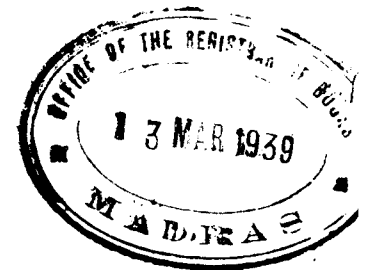


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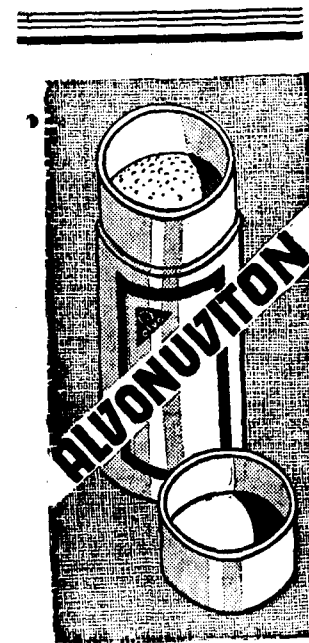
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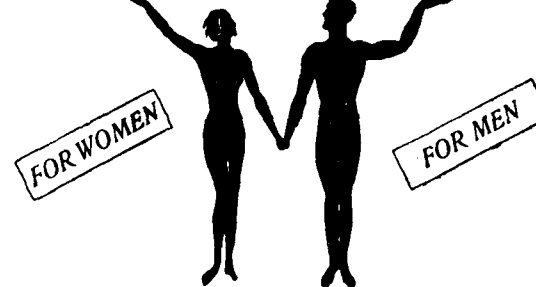
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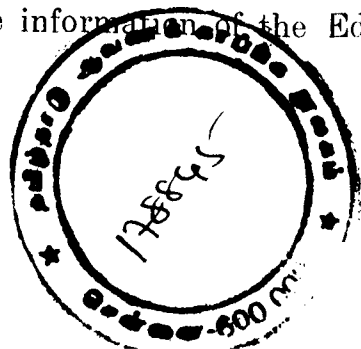
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January, 1939.

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OF THE

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FOR CIRCULATION AMONGST MEMBERS ONLY

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[No. 1

Clinical Application of Renal Efficiency Tests

BY

JAMES V. WILSON, M.D.

(From the Practitioner)

THE object of this article is to review the modern conception of renal function in health and disease with especial emphasis on the measurement of renal function and the impairment which occurs when a renal lesion is present.

Renal Anatomy and Physiology

Just as the central nervous system is regarded as consisting of a system of neurones, so the kidney must be accepted as being built up of nephrons. The nephron is the functional unit of the kidney and consists of:—(1) The Malpighian or renal corpuscle. (2) The tubule. (3) Blood vessels. Five important points emerge about the blood supply:—

1. Except for a small amount which nourishes the corpuscle and interstitial tissue of the kidney, all blood entering the organ passes through the glomerular tuft.

2. The renal artery breaks up suddenly into short branches and so the blood is delivered to the glomerular tuft at high pressure.

3. The tubules are supplied entirely by the blood which has first passed through the glomeruli.

4. There is a striking disparity between diameters of afferent and efferent vessels.

5. There are really two capillary fields in the kidney—capsular and tubular.

As Fishberg states, the dominant function of the kidney is excretion, and much of the earlier work on renal physiology was concerned with attempts to arrive at some conclusion as to how the kidney excreted urine.

In 1842 Bowman described the fundamental fact that the capsule surrounding the glomerular tuft is simply the expanded end of the renal tubule. He was so struck by this design and how admirably it fitted into the process of a filter, that the water passed through it into the tubules which in their turn secreted the solids present in normal urine. A few years later, Ludwig contended that the glomeruli were filters to remove from plasma, water and all substances except proteins, and that the resulting filtrate was concentrated in its passage along the tubules by diffusion. His theory was based on purely physical conceptions but is obviously not correct since the various constituents are not all concentrated equally, and hence we have the modern theory which is based on the work of Cushny. According to this worker, the modification of the glomerular filtrate as it passes down the tubules is due solely to a process of differential absorption and so he recognized two types of bodies—threshold and non-threshold substances. (1) Threshold substances which are of value to the economy of the body are reabsorbed into the circulation and so maintain their appropriate level in the blood. (2) Non-threshold bodies, which are waste substances are not reabsorbed by the kidney under any circumstances.

This theory has not wholly stood the test of time, as many now believe that the tubules themselves are capable of secreting certain substances. So that by way of summary it may be said that:—(a) All are agreed that the function of the glomerulus is that of simple filtration. (b) Those accepting the theory of Cushny believe that the sole function of the tubules is absorption but many workers ascribe to the tubules also a secretory function.

With regard to the other function of the kidney the ground is even less sure, but there is a growing realization of their existence and importance. In spite of opinions to the contrary, it seems impossible that the kidney elaborates an internal secretion but it is important to emphasize the metabolic functions of the kidney, and some of the recent work which has been done in this connexion.

The kidney performs work of a metabolic nature which is of the highest importance to the body; the manufacture of ammonia is the best known example of this function. How it does this is not exactly known, but it seems probable that by virtue of a phosphatase which can be isolated from the kidney, the renal epithelium elaborates ammonia from urea or amino-acids. The importance of this function will be realized when it is remembered that this is the chief mechanism by which the pH of the blood is maintained so constant, and by this mechanism the plasma is spared from giving up valuable base at times when the blood becomes flooded with acids. The Spanish school headed by Professor Jimenez Diaz, has gone further, and asserts that it is the failure of this metabolic activity of the kidney which is the cause of death in Addison's disease, and is a factor in other conditions such as diabetic acidosis. This conception of a functional renal failure, is one of the more important contributions to renal physiology of recent years, and opens up a new avenue to research.

Renal Lesions—Nephritis

It is now over a century since there was admitted into Guy's Hospital an intemperate sailor, John King. He was placed under the care of Richard Bright. The patient presented the features of oedema with pleural effusion and ascites, enlarged heart with pericarditis, and scanty urine with albuminuria. At post-mortem the kidneys were small and granular, and it was this organ that Bright described as being the primary seat of disease. He published his observations in 1827. Since then an enormous amount of work has been done, with but little advance in the knowledge of kidney disease.

In 1914 the modern trend of thought began to crystallize with the publication of the work of Volhard and Fahr, and

their introduction of an anatomical and pathological classification consisting of three main types of Bright's disease: glomerulo-nephritis, nephrosclerosis, and nephrosis. This was followed by Addi's work, in which he tried to correlate clinical findings with a quantitative study of the formed elements in the urinary sediment. Another step forward was made in 1930 when van Slyke and his co-workers published their monograph, in which they called attention once again to their test of renal function which they had published several years before.

The Modern Classification of Bright's Disease is as follows:

1. Inflammatory group Glomerulo-Tubular nephritis.
 - (a) Acute nephritis
 - (b) Subacute nephritis.
 - (c) Chronic nephritis.

The patient has an attack of acute nephritis, so severe that he dies, or on the other hand the condition may be sub-clinical. His recovery from the acute attack may be complete, or he may pass into the subacute stage with œdema, and finally, if he lives long enough, end up as the chronic nephritis with uræmic symptoms.

2. Vascular Group—nephrosclerosis

This includes:—

- (a) The senile kidney
- (b) The kidney of essential hypertension

These conditions are vascular rather than renal in origin, and death is rarely due to kidney failure, but is usually the result of cardiovascular accidents.

As the blood pressure rises the vessels throughout the body are affected especially in such places as the brain, kidneys, pancreas and spleen. In the brain this leads to cerebral hæmorrhage, in the kidney to albuminuria but rarely to renal failure except in malignant hypertension. Its effect on the pancreas may give rise to glucosuria. If the patient escapes death by other means he eventually reaches the final phase which is one of left ventricular failure.

Here the essential changes are organic states rather than organic diseases of the kidney. It is a difficult group to classify and some of these conditions, especially lipoid nephrosis, have given rise to much discussion. Death, when it does occur, is usually due to the primary conditions, inter-current infection or general causes. The renal lesion is but an incident in the clinical picture.

