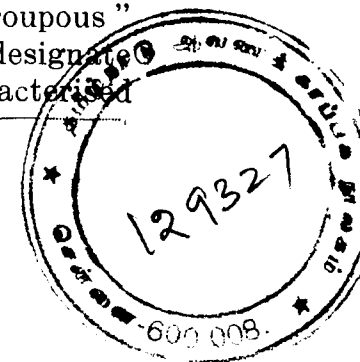
BY T. BHASKARA MENON, M.D., M.R.C.P.

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The term "colitis" has been used clinically to various obscure affections of the large intestine characterised by diarrhoea or by the passage of mucus or even blood in the motions. The underlying pathological condition has not been clearly described. The term itself presupposes an inflammatory affection of the colon. Varying grades are met with from mere superficial catarrh of the mucus membrane to actual ulceration and even total destruction and sloughing of the colon. Descriptions of the morbid anatomy of the lesions met with naturally vary in such a vague group. It must be understood that the condition called muco-membranous colic characterised by constipation, attacks of colicky pain and the passage of membranes is not included under "colitis" in agreement with the view that the condition is a hyper secretion of vagal origin very similar in its mechanism to asthma.

"Enteritis" is a similar term applied to an acute or chronic catarrhal affection of the bowel or more particularly the large intestines. The term "Follicular" ulceration connotes a type of ulceration confined to the lymph follicles of the small and also of the large intestine. "Stereocoral ulceration" is also another term that has been used to denote a type of ulceration thought to be due to the presence of hard scybalae. "Diphtheritic" colitis, "croupous" enteritis are terms that were commonly used to designate that type of acute inflammation of the colon characterised

* A paper read before the Indian Science Congress, January, 1930.



by the presence of a false membrane of fibrin and inflammatory cells. The term "ulcerative colitis" has been used to designate an affection of obscure aetiology but here in India at least most of the cases labelled as such show on autopsy well marked features of dysenteric ulceration. The term "dysentery" itself is rather loosely applied to ulcerative affections of the colon due to infection either with the *entamoeba histolytica* or of bacilli belonging to the shiga or Flexner-Y types. "Gangrenous colitis" is a name given to a type of severe ulcerative colitis characterized by the presence of dirty grayish black sloughs.

Colitis has been attributed to various causes such as chronic constipation, infection from various sources such as the food, mouth, the appendix, to mechanical irritation from kinks and stenosis, to intestinal parasites, etc., Excretion of uric acid through the colon in gouty conditions giving rise to a gouty colitis has also been described.

A study of the post-mortem records and material obtained from about 800 cases of various types of inflammatory and ulcerative affections of the bowel has shown the importance of what may be called the "dysenteric" factor in all these affections.

For a proper appreciation of this a description of types of dysenteric lesions we have met with is of interest.

A study of post-mortem material in Bacillary dysentery shows one great similarity in that the lesions are always inflammatory. It is not sufficiently recognised however that the reaction varies to a great extent with the type and virulence of the infection organisms.

Early lesions in severe cases are mostly in the crests of the folds of the mucous membrane and consist of a speck of necrosis covered by whitish grey slough—a diphtheritic reaction. This is surrounded by an area of acute congestion and hæmorrhage into the mucous membrane. However, it is quite common to meet with early lesions in which the hæmorrhagic reaction is more predominant and

the diphtheritic reaction is not so marked, so that the lesions found post-mortem are small serpigenous ulcers surrounded by hæmorrhage extending along the crest of the folds of the mucosa, the ulcers being mostly superficial while the whole mucous membrane shows intense congestion and points of hæmorrhage. Sometimes specks of hæmorrhage with a central area of necrosis are the first lesions that are noticed. Very often the whole of the sigmoid and the descending colon are covered with large irregular serpigenous ulcers with intense engorgement of the surrounding mucosa. The ulcers are covered with dirty yellow sloughs extending in sheets an appearance corresponding to the diphtheritic colitis of old writers. In very acute infections a "gangrenous" process may supervene the whole mucous membrane showing intense infiltration with inflammatory cells. It is to be noted that chronic cases of bacillary dysentery do not present this typical diphtheritic or hæmorrhagic reaction. Indeed in the great majority of cases late lesions can only be made out by an examination of sections which show the type of reaction. The lesions are invariably inflammatory even in chronic cases. Proliferative changes in the mucous membrane are quite common giving rise to polypoid elevations from the presence of adenomatous masses or cysts in the glands,—colitis polyposa. On the other hand atrophy of the bowel may supervene from extensive ulceration of the mucosa and the end result may be a thin pigmented and attenuated intestine which microscopically exhibits very little glandular tissue and in which all the coats show atrophy. The commonest type however is a thickened bowel presenting what may be called "moss covered" appearance of the mucous membrane with irregular scars in places. This is due to the superficial ulceration of the villi so that the uniform smooth texture of the mucosa is lost. It must be recognised that the commonest "follicular" ulcer that is met with here is in infections with the

dysentery groups and such ulcers form the basis of the so-called granular colitis of old writers. So called "Stercoral" ulcers identical in every respect with ulcers in the neighbourhood of strictures and kinks are met with in chronic infections with the dysentery bacilli. Then the question arises whether these types of ulcers are really dysenteric in nature. Histologically "stercoral" ulceration presents features indistinguishable from chronic dysenteric ulcers.

When we turn to amoebic dysentery we find a different reaction of the colon not inflammatory but degenerative and necrotic. This is obvious from a study of amoebic lesions. The lesion is a necrosis of tissue which involves the mucosa and submucosa and extends down to the muscle coat causing extensive and deep ulcers. Early lesions corresponding to Bartlett's first type where there are tiny pin point nodules without any breach of surface have not been met with in our autopsies. Minute nodules with yellow ulcerated margins corresponding to Bartlett's second type were also not met with. The usual type of amoebic lesion is a large irregular ulcer covered by gray and necrotic sloughs and with undermined edges, the surrounding mucosa showing a boggy appearance. Bartlett's classification of five types of amoebic lesions are not borne out by our records. In chronic cases ulcers have clear cut margins and a punched out appearance, while others have a honey-combed appearance. The wall of the intestine is usually thinned out and atrophied. In hyperacute infections the mucous membrane is extensively ulcerated and a gangrenous process is sometimes present. The ulcers have at their edges black or gray tags of necrosed mucous membrane and the base of the ulcers themselves is dark, discoloured and friable; The colon may be very much thinned out and friable and perforations sometimes occur. Here we have a different type of reaction with destruction of tissue. Very little

inflammatory changes are observed in uncomplicated infections with *E. Histolytica*. "Mixed" infections however occur in which the necrosis of tissue caused by the *entamoeba histolytica* and inflammatory lesions probably due to dysentery organisms are both present.

What are the lesions met with in chronic colitis and chronic diarrhoea? Mummery recognises three pathological types; (1) hypertrophic colitis where there is glandular proliferation, (2) granular colitis where there is follicular ulceration and inflammation; (3) chronic catarrhal colitis. Herschall and Abrahams recognize besides these (4) an atrophic form which is the late result of a chronic catarrh. An analysis of the post-mortem findings of 40 cases of chronic diarrhoea described variously as chronic colitis, enderocolitis, tubercular diarrhoea etc., have showed lesions indistinguishable histologically from that of chronic Bacillary dysentery in 18 cases and from amoebic dysentery in 6 cases. In the remaining 16 cases the only condition met with was atrophied intestine showing atrophy of the glands, atrophy of the mucous membrane and even of the muscle coat. Whether these are lesions met with as a result of previous dysenteric infection or whether they represent an atrophied state of the intestine as a result of chronic inanition or possibly vitamin deficiency or whether they represent atypical cases of sprue it is not possible to say at present. It seems probable however from a study of Post-mortem material that a large number of these cases of chronic diarrhoea are really the result of chronic infection with the dysentery bacilli or the *entamoeba histolytica*.

It might be asked why it is that cases of chronic colitis do not give definite evidence on culture of dysenteric infections. Bacteriologists have pointed out that dysentery organisms survive in the gut only in the very earliest stages of infection and that mild types of dysentery organism may cause progressive ulceration of the gut and

that cultures in typical cases themselves are most difficult unless they be undertaken sufficiently early before foecal organisms over grow the dysentery group. Thus Manson Bahr and Greig have pointed out instances of Bacillary dysentery of a mild type resulting in progressive ulceration of the bowel in which the specific dysentery bacilli play but the initial role. Probably the acute infection causes the destruction of the mucosa and the other organisms set up a chronic catarrh. Ledingham has pointed out on bacteriological evidence that the term colitis has been wrongly applied to conditions the dysenteric nature of which was not far to seek, and he brings forward strong evidence to prove the importance of *B. dysenteriae* in the aetiology of primary simple diarrhoeas. He calls the condition a simple enteritis of Flexner origin and also adds that in essence these flexner diarrhoeas are abortive dysenteries.

When we turn to *Entamoeba histolytica* infections Wenyon and O'Connor have pointed out that this infection is very often latent and that many have mild attacks of diarrhoea and only some have definite dysentery. This is borne out by Ledingham's observations on cases from 1922-24, which point to the fact that *E. Histolytica* is more often found in cases of diarrhoea than in dysentery.

