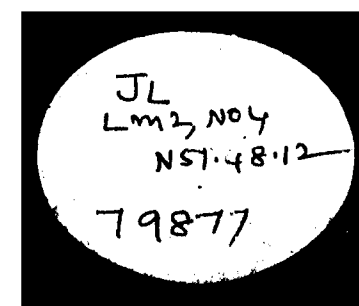




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Vol. XLVIII, No. 12

The Antiseptic

Estd. 1904

A Monthly Journal of Medicine & Surgery

Published on the 15th of every Month

Edited by: Drs. U. RAMA RAU & U. KRISHNA RAU, M.B., B.S.,

Asst. Editor: U. VASUDEVA RAU, M.B., B.S.

Editorial & Publishing Office: 323-24, Thambu Chetty St., Madras-1.

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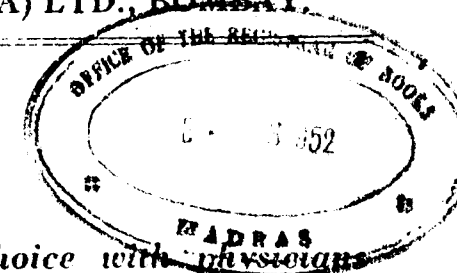
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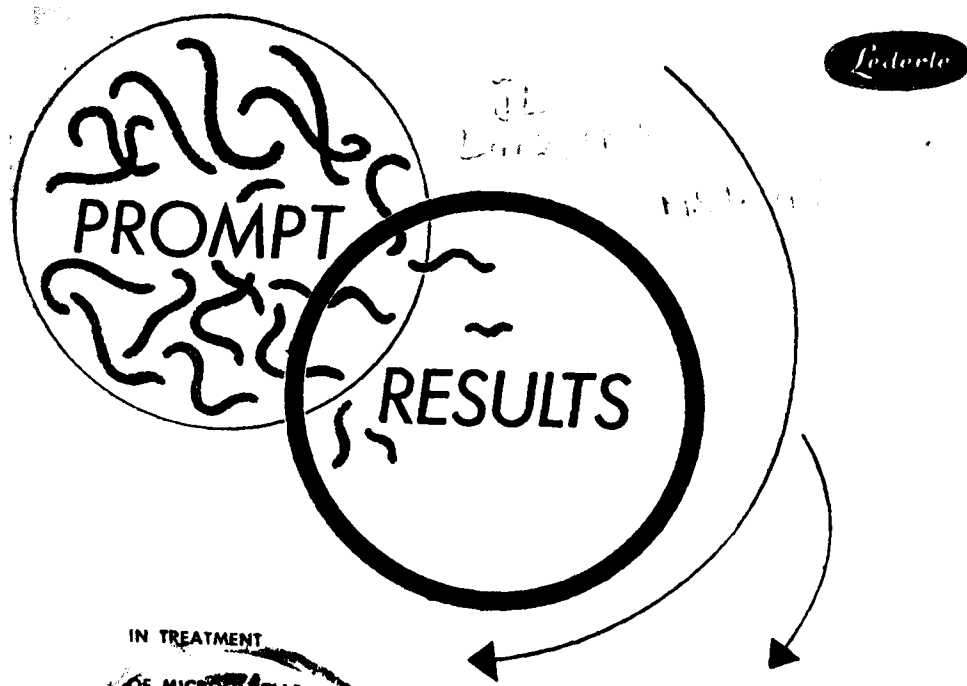
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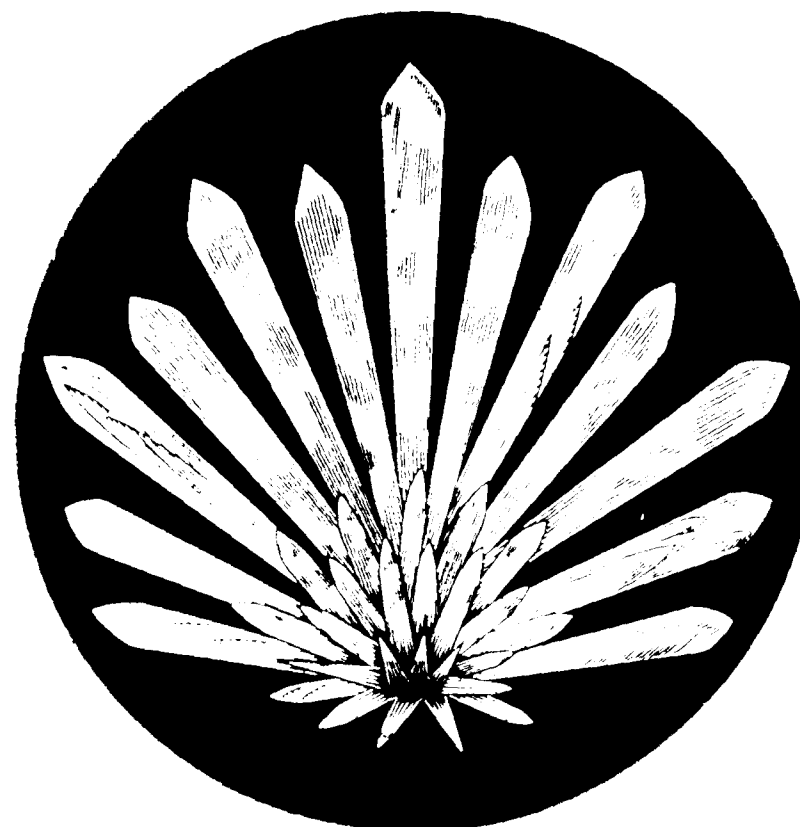
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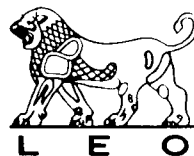
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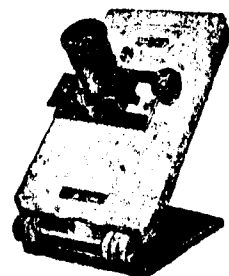