Renal Function Tests

On a basis of this classification, the problems of renal investigation may be approached from the point of view of clinical pathology, as follows:—(1) The measurement of the degree of renal impairment during an attack of nephritis and later the extent of permanent renal damage. (2) The clear differentiation between the organic disease of subacute nephritis and the organic state of nephrosis. (3) The separation of the patient with chronic nephritis, whose life will be short, from the patient with benign essential hypertension, whose chief danger is unwise interference. (4) The elucidation of the renal lesions in eclamptic states. It is obviously important here to differentiate the true chronic nephritic from the primary eclamptic.

Do renal function tests help at all? The aim of any test of biological function has been defined as the effort to determine the existing function of an organ in relation to the function, if the organ and organism as a whole were normal. A qualitative is never very satisfactory, but in the proper evaluation test of renal function an attempt to measure the amount of functioning tissue quantitatively must be the objective. In the case of the kidney, this offers tremendous difficulties.

Renal function tests are divisible into four main groups:—

1. Examination of urine and the ability of the kidney concentrate and dilute.
2. Examination of the blood chemistry.
3. Estimation of the ability of kidney to eliminate injected foreign substances.
4. Simultaneous examination of blood and urine, i.e. clearance tests.

This classification shows that there are two fields in which to work—the blood and the urine. To enumerate in any detail the various tests that have been devised for the estimation of renal function would take too long and it is proposed to deal only with those in common use.

1. FIRST GROUP.—This includes the naked-eye examination and careful analysis of a twenty-four hour specimen of the urine, with especial emphasis on the specific gravity, the presence or absence of albumin, and the search for casts. The question of specific gravity must be especially emphasized. A characteristic of a healthy kidney is its ability to vary its work according to the needs of the body. When a normal individual drinks freely of fluids, the urine produced during the subsequent period should be of large volume but of low concentration. When his intake of fluid is low, as during sleep, then the volume is small but highly concentrated. This ability to vary the concentration is impaired in the presence of renal disease, the kidney can no longer accommodate itself to the needs of the body and so the range of variation in the specific gravity becomes diminished and finally—fixed—in other words when there is a renal impairment the molecular concentration of urine tends to approach that of blood, and varies very little throughout the twenty-four hours. Finally there comes a time when the specific gravity becomes fixed about 1010. The fixation of the specific gravity of the urine to a range between 1008-1012 in all specimens passed by a patient is sure indication of severe renal damage and this fixity has been used as a measure of impairment of renal function.

In the application of this principle two tests are very useful:—

MACLEAN'S UREA-CONCENTRATION TEST, in which the amount of urea in the urine is examined after fifteen grammes of urea by mouth. It is not a delicate test, but it shows renal impairment a considerable time before there is any rise in the blood urea.

CALVERT'S CONCENTRATION TEST, a much simpler test than above, and in many ways more valuable. The patient has no fluid after 5 p.m. and at 10 p.m. empties his bladder and

discards the urine. At 7 a.m. he empties his bladder again and collects this specimen, labelling it "A". He then drinks at least two pints of water of weak tea, and two hours later collects another sample labelling it "B". The specific gravity of each specimen is then examined and normally that of A should be high 1025 or so while B should be under 1005. Any degree of approximation of specific gravity of the two specimens denotes renal impairment.

With regard to albuminuria it is unwise to pay too much attention to the presence of traces in the case of an ordinary specimen, especially in a female patient. If albumin is found then it is always well to repeat the test using a catheter specimen.

A simple classification of albuminuria is as follows:

- (i) Physiological, such as occurs after exercise or heavy protein meals.
- (ii) Benign or orthostatic.
- (iii) Cardiac or circulatory.
- (iv) Toxic or febrile.
- (v) True renal albuminuria.

Casts should be searched for in a fresh specimen of urine, as they rapidly disappear and their significance varies with the type found. There are three main types:—(a) Hyaline, (b) Epithelial, granular and fatty, (c) Waxy.

(a) *Hyaline casts* are the most common and the least important as they appear in many slight and transitory conditions. They are probably formed by the coagulation of the traces of protein which must always be present in the renal tubules. Their frequent presence in icteric patients may be explained by the fact that the bile acids which are present easily precipitate protein.

(b) *Epithelial, granular and fatty casts* are more important and probably represent varying stages in the same process, the epithelial being the precursor. They are composed of cells from renal tubules and their presence in considerable numbers is an indication of severe tubular degeneration. Beginning life

as pure epithelial casts, by a process of degeneration they become granular and later fatty droplets appear.

(c) *Waxy casts*. Little is known as to their origin and composition, but they only appear when severe degenerative lesions are present and consequently are of prognostic importance.

The value of emphasizing this group of renal function tests is that they can all be carried out by the general practitioner with little apparatus at his disposal. He can examine for albumin using either the heat test, or, better still, the salicylsulphonic acid test. He can examine the deposit microscopically and look for casts, and by the aid of simple apparatus do either of the concentration tests. The information thus derived is always significant, and often of diagnostic and prognostic importance. With regard to specific gravity, it may be said that both temperature and albuminuria have very little effect on it. For every three degree above 15° C. one should be added to the figure obtained, and it has been estimated that so massive an albuminuria as one per cent would raise the specific gravity by less than three.

2. EXAMINATION OF BLOOD.—This includes the estimation of the urea, non-protein nitrogen, uric acid, creatinine, and cholesterol content of blood. Unfortunately in this group of tests a normal figure gives little information as regards the state of renal reserve. For example a raised blood-urea suggests that the kidney function is impaired, but a normal figure does not exclude serious renal damage. As has been often shown, the blood urea may be quite normal after a unilateral nephrectomy, and so may be within the usual limits with half the renal tissue destroyed. Any figure over 40 mgm. per 100 c. c. of blood is usually regarded as pathological. It is to be noted that the blood urea is low in the later months of pregnancy owing, it is thought, to the demands of the growing foetus from the protein of the maternal diet. I have quite often obtained figures below 12 mgm. per 100 c. c. in pregnancy. Again, it may be raised by extra-renal factors and this so-called extra-renal azotaemia is very important, although it is only within recent years that its significance has been grasped. To-day,

however, the clinician recognizes it as occurring in the course of a group of conditions which may be classified as follows:—

- (1) Long continued loss of body fluids.
 - (a) Vomiting due to pyloric stenosis or intestinal obstruction.
 - (b) Diarrhoea.
 - (c) Haemorrhage, especially haematemesis.
- (2) Acute febrile conditions.
- (3) Addison's disease Diabetic coma. Following coronary thrombosis.
- (4) Severe hepatic disease, such as acute yellow atrophy.

The condition to which most attention has been called recently is haematemesis. Here a definite rise in the blood urea occurs, and there is general agreement that the height of the rise is closely correlated with the severity of the haemorrhage. It is thus important in all cases of intestinal bleeding to estimate the blood urea in attempting to arrive at some idea as to the extent of the blood loss. After operation there is also a slight rise in blood urea; the extent of this depends on the severity of the operation and the presence or absence of complications.

A surgical colleague and I investigated a series of cases operated upon for appendicitis. We estimated the blood urea every two hours over a period of several days after operation, and it was dramatic to see how the blood urea rose slightly, to fall again in thirty-six hours in normal cases but in those cases in which complications arose the rise was maintained and this was shown often before other signs were at all obvious.

Thus there must always be care in the interpretation of a single reading of the blood urea and no hasty conclusion that elevation means defective renal function. Realizing this danger of taking a single reading of blood urea as an index of renal function, Mosenthal and Hiller in 1917 suggested the ratio of the urea nitrogen to the non-protein nitrogen of the blood as an index of the amount of effective functioning renal tissue,

irrespective of the level of the blood urea. This gives the "azotæmic ratio" or urea ratio, and is expressed according to the formula:

$$\frac{100 \times \text{urea nitrogen}}{\text{non-protein nitrogen.}}$$

When renal function fails, the concentration of all forms of nitrogen in the blood rises, i.e. urea, uric acid, creatinine. Urea nitrogen makes up sixty to seventy per cent of the total non-protein nitrogen in the normal blood, but in disease the urea nitrogen in the blood increases faster than the total amount of nitrogen contained in other substances, and so the urea ratio is elevated when renal function is impaired. This estimation has the advantage of being in some respects a quantitative test and yet can be carried out on a single sample of blood. The normal figure for the azotæmic ratio is usually taken as 40 or less, whereas any figure over 80 is pathological. As far as I have been able to test it out, this test has no special advantage over other tests to be mentioned later, but it does give a rough quantitative estimation of the amount of functioning renal tissue.