It might be argued that in the absence of the demonstration of the infecting agents we have no real evidence as to the dysenteric nature of these diarrhoeas but my contention that is the morphological appearances of the lesions are characteristic and show striking resemblances to dysenteric lesions either caused by the dysentery bacilli or the *Entamoeba histolytica*. Bacteriological evidence of this as I have pointed out is extremely difficult if not impracticable.

SUMMARY.

1. The different types of dysenteric lesions are described from a study of 800 autopsies.
2. It is put forward that in about 60% of fatal cases of chronic "colitis" or "chronic diarrhoea" of inde-

finite origin show morphologically strong resemblance to lesions of chronic dysentery.

3. Some cases show an atrophied state of all the coats of the large and small intestine the exact cause of which has not been determined.

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Psychology and Medicine

BY T. C. THOMAS, IV YEAR.

No other science is more connected with medicine than Psychology. It might sound strange when we say that psychotherapy was the oldest form of medical treatment. If we look back into ancient medicine we find disease regarded as the work of demons or of magic and corresponding treatment by the aid of incantations, charms and excursions adopted. Rivers classifies the causation of disease under (1) human agency (magic), (2) supernatural agency, (3) natural causes.

Preventive medicine, etiology and therapy are built almost exclusively on magical and religious conceptions. The Greeks were more philosophic in their approach to medicine. They introduced the doctrine of the four elements—earth, air, fire and water. Later they postulated the theory of the essential humors which our Hakims and Vidyans share to a certain extent, *viz.*, blood, phlegm, yellow bile and black bile. We see genuine psychology in such writers as De-mon-divelle of the 13th century: "Keep your patients' spirits by music of viols and ten-stringed psaltry or by forged letters describing the death of his enemies or by telling him that he has been elected to the bishopric if a churchman".

The notion that magnetism was a force capable of influencing the human body and of being used for curative purposes is found far back in the history of medicine. The personal power of healing is also a doctrine of antiquity. Mesmer believed in the influence of planets on the human body which was believed to be due to the universal magnetic fluid. He opened a clinic and treated by "animal magnetism". The patients were seated round a tub in baquet containing various chemicals and protruding from it are iron rods which were applied to the portion of the body affected. The room was darkened, music was provided and mesmer appeared in a lilac robe, passing

from one patient to the other touching them with his hands or with the wand. Whatever we may think no doubt results were produced. Later workers found magnetic phenomena and curative results were best induced in neuropathic subjects.

Later on Braid and others showed that the power of magnets and metals were due to the suggestive effect of the ideas aroused in the patient's mind by the operators, words and actions. Charcot and later Bernhiem took up the work and showed that suggestion can be produced without hypnosis and this has produced a differentiation of methods into hypnotic and non-hypnotic, the latter comprising of frank verbal suggestion, with or without the use of drugs, electricity, etc., not for specific purposes but as a means of introducing suggestion into the patient's mind.

Psychological medicine is used with advantage in neuropathic cases. But there are other instances also when this was said to be used. Dr. Cotes gives a series of cases where major operations were performed under hypnosis and the patient thus escaping the evil effects of chloroform but these may be considered as gallant deeds but the main use to which it is put is in the cure of mental diseases though other branches also are equally dependant on it in preparing the patient to receive the medicine and produce the required result. Psychology was built as it were on the study of these mental diseases. The best known disease perhaps is Hysteria which shall be our main theme in the following few pages not to speak of Dementia Precox and other psycho-neurosis.

Hipocrates thought it was due to the wanderings of the uterus about the body in search of humidity and the pressures exerted upon the organs explained the various symptoms as coma and lethargy to pressure on the vessels going to the head, suffocation due to pressure on the bronchial tubes and so on. This theory was exploded before it could gain ground by others who looked into the anatomy too.

In hysteria according to one view we have a case of dissociation of certain parts of the mind. Certain powers and

certain memories are lost to the main consciousness but still exist somewhere or other in some form or other and can be restored under appropriate conditions. The anaesthesias of hysteria are very peculiar. They more or less correspond to the ordinary (or layman's) concepts of nerve distribution than to anatomical. The so-called glove anaesthesia is a good example. Janet says in hysteria there is a weakness in mental synthesis and well illustrates it by an example. An old lady goes a shopping and return laden with parcels, some of them fall to the ground and when she stoops to pick them others fall. According to him this is what happens in hysteria. There up the is a lack of synthesis of mental elements which make personality and in trying to recoupe it is deranged again.

Freud is famous in his sexual theory that in earlier days of childhood the sex energy of the child is directed towards the parent of the opposite sex with a repulsion or hostility for the parent of the same sex and later this is repressed. The hysterics suffer from a repression of perverted sexuality. When the repressed libido in hysterics fails to find an outlet in Physical symptoms—the paralysis, contractions, anaesthesias, etc., it appears in the conscious state as an anxiety state. Thus Freud says the *earlier* inauguration and memories are directly responsible for hysterical symptoms while Jung says it is due to a failure, of adaptation to the environment at *the present movement* which causes regression and this calls back earlier memories and the hysterical symptoms are produced. The hysterical state represents a compromise between a sub-conscious complex and its regression. The hysterics can be classed according to the degree of mental dissociation that has occurred. Thus the psychasthenic where there is a weakness in synthesis. The neurasthenic where the dissociation is on a higher level the patient worries about something or other and spoils his health and this acting as a vicious circle. The compulsion neurotic, where the patient is obsessed with the idea, that he must do certain things.

The multiple personality where there is a complete disintegration. There may be an alternation or doubling of personality we hear of so many queer cases which are often said to be due to some spirits or evil influence. The patient is usually an young girl in her teens posing herself as some other personality and giving out many wonderful things.

To understand this we must have an idea of what they call the sub-conscious. Ideas are said to exist in independence of the main personality and along with it and this was called sub-conscious or better co-conscious. This is below the threshold of consciousness and activities of every day life. This sub-liminal self is capable of development to a great extent. This sub-liminal and supra-liminal (every day life) is separated by a semi-permiable "psycho-physical diaphragm" through which ideas and mental processes pass in either direction. In anaesthesias and paralysis the sensation fall from their supra-liminal to the sub-liminal and in other cases then may be an uprush from the sub-liminal which are thought to be super-normal actions of soul. Through the sub-liminal we have close association with spirits.

Freud's theory of dreams is another great factor which has lent much in the study of hysteria. According to him every dream is a wish fulfilment which is sometimes disguised and varied. There is an endo-psychic censor which is the repressing factor in waking consciousness but in sleep this slackens a bit and the disguised wish succeeds in slipping past the censor. But in anxiety dreams the censor is completely overpowered. This is the same factor which happens in hysteria. The wishes and emotions are disguised and the censor is in a weak state and there is a rush outside.

Psychology has advanced a great deal after the war. Certain derangements which they called shell shock or War Psychoneurosis have been carefully studied. Of course this is another form of hysteria. Dr. Brown gives a few cases with full notes one of which is given below.

Case I. **Total loss of memory.**

No. 2993. Private J. M., 6th Brigade Machine Gun Coy., A. I. F., aged 32, single, seven months exposed to shell fire. Previous occupation surveyor.

Admitted to No. 22 C. C. S. on the evening of 5-12-1916 with complete loss of memory unidentified. Could speak but could not give any account.

Physical examination.—Plantar reflexes, flexor, Knee Jerk +; arm reflex; biceps and triceps +; spasticity of limbs and slight tendency to katatonia. Hypertonicity of leg muscles; eyes normal. Tongue clean; pulse steady 68. Heart normal. Abdominal reflexes normal. No ankle clonus; patient complains of acute pain at the back of head.

5th Hypnotized him. Got the following—was pushed out of a car by somebody who was drunk. Fell on back of head and was stunned. He gave address, name and his father's name.

6th again hypnotized him and he gave the description at different periods of life. 11th again hypnotized him he was asked to live through his experience in trench, he did so; gave a vivid description.

16th gave some suggestions under hypnosis and some post-hypnotic suggestion. 19th discharged.

Till now we have said nothing about the therapeutic portion. The main factors are:—

Reassociation and Psychocatharsis.—This is done under light hypnosis rest explanation and rational persuasion are the most important but in ordinary cases waking suggestions persuasion and mental analysis are sufficient. Reassociation is slowly filling up the gaps in the memory.

Psycho-catharsis.—Here the patient is asked to live through the incident which has upset him and thus work off his emotions.

Autognosis or self knowledge. In practice it is long talks between the patient and the physician. He is led back in his memories and asked to describe minutely all his feelings and thoughts and encouraged to discuss it especially the emotional

memories where he failed to adapt himself. This is distinct from psycho-analysis.

Suggestion is the most important part in the treatment here the personal influence of the physician comes into play. The patient must feel an interest in the physician either of fear or confidence. There is always an emotional element in this.

Hypnotic suggestion may be used but some cases may not be amenable to this treatment.

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"Musings of a Surgical Dresser".

II. DRESSING IN THE WARDS.

(Continued from page 210.)

We see how anxious the passed out students are to get the post graduate house surgeon's training. There is an unquenchable thirst among the unemployed medical men at large, to be allowed to work once more in the hospital, lest they forget what they have already acquired. Some of our Colleagues comfort themselves saying that a year's training as House Surgeon will make up all the wants. How delusive is the future remains only to be experienced, if we are not going to accept as truth what many a House Surgeon has been describing about his career to us. When time and opportunity is within our grasp let us not waver in our duty. "He that hath set his hands to the plough shall not turn back" we may have to repent and long for the chances later on, when **duty** calls us away from such a good institution as our Hospital. Besides the opportunity, we are now working entirely under a different atmosphere, the doctors who actually come in contact with us every day are so sympathetic, kind and willing that the ardent student need not be afraid to work.