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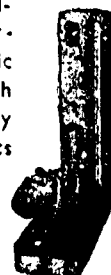
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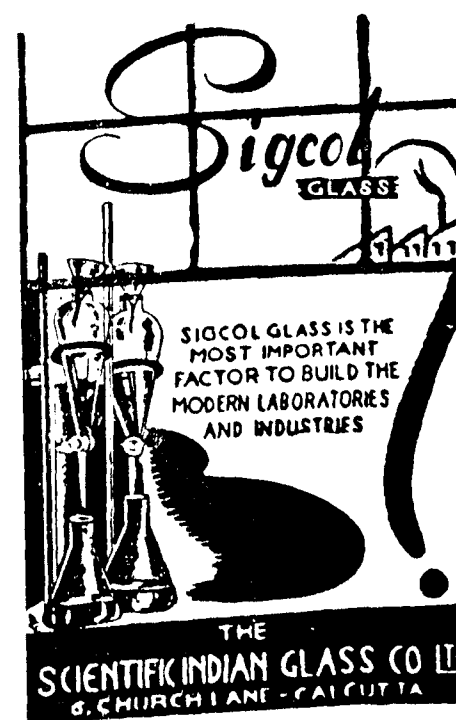
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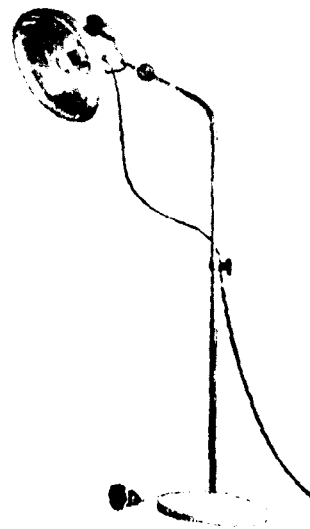
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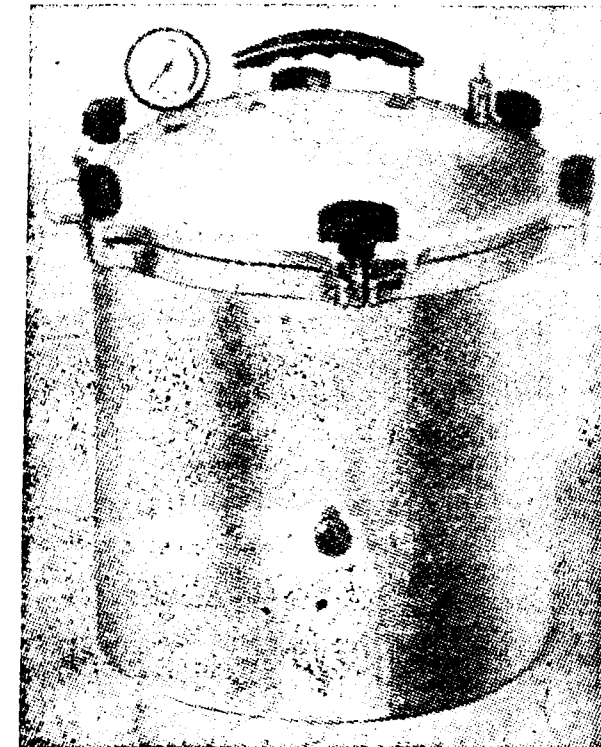
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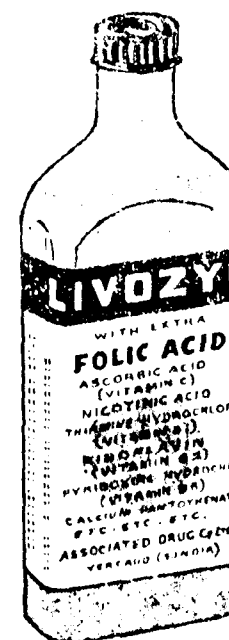
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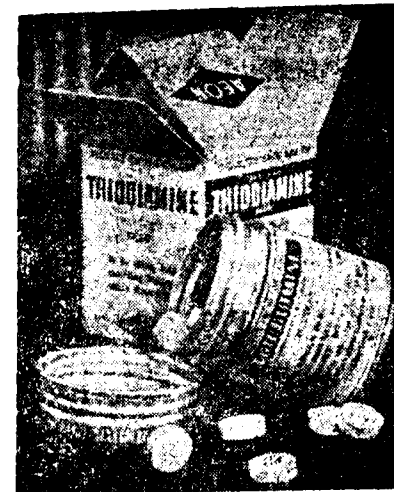
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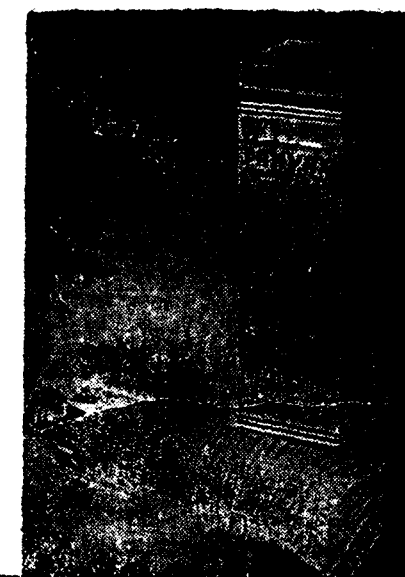
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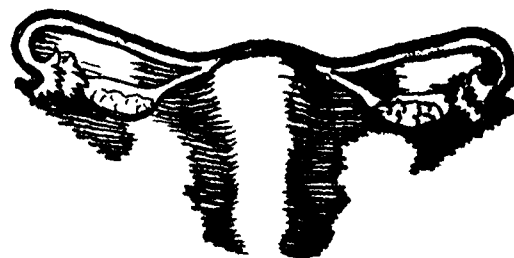
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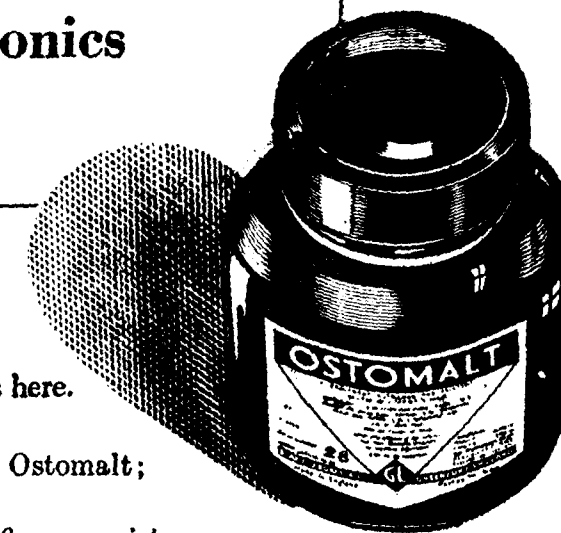
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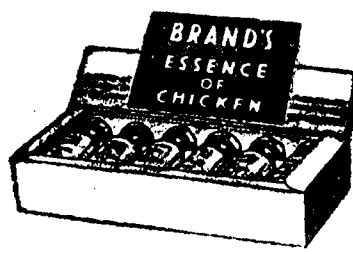
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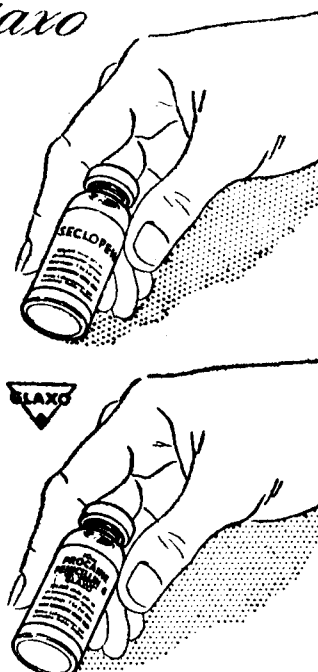
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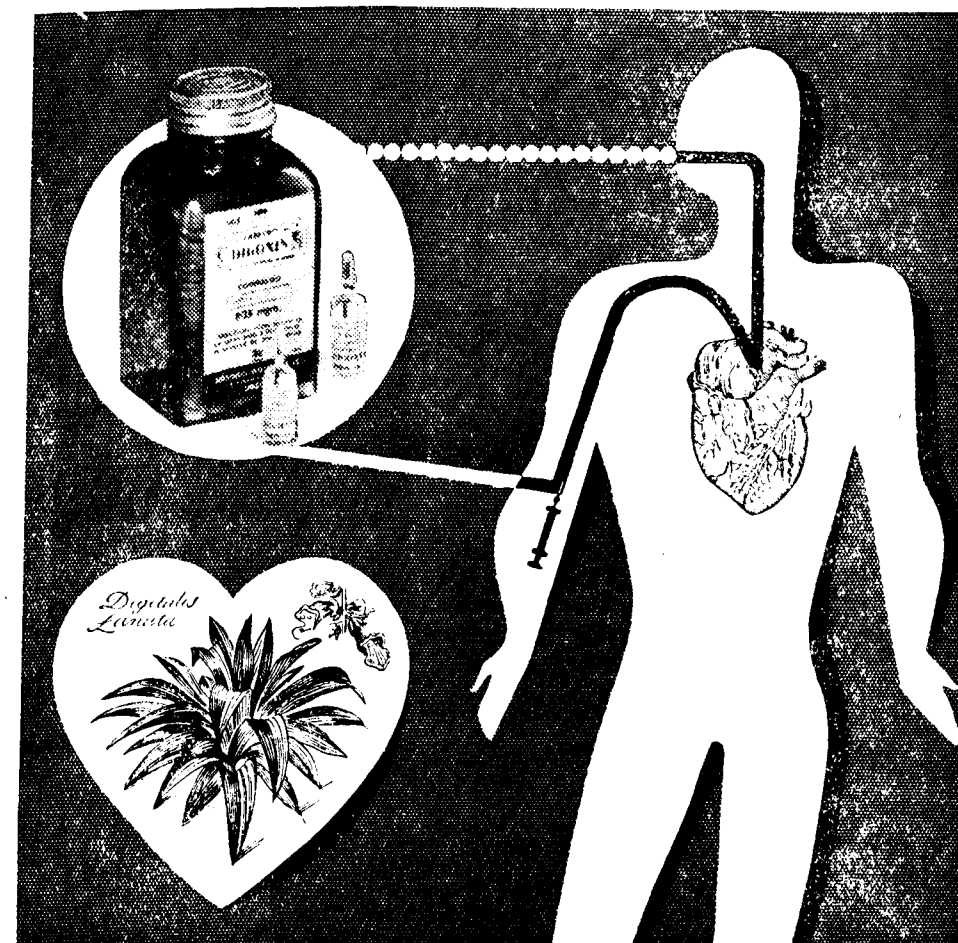
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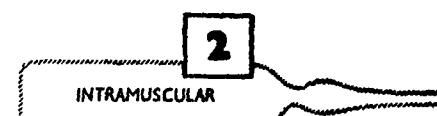
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Med. J. Australia, 11, 502, 1949.

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The Antiseptic

ESTD. 1904

A Monthly Journal of Medicine and Surgery

Published on the 15th of every month.

Editors: U. RAMA RAU & U. KRISHNA RAU, M.B., B.S.

Asst. Editor: U. VASUDEVA RAU, M.B., B.S.

Editorial & Publishing Office:

"RAMARAU BUILDINGS," 323-24, Thambu Chetty St., Madras-1.

Annual Subscription: Rs. 7-8-0: Foreign—Rs. 10—Post Paid.

Vol. 48

DECEMBER, 1951

No. 12

Original Articles

CHOLERA—ITS TREATMENT*

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R.M.O., I. D. Hospital, Madras-21.

As it is not possible to speak on the subject of cholera in detail in such a short time, I confine myself only to one aspect of it, viz., the treatment, and in that too, only the most important aspects of it. Apart from that, a few things worthy of mention, which have been observed in our experience, I think, will not be out of place.