The test in this group that is performed most frequently is the simple estimation of blood urea. It is a non-toxic substance and so the test is merely using the accumulation of a waste product in the blood as a measure of the degree of renal inadequacy. The creatinine output, however, is much more constant, and so the blood creatinine estimation has been suggested as a more delicate index of the degree of renal failure. Actually it is doubtful if the information so gained is of any greater value than the blood-urea figure. It has, however, a certain prognostic value, in that any figure higher than 5 mgm. per cent in a chronic nephritic indicates that death will probably take place inside a year.

3. THIRD GROUP.—This group includes the various dyetests and also the use of uroselectan as an index of renal function. They have been extensively used in America, but much less so in this country. The general consensus of opinion is that they are no more delicate than other much simpler estimation. Their chief scope is in surgery.

In the interpretation of these tests it is essential that extra-renal factors should be carefully considered. That there is delay in the excretion of, say, phenolsulphonphthalein does not necessarily indicate renal damage; it may mean that there is circulatory failure and the kidneys are not having a chance to excrete properly. Thus it is unlikely that they will ever have a wide application in medicine since they never show definitely whether or not the delayed excretion is due to renal impairment.

4. FOURTH GROUP.—In this group the blood and urine are examined simultaneously. The substances most easily studied are urea and creatinine, and thus there have developed so-called clearance tests dealing with one or other of these substances. The creatinine clearance test has little to recommend it, as it means giving an intravenous injection, and the results given by the test are in no way superior to that of the urea-clearance test which is much simpler and in many ways the best test of renal efficiency yet devised. During several years' work in laboratories where this test was carried out as a routine, I have had abundant opportunity of examining its usefulness, and when properly performed and correctly interpreted it has always given useful information. The test is founded on pioneer work of Ambard and later by Addis. It was van Slyke, however, who devised the present test in its modified form. It combines, as no other test does, the three essential data necessary for any accurate measure of renal efficiency, i.e. the concentration of urea in blood and in urine, and the volume of urine excreted in unit-time. The measurement of the weight of urine excreted is most important in any accurate estimation of the functioning kidney and its omission is a serious criticism against such tests as Maclean's urea concentration test.

The principle of the test is simple, and its interpretation direct. Addis and others have shown that, provided the volume of urine was above a certain standard, then the urea excreted is directly proportional to the blood-urea content; in other words, the urea excretion per minute was equal to the amount of urea in about 75 c. c. of blood. This standard of urine-volume Van Slyke has called the "augmentation limit," and found that it was about 2 c. m. per minute for an adult. This is what he calls the maximum blood-urea clearance, and, when

the output of urine is above the limit, it represents the amount of blood cleared of urea per minute.

$$\text{Maximum clearance } C_m = \frac{U}{B} \times V$$

When U.....urine urea in mgm. per cent.

B.....blood urea in mgm. per cent.

V.....volume of urine.

When the volume of urine falls below the augmentation limit, the formula becomes:

$$\text{Standard clearance: } -C_s = \frac{U}{B} \sqrt{V}$$

It represents 54 c. cm. of blood cleared of urea per minute. For the sake of comparison, it is usual to express both as a percentage thus:

$$C_m = \frac{U}{B} \times V \times \frac{100}{75} = \frac{U}{B} \times V \times 1.33$$

$$C_s = \frac{U}{B} \times \sqrt{V} \times \frac{100}{54} = \frac{U}{B} \times \sqrt{V} \times 1.85$$

In carrying out the test, no special preparation of the patient is needed except that since the results are probably more accurate if the urine volume is high, it is an advantage to give the patient a glass of water before and/or during the test. Van Slyke states that, except in fairly advanced cases of nephritis, it ordinarily makes no difference whether the patient is in bed or walking about. Clearance readings below fifty per cent. usually show lower readings if the patients are up and about than if they were in bed. It is usual to perform the test during the morning.

The patient has an ordinary breakfast, except that he should take no coffee or strong tea. The patient completely empties his bladder, say at 10 a.m. and this specimen is discarded. Specimens are again collected at 11 a.m. and at noon, great care being taken that the nurse has been accurate as regards the time interval. Blood is taken off for urea estimation some time towards the end of the first and beginning of the second hour.

It is not desirable in an article such as this to go into the details of laboratory technique of the test except to state that it is essential that the blood urea should be done accurately

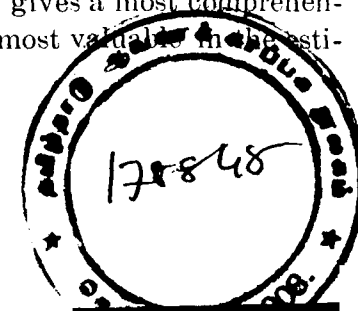
that the volume of urine in unit-time should be carefully measured and that it is quite satisfactory as well as being much simpler to estimate the combined urine urea and ammonia and not the urea alone. If the ammonia is formed by the kidney from the urea of the blood, then this is also a theoretically more correct reading besides being a great technical convenience. The van Slyke's rapid hyper-bromide method may be used both for urine and blood, using of course his improved hyper-bromide reagent. It is usual to estimate the clearance on each specimen of urine and then take the average figure as the result.

Anything above eighty per cent. is usually taken as a normal figure, anything below fifty per cent. as indicating impaired renal function. But it must be imagined that the figures given by this test are mathematically exact as regards renal impairment. Blood-urea concentration and urine volume are two important factors in urea output, but they are by no means the only ones acting. If this is realized, the test is one of the most useful now available and gives a lowered reading long before other renal function tests show any defect, and shows improvement when other tests give no such hopeful indication. A useful modification has been introduced by Fowweather of Leeds. He measures the clearance after giving 15 gm. of urea, and finds the range of normal much narrower than in the original method, which thus makes the test more delicate and reliable. The technique he suggests is as follows:

At 7 a.m. and 11 a.m., the bladder is again emptied a specimen of blood being taken at about 10.45 a.m. With this material a test can be performed using the third urine specimen and the blood taken at 10.45 a.m., and a urea-concentration test can also be made using the first and second urine specimens. The urea-concentration factor may also be calculated, which is,

$$\frac{\text{Blood urea}}{\text{Urine urea (using the third specimen)}}$$

In my experience this combination gives a most comprehensive survey of kidney function and is most valuable in the estimation of renal impairment.