The student who had a preliminary course in medicine will clearly understand and appreciate most of the post operative complications. They are generally due to acute or chronic pathological conditions of heart, lungs or kidneys; diseases of thyroid, blood or blood vessels; acidosis, jaundice, diabetes and a host of other things. One should never forget the fact that the patients who require *surgical* treatment may not be otherwise healthy. The *Malarial patient* may get an occasion to break his tibia and get admitted in the Surgical wards. It may be a sure case of piles, but it does not exclude the person from getting an attack of dysentery as well to aggravate the matter. Friends err when they say that there is not much danger of infection from the surgical wards. The usual precautions to ward off infection which we preach, we should also practice with absolute faithfulness in all its details wherever we may work.

The work of a surgical dresser is not a monotonous job as it may seem to be. It gives us the rare opportunity to study the effects

of various remedies that are placed at our disposal and to adopt the proper methods of treatment for the various cases which we are privileged to follow very closely for many days together in the wards. Thus the student becomes very familiar with the art of dressing. Before actually beginning to work in the wards it is essential for the student to learn certain general things. He is supposed to know the names of the wards where such and such a ward is and the shortest way to it. The hospital admission days whether surgical or medical, he must be in a position to say on which days and in which wards the patients are likely to be admitted. In the surgical wards he must study the contents of the dressing trolley and ask the seniors to explain how they are applied and when they are to be used and should watch the seniors dressing and bandaging the cases. The correct method of bandaging may be acquired mostly by practice aided by a light study from books. If anything you observe is not understood, ask the seniors to give you a clear explanation. Do not be satisfied with vague incomprehensible explanations which the seniors try to give when they themselves are not sure of the correct answer to your question. Use the House Surgeon and Assistant Surgeon to pick up knowledge, "with an inquiring mind work as hard as you can in the wards for it contains the building material for your knowledge and future success".

The student should enter the wards punctually at an hour conveniently early. He should then pay a hurried visit to all the cases that are entrusted to him. This is very necessary, for the patients are ever anxious to meet him, sometimes they have long reports to narrate. They may ask him to help them by doing slight adjustments in posture or the student may have to go here and there or do certain things which he will not be able to do, if he begins to dress the cases.

If any of the student's cases requires any advice or special instructions from the seniors, he must go at once to them and get all the help possible. He must study the daily chart of the patient, look for any special report by the night nurse. If urine is to be examined he should ascertain from the nurse in-charge, whether the urine of the particular patient is sent to the laboratory. If not he must see that the urine is taken by the boy and kept with the label in time. Otherwise he will have to go unnecessarily to the laboratory to find that nothing is kept. If blood, motion or urine is to be examined, the laboratory work must be finished first and the result of the examination must be written on the case sheets of the patients immediately. If any report

merits the attention of the superior Officers he should *never* fail to intimate the same to them.

Preparation for dressing.—Take off your coat and hang it in a *safe* place. Tuck up the sleeves of your shirt and fix them up *securely* at the arm's level. Hands and arms must be thoroughly and effectively purified they are scrubbed with soap and brush in a running stream of tap water. See if your nails are long. If so, they must be cut but not too close. Clean again for the second time in a similar manner and then bathe your forearms completely in the lotion provided in the wards. Ask the nurse to give you a sterilized overcoat. This must be put on and tied by means of the fasteners provided. The sterilized dressing coat may hide the costly shirt and its matching tie but it will protect them from getting soiled and also prevent dirt falling from them into the wounds of the patients. Our clothes however cleanly kept are supposed to gather dust and dirt on the way. If you have a wound in your finger you must bear in mind that infection from the patient may be virulent. So proper sterile rubber gloves or finger stalls must be worn. These of course, would have been sterilised and kept ready. By the time you go through all these ceremonies and get ready, the nurse would have brought all the necessary things to the bed of the patient. Start dressing the clean cases first and then go to the septic cases.

The Dressings.—Ask the nurse to remove the bandage. The dressings put on the wounds must be removed with the dressing forceps by the dresser. The appearances of the wound and the soiled dressings may be nothing to a casual onlooker but to us every bit of it will present nothing short of a detailed true report of the previous days labour. **The pus**, like a tongue-tied creature will explain the actual condition of the wound by its characteristic appearances and variations. "Thick creamy pus of a yellowish colour is generally considered as a sign that the patient is practically out of danger of 'blood poisoning' and the pus is termed "Laudable or commendable pus". The Condensed milk looks very nearly the same as laudable or normal pus in its colour, and consistency. The healthy pus has no smell practically. The pus noticed discharging from inflamed mucous membranes, is sticky or glairy due to the presence of mucour. The pus is called *Muco-pus*. If the pus is thin and tinged with blood it is called *sanious pus*. *Sero-pus* is thin and more liquid due to an excess of serum." Sometimes the pus is thick, coloured, and emits foul odours. These appearances show

that the wound is not properly drained. In certain cases where the wound holds a communication with a deeper cavity or in cases of gangrene, the pus on close examination may manifest a number of small gas bubbles. This shall be to you a danger signal for a careful attention to the case in future. This condition should be reported to the ward Assistant.

The *dressings* put on a previous occasion if found sticking in certain parts they must be will moistened with the Eusol lotion squeezed out from a *wiper* before being removed. With the Eusol lotion wash out the excess of pus and debris from the wound. The wound must be gently wiped with a fresh surface of the *wiper* each time. The surface of the wound must be examined. The healthy granulating surface will present a velvety appearance and bright pink colour. It is composed of large number of newly formed blood vessels embedded in a mass of leucocytes and actively proliferating tissue cells. *The skin* around the wound must be cleaned carefully. The direction of wiping must always be away from the centre of the wound. Otherwise dirt may enter from the surrounding skin into the open wound. Next with a delicate touch the wound is gently pressed to squeeze out the pus from the tissues. Gentleness is the essential merit in cleaning the wound. Rough handling devitalises the tissues and renders it more liable to succumb to the toxic action of the germs. While squeezing out the pus, the wiper must be directed from the limits of the wound towards its centre. The discharge must be mopped away each time with a fresh surface of a wiper. If the wound requires Hydrogen Peroxide to complete your cleaning, *i.e.*, in cases where there are many crevices in the wound, which you feel are not efficiently cleaned, you apply a liberal amount of fresh Hydrogen peroxide with a cotton swab on a kutchie. Wait for a few seconds to allow the hydrogen peroxide to act. Next wash away the froth. The wound will look fresh and clean. Then gently dab with a clean and squeezed out dry wiper. Bleeding from the surface of granulating wound should not be induced. But blood serum possesses a high curative value.

Sloughs.—The central necrotic mass noticed in the structure of an abscess or a suppurating wound is the slough. The slough is pearly white in colour and may be adherent to the inflamed tissues subjacent to it. In a small abscess it is called 'core of the abscess.' It may be thin or thick. It may be cylindrical or sheet like or it may be in bits like pieces of white membrane scattered here and

there. In all cases, to ensure quick healing, the ulcer must be freed off the slough. The sloughs are easily removed in the later stages than in the early stages. The bacteria find an abundance of suitable pabulum and usually multiply at an alarming rate in the sloughs spreading widely into the damaged tissues and causing widespread inflammation. Hence the sloughs must be removed for they provide culture material of Bacterial activity.

Appearances of the wound.—The essential knowledge of the differences between a syphilitic ulcer, herpes, scabies, leprosy and accidental wounds must be acquired by practice. It is difficult to convey an exact idea by mere description. A healing ulcer, a callous ulcer and inflamed ulcer can be appreciated by careful attention to individual cases.

When to plug a wound and when to put in a drainage tube?—This question still presents at times even to the House Surgeons, a difficult problem. To make matters worse, one sees drainage tubes fixed in some cases quite contrary to laws of gravitation. One may be puzzled to find out the object of drainage tube sticking up from the pelvis to a higher level. In certain cases we plug into the wound antiseptic gauze and in many we do not plug anything into the wound. J. S. Horsely is of opinion that any foreign material introduced into a wound causes a reversal of the lymph circulation in the tissue, which is due to the response to the irritation. There is, in the damaged tissue of an abscess or carbuncle, high tension due to blocked up lymph. The exudating serum therefore is stale and has no active Bactericidal properties. It gets partly autolysed and the Trypsinized serum is an excellent pabulum for bacteria. In such cases where the absorption of injurious material from a wound has to be prevented a sterilized antiseptic gauze is put into the wound as a drain. Thus we note the necessity of introducing fairly good amounts of antiseptic dressings within the spaces of big abscesses and carbuncles. Thus *plugging* in to the wound tends to extrude some of the toxic substances over the irritating foreign body. There is an increased flow of lymph in an effort to expel the foreign body. Thus it washes out any toxic material stagnant on the way. *Plugging* a wound is beneficial when the focus is deep seated and when there is a greater chance of the toxic material getting absorbed. The healing of such wounds must be encouraged from bottom upwards. If no plugging is done the superficial contiguous surfaces of the wound granulate and occlude the

passage for the pus. This is what happens in heel abscesses in the casualty department. Patients may not allow the plugging but it has to be done to avoid a second operation. The infective focus will remain concealed and cause a series of similar troubles. The pus may be absorbed and the bad effects may manifest once again.