During the period under review, viz., 1943 to 1950, 8,333 cases of suspected cholera were removed to the I.D. Hospital, Madras, from the City, of which only 3,898 i.e., 47.7% were declared bacteriologically positive. Of the remaining, gastro-enteritis of all varieties and dysenteries, acute and chronic, amounted to 4,158 and 277 cases belonged to other groups of cases such as myocarditis, anaemia, starvation, pneumonia etc. There was an instance when, even a case of strangulated inguinal hernia was sent by one of the City State Hospitals as a case of cholera. This gives you a picture of how an Infectious Diseases Hospital, becomes, so to say, a dumping ground during epidemics—I am sorry to say—for all cases who complain of diarrhoea and vomiting, irrespective of whether or not there is ever the slightest suspicion that they may be cases of cholera. It will not be out of place, though it is beside the point, when I say, any destitute lying on a road becomes a case of cholera for an onlooker who passes by, since no other ambulance is

* Read before the Annual Conference of the Corporation Medical Officers' Association.

so very obliging as to pick up such cases freely and readily and no other hospital accommodates them so ungrudgingly as our ambulance and hospital.

I do not profess to know anything about the epidemiology of this disease, but from the figures available with me regarding the admission of cases of cholera during the last 20 years into the I.D. Hospital, Madras, there does not seem to be any periodicity in the occurrence of the cholera epidemic, though it has been the general impression that this disease takes an epidemic form once in five years. But it is true that it occurs seasonally, though stray cases are admitted throughout the year. Yet, either localised or generalised epidemics occur only during the period of August to February. Nearly half the total number of cases occurred during the months of September and October alone, and the months of March to June have been comparatively free, the incidence in those months being only 5%. This statement may be quite at variance from that of Rogers who said, "In Madras the maximum cholera season occurs during the hot months and the next highest rate being in the comparatively warm and dry season, i.e., from December to February". Perhaps it might have been true in his days. As with everything, seasons too have changed and, with them, the incidence of diseases.

Cholera is a disease characterised by a sudden onset of severe diarrhoea followed, after a few hours or less, by vomiting, resulting in severe dehydration and collapse with cramps and suppression of urine. I don't propose to deal with these clinical factors, but there are two or three points requiring special mention. Firstly, all cholera motions are not typically "rice watery" though a majority of them are. In a small percentage of cases, frank and pure blood was noticed in large quantities both in the motion and vomit and these were bacteriologically positive and a majority of them proved fatal.

Another important thing that should be borne in mind is that in cases of cholera, skin temperature is most unreliable. Sometimes it was found that axillary temperature is lower by even 8 to 10°F. than the rectal temperature and this is due to peripheral vascular failure and vaso-motor disturbance, with the result that only rectal temperature should be recorded and depended upon.

Collection of specimens of the motion for bacteriological examination should be done carefully. The method employed in our hospital is this: As soon as a case is admitted, a sterilized rubber catheter is introduced into the rectum and the motion is allowed to flow into an autoclaved test tube containing about 5 cc. of 1% alkaline peptone water of P.H. 8.2. Such inoculated peptone water medium is sent to the Public Health Laboratory for culture and report.

Clinically these cases of cholera have to be differentiated from so many other conditions which simulate cholera and are removed in large numbers as suspected cases during epidemics.

They are:

- (1) Acute gastro-enteritis;
- (2) Acute bacillary dysentery of choleraic type;
- (3) All cases of food poisoning;
- (4) Heavy metal poisoning like arsenic etc.;
- (5) Algid malaria;
- (6) Toxic and infective conditions associated with diarrhoea and vomiting as pneumonia, typhoid, influenza, pleurisy, meningitis etc.;
- (7) Acute abdomen associated with vomiting and diarrhoea;
- (8) Ascariasis;
- (9) Chronic enteritis as tubercular, senile etc.; and
- (10) Lastly other conditions as starvation, deficiency diseases etc.

Excepting the acute gastro-enteritis and choleraic type of bacillary dysentery, all other conditions can easily be distinguished from cholera. But these two may have to be diagnosed only bacteriologically.

The treatment of cholera mainly falls under two heads:

I. Replacement of fluids and salts, lost through evacuations.

Immediate attention has to be paid to this most important aspect of the treatment of cholera since, almost only on this, the success of the treatment depends. No time should be lost in starting the saline transfusions whenever indicated. Initiation and maintenance of the blood circulation, which is impaired as a result of severe dehydration and consequential rise in the viscosity of blood, should be the first aim in the treatment of this disease and that can be done only by diluting the blood with saline. The replacement of fluids can be done in several ways and they are:—

(1) *By mouth*:—Perhaps it is the simplest but not always successful. Sometimes it may be positively harmful too. There have been many instances in our experience, when a few ounces of water, given as a feed, to an acute case of cholera with mild collapse, has induced severe vomiting of one or more pints of fluids and made the patient collapse more. But in cases where there is not much vomiting, or the vomiting had subsided, a few sips of water, glucose water, or barley water, preferably iced, will certainly not only help the replacement of fluids but also satisfy the patients' thirst.

(2) *By rectum*:—Normally the large intestine absorbs small quantities of water and a few drugs. In cases of cholera where the diarrhoea is severe, it is rather doubtful whether anything can be absorbed per rectum. Yet in mild cases rectal normal saline given by drip method has helped many patients to tide over further collapse.

14.3% three Salines, 3.45% four Salines and the remaining 2.3% had five Salines. Each adult, on an average, required 2.02 Saline transfusions and a child 1.68. An adult required 5.96 pints and a child 2.81 pints of Saline on an average for a cure. The maximum total amount of Saline was 15 pints per case and there were four such cases in this series and all of them survived. During the course of these 8 years out of these 3,898 cases the maximum number of transfusions to a case was 8, with the total amount of 28 pints of Saline and he survived.

Very little purpose is served if the Saline transfusion is given slowly by *Drip* method when the blood circulation is impaired and has to be initiated with minimum waste of time. Our experience here showed that the danger in giving saline transfusion rapidly is very much exaggerated and no case has died of cardiac embarrassment or mediastinal flutter. With the Saline bottle at about 3 feet high from the bed level, the Saline is allowed to flow without a regulator. This will come roughly to 3 to 4 oz. per minute. But if the patient shows any signs of cardiac embarrassment as evidenced by heaviness in the chest and restlessness, or other signs as severe headache or vomiting etc., the rate should be regulated. In cases with some cardiac or pulmonary complications, obese subjects, the very old, and the very young, the speed must be reduced and the Saline be run rather slowly so as to avoid some untoward complications.

II. The next important part of the treatment is the use of drugs acting against the vibrio. Various drugs have been tried and their effects were studied during the period. The original treatment of Rogers was in use continuously for a considerable time till the end of the year 1942 which consisted of:—

Mist pro-diarrhoea (Essential oil mixture) minims 10 every half hour for 24 to 36 hours, or till the nature and consistency of the motions change, and Pot. Permanganate pills 2 gr., Keratin or Salol coated, one every half hour, for 24 hours, or till the colour of the motion changes, whichever is earlier.

During the epidemic of 1942-'43 under the auspices of I.R.F.A. and under the guidance of Dr. Venkataraman, the late Director K.I.P.M., Guindy, Sulphaguanidine was tried against the control treated with Rogers' method. Out of a total of 731 cases 207 were treated with Sulphaguanidine and the remaining (524) with the Rogers'. Sulphaguanidine was administered in powders of 5 gms. stratum followed by 2.5 gms. every 4 hours upto a total of 20 gms. The results were rather disappointing, the mortality rates in both the groups almost being the same, viz., 28.8%, with the drug and 28.5% with the control. In subsequent years, till 1948, other drugs like Sulphathiazole and Sulphadiazine have been tried in a few cases with no encouraging results.

During September-October 1948, Phthalyl-Sulphathiazole (Thalazole of M. & B.) was tried on experimental basis on 196 bacteriologically positive cases of cholera. The dosage was 1 gm. every 4 hours for 48 to 72 hours. The control was once again the routine treatment of Rogers. The results were promising. Apart from the difference in mortality percentage which is lower by 5% in Thalazole treated cases, this new drug has the following advantages:—

- (1) It is easier to administer unlike R.T.
- (2) It does not produce nausea or vomiting unlike Mist pro-diarrhoea.
- (3) It makes the motions formed quicker than with R.T. On an average it takes 76 hours with Thalazole as against 102 hours with control.
- (4) Though no actual figures are available, it was felt in general, that the motions became culturally negative quicker with this drug than with the R.T., thus reducing the patient's stay in the hospital.

After this trial and since the beginning of 1949, the Rogers' treatment has been completely replaced by Thalazole, the dosage being 2 gm. stratum followed by 1 gm. every 4 hours for 48 hours. As I have already told you, there was a switch over to Glucose Hypertonic Saline from the original 'Rogers' at the same time.

Comparing the mortality figures of these two groups of years, viz. 1943 to '48, when the treatment was that of Rogers' both in Saline as well as in drugs, and 1949-'50 when the cases were treated with Glucose Hypertonic Salines and Thalazole, there was a marked decrease in the percentage of mortality. 543 persons died out of 2,238 positive cases treated during the six years ending with 1948 with a mortality percentage of 24.2, whereas 229 died out of 1660 cases during the years 1949 and 1950 with a mortality of 13.8%. This difference is considerable and certainly due to this drug as well as the Saline.