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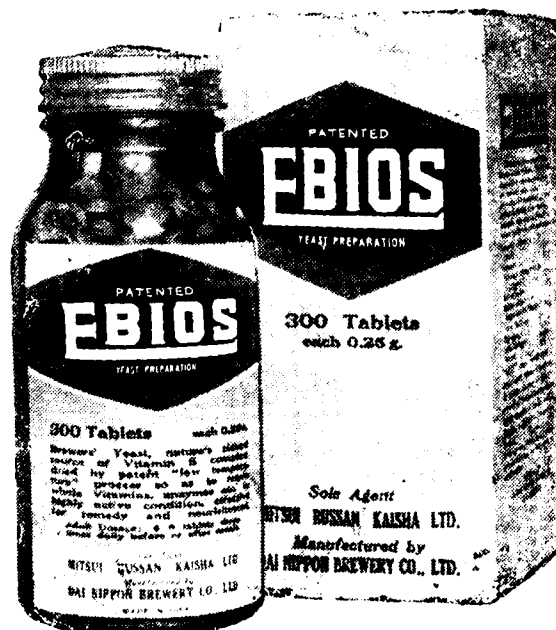
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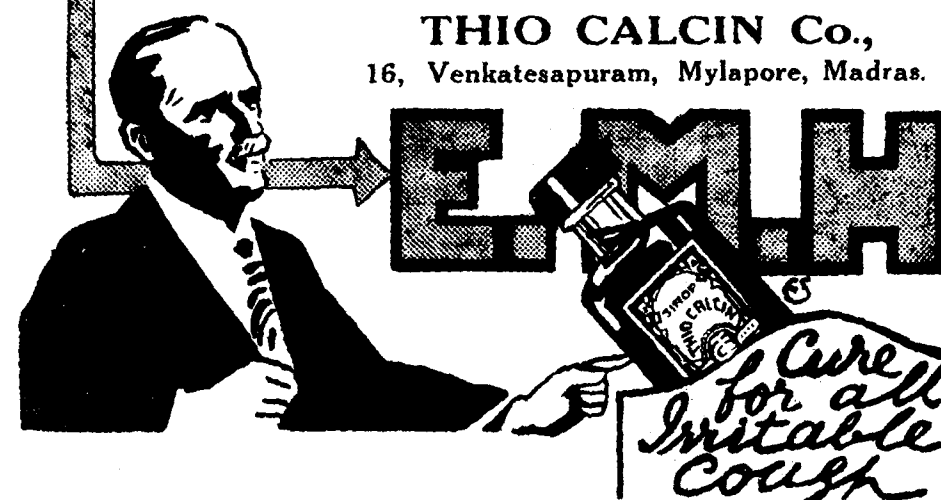
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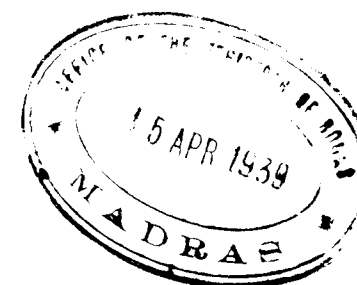
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 - (i) ANNUAL GATHERING - - - - -
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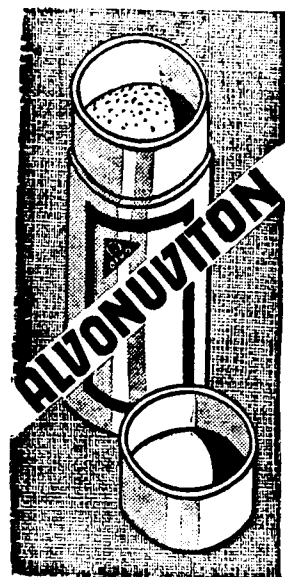
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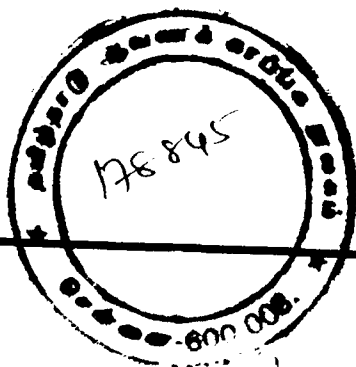
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Vol. VI]

FEBRUARY 1939

[No. 2

Toxicity of Sulfanilamide

(T. N. SUBRAMANIAN, L. M. & S.)

THERE is no doubt that the introduction of Sulfanilamide to the armamentarium of the Physician is a potent weapon in the treatment of certain bacterial diseases. For a long time scientists have been searching for a drug that can cure all diseases without detriment either temporary or permanent to the human organism. When Sulfanilamide was introduced, it seemed as if that such a 'Therapeutica Magna' has been found. Consequently it has been used extensively and intensively in a variety of diseases. This extensive and intensive use of the drug has been much facilitated by the ease with which it can be manufactured as well as administered and also by the propaganda of the various manufacturing firms about the advantages of their particular Brand of the drug. The claims of the drug as a cure for many diseases have been published in the lay press to the extent that the public have begun the use of it without the guidance and control of a doctor which is necessary for such a potent drug of Sulfanilamide and which may not after all be free from dangerous immediate and late after-effects on the human system. Although the exact mode of action of the drug has not yet been determined, in spite of its low toxicity, sufficient experience has been gained about its immediate and late toxic effects on the human system as to warrant a more discriminate use of the same than in the past so as to avoid certain definite, though not necessarily, unavoidable or serious hazards.

Clinically, the toxicity of Sulfanilamide may be considered under the following classification:

(a) Mild. Cynosis, symptoms of gastro-intestinal tract and of the central nervous system, fever, precordial and abdominal pain and acidosis.

(b) Severe. Rash, Jaundice, Anaemia, Granulocytopenia, and Hemaglobinuria.

(c) Secondary reactions and late manifestations.

A. Cynosis

The development of cynosis of varying degrees in patients receiving sulfanilamide is common. It usually begins with pallor progressing to deep cynosis especially marked in the lips. In some cases cynosis develops within twelve hours while most persons become blue before the third day of the drug therapy. The intensity of the colour seems to depend on the amount of drug in the circulation rather than on the amount of drug administered. This manifestation does not seem to be dangerous in most cases since it disappears rapidly following withdrawal of the drug. However, in patients with diseases accompanied with lowered arterial oxygen either systemically or in a localized area the question arises whether the development of the condition may aggravate the underlying disease. This problem presents itself in Pneumonia, in Cardiac insufficiency and in recovery of compromised tissues following local inflammation or injury.

Gastro-intestinal symptoms. A great many disturbances in physiology of the gastro-intestinal tract particularly anorexia, nausea, vomiting and abdominal pain occur especially among adults. Diarrhoea may be severe in a few cases. Like cynosis, these symptoms disappear rapidly when Sulfanilamide therapy is discontinued. This problem is particularly acute in infants in whom essential maintenance of nutrition will be made difficult by severe anorexia.

Central Nervous System. Dizziness and headache are fairly common symptoms of sulfanilamide toxicity among adults. Mild

personality changes, especially light-headedness, loss of ability to concentrate, and depression may be noted in many persons shortly after beginning the drug. A few may become disoriented and even maniacal. All these symptoms subside rapidly when the drug is withdrawn.

Fever. Development of fever after the administration of Sulfanilamide is rare in children but frequent in adults. Most persons who develop pyrexia experience some of the other symptoms mentioned. A few, however, develop pyrexia without symptoms. There are three different types of febrile response to sulfanilamide. The most common of these is a slowly rising temperature, occurring after the fourth day of therapy, progressing by stages each day and falling in about twenty-four hours after the drug is stopped. In some cases the patients develop high fever and chills in about twenty-four hours after the institution of therapy. These patients seem to have a low tolerance for sulfanilamide. Some who have previously experienced sulfanilamide sickness may show a 'Hypersensitive reaction' on subsequent administration of the drug from one month to one year later. This 'Hypersensitive reaction' characterized by aches, chills, temperature shooting up to 104 F., headache and leucocytosis may be profound within six hours of the administration of the drug.

Precordial and abdominal pain. A few patients may show unexplained abdominal pains during the course of treatment, located chiefly in the upper quadrants. This may occur mainly in patients who show other evidences of toxicity from the drug such as cynosis or gastro-intestinal complaints.

Acidosis, clinically as evidenced by combination of hypernoia and a lower carbondioxide combining power, may occur rarely and prompt recovery will follow the cessation of the drug.

B. Severe Manifestations

Rash. A maculopapular rash resembling confluent measles may develop during or just after sulfanilamide therapy. This may be preceded by formication. Itching may be severe in some cases. The drug should be stopped immediately as otherwise it may progress to toxic dermatosis or exfoliative derma-

titis. The rash may persist for two days or longer after the withdrawal of the drug, finally clearing completely.

Jaundice. Two types of jaundice may develop; one a severe hemolytic jaundice, the other presumably a result of hepatitis. The jaundice is usually accompanied by secondary anaemia. In view of the possibility of permanent damage to the liver, the drug should be withdrawn whenever jaundice appears. Transfusions may be necessary in some cases of jaundice though it subsides when the drug is stopped.

Anemia. Mild anemias may develop in a number of cases, but it is difficult to determine whether they are due to the infection or to the drug. A few may develop a severe hemolytic anaemia with intense icterus or a less severe form of progressive anaemia even only after a moderate dosage of the drug. Spleen and Liver may become enlarged. As the most serious toxic manifestations are those associated with the blood or hematopoietic system, the drug should be stopped immediately and transfusions of suitable blood may counteract the progress of anaemia to some extent.

Granulocytopenia. In a few cases marked depression of Leucocytes may follow sulfanilamide therapy.

Hemaglobinuria. In a few cases hemaglobinuria may occur with an alarming drop in the hemaglobin level even with a moderate dose of the drug.