Again in all wounds where fat bounds, it is good to put in a sterilized antiseptic gauze packing. This prevents accumulation and absorption of liquified fat produced by bacterial action. This liquified fat mixed with the toxins of the bacteria interferes with the healing of the wound for it forms a good culture medium for the micro-organisms.

There is no danger in light packing of a wound in every case where doubt exists as to the efficient removal of infective material. You may be erring on the safe side and it is better.

Drainage is indicated where it is impossible to avoid the existence of an actual or potential cavity in the deeper layers of the diseased tissue. For blood, serum, or pus may tend to accumulate within the spaces. The drainage tube must be placed in such a way that gravity assists in draining away the discharges from the depth into the dressings.

Rolled rubber tissue, a rubber tubing with side openings or a cigarette drain (a slit rubber tubing with gauze-wick inside) properly sterilised are used for this purpose. In most of the cases where this principle is adopted to drain off the infective material from the tissues an abundant amount of pus will be exuded into the dressings. But there is no need for alarm. The patients get a little frightened when they see it, but the *Dresser* feels satisfied to note the proper response to his rational dressing of the wound, as shown by temperature coming down to normal and patient improving.

We should *not plug* any wound which is healing by first intention, when a wound is rapidly healing from the bottom upwards, when doubt exists about tubercular infection of the wound or the neighbourhood. In tubercular abscesses the danger of pushing in any secondary infection is great hence plugging is not indicated.

Persistent discharge, from a wound or scar of an old wound, may mean either the opening is too small to let out the discharge completely or some deep seated undrained loculus is present. This is also present in cases of mixed infection of the wound or in cases where the part is not kept at rest and in cases where the general condition of the patient is very low. The passage through which the purulent or serous dis-

charge is passed out is known as the sinus. It is a narrow track lined with granulations and closed at one end.

Fistula is an abnormal communication congenital or acquired between two cavities or a cavity and the exterior. It is a difficult task to secure the healing of a sinus or fistula. Any amount of dressings applied to the external orifice of the fistula or sinus will never stop the persistent discharge until the various primary causes for their non-closure are effectively attended to. But if the orifice presents prominent fungating granulations we can say that it may contain a foreign body lodged within.

"When a pyogenic abscess, acute or chronic, (tubercular abscess should not be incised for fear of secondary infection) is opened and treated antiseptically the formation of pus quickly ceases. If the wound does not heal for months and the discharge is merely serous the underlying cause, constitutional or otherwise should be searched for. Should a tuberculous abscess be infected with pyococci or mixed infection occur in a pyococcal abscess, the discharge of pus continues or reappears and fever to a varying degree develops.

Long continued suppuration is always an evidence of persistent infection."

Post operative infected wound.—Generally there is no need for any special treatment for the aseptically finished operations. The site of incision must be examined daily and Alcohol picric must be liberally painted over the whole length of the incision. The direction of smear must always be away from the middle of the wound. But in some cases due to so many causes the operative wound does not heal by the 1st intention, (i.e., healing within a short period without pus formation and loss of tissue). In such cases one must be on the look out for the early detection of what are known as stitch abscesses. These are collections of pus in the inflamed site due to the stitches damaging the tissues. "It may be due to faulty technique" or due to the stitches strangling the tissues within its grasp as a result of increased pressure within the part gets inflamed.

Can I remove the stitches? This is a question which the dresser asks. The seniors must respect the sincerity of the questioner and should answer only after looking at the stitches in question. When all is well there is no harm in allowing the stitches to remain longer than a week for the stitches are put to aid healing by first intention, in minimum time, to preserve the form and to reduce the size of the scar. But when

there is a tendency for the formation of abscesses the stitches must be removed early. In one instance, the house surgeon went to ascertain the correct answer from the senior nurse. She of course said "Oh the stitches ought to be removed only after the eighth day". The house surgeon gave that as the answer to the eagerly waiting student. But they were wrong. The stitch abscesses had already developed considerably, one or two stitches had given way due to high tension. So the exact time which ought to be allowed for the stitches to remain varies according to the nature of progress of the wound.

In all cases of post-operative infection of the wound, the stitches must be removed to give relief to tension and fomentation must be applied to encourage local reparative activity. The wound is opened wide enough to give free exit to the pus. Undue interference as by scraping, rubbing, squeezing or syringing is to be deprecated. Sloughs if present must be cut away. Wound is sterilized as far as possible. Light packing of the wound assists the lymph to flow back into the dressing. In very bad cases where there is a lot of offensive discharge Carrel-Dakins treatment is to be adopted, i.e., continuous irrigation.

The bruised or damaged parts must be removed and a Secondary suture may be necessary, to keep the opposing parts together. The wound may be treated just as any ulcer, with antiseptic dressings.

Fomentations.—In every case where there is acute inflammation in the tissues application of moist heat has been found to be good. The heat is of great use in the treatment on account of the active arterial hyperæmia it produces whereby the part to which it is applied is bathed with fresh blood containing defensive agents such as leucocytes opsonins, anti-toxins and other anti-bodies by means of which the activities of harmful bacteria and their toxins are neutralised. Moreover when applied to a part which is infiltrated and brawny it assists in the restoration of a healthy circulation by softening and relaxing the tissues." The fomentations must be aseptic if not antiseptic. It is the duty of the nurse or anybody who is assisting the dresser, to prepare the fomentation. The student with his sterilised hands shall not touch the dirty kettle or the utensils when he has not finished dressing the cases. He should maintain his hands clean till all the dressings are finished. There are instances in which the seniors without much thinking order that the fomentations must be prepared by the students themselves who are actually engaged in the dressing. The fomentation must be as hot as

one could bear. There is great danger in applying very hot fomentation to the inflamed part. The hot lint used for fomentation must never be placed direct on a raw wound. The wound must be covered with antiseptic dressings over which the fomentation has to be applied. Spread a bigger sized oil paper over the fomentation without much loss of time put enough cotton pads to prevent loss of heat through dissipation.

Tincture Iodine.—Nearly every patient desires that iodine may be painted for his case. The poor ward boys in their eagerness to earn a little to meet the needs of their humble life, ignorantly apply this curious remedy day after day liberally on all their paying patients, little suspecting that they thereby cause dermatitis. To have an efficient action the part must be as dry as possible. As iodine is a powerful irritant it should be applied cautiously in patches which should resemble the chess board if the area is extensive. The first day you paint squares corresponding to the black ones, the next day the squares corresponding to the white ones. Thus the dermatitis may be avoided. For tubercular and syphilitic affections of glands iodine painting is good and in cases where you desire to increase the tissue activity to cause a healing, iodine may be painted all around the ulcer. This drug may be applied over wounds which are suspicious or dirty to prevent the progress of infection. As a rule put no wet dressing over the part where iodine is applied.

Iodoform.—The smell has a good effect on the minds of the patients. They feel that the proper medicine in a hospital for wounds, is Iodoform. This drug therefore works psychologically wonders. Iodoform masks or deodorizes the foul smell. Works in the same way as iodine in increasing the tissue powers of repair, while coming in contact with the tissues.

Sealing of a wound or healing under a sterile scab.—Some cases which by careful observation may be found to be healthy but healing very slowly, for which the 'seal' may be put on. (This method is suggested for cases treated elsewhere). In the usual way clean the wound and dry it by blotting the wound with an absorbent swab. Then dust over the wound, finely powdered Bismuth Carbonate or Magnesium carbonate. When the powder has been applied liberally over the entire wound, a dry sterilized gauze must be taken and similarly dusted with the same powder, and the gauze must be carefully placed over the wound and kept in position by gently pressing it. With plenty of cotton pads spread over it, the part may be bandaged. The limb must

be kept at a higher level. Least movement must be given to the part next day and the bandage must be removed leaving the gauze sticking to the wound. With the educated touch of the finger see whether there is lot of moisture due to pus. If so remove the gauze and clean the part again and put on the same dressings as on the previous day. If the gauze is fairly dry put fresh Bismuth or Magnesium carbonate over the gauze. This method of providing a crust for any wound must be applied to all wounds where the formation of crust will help the healing of the wound. This 'seal' must be examined every day and it must be kept disinfected. Otherwise micro-organisms will find the exudate, a favourable medium for growth and mischief and the seal may begin to smell bad. The following recipe for the disinfectant lotion has been tried and found effective.

Rx.

Mercuric Bichloride Gr. 3
Alcohol Absolute 1 oz.
Aqua distil. ad 2 oz.
Fiat Lotio. For external use only

(G.M.T.)

The lotion is sparingly applied over the seal. If too much is put on at one time the 'seal' may come off. This ensures a thorough disinfection and there will be no harm in allowing the seal to remain for a long period provided that there is no adverse sign. The disinfection of the seal need not be done daily after the first week. The 'seal' serves as a sterile scab over the wound. Unless there is a necessity never yield to curiosity or to itching sensation which might induce one to break the seal only to find the ulcer not completely healed. The 'seal' falls off when the wound has healed completely.

In conclusion, let my friend trust not too much his own judgment especially when he is treating difficult cases but seek the advice of physicians and surgeons whose long experience enables them to instruct him further in preventing accidents and to encourage him to go on in the work or forewarn him of the dangers. After thus protecting himself from the censure of rashness let him proceed boldly and let his sincerity in his work be his warrant to hope for God's blessings on his endeavours. "The world may sound no trumpets, ring no bells but a child's kiss set on thy sighing lips shall make thee glad. A poor man

served by thee shall make thee rich. A sickman helped by thee shall make thee strong—thou shalt be served thyself in every sense of service which thou renderest" Mrs. Browning.