During the latter part of the year 1949, once again, under the auspices of I.R.F.A., their Cholera Unit worked in the hospital for about two months, studying the effects of Forma—Cibazol of Ciba against a control treated with Rogers' treatment. Though their detailed report is not available, from their preliminary note on the investigation, it was found that the drug was not so effective as it was claimed to be. Dr. Bhatnagar in his article in *B.M.J.*, (1948) says that 57 out of 85 cases of cholera treated with this drug, were in a critical condition with anuria, marked dehydration, semi-comatose condition, algidity, imperceptible pulse and body cramps, and none of them were given any Saline transfusion and in spite of that the mortality rate was only 3.52%. This is certainly surprising and it is a great wonder how a case with severe dehydration and collapse could be cured so easily without I.V.S.

And lastly coming to another drug which has come into the field of cholera recently is 'Thiodiamine.' This *Thiodiamine* is derived from the decomposed bark of a tree—*Cratæva Roxburghii*—found in some of the plains of India but grown abundantly in East Bengal. This was first isolated in a pure crystalline form by Dr. J. S. Chatterjee and put up in the form of tablets, each tablet containing $\frac{1}{2}$ mgm. by Aeon Chemical Industries Limited, Calcutta. This has an empirical formula $C^{15}H^{16}N^{23}$.

Under the instructions of the Director of Public Health with the Government of Madras and with samples supplied to us by Messrs. Raka Corporation Limited, (39 Second Line Beach), Madras, this drug has been tried in a few cases. Unfortunately the number treated is too small to help me to draw definite conclusions about its efficacy. For the sake of convenience, only male cases, irrespective of the severity and whether they were likely to be positive or negative, were taken up for the trial. Every other case was put on Thiodiamine, the other being the control treated with our routine Thalazole. For purposes of this investigation, only cases that were positive for cholera are taken up. Motions of these cases were sent for bacteriological examination daily and after getting three consecutive negative results, these cases were discharged. During the period between 26th June to 21st August, in all 91 males were admitted as suspected cases of cholera of which only 30 were declared positive. Exactly half of them were treated with Thiodiamine and others with the R. T. and the following are the results:—

(1) On an average each case required 12 tablets for a cure as against 16.6 tablets with Thalazole.

(2) It took 3.2 days with Thiodiamine as against 1.9 days with the R.T. for the motion to become culturally negative. Perhaps this difference would not have been so much if we had given a heavier dosage of Thiodiamine. In the beginning a maximum of 10 tablets of Thiodiamine only were given but later on it was found inadequate and it was increased. There is a possibility for the motion to become negative quicker with an increased dose.

(3) Cases treated with Thiodiamine required on an average 3.7 $\odot$ of Saline as against 4.5 $\odot$ with the group under control. It took 1.7 days with Thiodiamine for the motion to become 'formed' as against 1.4 days with the control. This slight difference is not of much significance, since 26% of the cases treated with the control were mild even at the time of admission.

(4) 3 cases with Thiodiamine and 3 with Thalazole died out of 15 with a mortality of 20% with each group. But the number of cases treated is too small to attach any importance for these mortality rates.

(5) No complications have been noticed on either side.

Though it is difficult to conclude definitely about the efficacy of the drug, from these results, one can say that Thiodiamine is:—

(a) Non-toxic; (b) It has got a definite action against the cholera vibrio; and (c) It is as good as Thalazole.

To arrive at any definite conclusions, a thorough trial should be made on at least 500 cases on each side with an increased dosage, say 4 tablets every 8 hours for 48 hours for both the drugs.

I would like to conclude this paper with a note of warning that the value of a drug cannot be estimated from the mortality rate as the prognosis of cholera cases depends upon several factors, viz.:

- (1) The delay in the removal of the case.
- (2) The degree of severity of dehydration.
- (3) The age of the patient.
- (4) And lastly, associated conditions.

What we should expect in a good drug are:—

- (1) It should be non-toxic.
- (2) It should be in an easily administerable form.
- (3) It should make the motions formed and *negative-bacteriologically—quicker* so as to minimize the quantity of saline and the stay of the patient.

Control of Pulmonary Hæmorrhage by Pituitrin

Thirty-two patients with pulmonary hæmorrhage were given 10 international units of pituitrin with 10 cc. of normal saline intravenously. The injection was made slowly over a 10 minute period. The hæmorrhages were due to various causes, such as tuberculosis, bronchiectasis, tumours, coccidioides, heart disease etc. Hæmorrhages were severe, were not immediately fatal, but if allowed to continue might have produced other complications, such as atelectasis and other interference with the bronchial drainage or eventually ex-sanguination. Dizziness, slight abdominal cramps, an urge to empty the bowels and bladder, and nausea were transitory effects which rapidly disappeared. Prompt control of the bleeding was obtained in 31 of the 32 patients. In the single case which terminated fatally, the dose was only 5 international units instead of the 10 usually recommended and might have been a contributory factor to the patients' continued bleeding and death. In general one intravenous injection will control the severest pulmonary hæmorrhage from whatever cause.—*Dis. of Chest*, 18, pp. 345-352, Oct. 1950.

HEMOLYTIC DISEASES OF THE NEW-BORN*

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THOUGH the primary process is hemolysis of R.B.C in the new born and erythroblastosis—presence of a large number of immature nucleated red cells (erythroblasts) in peripheral blood is merely a response to this destruction, this group of diseases is called *erythroblastosis foetalis*.

There are 3 pathological groups :—

I. Hydrops foetalis.—This is a most severe form ; the infant is born moribund or is still-born. There is generalised oedema, effusion into serous cavities, severe toxæmia and jaundice.

II. Icterus gravis neonatorum.—Jaundice may be present at birth or may develop within 48 hours. This is the most common type. Kernicterus may supervene later.

III. Congenital hemolytic anæmia.—The infant may be normal at birth but develop severe anæmia in a few days (about 10).

To understand the etiology and pathogenesis of this disease (E.F.) it is quite essential to have a comprehensive idea about the Rh factor and its variants and about iso-immunization. The recent outstanding discovery of Rh factor in 1940 by Landsteiner and Weiner has provided not only an explanation for certain hitherto obscure hemolytic accidents in blood transfusion but also has made clear the pathology of E.F.

HISTORY.—In 1900 Landsteiner attributed the hemolytic reaction occurring in some blood transfusions to the existence of 4 different blood groups (ABO system) i.e., the presence or absence of iso-agglutinogens A, B in the red cells and of iso-agglutinins in the serum and to the action of the agglutinins of the serum of the recipient upon the agglutinogens of RBC of donor. In 1927 the presence of 2 new hemagglutinogens M and N, quite unrelated to A and B, in the red cells, were revealed (Landsteiner and Levine) and in 1930 it was discovered that blood group A contained 2 subgroups A_1 and A_2 and thus 18 distinct types of blood became known. Further variants in M & N corresponding to the subgroups in Group A as well as another antigen S were recently discovered (Weiner 1949). The M—N types are of no importance in the selection of blood donors as there are no corresponding agglutinins anti M and anti N in human sera, but their determination, though difficult, is useful in medico-legal work, because M and N are inherited according to Mendelian laws. Some more hemogens such as P, Q, G, H have been found in human blood but have not been studied fully.

* Specially contributed to THE ANTISEPTIC.

[972]

Rh factor.—During the last decade it was seen that hemolytic reactions occurred occasionally in blood transfusions even after matching properly according to the ABO groups. Landsteiner and Weiner discovered the cause in 1940 when they were attempting to prepare anti-M serum. They injected the RBC of *Macacus rhesus* monkey (Brown Indian) into rabbits and an antibody developed in the latter's serum (anti Rh). When that rabbit's immune serum was again mixed with red cells of the rhesus monkey or with RBC of majority of human beings (85% of American and European whites, 93—95% of Indians, 98% of American Negroes) there was agglutination. This showed that these people, irrespective of their ABO group, possessed this agglutinin of rhesus monkey in their RBC. Thus came to light this new factor, the most commonly observed cause of intra-group incompatibility ; it was called *rhesus factor* (Rh) because it was first discovered in the RBC of rhesus monkey. Those persons whose RBC were agglutinated by this serum were called Rh +ve and the others were Rh —ve or rh when their red cells lacked this Rh hemogen. These Rh —ve people should not receive transfusions of other than Rh —ve blood if severe and fatal reactions are to be avoided. With Rh +ve individual the Rh group of donor does not matter. Rh factor is thus an antigenic substance present in RBC and tissue cells of majority of people and capable of giving rise to antibody formation when introduced into the body. It is protein in nature and is inherited as a Mendelian dominant. Normally human plasma does not contain the anti Rh agglutinin.