Secondary reactions and late manifestations. So far the symptoms mentioned may develop during the course of sulfanilamide therapy. But some of them as pyrexia, enlargement of spleen and liver, rash and jaundice may appear even more than two weeks after the last dose of the drug. Inhibitory action on spermatogenesis has been recorded. Probably toxic peripheral neuritis with a progressive muscular dystrophy-like picture may develop.

The toxicity of the drug has no observable relation to its effectiveness in overcoming infection.

The striking differences in the response to sulfanilamide therapy seems to be related more to the character of the lesion

than to the identity of the bacterial infection. The protective action of sulfanilamide seems to be greatest in the circulating fluids by depressing the invasive properties of the organism. It is effective in conditions of rapidly spreading infection in which there has been little tissue destruction such as bacteremia, lymphangitis, erysipelas and cellulitis by preventing the further spread of the infection. But its effect is diminished in the presence of necrotic tissue and it is ineffective when abscesses are well established except perhaps in limiting their further spread and protecting normal surrounding tissues against invasion when drainage is used. Hence sulfanilamide should be considered as an agent which supplements but in no way supplants other measures of treatment such as antibacterial immunity, drainage of localized abscesses etc.

Ref.: Mechanism of action of sulfanilamide by John S Lockwood, M.D., Alvin F. Coburns, M.D., H. E. Stokinger, Ph.D., New York. The Journal of American Medical Association, Vol. III, No. 25, 17-12-'38.

The Intravenous Use of Hypertonic Glucose

ONE of the most confusing syndromes encountered in all branches of medicine is shock. This syndrome is generally recognised to be a group of symptoms and not a disease. It is used to denote a condition of acute peripheral circulatory collapse. It does not resemble cardiac decompensation as it lacks the characteristic signs of edema, visceral congestion, and the accumulation of fluids in the serous cavities. The more common predisposing causes of surgical shock are the following: (1) acute hemorrhage, (2) trauma of extensive wounds, (3) Prolonged anesthesia, (4) excessive manipulation of abdominal viscera during operation; while those of obstetrical shock are (1) acute-antipartum and post-partum hemorrhage, de-hydration and acidosis, (2) the severe toxemias of pregnancy, (3) extensive trauma of difficult delivery, (4) prolonged anesthesia. Two types of shock resulting from injury and or loss of blood may be recognized (1) primary shock or collapse in which the low blood pressure follows immediately and (2) secondary shock in which the development of low blood pressure is delayed.

In the condition of shock there is a stasis of blood in the volume of circulating blood and a consequent fall in blood pressure. It therefore follows that any treatment of this condition must increase circulating blood volume and blood pressure. An increased blood pressure with no increase in blood volume is undesirable but an increase in blood volume which naturally increases the blood pressure is highly desirable. Though Vasomotor drugs temporarily increase the blood pressure they actually diminish the quantity of blood reaching the organs. The temporary rise in blood pressure is never sustained. For example adrenalin produces a more prolonged increase in the blood pressure and in the output of the heart than the other drugs commonly used for this purpose. However, it is a well-known fact that a normal animal can be placed in a state of shock by the frequent introduction of large amount of adrenalin. When continued vasoconstriction is produced without increasing blood volume, the amount of blood reaching the organs is decreased rather than increased. A lack of oxygenation of tissues results, producing further damage. Likewise other vasomotor drugs cause similar reactions.

Hence the ideal method of treatment is to introduce some fluid into the blood stream that will cause an increase both in blood volume and blood pressure. Whole blood by transfusion is the ideal therapeutic agent for this. When whole blood is used there is a dilution of red blood cells in the blood stream due to the passage of fluid from the tissue spaces back into the blood vessels. Therefore it will increase blood volume, although when injected intravenously in the presence of circulatory stasis, protein is lost through the capillary walls. This, however, is at least partially compensated for in as much as a very large amount of additional protein is being put into the circulation by the intravenous injection of blood.

As an emergency measure in the actual absence of blood or because of delay in obtaining the proper donor, it is usually necessary to use a substitute for blood in order to tide the patient over this waiting period. Isotonic saline will temporarily cause an increase in blood pressure, but as the fluid injected passes into the tissue spaces, the blood pressure will be

as low as before and usually, lower. Besides, with the injection of normal saline there is a decrease in the total amount of plasma and at the same time a decrease in the percentage of protein in a unit volume of plasma. Therefore isotonic saline will not cause an increase in the volume of blood in the circulation, because with the diminished osmotic pressure only a diminished quantity of fluid is attracted back into the blood vessels from the tissue spaces. Weak solutions of glucose have proved to be no more efficient. Accidents have been reported following the use of solutions of gum acacia with or without glucose.

Glucose may be designated as "the universal therapeutic agent" considering that (1) it is a food for the vital organs, especially the liver and heart muscle. (2) It requires no digestive processes to prepare it for absorption; being a monosaccharide it is spontaneously and completely metabolized to carbon dioxide and water. (3) Its use temporarily raises the systolic blood pressure and produces a peripheral circulation by its osmotic effect on tissue fluids. (4) Its use temporarily improves the quality of the pulse by improving the ventricular filling of the heart. (5) It promotes diuresis and combats acidosis. (6) It temporarily lowers body temperature. (7) It increases the protective power of the blood and according to Hofbauer, it stimulates the production of myelocytes. (8) It increases the coagulation time in jaundiced patients but increases the clotting period in other patients. (9) In high concentration it prevents agglutination of the blood, and in diabetics glucose and insulin promote glycogen storage in the heart.

The ideal intravenous solution in the treatment of shock and its allied conditions should act (1) to increase blood volume in the cardiovascular system without overloading the heart, (2) to provide easily accessible nourishment for the tired myocardium and liver, i.e. replacement of glycogen in these organs, (3) to help restore the circulation in general and (4) to tide the patient over the critical period of shock, dehydration and acidosis, profound toxemias and critical infections until suitable measures can be instituted. Furthermore it would seem that the intravenous addition of large quantities of any nature

except perhaps blood, is inadvisable and often actually dangerous for it might very well mean the addition of an insurmountable load on an already impaired circulation and weakened myocardium which might result in cardiac dilatation and failure. Therefore the use of Hypertonic glucose solution will meet the situation though it is not a perfect substitute for suitable whole blood in hemorrhage or in shock.

Ref.: The Intravenous use of Hypertonic glucose in Obstetrics and Gynecology by Harvey B. Matthews, M.D., F.A.C.S. and Vincent P. Mazzola, M.D., F.A.C.S. New York, in Surgery, Gynecology and Obstetrics, Vol. 62, No. 5.

Practice and Prescription

IN the daily routine of our life some have no time and others have no inclination for introspection. If we do not occasionally examine ourselves, our motives and actions, we will fall into a rut and move on without improvement. Perhaps the examination is irksome or unpleasant, and as human nature is always ready to take the line of least resistance we avoid such examination. Hence I shall put here one proposition for your thought.

1. Regarding the multitude of pharmaceutical combinations and our use of the same.

Month by month, nay day by day new pharmaceutical combinations are pushed into the market; some are really good and many are really worthless. In bringing these goods to the notice of the Doctors the agents learn by heart a set of speech with high-sounding scientific terms and repeat the same to each and every doctor he calls upon, and as a token leaves generously on the table a sample for clinical trial. Then we begin writing out to our patients whether it will have the desired effect or not and whether it is worth the money it costs. The patients also are satisfied because the Doctor had prescribed the latest in the medicine market perhaps so latest that it is not even available in the local market. Perhaps it may be thought that this picture is too much coloured. But as I said in the beginning if we honestly examine

ourselves I am sure you will find that this is not exaggerated. Now the question arises whether we are justified in doing this with the scientific background of our education and training and with the general poverty of the masses of our country. Please answer yourself.

ACKNOWLEDGMENT

We gladly acknowledge receipt of the February number of the magazine "The Indian World" edited by Sri S. G. Warty, M.A. It contains articles from the pen of such distinguished people as H. H. The Yuvarajah of Mysore, Sir P. S. Sivaswamy Iyer, Mrs. Sarojini Naidu, Raj Mitra B. D. Amin etc. besides many interesting features. Further, the printing and get-up is so good as to make it a pleasure to read the magazine. We are sure that anyone who reads the magazine will derive profit and pleasure.