I cannot but express my thanks to the kind and instructive supervision of Dr. R. V. Rajam during my work in the wards which was a source of real encouragement and pleasure during the entire period of my working as a surgical dresser in his wards.

G. M. THAMBIDURAI.

Fourth Year.

Case Reports

A Case of Cerebral Diplegia.

Name.—S. Age—6 years. Residence.—Mangalore.

Caste and sex—Hindu-female. Admitted.—28-12-1929.

Complaint.—Inability to talk or use the limbs—congenital.

Family History.—Nil of importance.

Previous History.—The child was born at full term after a normal labour. She has had no disease all these years, except an attack of dysentery which came on about the age of 2½ years and lasted for a few days.

History of Present illness.—The child has never walked or talked till now. General development has been normal. Teeth were cut at the usual time; she is able to understand what is said and obeys alright. Movement is accomplished by dragging of body with hands twisted. Left hand is more readily used than the right in all actions. She is able to stand when the trunk is supported, but reels away when the support is withdrawn. She walks with great difficulty when helped but soon legs cross-over—(cross-legged progression.) At first she was unable to sit up but now she can be made to sit with some help but the slightest disturbance or shake makes her fall. When the child sits up, there is too great a curvature of the spine. Her intelligence appears below normal. She smiles and cries normally and expresses her desire to answer the calls of nature by means of gestures.

Physical signs and condition on admission.—Patient is a well-nourished child, not anæmic, pupils reacting to light but slightly dilated. No nystagmus.

Cranial nerves—all normal.

Abdominal reflex—Brisk.

Knee jerks—exaggerated.

Ankle clonus, patellar clonus—absent.

No apparent wasting of muscles.

Rigidity present generally and in the wrist joints. The hands can be manipulated into the normal position but when let go, resume their usual position which is one of apparent wrist drop with hands twisted outwards. There is a slight lateral curvature of the spine in the lumbar region, the convexity directed to the left. No tilting of the pelvis to any side, no shortening of any limb. Plantar response—Flexor.

Circulatory System.—Apex beat visible and palpable in the V left space in the nipple line. Heart boundaries and sounds normal.

Respiratory System.—Breath sounds harsh. Otherwise nil abnormal.

*Investigations.**Radiograph Skull.*—Nil abnormal.*Wassermann reaction.*—Negative.*Blood.*—Nil abnormal*Urine.*—S. G. 1010.

Sugar	} Nil.
Albumen	

On 29-12-1929 the child vomited twice. Had a fit of convulsion and slight temperature and was a bit drowsy. She was put on :—

Rx

Bismuth oxycarb Gr. xxx.

Sodi bicarb Gr. xx.

Acid Tannic Gr. x.

Salol Gr. iii.

F. P. 6. one every 4th hour.

On 18-12-1929 ;—temp. was noticed rising. On examination, blood showed no parasite and the following effectively brought down the temp. to normal.

Rx

Soda salicylas Gr. x.

Sodabcarb Gr. x.

Lqr. am. acet. i dr.

Spts am arom ½ dr.

Syrup i dr.

Aqua ad. iii oz.

Oz. 1 T. D. S.

Treatment.—

1. Rx

Ext. Thyroid Gr. i.
B. d.s.

2. Rx

Liq. paraffin dr. ii.
at bedtime on alternate days.

3. Rx

mist Ol. morrhuae 1½ oz. ½ oz. T. D. S.

4. Educative exercises.

The child tries to answer simple questions.

I am grateful to Lt.-Col. G. E. Malcomson, M.D., M.R.C.P., I.M.S., for his kindness in having permitted me to report the case.

DR. MD. ABDUL HADI, L.M.S.,

H. P. M₁ WARDS, G. H.

In Lighter Vein

Patient.—(Sarcastically after being kept waiting) "I'm afraid I have almost forgotten what I came to see you about Doctor !."

Doctor.—(Brightly) Ah ! Loss of memory obviously.

Child.—(to a young man who has called) "Sister told me to entertain you until she comes down.

Youngman.—"Oh, did she ?"

Child.—Yes and I'm not to answer too many questions.

Friend.—"Doctor, why not wait and have tea with me ?"

Thambi.—Sorry I'm in a hurry to attend my patient's funeral.

Dentist.—(who was just saved from drowning in the Cooum River) Say friend I'm greatly indebted to you. Sorry I don't have my instruments with me now. Will you please call at any clinic I shall pull all your teeth and charge you nothing !

Frank.—You know when K... ..arrived home from the west he fell on his face and kissed the pavement of his house.

Friend.—"Emotion."

Frank.—"No Banana skin".

At a College tea party a lady student had a doctor on either side, one of whom remarked that they were well served since they had a duck between them. 'Yes' she broke in "and I am between two quacks".

Isaacs.—"Please have you any dry biscuits ?"

Grocer.—"Yes, as many as you want, I dare say."

Isaacs.—Have many of them? Are you sure they are quite dry ?"

Grocer.—"Quite dry." Isaacs walked towards the open door and shouted "Then why not give them a drink !"

Cricket Explained.

(First time seen witnessing a match).

Doctor.—Oh ! I came to see my boss playing otherwise I never go to witness these matches.

Lady Student.—Who are all playing?

Doctor.—Why don't you see the batsman on this side representing the house surgeon team? Then the other batsman must be one of the fourth year class team.

Lady Student.—Does it means only two play at a time? Who are all these men in the field.

Doctor.—Those are the stalwasts of the hospital staff.

Lady Student.—I never thought cricket is such an unfair game after all. Eleven against one!

Doctor.—No, the batsman by turns try and score goals while others are there only to pick the ball and to return it with minimum loss of time.

Lady Student.—Sometimes the man with the bat takes short runs.

Doctor.—So sorry you don't understand the game the principle of cricket is to hit and run!

Lady Student.—(one batsman has been bowled out). How's that the man is returning.

Doctor.—That means he is *bold*. He ought not to have allowed the ball to touch his wicket. It is a gross mistake so he is out.

Lady Student.—Well I don't see why they should send him out of the field for that everybody makes mistakes.

Dr.—Ever have any trouble with Dyspepsia?

Pt.—Only when I try to spell it.

Unilateral Nephritis.

Dr.—(Showing a patient with swelling in the right leg). Students note this case, this is a Typical case of unilateral nephritis where you get the characteristic swelling of the leg corresponding to the affected kidney.

G.—I have decided not to enter for the sack fight in future.

Sports Secretary.—But why?

G.—“Last time a somersault was forced on me, I could not avoid it and somebody from the ladies group remarked ‘what a fall was there.’”

D.—I say how is it they keep only female cows in the Hospital Milk Sheds?

Friend.—Ah! Better inform the Superintendent about it.

Pathology Howlers.

Bacilliary dysentery produces spores. There is occult blood in the stools in a case of Dysentery.

The “Soul force” in the causative of Cruentis.

The *causating* factors for enlarged spleen or Bunly spleen are:—

1. Gin drinkers wine.
2. Anthracosis.
3. Haemorrhagic small-pox causes enlarged spleen with elements of bone in it.
4. Malarial bodies found in the Kala Azar spleen causes an enlarged spleen.

Professor.—If the Students writes like these what does the examiners thinks?

Doctor.—You are looking much better.

Patient.—I expected, that's because I followed your directions on the bottle.

Doctor.—What were they?

Patient.—Keep in a cool places tightly corked.

Somebody remarked A Doctor is really a strong willed man. “But how?” I asked. “Because he can ask the *women* to close their mouths, or to open and to get away with it” was the reply.

Doctor.—(Standing before an interesting case) “Can I have a look at it please?”

Nurse.—(who has just bandaged the patient for the 4th time for she has been obliging others many times) Sorry I cannot be undressing once for him and once for you.

Student.—Why did not *Macaulay* write about Johnson.

Tutor.—Perhaps he forgot about it.

Student.—Why he lived in 17th century and Johnson in 18th century.

Tutor.—Perhaps that might be another reason.

Editorial.

It is very regrettable that the General Medical Council has come to a hasty decision in refusing to grant recognition to Indian Medical degrees. The decision is apparently based on the refusal of the Indian Legislature to appoint a permanent commissioner of Medical qualifications as recommended by the General Medical Council. It will be remembered that the Council sent a commission consisting of a well known dermatologist and an army doctor to examine and report on the various subjects of the medical curriculum in India. Probably their report has something to do with this decision though we fail to see how the council has thought it fit to base its refusal on such general lines without bringing forward specific reasons where each University is concerned.

The reasons put forward for this decision are surprisingly vague. "The growth of Indian Nationalism" and its "suspicion of foreign ideals" certainly cannot possibly stand in the way of scientific learning especially on a subject like medicine. Another reason put forward, *i.e.*, "the differing languages and differing civilizations" have so far not stood in the way of other countries attaining such high standard of proficiency in the study and practice of the healing art.

Deficient instruction in obstetrics is put forward in spite of the fact that Comyns Berkely has pointed out that the standards insisted on by the General Medical Council do not obtain even in London. No unprejudiced observer who has studied the question will be prepared to admit that the training given in obstetrics in Madras at any rate is inferior to that in British Universities.