Weiner in 1942 showed that Rh factor was not a single entity ; he also studied the question of its heredity. Davidson and Tharskey ('42) also stated that the subdivision of Rh factor was complex. In 1943 Race and Taylor stated that Rh was of 2 types inducing the formation of 2 types of antibody. Weiner also came to a similar conclusion. Rh_1 (70%) are those whose red cells reacted with his new anti serum anti Rh_1 (containing rh_{01} agglutinin) as well as anti Rh serum, Rh_2 (15%) are those whose cells reacted with anti Rh but not with anti Rh_1 and rh (15%) are those who reacted with neither. Thus first 3 allelomorphs Rh_1 , Rh_2 , and rh were described giving rise to 6 genotypes Rh_1 , Rh_1 (20%), $Rh_1 Rh_2$ (14.4%), $Rh_1 rh$ (35.1%), $Rh_2 Rh_2$ (2.6%), $Rh_2 rh$ (12.5%) and $rh rh$ (15%). The third anti-serum anti Rh_2 was detected in 1943, (Race *et al*). Four more rare allelomorphs Rh' , Rh'' , Rh_o , Rh_y (1% or less) were found out later (Weiner, Race and Taylor) ; with these 7 allelomorphs there are 28 genotypes. The eighth subtype Rh_z was found out in 1945 (Race *et al*). Two more sera anti Rh' and anti Rh'' were discovered afterwards but they were found out to be respectively only anti Rh + anti Rh_1 serum ($rh_o + rh_{01}$) and anti Rh + anti Rh_2 ($rh_o + rh_{02}$). As anti Rh' serum is very common, use of Rh' blood is risky in transfusion ; but Rh'' blood is much less risky. Weiner, using the 3 antisera anti Rh, anti Rh' and anti Rh'' recognised 8 Rh antigens Rh_o ; Rh_o'

or Rh₁; Rh₀" or Rh₂; Rh₀ Rh' Rh" or Rh₁ Rh₂ (Weiner) or Rh₁₋₂ or Rh₂ (Taylor); Rh' (now rh'), Rh" (now rh"), Rh' Rh" (Weiner) or Rh' Rh" or Rhy (Taylor and Race) and rh. The R.B.C. containing these 8 antigens are agglutinated by the following sera: (1) anti Rh₀, (2) anti Rh₀ and anti Rh'; (3) anti Rh₀ and anti Rh"; (4) anti Rh₀, anti Rh' and anti Rh"; (5) anti Rh'; (6) anti Rh"; (7) anti Rh' and anti Rh"; and (8) none of the 3 sera respectively. Of these the first 4 types are Rh + ve and or dominant while the remaining 4 are Rh -ve and are of recessive character.

R. A. Fisher used his own terminology and nomenclature to describe these Rh allelomorphs and antibodies, basing on his assumption that the Rh antigens were inherited from 3 genes occupying a chromosome and that each of these 3 genes had 2 attenuate forms C or c, D or d and E or e so that the 8 Rh antigens came to be known as cDe, CDe, cDE, CDE, Cde, cdE, CdE and cde respectively. Race in 1944 described this scheme of nomenclature. However this is not as simple as the old terminology of Weiner which is based on the genetical conception and on the assumption that each chromosome contains only one out of these 4 elementary and 4 compound genes.

Now the Hr factor, the new antigenic gene in the R.B.C. came into the field (Fisher and Race) and complicated the matter. Corresponding to the 3 Rh antigens Rh₀, Rh' and Rh" there are 3 antigens Hr₀, Hr' and Hr". Genetically Rh₀-Hr₀, Rh₀'-Hr' and Rh"-Hr" are the 3 pairs of contrasting characters and Rh₀ is the allele of Hr₀, Rh' of Hr' and Rh" of Hr". According to the composite gene theory of Fisher and Race which states that each chromosome contains either one or two or all the 3 Rh genes together with the noncontrasting Hr genes or gene, a person having only Rh₀ antigen in R.B.C. will also have Hr' and Hr" i.e. his phenotype will be Rh₀ Hr' Hr", a person with Rh₀ Rh' is phenotypically Rh₀ Rh' Hr" and so on. So the 8 phenotypes are Rh₀ Hr' Hr", Rh₀ Rh' Hr", Rh₀ Rh" Hr', Rh₀ Rh' Rh", Rh' Hr₀ Hr', Rh' Rh₀ Hr', Rh' Rh" Hr₀ and Hr₀ Rh' Hr". According to this theory rh gene is not true recessive as it has got Hr₀ Hr' Hr" while according to the multiple gene theory of Weiner, rh gene is recessive and has no antigen.

Rh testing.—It is necessary to test the blood when multiple transfusions have to be given or when pregnant, puerperal women and newborn have to be transfused. The Rh factor requires special tests for its detection. With animal serum one can determine the Rh +ve or Rh -ve states of blood i.e. rhesus grouping and with human sera one is able to determine the Rh phenotype i.e. rhesus typing blood.

(1) *Tube technique*:—3 standard drops each of anti-Rh, anti Rh', and anti Rh", sera in each of 3 Wassermann tubes and to each 3 drops of 2% suspension of R.B.C. to be tested are added; contents are mixed by agitation and then they are placed in an

incubator for 2 hours at 37°C and the result read by noting the appearance of the cell deposit in comparison with a control tube containing only the R.B.C. in isotonic saline without any serum. Agglutination is present if the edge is irregular or takes the form of scattered dots while it is absent if edge was clear cut.

(2) *Slide technique*:—Into each of 3 compartments on a slide one standard drop of each of the 3 sera are put and then a standard drop of R.B.C. suspension (50%) and the drops mixed. The slides are placed in an incubator for 1 hour and the fluid agitated every 15 minutes. After 1 hour the result is read with a hand lens or using a microscope for agglutination.

(3) *Biological test* which is practical and simple should be done if Rh testing could not be done. First 10 c.c. of recipient's blood is drawn into citrated and non-citrated tubes. Then 50 c.c. of donor's blood is injected into the recipient slowly; after 1 hour again 10 c.c. of recipient's blood is drawn out and compared with the previous tubes for hemolysis, which, if present, meant that there was antibody in recipient's serum.

Iso-immunization.—If any Rh antigen is introduced into the circulation of one who does not possess it the production of corresponding antibody is stimulated in the recipient. As plasma does not usually contain the anti-Rh agglutinin the transfusion of Rh+ blood in Rh -ve persons does not at first cause hemolysis but the formation, as a slow process, of antibody (i.e.) anti-Rh agglutinin in the serum of more than 50% of recipients but not in all, in about 3 weeks to 3 months (i.e.) *Iso-immunisation or Rh sensitization* occurs. However if such a recipient in whom this antibody production has been sufficient, is subsequently transfused with the blood of Rh +ve donor again, there is danger of an antigen antibody reaction, (i.e.) of hemolysis. The danger of this accident increases with each subsequent transfusion as the anti Rh agglutinin titre rises higher and higher. A few had these intragroup reactions at the first transfusion itself of apparently compatible blood and they were found to be either Rh -ve pregnant or women with children. In 1940 Levine and Stetson studied this and found that these Rh -ve mothers carried a Rh +ve foetus, which had inherited the Rh antigen from the Rh +father during the third month of its intrauterine life, and during that pregnancy developed anti Rh antibodies in response to the foetal Rh antigen and thus became iso-immunized. Antibodies were demonstrable in the last trimester though absent 6 weeks previously; the titre increased even after delivery and then after some period began to fade away. So when these women received transfusion with Rh + blood, hemolytic reactions occurred. Else when sufficient anti-bodies were formed in the mother they passed back to the foetus and acted on its Rh agglutinin causing a severe reaction and *hemolytic disease of new-born*. Thus the foetus in its turn may suffer from the action of mother's

agglutinins provoked either by its own Rh factor or by previous transfusions with Rh+ blood. Even Rh+ persons may be sensitized; iso immunization among the various Rh -ve types also is possible. There is evidence that the antigens in foetal blood other than Rh (such as A, B) may also immunize the mother and be responsible for foetal morbidity. Thus the Rh -ve persons may acquire sensitivity to Rh factor in one of 3 ways:—

- (1) In both sexes by transfusion of Rh+ blood.
- (2) In women when they become pregnant with Rh+ foetus.
- (3) In both sexes by I.M. injection of Rh+ blood for hemotherapy.

Mechanism of iso-immunization.—Levine's theory for the transfer of the antigen across the placenta is that there is only a single layer of syncytial cells separating the foetal blood from the maternal sinuses and these surface areas afford ample opportunity for the passage of the antigen in the last trimester of pregnancy. This idea is compatible with clinical observations made. But Weiner's theory is that iso-immunization occurs only during labour (*i.e.*) at the time of separation of placenta. Morgenroth's observation is that theoretically maternal immunization could be brought about either by the escape of foetal RBC or by the diffusion or group-specific, water-soluble substances across the placenta into the maternal circulation. It has been proved that latter is the case.

Pathogenesis of erythroblastosis.—The observation by Levine in 1939 that foetal blood may in some cases iso-immunize the mother paved the way for the correct description of pathogenesis of E.F. It has been found from statistical analysis of blood of pregnant women (Rh factor, blood groups) that the incidence of abnormality, in new-born (still birth, premature birth, E.F.) occurred much more when there was Rh incompatibility between mother and foetus. Some hold that Rh incompatibilities do not cause abortion, others however hold that though they are not major causes yet may play at least minor etiological roles in causing repeated miscarriages. However, it is definitely known that the highest incidence of hydrops and icterus gravis occurred in groups of Rh incompatibility and of congenital anaemia in groups of ABO incompatibility. It is possible that progressive lenticular degeneration (Wilson's) may be related to Rh incompatibility which also caused in some children low grade mental deficiency (Yannet and Lieberman).