The Trichinopoly District Medical Association

(REGISTERED)

THE Ninth Annual Gathering of the above Association was held on Saturday the 21st January 1939 in the premises of the National College, Trichinopoly, when about 75 doctors were present. Lt. Col. T. S. Shastri, I.M.S., District Medical Officer, Tinnevely, presided. Proceedings commenced at 12 a.m. Dr. C. E. R. Norman, President of the Association, welcomed the guests and members and in reviewing important activities of the Association he hoped for the better working during the coming year. Dr. O. R. Balu, Secretary of the Association, presented the Annual Report which showed that there were regular meetings of the Managing Committee every month and that the interest of members in the ordinary meetings of the Association was declining. Though the Miscellany continued to be run profitably yet the finances of the Association was at low ebb as there is an outstanding of nearly Rs. 700/-

in members' subscription. This prevented the Association from becoming a branch of the Indian Medical Association as resolved at the last Annual Gathering. Dr. T. S. Balasubramania Iyer proposed and Dr. N. C. Subramanian seconded that the Annual Report and the statement of accounts be passed and it was carried unanimously.

The following office-bearers were elected for the year 1939 :—

President—Lt. Col. N. K. Bal, I.M.S.,
District Medical Officer, Trichinopoly.

Proposed by Dr. C. E. R. Norman and seconded by Dr. P. A. S. Raghavan and elected unanimously.

Vice-President—Dr. C. E. R. Norman, F.R.F.P. & S., D.P.H.,
Chief Medical Officer, Golden Rock.

Proposed by Dr. O. R. Balu and seconded by Dr. P. A. S. Raghavan and elected unanimously.

Secretary—Dr. P. A. S. Raghavan, Z.M.R.E. (Vienna) Radiologist, Trichy X Rays Ltd., Trichinopoly.

Proposed by Dr. T. S. Balasubramania Iyer and seconded by Dr. O. R. Balu and elected unanimously.

Treasurer—Dr. T. N. Subramanian, L.M. & S.,
Trichy Medical Union Ltd., Trichinopoly.

Proposed by Dr. P. A. S. Raghavan and seconded by Dr. T. S. Balasubramania Iyer and elected unanimously.

Committee Members :—

Dr. M. Balammal, M.B.B.S.
Dr. R. Sambasivan, M.B.B.S.
Dr. N. C. Subramanian, M.B.B.S.
Dr. T. V. Srinivasan, M.B.B.S.
Dr. D. Srinivasan, L.M.P.

They had sent in their names agreeing to serve as office-bearers and were elected unanimously.

Dr. O. R. Balu then read messages of good wishes from Hon'ble Dr. T. S. S. Rajan, Minister for Public Health, Major

Sangham Lal, District Medical Officer, Salem, Dr. D. V. Venkappa, President, of the Madras Provincial Branch of the All-India Medical Licentiates' Association, the District Medical Associations of Ramnad and Tanjore and various medical firms. He also announced the names of various firms who are participating in the day's function by exhibition of their products and some of them with monetary contributions also. Dr. C. E. R. Norman proposed that the rules of the Association passed at the extraordinary general body meeting of 19-1-38 be passed with the following amendments.

“Rule - 29—Instead of a quorum of half the number of members for an extraordinary meeting the quorum shall be one-third.

Rule 31—Four members instead of five members shall form the quorum for the meeting of the Managing Committee and any member failing to attend three consecutive meetings shall cease to be a member—The amended rules were passed unanimously.

Dr. T. S. Balasubramania Iyer proposed and Dr. R. Kalamegham seconded the following resolution which was passed after being fully discussed by the members present.

“This Association resolves to nominate the executive committee of the Trichinopoly District Medical Association to arrange with the Trichy Branch of the Provincial Rural Medical Practitioners' Association to arrive at an arrangement which will facilitate their affiliation with the District Medical Association and authorise the Managing Committee of the Trichinopoly District Medical Association to bring into force such an arrangement at an early date.”

The members then adjourned for lunch. The afternoon session commenced at 2 p.m. when Lt. Col. T. S. Shastri addressed the gathering tracing his connection with this Association and about the efficient working of the Tinnevely District Medical Association. He suggested that the meetings might sometimes be held in Taluk Headquarters and sometimes conjointly with the neighbouring districts. He also deprecated the idea of forming smaller Associations and hoped that the District Medical Association will be the Primary unit and be a

branch of an All-India Medical Organisation such as the Indian Medical Association. After the address the following lectures were delivered.

"Vitamins in relation to S. I. diet" by Dr. S. Gurupatham of Coimbatore.

"X Rays in Diagnosis" by Dr. P. A. S. Raghavan,

"Some Aspects of Injuries of the upper Extremity" by Dr. K. Raman Menon, A. D. M. O., Trichinopoly.

"Abdominal Emergencies" by Dr. C. P. Viswanatha Menon, Hony. Surgeon, General Hospital, Madras.

"Bio-Chemical Tests in Diagnosis" by Dr. V. Iravatham, Trichinopoly.

After the lectures the Secretary brought forward the following resolutions which were seconded and carried unanimously.

1. This Association invites the attention of the Government of Madras to the December 1938 issue of the Journal of the Indian Medical Association and requests that the Local Self Government may accord recognition to the Indian Medical Association and consult the Indian Medical Association branches both pucca and affiliated in all matters of medical and Public Health administration as is being done by the U. P. Government.

2. This affiliated branch of the Indian Medical Association resolves that to render possible the formation of more branches in this Presidency the Central Council of the Indian Medical Association be requested to reduce the Annual subscription per member of the branch from Rs. 3 to Rs. 2.

In summing up the proceedings of the day Lt. Col. T. S. Shastri referred to the salient points in the various lectures and was glad to find that the function was a great success and hoped that the Association would have a useful future and thanked the Association for giving him the opportunity to preside and renew old acquaintances with the members. Lt. Col. N. K. Bal proposed vote of thanks to the President and various lecturers and to the various firms that participated in the exhibition and the authorities of the National College for

lending the premises. After dinner there was an interesting cine show on Diphtheria by Bayer Remedies Ltd., through their representative Mr. L. S. P. Sarathi and the meeting dispersed at a late hour.

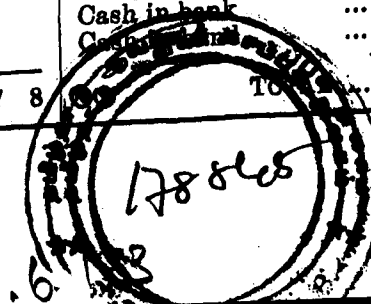
The Trichinopoly District Medical Association, TRICHINOPOLY.

Statement of accounts for December 1938 and January 1939

RECEIPTS				PAYMENTS			
December 1938							
	Rs.	A.	P.		Rs.	A.	P.
Cash in bank	176	13	2	Return of previous clerk's security	50	0	0
Cash on hand	24	13	6	Clerk's pay, Nov. and Dec. (2 days)	19	0	0
Members' Subscription.	35	0	0	Gall bladder operations			
Clerk's cash security...	50	0	0	film hire charges (in May)	9	6	0
Advertisement charges.	91	9	0	Postage	9	8	0
				Miscellaneous charges..	6	7	0
				Miscellany printing	60	0	0
				Auditor's fees	25	0	0
				Secretary's expenses to go to Madras for Annual Gathering	50	0	0
				Balance in bank	60	5	2
				Balance in hand	88	9	6
TOTAL	378	3	8	TOTAL	378	3	8

January 1939							
	Rs.	A.	P.		Rs.	A.	P.
Cash in bank	60	5	2	Clerk's pay (Dec. '38)	18	0	0
Cash in hand	88	9	6	Office Rent for Dec. '38 and Jan. '39.	10	0	0
Advertisement charges.	32	13	0	Printing	25	0	0
Contribution to Annual Gathering	154	12	0	Postage	12	1	6
Members' Subscription..	40	0	0	Annual Gathering expenses	5	0	0
Suspense a/c	15	0	0	Miscellaneous expenses	1	4	0
				Stationery	1	11	6
				Cheque under realization	2	15	0
				Cash in bank	315	6	2
				Cash on hand	0	1	6
TOTAL	391	7	8	TOTAL	391	7	8

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(REGISTERED)

The Secretary requests contribution on Medical, Topical and News Subjects from Members of the Association and other medical men. Contributions should be sent typed in duplicate and signed by the contributor. In case where it is desired to write under a nom-de-plume the signature must be included for the information of the Editor as a token of good faith.