The unfortunate part of the whole decision as it appears to us, is that it is bound to create bad blood. Though we

are not prepared to admit that the standard of training here is in any way inferior, it is obvious that it can certainly be improved upon. It may be that the course of instruction in obstetrics could be modified after that in Holland where 4 years of practical training is insisted. One thing however is evident from the decision of the General Medical Council and that is, we should endeavour to raise our standard much higher than that laid down by the General Medical Council for British Universities and for that Madras should take the lead. We should further be in a position to give post-graduate training to students from other Universities and the time is not far distant when we could afford to do so in tropical medicine and in our new pharmacopœa. We would thus make it impossible for any medical council or University to dare question our standards of teaching.

Another result of the General Medical Council's unfortunate "faux pas" would be that Indian students would not care to proceed for post-graduate instruction to British Universities and would turn to other foreign universities. The result would be that the value of the British qualification in medicine in India would tend to go down and popular opinion would turn against the doctor with such a degree.

If the General Medical Council is wise it would bring forward constructive proposals if its desire is to help, and not to create impossible situations which would cause further trouble and disharmony among the medical profession in India besides making its own position untenable. In our opinion the formation of an All India Medical Council would go far to remove the anomalies of the present system besides bringing greater co-ordination among the Indian Universities. The question of reciprocity with the General Medical Council would only be then a matter of detail.

Among topics of local importance the appointment of Col. C. A. Sprawson, I.M.S., as Surgeon-General to the

Government of Madras is a matter of great interest to us all. His long association with the Medical Faculty in Lucknow both as Professor of Physiology and later as Professor of Medicine and Principal of the College is of great importance since he must be quite familiar with the problems of Medical Education.

We extend our hearty congratulations to Col. Megaw, I.M.S., in his appointment as the Director-General of the Medical Services and wish him every success in his new office.

We are very glad to hear that the term of service of Dr. Sundara Reddi, Professor of Anatomy has been extended for a year. It is but right that age and service considerations should not arise in the case of a distinguished anatomist with such a wealth of experience as a teacher. Anatomy is a subject which could not be properly taught except by an anatomist and that only one with considerable experience, apart from the question of purely academic attainments.

It is indeed gratifying to observe that the Medical College still holds a premier place in athletics. We have this year wrested the Inter-Collegiate trophy from the Engineering College in the Inter-collegiate sports. We offer our hearty congratulations to the students we took part and to their enterprising Secretary.

The question of a "Hostel" for the Medical College has been engaging our attention for many years. It is regrettable that Medical students still continue to live a sort of nomadic life migrating to different localities in Madras for want of a suitable hostel. Let us hope that the New Surgeon-General will take the initiative in the matter and see that the "hostel" is an accomplished fact.

Before concluding we wish to draw the attention of our readers to an article entitled "Obscure fevers in child-hood" in our last issue. This is only an abstract of a clinical lecture given to the students by Col. Malcolmson. We are sorry that a foot note to that effect has been omitted.

Madras Medical College Athletic Club.

Our Athletic Club has been passing through a very busy season in the varied features of sports during the current year. It is not for me to comment on or criticise the various endeavours of our teams and our athletes. Anyhow, it is quite obvious to us that all of them did their best in the most sportsmanly fashion. On the whole, we have been having very interesting and exciting matches and contests. I shall leave it to others to have their say further in the matter. I take this opportunity in offering my sincerest thanks to the members of the athletic committee and all my fellow students without whose help I would not have been able to carry out my work as the sports secretary. Before I conclude I must specially thank the following for presenting cups for the chief events of the college sports. We hope we shall be able to acquit ourselves more and more creditably in the future in athletics trusting that their co-operation and interest will be sustained as warm as ever.

Lt.-Col. E. W. C. Bradfield	...	120 yds Hurdles.
" J. M. Skinner	...	Open Mile.
" R. E. Wright	...	Tennis Doubles (Mixed).
Mrs. Pandalai	...	Championship Cup.
Maj. Pandalai	...	Long Jump.
Dr. Rao Bahadur Lakshmana-	...	Mile Cup for College.
swamy Mudaliar	...	100 yds. Dash.
" A. S. Mannadi Nayar	...	Half Mile.
" B. T. Krishnan	...	Ladies' Singles Tennis.
" A. Srinivasulu Naidu	...	Throwing the Cricket Ball.
" Rao Bahadur M. Kesava Pai	...	220 yds Dash.
" W. Happer	...	Putting the Shot.
" S. Ramakrishnan	...	High Jump.
" P. V. Cherian	...	440 yds Run.
Capt. T. W. Barnard	...	
Dr. Khan Bahadur Muhd. Azizullah	...	Pole Vault.
Sahib Bahadur	...	
" Rao Sahib T. Sundara Reddy	...	Tennis Singles (Men's).
" Garu	...	Tennis Doubles (Men's).
" J. B. Shenoy	...	

V. O. ZACHARIAH,
Hon. Secretary.

A "PASSIVE OBSERVER" ON MEDICAL COLLEGE ATHLETICS.

The results in athletics during the past year, though not very satisfactory, are full of encouragements for better efforts in the future. The college has taken lively interest. Not only the leading athletes but all the students, including the women students, and some members of the staff by their co-operation augmented the interest in sports.

During our strenuous course in the college keen interest and participation in sports alone can keep us afloat from sinking into despair, dyspepsia and death. Excluding the occasional social functions and the U. T. C., athletics is the main factor that brings the different classes together. It is in athletic fields that we make new friendships, deepen old friendships and sink our differences and play and rejoice in healthy exuberance.

Let us hope that in the future we shall take more and more interest in sports. A little more practice and interest will make us second to none. With the material that we have we ought to be able to gain the first rank in everything.

INTER-COLLEGIATE TOURNAMENTS.

Unfortunately we have been unsuccessful in all the Inter-collegiate Tournaments. We had high hopes, and sure we did display splendid games, but we failed. We have learnt a lesson this year that good material alone brings no success. We have the best of material in hockey, foot-ball and cricket. I am afraid we are suffering from hydrocephalus (swollen head)! Success is possible only out of harmony and team work. Only by regular training we can achieve this. We have to work in unison. Lack of time is an excuse without foundation. The captains and players ought to come to the grounds at least once a fortnight for practice and arrange for friendly matches. If the college sports is held at least a month before the Collegiate sports we could have regular coaching for our competitors, O, for a hostel! It will carry us very far ahead in sports. But even under the present circumstances we ought to be able to do much better. The captains must ease the burden of the secretary, and the players must help the captains by their co-operation. Anyone who is not prepared to do so, even if he be the best athlete, does not deserve to be in the team. All the students can help by attending the athletic meets in large numbers and yelling the college to success. Then again, it is better to have inter-class tournaments early in the

year (August) so that we can choose the best teams for the Inter-collegiate Tournaments. It will also leave the students free to have practice for the Collegiate sports during the short term.

INTER-CLASS TOURNAMENTS.

Extraordinary interest was evinced in the inter-class matches this year. Especially in cricket we had large crowds to witness. Rows of women students were found clapping their hands and yelling musically and sending forth peals of roars into the firmament!

The First Years were the most enthusiastic, and wanted to win through in everything. Above all, they set their hearts on cricket. So did the IV years. The hospital team was not behind hand. Col. Bradfield's presence in the team fired them to a high pitch of enthusiasm. They fought tooth and nail against the Fourth Years but lost, not by anyway a large margin. IV and I met for the finals. There was great excitement over it. There was a huge crowd to witness the match. The 1st year, won. They deserved to win, more so for their enthusiasm. Col. Bradfield's participation in the tournament raised the interest and the spirit in the game.

The Tennis tournaments except the ladies' singles were postponed to March due to want of time. Very many interesting matches were played. I would add here that it is sad that we failed to put up a good fight in the Inter-collegiate tennis.

Hockey.	Football.	Cricket.
(1) II years.	(1) III years.	(1) I years.
(2) I years	(2) I years	(2) IV years.
(Runner up)	(Runner up.)	
Tennis.	Badminton.	Badminton.
	Winners.	Runners up.
Ladies Singles.	Miss Mahadevan	Miss Bower.
	Miss Vedavanam.	Miss Chacko.
(1) Miss A. Chacko.		
(2) Miss Grant		
(Runner up.)		
TENNIS.		
Men's Singles.	Men's Doubles.	
P. P. Paulose—Winner.	G. Kuruvilla and P. P. Paulose—Winners	
G. Kuruvilla—Runner up.	Drs. Cherian and Chandra Rao—Runners up.	
	Mixed Doubles.	
	Miss Wagenmaker and Dr. A. J. Eapen—Winners.	
	Miss Cama and Kuruvilla—Runners up.	

COLLEGE SPORTS.

The college sports went off most successfully under the able management of the president Major Pandalai, and the Secretary Mr. V. O. Zachariah who is endearingly called as "Zach", "Zachy" or "Karrya". The only complaint was caused by the absence of a Pandai where *together* we could howl to our hearts' content. I hope in the future we will not be denied this pleasure. It is also suggested that we should have more fancy items which will add fun and interest to the sports.