If the antigen-antibody reaction due to Rh incompatibility is very severe and occurs in early months of pregnancy, the foetus may die and abortion caused; if it is severe and is in later months, the foetus may be still-born and with Hydrops; if moderate there may be severe jaundice and if slight, there may be congenital hemolytic anaemia. Usually (in 90%) the mother is Rh -ve;

foetus Rh +ve the father being Rh +ve; in the rest 10% the mothers were Rh +ve and their iso-immunization was attributed to the new Hr factor or to finer differences within the Rh complex or to the several other blood factors (other than Rh). Thus in a few cases of EF no Rh incompatibility could be detected but there was present ABO incompatibility and the causative factors were anti A or anti B, anti M or anti N or some other unidentified agglutinin. The first baby usually escapes the disease, as the mother would not have developed sufficient antibody titre, unless blood transfusion has already caused antibodies in mother previously. The titre becomes progressively increased with each succeeding pregnancy and with it the severity of hemolytic disease as well.

(1) The finding of iso-immunisation in a woman during pregnancy does not mean that the unborn child will suffer from E.F. even though the father is Rh +ve, as if the latter is heterozygous the foetus may be Rh -ve and unaffected. The *phenotype of baby depends on whether father's genotype is Rh Rh or Rh rh*; about 47% of Rh +fathers are heterozygous (Rh rh) *i.e.*, dissimilar genes are contained in the chromosomes and there is only 50% chance of foetus being Rh+. If the Rh +ve father is homozygous (38% cases) *i.e.*, Rh Rh—similar genes being present in chromosomes, the foetus will always be Rh +ve. The St. serum obtained by Race and Taylor ('43) reacted with blood of heterozygous people and the homozygous were St -ve and of genotype Rh₁, Rh₁. In the remaining 15% the father is Rh -ve.

(2) The *low incidence* of EF is also due to the failure of Rh -ve women to respond to iso-immunisation, though there is Rh incompatibility. The *mother's response*, to the call of the antigen, however strong, may be poor and antibody production low. This capacity to produce antibodies may be determined by some genetic factors.

(3) Another factor responsible is the *strength of foetal antigen* to stimulate sufficient antibody formation.

(4) The *extent and type of antibodies* formed by the mother.

(5) Variations in the permeability of the placenta to the passage of Rh factor and the anti Rh. Thus due to any of the above causes many normal babies may be born of Rh -ve mothers and homozygous Rh +ve fathers and variations in the severity of E. F. disease from baby to baby may be present.

Blocking antibody.—Only in 50% of Rh -ve mothers with history of repeated erythroblastic babies the presence of agglutinins which is direct proof of iso-immunisation, could be demonstrated even if tested soon after delivery; the remaining mothers did not show anti Rh by the routine procedures, though they also were actively iso-immunised but they had in them antibodies called BLOCKING

(Weiner) or incomplete (Race) or hyperimmune antibodies which are mostly of Rh₀ specificity and which can be demonstrated by the Blocking Test. In the latter women the agglutinin titre had gradually increased due to repeated exposure to the foetal antigen and then had disappeared altogether. Weiner as well as Race '44 recognised this blocking antibody, which united with homologous rh +ve red cells in vitro and coated the surface without inducing the visible effect of agglutination.

In 1937 Brand found atrophic cirrhosis of liver in a child which died of icterus gravis. Henderson '42 found intralobular connective tissue increase in the liver of many still-born infants and of those with E. F. Drummond and Watkins '46 found enlargement of liver and spleen in children whose mothers had Rh iso-immunisation and in children with E. F. Jaundice appeared in the absence of or before the development of anæmia in many cases of E. F.; also in many, parenteral vitamin K had little effect on the prothrombin level. These two things showed that there was hepatic dysfunction and damage in erythroblastosis. Perhaps it is the arrested development of the liver in an immature phase or the direct effect of antigen antibody reaction. However it is worthwhile further investigating whether Rh incompatibility has any role in the causation of parenchymatous damage of the liver.

Pathogenesis of kernicterus.—Vaughan '46 has shown that kernicterus is not found in cases of severe jaundice due to other causes. In E. F. the basal ganglia are affected; the nerve cells undergo atrophy and degeneration manifested clinically by mental impairment and spastic paresis as the child grows up. There is discoloration of hippocampus, basal ganglia, mid-brain, floor of 4th ventricle and medulla. In regions grossly bile stained the nerve cells are shrunken and readily stain with eosin; some have poorly staining cytoplasm and some vacuolization. There was no alteration in the glia cells, microglia and connective tissue. There is no relation between kernicterus and the degree of blood destruction; probably the former is the result of cell destructive process similar to that by which R.B.C. are destroyed i.e., antigen-antibody reaction in the tissues.

SYMPTOMS AND SIGNS.—In the pregnant mother the movements of the foetus may be sluggish. Icterus gravis is seen in several children of the same mother; usually the first born is spared. Jaundice is present at birth or appears within 24-48 hours; there is an associated progressive anæmia in many. At first the infant is not ill but soon becomes lethargic, drowsy and sucks poorly; it may have a shrill cry—liver and spleen are enlarged.

Usually jaundice is less severe and anæmia severe in cases of congenital hemolytic anæmia and in these there is severe disturbance of hemopoietic system; there may be no obvious jaundice even.

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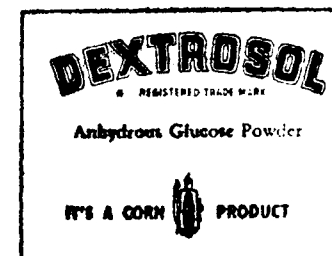
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In blood film there are many nucleated R.B.C. (erythroblastæmia) and reticulocytes. Urine contains excess of urobilin and bilirubin; stools is bile-coloured. There are certain radiological signs of E.F. in the skiagram of foetus (Javert '42). A hæmorrhagic tendency may be present due to hypoprothrombinæmia. Clinical signs varied greatly in degree and the typical picture of E.F. is neither always present nor essential for diagnosis. Death follows in a few days or weeks. Some get purpuric hæmorrhages and die; some are less severely ill and some others get lesions in the basal ganglia. *Kernicterus* is the most serious complication. Cerebral signs such as shrill cry, restlessness, head retraction, twitching and convulsions develop. *Kernicterus* is often fatal; a few that survive may become mentally defective.

DIAGNOSIS.—Diagnosis can be made in the ante-natal clinic.

1. History of still-births or death of previous babies from neonatal oedema, jaundice or anæmia. History of multiple blood transfusions should arouse suspicion. Usually the first born of the mother escapes the disease.

2. Rh heterospecificity of mother and baby. The child is Rh + and mother is Rh -ve, with anti Rh agglutinin or blocking antibody in her serum. The agglutinins of mother have specificity to the baby's R.B.C. If the antibodies are present in the mother and are in the increase, one can make the diagnosis of E.F. well in advance of the delivery. These mothers show high titres early in pregnancy.

3. Onset of jaundice on the day of birth and its progressive increase associated with anæmia.

4. Presence of many erythroblasts in the blood of infant.

5. In all cases complete blood studies of mother, father, and surviving children have to be carried out. First the blood group (OAB system), then Rh grouping (Rh+ or Rh -ve state) of wife as well as husband should be determined, testing their R.B.C. with animal's testing serum. The Rh should then be typed (whether Rh₁, rh etc.) by testing their R.B.C. with human testing sera (anti Rh₀, anti Rh' and anti Rh''). From the 3rd—4th month of pregnancy the serum of wife should be weekly tested with her husband's red cells suspended in saline and *agglutination* noted for evidence of iso-immunization and with her husband's R.B.C. suspended in his own serum (*conglutination*); the time taken as well as the titre of agglutinin should be recorded. The tests should be done on slides as well as tubes.

6. When the child is born sample of the cord blood may be examined for erythroblasts and for Rh group and type and a sample of colostrum for Rh antibodies. Dahr places on a slide one drop each of AB serum and isotonic saline by separate pipettes. A trace of blood from umbilical cord of new born is brought into the drops

separately; the drops are stirred and then spread out until a uniform mixture has been obtained. After 2 minutes the slide is rocked to and fro and again from time to time; the reaction is read at the end of 20 minutes. If the blood of the new born contains univalent Rh antibodies they become activated by the X-protein contained in AB serum and as a result there is fine granular agglutination in the serum but none in the saline drop. If there is no agglutination either the new born does not have E.F. or, if it has, it has no univalent Rh antibodies; so this test is useful only if it is positive.

DIFFERENTIAL DIAGNOSIS.—A. Jaundice.

1. Physiological jaundice of new-born or *icterus neonatorum*.
2. Pathological jaundice: (a) Hemolytic: (i) Acholuric jaundice.
 - (b) Obstructive: (i) congenital narrowing of bile duct.
 - (c) Toxinfective: (i) congenital syphilis, (ii) sepsis.
 - (d) Catarrhal jaundice.

B. Anæmias in infancy:—1. Acholuric jaundice.

2. Cooley's or Mediterranean or target oval cell or Von Jaksch anæmia.
3. Sick cell anæmia.
4. Congenital anæmia.

Lederer's anæmia (acute febrile) never occurs in infants.