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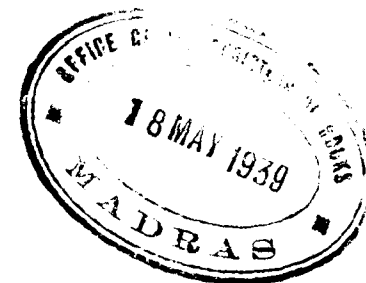
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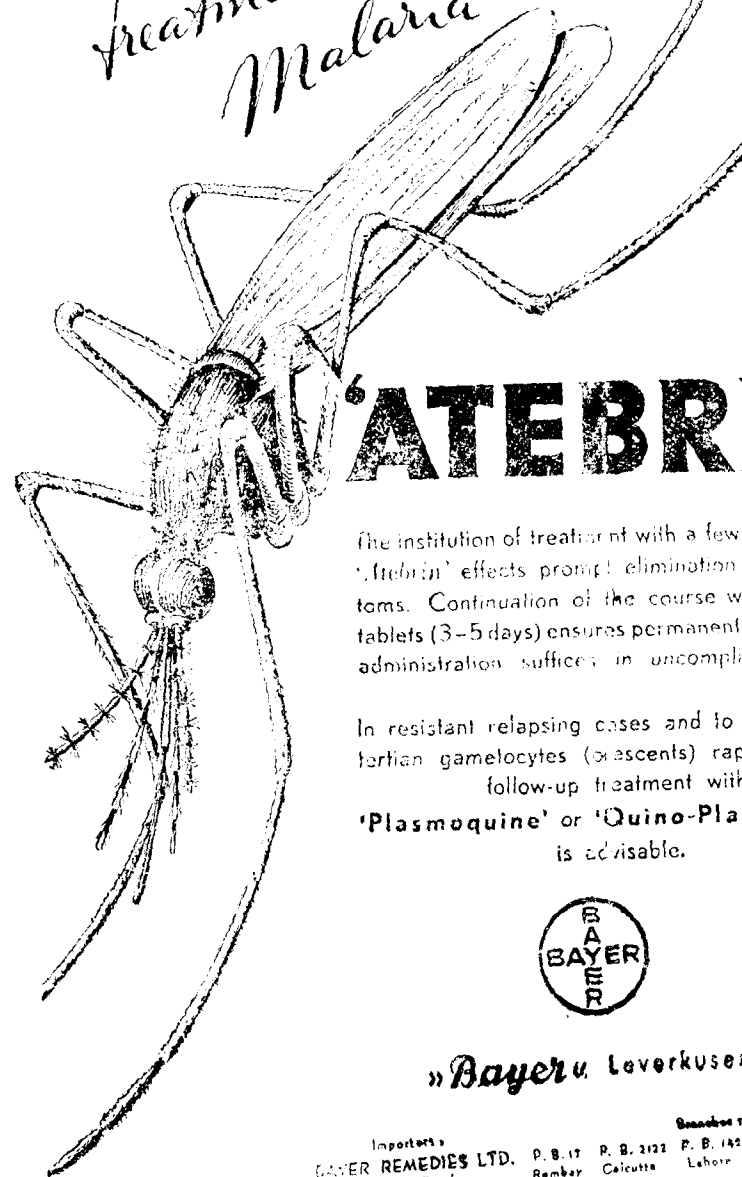
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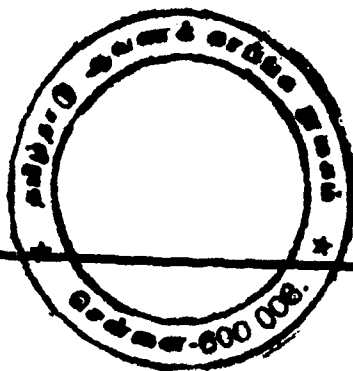
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(REGISTERED)

FOR CIRCULATION AMONGST MEMBERS ONLY

Vol. VI]

MARCH 1939

[No. 3

Practice and Prescription

BY

(DR. T. S. BALASUBRAMANIA IYER)

HERE is another proposition for your thought. Do you realise your serious responsibility when you advise or undertake the operation for the removal of tonsils and adenoids in children?

Mr. P. W. Leatheart in the B. M. J. 1938 ii 835 says that the tonsils and the adenoid masses and the Payers patches are "external lymphoid masses" one surface of which is in contact with food or air, their lymphoid tissue is so arranged that deep crypts lead inwards from their surface in which living micro-organisms are always found which they collect from food or air and grow only those to which an immunity is required by the individual, by repeated infections, enlargements and recoveries of these masses "auto-immunization" of the individual takes place, and hence the tonsils and the adenoids are valuable organs with a highly important function. Their removal is often performed without a thought as to the function of the tonsil and the adenoids or to the ill-effect on auto-immunization. In view of these facts, at the Royal Liverpool Children's Hospital conservative treatment has been adopted in preference to the radical removal and had yielded excellent results.

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Dr. Georgio Calderoni of Rome is of the opinion that the retention of the tonsils is of the utmost importance in the preservation of the human race and their removal a tragic blunder. Investigations have shown that fifty per cent of the students whose tonsils have been removed before the age of fifteen become "bad subjects," they abandon their studies and become vicious in their morals and even take to crime in some cases.

The official report of the British School Medical Service has the following remarks about the operations for the removal of tonsils and adenoids. Many unnecessary operations are performed and one of the most cogent arguments in favour of a reduction in the number of tonsillectomies is afforded by the examples of drastic reduction in the operation rates, which reductions have been unaccompanied by the slightest evidence that any thing but good has resulted and on the other hand there is evidence that, at the present time, operations are often performed without adequate reasons and sometimes without full precautions against dangers and complications.

The Incidence of Tonsillectomy in School-Children

(From the Clinical Journal of December, 1938)

THE rise of the incidence of tonsillectomy is a major phenomenon of modern surgery. It has been estimated that 200,000 tonsillectomies are performed annually in this country, and that they form one third of all the operations performed in the United States. There are, moreover, remarkable features in the age, geographical and social distribution. The rising tide of the incidence of tonsillectomy is shown by the official figures of tonsillectomies on public elementary school-children, which increased from 42,004 in 1919 to 110,239 in 1931. In 1923 Sir George Newman issued the first of his many warnings against premature resort to operation. During that year the number was nearly 48,000. But notwithstanding his warnings, which were reinforced by a memorandum from the Section of Laryngology of the Royal Society of Medicine, the number mounted steadily. These admonitions, combined with those of Tilley, Layton, Paton and others began to take effect, and the number fell to 73,259 in 1934 but has since risen to 84,414 in 1937. The highest age-incidence of tonsillectomy is between the ages of 5 and 7 years. This period is a time of great change in the life-history of the child, both in his oral cavity and general development, and he then also exchanges for the first time the familiar "herd" infections of the home for the much larger and more varied infections of the larger "herd" of the school.

Function and Physiological Enlargement of the Tonsil

If the tonsils have a protective function, as some believe, "absorbing small numbers of organisms and so establishing immunity by gradual dosage," it would seem that they would be useful on the child's entry into the new environment of school. "To me," says L. W. Dean, "it is certain that the tonsils in infancy and early childhood are part of the defence

mechanism of the body. They protect the organism against those factors which cause them to become acutely swollen." If, again, there be a period of physiological enlargement between 4 and 6 years, tonsillectomy at this age seems open to question. It may remove tonsils which are enlarged physiologically, or in response to their protective function. Some of the improvement ascribed to tonsillectomy may be due to the normal physiological changes of this "critical age" as Hippocrates called it.

In the report of an inquiry into the catarrhal child by the Chief Medical Officer of the Ministry of Health, "unselected" control children showed a higher percentage of enlarged tonsils and adenoids than did "catarrhal" children. This supports the view that some physiological enlargement of the tonsil occurs between the ages of 4 and 6 years. Other investigations have shown that children with "hypertrophic" tonsils have a greater average height and weight than those with "normal" tonsils.