Beetles carried off the championship. It was a treat to watch him heading to the winning post in the forefront item after item. Denton has broken our former record in shot put. He has left many a dent in our grounds and is liable to be sued for damages. At the heats for the Collegiate sports it is reported that in putting his shot fell on a mango tree and brought down a shower of mangoes and a huge branch. It was great to watch Morrison pedalling, and it was inspiring to see Rayan propounding the doctrine of "Non-violence" and "Non-co-operation" in sack fight! The ladies 100 yards dash caused great excitement due to the fact that our Veteran Miss Cama was beaten by a Fresher Miss Chacko, and there was a tie between them for the first place. It is a happy sign that more of our girls are coming forward for such contests. In a few years they are sure to race with us not separately, but together neck to neck with our boys. There is every chance that one of them might carry off the championship cup and throw into the shade Ghani, John, Narayanaswamy, Beetles and even the like of Lakshmanan!

CHIEF RESULTS OF THE SPORTS

(1st place, 3 points; 2nd, 2 points; 3rd, 1 point.)

Names.	100 yds.	220 yds.	440 yds.	880 yds.	Mile.	Hurdles.	Long Jump.	High.	Shot Put	Total.
Beetles	...	3	3	3	...	...	3	...	2	17
Ghani	...	2	1	...	...	2	2	3	...	10
Balan	...	1	...	...	...	3	1	...	...	5
Sambandam	...	...	2	2	2	3	...	...	...	9

Items.	I year.	II year.	III year.	IV year.	V year.
Hurdles	...	1	...	5	...
100 yards	3	...	...	3	...
220 yards	3	...	...	3	...
Cricket Ball	3	1	...	2	...
440 yards	...	1	1	...	3
Pole Vault	...	...	3	3	...
High Jump	...	...	...	3	...
Long Jump	3	1	...	2	...
880 yards	2	...	3	...	1
Shot Put	3	3	...	...	...
Fast Cycle	2	1	...	3	...
Relay	2	1	...	3	...
Mile	...	2	1	...	3
Tug	...	...	...	...	...
Total	27	14	8	27	7

INTER-COLLEGIATE SPORTS.

We reached the climax of the sports season at the Inter-Collegiate meet when we beat the Engineers by two points and regained the Championship shield which had been with us for very many years in the past. Our excitement during the sports reached the boiling point. His Excellency Sir. Stanley who presided and gave away the prizes told us in his speech that he judged at the outset that Medicoes were sure to carry off the shield. Yes, we did make *some* noise. Ask the O. P. nurse how many bottles of Mandle's solution we emptied and how many "kutchies" we used the following morning!

Denton broke the Inter collegiate record for putting the shot. We lost the relay unexpectedly. The competition was most feverishly exciting. "Black and White" was leading for three rounds but during the last one it fell back and it was little beyond even for Ghani in the last lap to regain the lost ground. In Tug, for the first time since many years, we lost, pulling against the Lawyers each of whom weighed according to Major Hesterlows calculation 300 lbs a-piece. It seems, they had regular practice at "Komala Vilas."

After sports we carried the shield in procession to the residence of our president who heartily congratulating the athletes supplied us with bank-notes to have a liberal treat at "Clarence". The women students next door invited us inside their compound and gave us hearty and gentle cheers which thrilled our Heroes not a little! Col. Hingston rose to the occasion and declared the following day a holiday. Sure he is the most popular principal.

RESULTS OF INTER-COLLEGIATE SPORTS.

College.	Swim.	Hurdls	100.	440.	880.	Mile.	Cricket Ball.	Long.	High.	Put.	Relay.	Tug.	Total.
Christian	...	...	...	2	3	...	...	...	...	...	3	...	8
Engineers	...	3	...	2	...	1	2	5	2	...	2	...	18
Law	...	...	...	...	...	...	...	...	...	...	...	3	3
Loyola	...	3	1	1	1	2	...	...	...	...	...	...	3
Medical	...	...	2	...	...	2	4	1	...	4	1	2	20
Presidency	...	...	3	3	3	...	...	...	4	2	...	...	10
R. M. S.	...	...	3	3	3	...	...	...	...	...	...	...	9

Shield :—Medical College; Champion Cup :—Raju, R.M.S.; Swimming :—Chacko;
Cricket Ball :—Abraham; Shot Put :—Denton.

ISAAC DAVID.

U. T. C. Notes.

Our activities for this collegiate year have come to a close after finishing up the annual musketry course both for trained men as well as the recruits. Our new officer Dr. S. Ramakrishnan is a very keen and strict disciplinarian. Hence the attendance at company parades has been very good and the efficiency of the company is steadily increasing. We hope to do very well under him at the next camp and are sure to make a bold bid for the Willingdon Shield, not to speak of the numerous other cups which are to be won along with the shield.

Our U. T. C. day came off in great splendour on the 19th of February in the college premises. We had invited a large number of distinguished ladies and gentlemen for the function. After tea in the college grounds where speeches were made by our officer and the commandant the party adjourned to the Biology hall where a fine stage was erected. A very comic farce named 'My Turn Next' was staged by the members of the company which kept the audience in laughter from beginning to end.

The entertainment began with an English song named 'Leander' from the famous play 'Katja the Dancer' by Sgt. G. Verghese, very ably accompanied on the piano by Pte. J. Jacob. During intervals the audience was kept engaged by instrumental as well as vocal music. Our thanks are due to Mrs. Chandrasekharan and Mrs. Kuppuswamy for the fine way in which they sang and played on the violin. They were encored time and again. L/ Cpl. Sambandam also gave a good account of himself on the veena.

THE CHARACTERS IN THE PLAY.

Mr. Taraxicum Truitlers—Sgt. G. Verghese—(a country doctor).
Tim Bolus—L/ Cpl. Gangadharan—(His chemical assistant).
Tom Trap—Pte. Dharmapal—(a commercial traveller).
Farmer Wheatean—L/ Cpl. Chandrasekharan.
Lydia—Sgt. Ragavachari—(The doctor's wife).
Cicely—Pte. P. I. George—(Her niece).
Peggy—Cpl. Sriramulu—(The maid-servant).

Col. Hingston who presided congratulated all the actors at the end of the farce in a short and humorous speech. He specially made mention of Sgt. Verghese who played the part of the hero.

The success of the show was due to the untiring enthusiasm of our popular Sergeant-Major Kuppaswamy and the co-operation of all the other members of the corps.

The officers and N. C. O.'s of the Battalion were 'At Home' to Captain K. R. O'Brien our commandant who is leaving us at the expiry of his term of four years to join his own regiment, the Royal Ulster Rifles. The success and the efficiency of the corps is in no small amount of measure due to his unwonted spirit and soldier-like qualities. We hear that Captain Bacon of the Bombay Grenadiers is going to succeed him. We welcome him with all enthusiasm and pleasure.

MADRAS MEDICAL COLLEGE ASSOCIATION.

Receipts and Payments Account of the Madras Medical College Association and Magazine
for the period from 1st July 1929 to 31st March 1930

RECEIPTS.	RS. A. P.	PAYMENTS.	RS. A. P.
To Balance on 1-7-29	121 12 3	By Association	10 14 0
Association Subscriptions from Students at Re. 1 per head	458 0 0	" Magazine :-	10 5 3
Magazine :-		" Conveyance, etc.	47 3 6
From Students at Rs. 2 per head	559 0 0	" Postages	803 8 6
" Staff	99 0 0	" Printing	6 6 0
" Mofussil Subscribers	328 8 0	" Distribution of Magazines	6 3 0
" Advertisers	112 0 0	" Commission on collections	686 11 6
Association :-		" Graduates' reception	99 0 0
Graduates' reception	115 0 0	" Staff payments	128 0 6
Miscellaneous	5 0 0	" Balance on 31-3-30	
Total	1,798 4 3	Total	1,798 4 3

MADRAS,
1st April 1930.

Examined and found correct.

K. JOHN & Co.,

Government Certified Auditors.

*Receipts and Payments Account of the Madras Medical College Magazine for the period
from 1st July 1929 to 31st March 1930.*

RECEIPTS.	RS. A. P.	PAYMENTS.	RS. A. P.
To Balance as on 1-7-29	... 25 0 0	By Conveyance	... 10 5 3
Subscriptions from Students	... 559 0 0	" Postages	... 47 3 6
Do. Staff	... 99 0 0	" Printing	... 803 8 6
Do. Mofussil	... 328 8 0	" Distribution of Magazine	... 6 6 0
Advertisers	... 112 0 0	" Commission on collections	... 6 3 0
		" Graduates' reception	... 22 13 3
		" Staff payments	... 99 0 0
		" Balance as on 31-3-30	... 128 0 6
Total ...	1,123 8 0	Total ...	1,123 8 0

MADRAS, }

1st April 1930.

Examined and found correct.

K. JOHN & Co.,

Government Certified Auditors.

Medical College Magazine

*Receipts and Payments Account of the Madras Medical College Association for the period
from 1st July 1929 to 31st March 1930*

RECEIPTS.	RS. A. P.	PAYMENTS.	RS. A. P.
To Balance on 1-7-29	... 96 12 3	By Expenses of organising meetings.	5 0 0
" Subscriptions from students at Re. 1 per head	... 458 0 0	" Printing charges towards the election of Association and Sports Secretary	... 4 12 0
" Miscellaneous	... 5 0 0	" Conveyance charges	... 1 2 0
" Graduates' reception	... 115 0 0	" Graduates' reception	... 686 11 6
" Loan from Magazine Account	... 22 13 3	Total ...	697 9 6
Total ...	697 9 6		

MADRAS, }

1st April 1930.