Icterus neonatorum.—Nearly 50% of new born babies suffer from slight or moderate jaundice from third or fourth day after birth and it continues for another 4–5 days, commonly fades in the second week but may last for 4 weeks. It is noticed first in the face; conjunctiva are not much coloured. This disease does not cause symptoms except slight lethargy and reluctance for feeding. Urine is dark from excess of urobilin; stools normal in colour. No treatment is essential. Two factors at work are:

(1) Blood destruction; and (2) Immaturity of the liver. The infant at birth has excess of R.B.C. because in utero it has been living in a condition of anoxæmia; after birth when pulmonary respiration becomes established the need for polycythæmia ceases and the excess red cells are hemolysed and increased amount of bilirubin is produced causing jaundice. It is marked in premature infants. This blood destruction occurs in all new-born but the variable factor is the adequacy of infant's liver to excrete excess bile pigment. The suggestion that all neonatal jaundice was hemolytic in origin is only partially correct. Fashena investigated the problem and found by direct assessment of hepatic function impairment of liver in such babies (*Am J. Dis. Ch.*, Aug. '48) showing that liver immaturity is at least one factor in producing *icterus neonatorum*.

Acholuric jaundice.—This is congenital and familial. Two forms: (1) *Congenital type* commoner and may occur in neonatal life. (2) *Acquired type* comes on later in life; more severe; anæmia is more marked. This disease is a condition of slight persistent jaundice with periodic exacerbations (crisis). Though jaundice is the most striking clinical picture this is really an hemolytic anæmia. It is characterised by attacks of biliary colic, moderate anæmia usually but severe during the crisis, enlarged, firm spleen. Liver is not enlarged. The cause of excessive fragility of R.B.C. is some congenital defect. Blood film shows smaller but thicker R.B.C., C.I. is less than one; there is leucocytosis especially polymorphonuclear; V.D.B. is indirect +ve. Urine is dark, contains urobilin but no bilirubin. (Hence the disease is called acholuric jaundice). Motion is normal coloured.

Congenital narrowing or obliteration of bile duct in new born.—Perhaps an unknown toxin from maternal blood acts on foetal liver causing portal and biliary cirrhosis and narrowing of the bile duct or there may be a developmental abnormality or a pre-natal obliterative cholangitis. Jaundice starts within the first few days of birth and is persistent and progressive; no symptoms are present except the failure to thrive. Liver is enlarged and spleen also secondarily. When obstruction is severe no bile passes into the gut and so the stools are clay coloured. Urine contains bile salts, pigments but no urobilinogen. V. D. B. is direct +ve. Death follows in a few weeks.

Congenital syphilis.—Jaundice may be present even at birth; more often the onset is in 2nd or 3rd week. It means there is severe infection and so prognosis is poor. Other signs of syphilis are present; liver is enlarged and tender; urine contains bile and motion is of normal colour.

Sepsis.—Jaundice may occur as a complication of any severe sepsis in the neonatal period (umbilical sepsis, septicæmia, pyæmia and gastro-enteritis). Usually the clinical picture is that of the infection, jaundice being but an incident though denoting a grave prognosis.

Cooley's or erythroblastic anæmia.—This has a familial and racial incidence. Though confined mostly to the Mediterranean races it may also occur in Indians. Mostly it occurs in 1–2 years of life but also rarely in the new born; it is initially very mild. Earliest manifestation is pallor which increases; there is also increasing weakness. There is a typical diagnostic Mongoloid facies (head enlarged with high molar bones, prominent eyes, short nose with depressed bridge); growth is retarded, stature stunted but mentally normal. Abdomen is protuberant; spleen enlarged; liver becomes palpable; lymph glands are enlarged but not tender.

Blood:—Microcytic, hypochromic anæmia, high leucocytosis and increased platelets. There are numerous nucleated R.B.C., normoblasts and megaloblasts.

Urine contains excess of urobilin.

Skigrams of skull and limb bones are characteristic. Skull is thickened; diploic space is wider. Vertical striations to the inner table produce 'hair on end' appearance. The limb bones show typical mosaic appearance due to hyperplasia of marrow. This disease ends fatally in a few years.

Sickle cell anaemia.—This disease also is familial and hereditary. It occurs usually in persons with Negro blood but at times in others as well. It has 2 phases. The active phase is marked by severe hemolytic anæmia, jaundice, fever, joint and muscle pain, vomiting and diarrhoea; there is increased urobilinogen in urine. In the chronic phase there are greenish yellow sclera, enlarged liver, spleen and lymph glands. In both the blood film shows number of crescentic or sickle shaped R.B.C. and reticulocytes.

PROGNOSIS.—It has recently been proved that there is correlation between the time of appearance, the maternal antibody level and the severity of the disease in the infant. Hydrops foetalis is universally fatal. Unless cases of icterus gravis and congenital hemolytic anæmia are promptly and efficiently treated immediately by replacement transfusion with Rh—ve blood there is very high mortality and with treatment mortality is still around 50%. Female babies with E.F. did significantly better when treated than males particularly with regard to the incidence of kernicterus. Even if the infants escape death, cerebral manifestations develop later due to anæmia and anoxia of cerebral tissues and cirrhosis may come on due to damage to liver.

TREATMENT:—*Preventive treatment:*—Damage to the infant occurs also during intra-uterine life and tissue cells are damaged as well as the red cells. Hence the best thing will be to prevent the maternal antibody from being formed, or prevent the silent processes of transplacental transfer from and to the infant. There are at present no measures that will positively prevent either. So it will be enough if by any method the severity of E.F. can be reduced and the disease made amenable to treatment. Kernicterus cannot be prevented by any treatment. Premature induction of labour in selected cases to shorten the period of intrauterine hemolysis seems logical but its value is yet to be investigated. Thus specific preventive measures must await the extraction of "Haptene," which will neutralise the anti Rh antibodies without stimulating their production.

Basing on the idea that if antigens of unequal potency are injected into an individual, the antigenicity of weaker antigen may be suppressed by stronger one. Weiner ('45) suggested counter-

immunisation of the mother by a more potent antigen such as typhoid or pertussis vaccine early in the course of pregnancy. Probably the stronger vaccine would suppress or delay the formation of anti Rh agglutinins, if the former is given early enough before antibodies are seen in maternal blood. Dr. K. S. Ranganathan tested mother's blood for both agglutinating and conglutinating anti Rh antibodies at monthly intervals from 20th to 30th weeks of pregnancy and their titre and gave $\frac{1}{2}$ c.c. T. A. B. Prophylactic vaccine (1000 millions E. Typhosus and 500 millions each of para A and B per c.c.) subcutaneously during the 31st week and increased the dose by $\frac{1}{2}$ c.c. weekly until 1 c.c. was reached. The 1 c.c. dose was repeated 4 or 5 times till delivery. Each week after the injection, the blood serum was tested for Rh sensitization using at least 3 different Rh +ve group O cells. (Chown's test, agglutination and conglutination tests); if sensitization was present, the titre of the antibodies was estimated, the antityphoid titre in the mother's serum was also determined by Widal's test. He holds that in his experience antepartum immunization with typhoid vaccine had no effect apparently in preventing E.F. Kariher ('47) has claimed success in preventing hemolytic diseases by repeated I.M. injection to the mother of Ethylene Di Sulfonate (Allergosil Brand) 2 c.c. (each c.c. containing it in a dilution 1 in 10-15 in triple distilled water). However, this has not yet been properly assayed.

GENERAL TREATMENT.—1. Pregnancies to be avoided until 1 or 2 years elapse after the disappearance of all residual agglutinins and incomplete antibodies.

2. The infant should be artificially fed as mother's milk is believed to contain the offending antibodies. Some think that the child may be suckled after a few weeks, that the breast milk should be boiled before it is given, but others think it unnecessary.

3. *Treatment of hypoprothrombinaemia.*—Some hold that the hemorrhages occurring in E.F. responds to administration of vitamin K. One injection of water-soluble preparation is given to the infant I.M. for immediate effect followed by oil soluble preparation 1 mgm. for sustained effect, then 1 mgm. is repeated daily 2-3 days and later oily liquid or tablets can be given orally (5 mgm. daily) for 8 days more. However it is better, to give prophylactically to pregnant mothers at the commencement of labour, an injection of 10-25 mgm. not earlier than 12-16 hours and not later than 4 hours before delivery; else give orally 10 mgm. tablets daily during the last week of pregnancy.

Curative treatment.—The discovery of Rh factor, its variants, iso-immunization etc. has placed the treatment of congenital hemolytic diseases on a rational and efficient basis. Mild cases recover. American workers claim better results with exchange transfusion, though blood transfusion treatment has effected a considerable

reduction in case mortality. The grave risk is that of those who survive, many suffer from neurological sequelæ such as mental deficiency, spasticity and damage to the liver.