Drastic reductions in the numbers of operations performed in elementary school-children in certain areas (for example in Derbyshire, where owing to the reduction in the rate of operation, some 13,000 children have been spared the operation in six years), have had no unsatisfactory results.

Although tonsillitis seems common to all classes of society, the incidence of tonsillectomy is at least threefold heavier in the children of the well-to-do.

In 1932 Layton pointed out that the mortality directly due to tonsillectomy was greater than is usually appreciated. The Registrar-General's Statistical Review for 1935 includes a review of this mortality for the years 1931-1935. Enlarged tonsils or adenoids were given as the cause of 60 deaths, and tonsillectomy—without specification of the disease for which the operation was performed—was given as the cause of 513 deaths, 369 in children under 15. These numbers do not represent all the deaths following tonsillectomy in the five years, since there were 231 deaths with mention of tonsillectomy in conjunction with the disease of the tonsils necessitating the operation classified in tabulation to the particular disease.

The strange facts of incidence support the opinion given on other grounds by the Schools Epidemic Committee of the Medical Research Council that there is a tendency for the operation to be performed as a routine prophylactic ritual for no particular reason and with no particular result.

SULPHANILAMIDE IN INFECTIONS OF THE HAND

Mr. W. H. Ogilvie states that sulphanilamide has entirely altered the outlook in some of the commonest and most distressing problems, notably infections of the hand. A tendon-sheath or tissue-space infection formerly meant a ruined hand, usually a ruined life. Following Kanavel's anatomical researches the operative treatment was put on a sound basis, and many hands were saved with perfect function; but in all except the earliest cases the result was still imperfect. By the use of sulphanilamide in combination with early and correct incision the period of active infection and necessary drainage is now reduced to a minimum, and the good function that can come only with early healing results.

The Trichinopoly District Medical Association

THE tenth inaugural meeting of the Trichinopoly District Medical Association was held at 4 P. M. on 19th February, 1939 in the Government Headquarters Hospital, when Rao Bahadur T. S. Thirumurthi, Principal, Stanley Medical College, spoke on "Some modern conceptions regarding the Pathology of Tuberculosis." Lt. Col. N. K. Bal, I. M. S., District Medical Officer, Trichy, Lt. Col. T. S. S. Shastri, I. M. S., District Medical Officer, Tinnevely, and Major Sangam Lal, I. M. S., District Medical Officer, Salem, and representatives from Madura and Pudukottah were among those present.

Dr. P. A. S. Raghavan was "At Home" to the guests.

The Secretary made a statement with regard to the recommendation of the Managing Committee that subsidised medical practitioners should continue to be regular members of the District Medical Association instead of being an affiliated branch and explained the ways and means suggested for converting the association into a branch of the Indian Medical Association.

Lt. Col. Shastri explained how his association was functioning and appealed to medical men in the District to be members of the District Medical Association.

Dr. Thirumurthi in the course of his address said that a sound knowledge of tuberculosis would give them a basic foundation for a proper treatment of many other diseases. It was a common disease in all parts of the world and in India one in every seven deaths was due to that cause.

Evolution in the knowledge of infection

The first knowledge about Tuberculosis was that the virus in different parts of the body were all of the one and the same nature. Ninety years ago they were not able to collate the manifestations in different parts of the body, but after a time a great advance was made when they found that the disease

was communicable to animals. Now they had not only the pathology of the disease but also its treatment and knew how it was easy to prove their clinical findings by the X Ray demonstration and how surgery more than medicine was useful in the treatment of advanced tuberculosis. Without surgical aid, it was possible to treat early infections, but in the later stages they had necessarily to resort to Pneumothorax and other measures and the lecturer saw no reason why a large number of practitioners should not specialise in the treatment of artificial Pneumothorax. The general consensus of opinion seemed to be that early infection was recovered from, but there was a later infection which produced eventually the disease. As age advanced there were more possibilities of infection and that all of them probably with rare exception, were all tuberculosis infected.

Epidemiology of Tuberculosis

The statistics showed that the more civilised a Nation was, the higher was the percentage of the disease. They had not yet developed a natural immunity to the disease and hence they were not as a nation well tubercularised. The greater contact with people from other places, the increasing industrialisation, the urbanisation, the diminishing vitality, etc. were the various factors for the disease. Whereas in all other progressive western countries the death rate in Tuberculosis had been brought down to the minimum, in India the disease was slowly advancing. The lecturer emphasised that tuberculosis was one of the most curable of diseases and that medical men should preach that gospel to the public. They had a very great responsibility to diagnose the disease in the very early stages without fear or favour to anyone. The source of infection was through air, dust especially in dwelling houses and contact with tubercular patient.

Immunity to Tuberculosis

Immunity was the watch word in Tuberculosis. There were the Natural and Acquired immunity to the disease. Their safety lay in strengthening the Natural immunity. Acquired immunity to tubercular bacilli could be developed

in the same way as one could develop a certain amount of immunity to other toxins. But they were unable to produce anti-toxins for the tubercular bacilli and so natural immunity alone could be increased first and next the acquired immunity which was possible only by minimal infection.

Tuberculosis is also being carried into rural areas because the factories attract large number of people from the rural areas in times when they are not employed in agriculture, and when they go back they carry this legacy of tuberculosis from the city life. They get the infection into the village and spread it to all the people round about.

The Tubercle bacillus is a fat individual easily to be seen under the microscope. It has got a fat envelope all round it, a thick lipid coat, a third of the bacillus being the coat. The tubercle bacillus is, due to this lipid coat, like a soldier well armed. It is also very lethargic. It has no agility of movement. So to speak the Tubercle bacillus does not fly or run. When it goes to a place it sticks on to it. It cannot go through a mucous membrane. When it lodges on the surface of the membrane the polymorphonuclear leucocyte goes there with the purpose of attacking it; sometimes the tubercle bacillus succumbs to this attack; but at other times becomes very friendly with the tubercle bacillus and offers a kind corner in its own body and the tubercle bacillus does not die. It goes to another type of cell and it is carried again by these cells through the lymphatics and through the lymphatics to the next lymphatic gland. People have tried to fragment the Tubercle bacillus. It contains two other materials in addition to the coat, one is of protein nature and the other polysachrine. The latter does not give any trouble. The protein is of great interest to the clinician. This protein gives rise to the allergic condition in Tuberculosis.

Immunity in Tuberculosis varies in different people in individuals and also according to the age. Tubercle prefers a lymphoid tissue for its multiplication. There are two lymphoid channels in the lungs—the intrapleural and the sub-pleural lymphatics. In children the lymphatic vessels are broad. The stream goes easily and so the tubercle-bacilli which are carried

do not stay in the lung. In cases of old age the lymphatic drainage becomes more and more obstructed so that the Tubercle bacilli are arrested in the lungs.

Source of infection is always airborne. The tubercle bacillus is killed within half an hour in the sun-light. Houses are the chief sources of danger.

P. A. S. RAGHAVAN,

Secretary.

The Trichinopoly District Medical Association,
TRICHINOPOLY.

Statement of account for the month of February 1939

RECEIPTS			EXPENSES		
	Rs.	A. P.		Rs.	A. P.
Cash in bank	...	315 6 2	Clerk's pay (Jan. 39)	...	18 0 0
Cash in hand	...	0 1 6	Stationery	...	2 4 6
Members' Subscription.	54	8 0	Printing	...	45 4 0
Advertisement charges.	29	3 0	Annual Gathering ex- penses	...	62 8 0
Contribution to Annual Gathering	...	25 2 0	Postage & Telegram	...	8 14 0
Suspense a/c	...	9 0 0	Miscellaneous expenses	...	4 5 0
Last month's cheque realised	...	2 15 0	Cheque under realization	...	3 3 0
			Cash in bank	...	285 11 2
			Cash in hand	...	6 2 0
TOTAL ... 436 3 8			TOTAL ... 436 3 8		



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THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION

(REGISTERED)

The Secretary requests contribution on Medical, Topical and News Subjects from Members of the Association and other medical men. Contributions should be sent typed in duplicate and signed by the contributor. In case where it is desired to write under a nom-de-plume the signature must be included for the information of the Editor as a token of good faith.

March, 1939.

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