Examined and found correct.

K. JOHN & Co.,
Government Certified Auditors.

*Statement of Liabilities and Assets of the Madras Medical College Association
and Magazine on 2nd April 1930.*

ASSETS.	RS. A. P.	LIABILITIES.	RS. A. P.
Bank	... 224 7 0	Staff payments	... 33 0 0
From Students	... 164 0 0	Commission on collections	... 30 0 0
" Staff	... 204 0 0	Postage	... 15 0 0
Advertisements	... 513 8 0	Arranging Association Meetings	... 10 0 0
		Conveyance	... 4 0 0
		Magazine	... 533 8 0
		Balance to be carried on to next year when all the bills are collected	... 480 7 0
Total ...	1,105 15 0	Total ...	1,105 15 0

MADRAS, }

2nd April 1930.

G. W. ROBERTS,

Honorary Secretary.

Madras Medical College Association

“IN MEMORIAM”

Born 31-7-1907.

Died 4-3-1930.

I find it a hopelessly painful task to write in memoriam of my beloved friend Mr. T. Chandrasekara Rao. Friends and college-mates for some years, we lived in adjoining rooms like brothers. His genial and gentle nature, great fund of humour and hilarity and unsurpassing spirit and enthusiasm had made him in the hostel the head of a family of medical students now left behind dull, dreary and disbanded. His father, a leading medical practitioner himself anxiously awaited his promising son to take his degree and enter the noble profession. But now his hopes are gone, his hair is hoary ; for his son's career is a matter of story

“ When beggars die there are no comets seen

The heavens themselves blaze forth.....”

It is too true. On the night Mr. Chandrasekar fell ill electric lights went off and the whole hostel plunged into darkness for some time. And there lay the youngman in that corner room with a small flickering candle rendering the sleepily silence that crept over the patient, dreadful and gloomy. The next day I carried him to the hospital and that was his last journey ; whoever thought he would succumb to that dreaded disease after having survived plague, typhoid, cholera and even small-pox ?

Mr. Chandrasekar's marriage was to come off very soon. The match was fixed just a few days prior to his departure. God loved him more than any girl or any of us could have possibly loved him. But what right had He to snatch away a precious child from those parents whose heart He now has broken !

Misery, O Misery.

This world is all too wide for thee.

B. SANJIVA RAO.

“IN MEMORIAM”

Mr. Krishnamoorty.

This is a very unfortunate year in which a number of our college-mates expired. Mr. Krishnamoorty who left us on the 3rd of March is the sixth student. He leaves behind, his parents, his elder brother and a large number of friends to bemoan his loss. He belonged to a respectable family. He had been always a perfect gentleman, unassuming not showy or clamorous, a very quiet and business like student. Though he was very reserved in his habits he had left some friends who miss him very much ; though weak physically he had always been hardworking and regular. He was one of our best students. His work in the wards was marked for regularity and careful attention to the rich and the poor alike. It is not an exaggeration to say that he is a loss to the institution and the medical profession. He had very high aspirations and had a good career before him. But God's ways are unsurmountable. All we can do is to convey our deep sympathies to the old parents and others affected by the loss and pray to God to give his soul peace—*Amen.*

V. S. JAYARAM.

"IN MEMORIAM"

When we feel the rude control of love, or pity or joy or grief we become tongue tied-words fail us and it is hard to smile when one would weep and to speak when one would be silent. I wish I could find language sufficiently powerful to convey the exact idea of late Mr. George Christadoss Nathaniel, son of Mr. Vedanayagam Nathaniel, Manager of the British Foreign Bible Society, Madras. My talents sink below my desire when I take up my pen to ink a few lines in memoriam.

"He was sincerely religious and found a great solace and pleasure in committing to memory religious songs which he used to be singing often.. In spite of the hard strenuous study in the college he found time to attend religious meetings and take a responsible part in conducting many. He was always simple in his ways and habits, very sociable and always willing to serve others and do all the good he could and above all he had a kind word and a smile for every one that made him beloved to all those who knew him.

On Wednesday the 22nd January he attended the Hospital and the College and in the afternoon he gave an address at the Christian Endeavour meeting held at the Chapel in the Davidson Street and went home with fever. Quite unconscious of the nature of his fatal illness he tried the C. A. Q. mixture for few days. Then he sent word to his brother-in-law at Mylapore. He came and took Nathaniel home and all possible medical aid was given. Doctor sent the blood for examination on Tuesday the 28th inst. Two days later the Diagnosis as Typhoid was confirmed. His doctor sister from Ongole arrived on Thursday and watched the whole night beside the restless sick brother. Friday the 31st his pulse was bad and about the midday he become delirious and at 3 a.m. Saturday he was taken to the General Hospital. He seemed willing to shake off his mortal coil and be with his parents who had gone Home before him to the presence of the Lord and Master."

Nathaniel! my early friend, I never thought that it should be my last look on you that you should smile no more. Your face, my faithful friend and my counsellor, I will never forget. If we had thought that you would die the grief would not have been so heavy to us. Sorrows we have had, severe ones too, but very painful it is to talk about the beloved ones when we shall not have them in our midst once again. You lay on the bed silently watching the progress of your fatal illness, quite helpless and ignorant of its end and of course after a fitful fever you now sleep—for ever?

G. M. THAMBIDURAI.
Student Editor.

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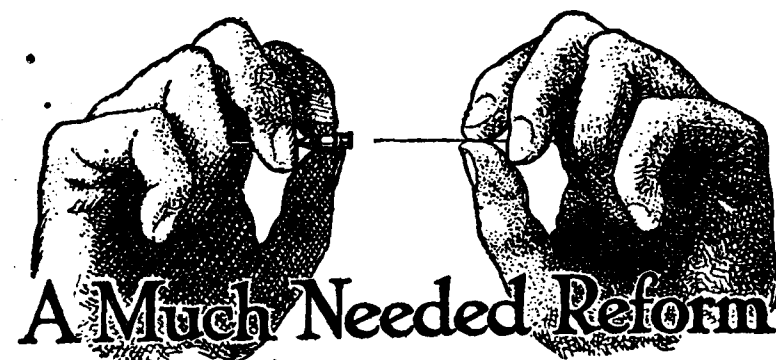


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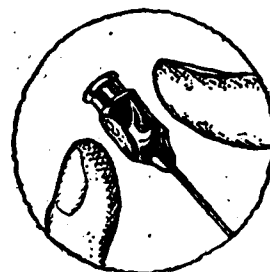
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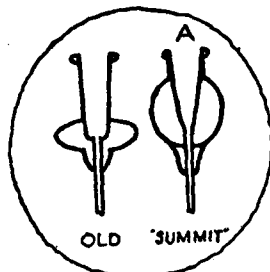
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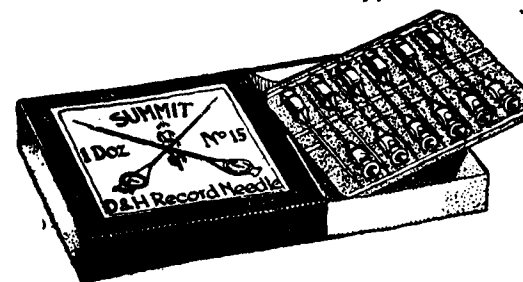
This diagram shows why the Summit Needle can be 'threaded' from the cone end. Note the converging bore. (A).

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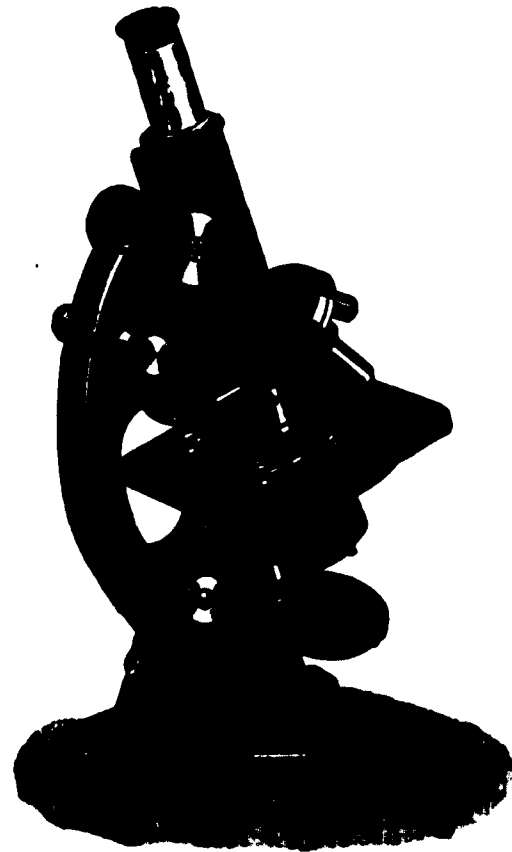
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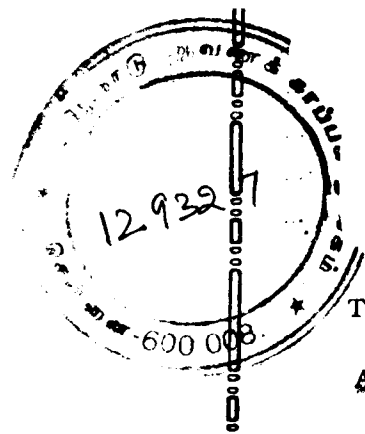
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