Treatment consists in *blood transfusion with (O group or child's own group) Rh —ve blood immediately (i.e.)* blood which is not agglutinable by the maternal antibodies which have entered the infant. Homologous blood is ideal, but this necessitates investigation of the exact nature of the Rh factor and antibody. Blood should not be taken from the mother as there are anti Rh agglutinins in her serum; transfusion of Rh₊ blood to infant is useless, as the blood will get hemolysed by the mother's agglutinins that persist in the baby's serum for a time. This treatment is still unsatisfactory and does not prevent kernicterus. 10-20 c.c blood per lb. body weight is the daily dose and should be repeated several times in neonatal period (10 or 15 times). For transfusion the umbilical vein (Mayes '44) within the umbilical cord may be used but should be done with-patent within the rapidly atrophying cord; this is relatively easy too. But Pinkus utilised that portion of umbilical vein which lies within the abdominal wall and is available even after the cord has atrophied (*J.A.M.A.*). Transfusion through bone marrow (upper and medial end of tibial medulla) is well tolerated and not followed by reaction, but cranial route through sagittal sinus is not a safe one. As a last resort blood may be given also *intraperitoneally*; it is useful though some degree of peritoneal shock may be encountered. When a suitable donor is not available, mother's blood may be used provided it is found compatible by direct matching test and the blood cells are first washed in saline (to remove the agglutinins), then resuspended in 5% glucose saline and used. A more promising method of treatment is by *exchange or replacement or exsanguino transfusion*. Wallerstein, Weiner, Diamond and others claim that severe cases have been treated successfully by this; it is not known whether this will prevent kernicterus. This treatment must be given *very early* and consists of alternate withdrawals of part of infant's blood and simultaneous replacement by transfusion of compatible blood till most of the R.B.C are Rh —ve. The whole procedure takes 1-2 hours and is a difficult one; it can be done only in special centres (Mourant and Race '48). Wallerstein's original technique was to remove the infant's blood by fontanelle puncture of the longitudinal sinus and to transfuse Rh —ve blood into an arm vein; he infused 75-100 c.c. more than he removed. Weiner found that by heparinising the infant with 500 or 1000 units of Heparin I.V. he could remove 150 or 400 c.c. blood before clotting. Allen and his coworkers had 15% mortality by this method and even much less in cases in which women were exclusively used as donors; however, the beneficial component of the blood from women is not known. Thus it would appear that for the present exchange

transfusion, using blood from a female donor *via* the umbilical vein, is the treatment of choice in babies with erythroblastosis foetalis, though this should not raise high hopes.

Treatment of kernicterus.—No treatment will cure the cerebral damage because the cellular changes involved are irreversible.

The only really effective measure can be the early neutralisation of the antibody and even this is not possible now.

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Choline in Coronary Arteriosclerosis

Morrison and Gonzalv treated 115 patients suffering from proved coronary thrombosis and myocardial infarction with choline—which was administered orally in daily doses of 12 grammes for one to 3 years after recovery from the immediate attack. Another set of 115 patients not receiving choline served as control. The results of the study showed that the lipotropic agent choline was of definite value in the treatment of coronary arteriosclerosis.—(*Am. Heart Jour.*, May, 1951).

Dipenicillin in Acute Otitis Media

Nielsen reports on 65 cases of acute otitis media treated with one daily dose of Dipenicillin (a mixture of penicillin Sodium G and Penicillin Procaine G). Patients under 7 years of age were given 200,000 units, those over 7 years 400,000 units. The treatment was continued for one or two days after the discharge stopped and was applied for at least 6 days. 96 per cent of the cases was cured by this treatment. If six or seven days' penicillin treatment is without effect penicillin resistant types of infecting organisms are to be suspected and streptomycin or aureomycin should be tried.—*Uges-Krift Laeger.*, Denmark, 112, pp. 1080-1089, 1950).

ON LEPROSY*

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LEPROSY is a disease characterised by sensory and pigmentary changes and nodular formations in the skin caused by bacillus lepra. The disease has been in existence from very remote times, possibly for several thousands of years. It is prevalent in practically all parts of the globe—Africa, India and other Asiatic countries, Europe and America. For this reason it is sometimes difficult to say from where it originated. Some writers believe that its original home was Africa^{1,2} and some hold that its cradle was Asia¹. Its existence in our country has been mentioned in the Vedas about 1400 B.C.¹.

To the European countries, the disease had been probably carried from the East, first by the Persian armies^{1,2} and later on to Greece and Rome between 400 and 325 B.C. by Pompey's soldiers returning from the East.

Then it began to appear in other parts of Europe. It was described by Galen in Germany towards the end of the 2nd century A.D. and by the 5th or 6th century it appeared in Spain, and hundred years later it appeared in most parts of the Northern Europe. The first Leper Hospital was established in England in Nottingham in 625 A.D. or 628 A.D.². About the 13th century the spread of the disease reached its height⁴ and leprosy cases began to occur practically everywhere—Italy, Spain, France, Germany, Russia, England, Norway etc. To Norway the disease was carried from Scotland.

When the disease was so spreading the rulers and priests started institutions for these lepers and laws were enacted to isolate the lepers. The Church in England at that time was largely responsible for the control of leprosy. The leper was isolated from the community, a funeral was performed as if he was dead to the society.

When "Saracens from Spain invaded France, Pepin (in A.D. 757) issued a decree making the marriage of lepers illegal and the disease a cause of divorce². In about 950 A.D. a similar law like that of Pepin was passed in England²." In France, when a leper was discovered, he was isolated and a burial service was performed, they sprinkled earth on his head as if for all practical purposes 'he was buried from the world.' He was then supplied with a special garment and with clappers to warn the healthy from approaching him too closely and he was taken to live in a hut in the fields outside

*An Inaugural Address at the S. N. Medical College, Agra, at the time of observing the "Leprosy Week" in U.P. from 11th December 1950.

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ON LEPROSY—K. N. GOUR

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the village or town, or to a leper house. He was forbidden to have a wife, to touch infants or young persons or to eat or drink with companions other than lepers. If a healthy person resolved to continue to live with an infected spouse the above service was held over both and it was necessary for them to separate from them any infants born to them if the latter showed no sign of the disease."²

After the so-called funeral service the priest addressed the leper like this, "while you are diseased you will enter no house, no inn, no mill, nor in the fountains will you drink water or wash your clothes. You will not eat except by yourself or with other lepers. You will enter no church during service, you will mingle with no crowd. When you speak to any one you will stand leeward. You must always use your gloves and will touch no rope without them. You will touch no children, not even your own, and you will return to your cabin every night."²

Such was the plight of the leprosy patients during those days in European countries. They were, so to say, an out-caste from the human society and had to suffer from 'the civil death.' He became a common figure and the subject of frequent references in the chronicles and romances of that period.¹ One may read the 'unforgettable passage in the Lunaburger chronik⁴, which Heine paraphrased: "living corpses they wandered to and fro, muffled from head to foot, a hood drawn over the face, and carrying in the hand a bell, the lazarus bell as it was called, through which they were to give timely warning of their approach so that every one could get out of the way in time."⁴

So by such strict legislation for isolation and control of these patients a great effort was made to stamp out the disease. This was an important step to eradicate it because at that time the cause of the disease was not known. But it was accepted that the disease was communicated from man to man and hence the preventive measures which were formulated aimed at segregation of those unfortunate patients to protect the healthy community. Leper homes were started—Laprodochia or Laprosoria were many in most of the cities of Germany in the 13th and 14th centuries.

While the disease was declining in European countries by the rigid and strict segregation, so much so that it had practically disappeared by the middle of 16th century⁴, it was still rampant where no such measures had been adopted. With the increase of communication it was transported to other places where virgin soil for it existed and it spread rapidly there. This was well seen in the Island of Nauru in the Pacific where leprosy was unknown till 1920 but several cases occurred after that year.

The epoch making discovery was made in 1871 by G. Armaner of Bergen in Norway where the disease was endemic when he described the lepra bacillus. He reported his discovery to the Medical

Society of Christiana in 1874 and our modern knowledge about the conception of disease dates back from that period.

In India, as mentioned above, leprosy has been present from several hundreds of years—1400 B.C. but it was described with several skin conditions and all these were grouped under the term '*Kusht*'. It is rather unfortunate that this term is even now used to include many skin conditions. It is now high time that this type be divorced from other diseases of the skin.

It is also very necessary that the social stigma which has been attached to the disease be removed. Now the etiology is known and the sufferer can be successfully treated. They can, when cured, mix with the society with impunity. They should no longer suffer 'the civil death'. The whole aspect of the disease should change. Every individual should know that it is a curable condition and if adequate precautions are taken it is a preventable disease. The old saying "Once a leper always a leper" no longer holds good. I feel that the word 'leper' for such patients should also be avoided as this word pinches them. They should be called leprosy patients as we use the terms with other diseases.

It is roughly estimated that in India there are about 10,00,000 leprosy patients and in the Uttar Pradesh there are about a quarter of this figure i.e. 200,000 to 300,000.

No age is exempt but its incidence is more common between 10 and 30 years though it may be found at a very early age and also beyond 60 years. Males are comparatively more affected than females in the proportion of 2:1. Racial factor does not play any important part. Social status is of utmost importance. Hutchinson said that 'it is a disease of semi-civilization, savages are exempt, the highly class people are exempt but when savages started putting on clothes they began to suffer from it. Indeed, social status of a large group of these victims is a cross section of the normal population as regards education, wealth and culture.'

Heredity is not of very great importance. The disease is not transmitted by the mother to the foetus though mycobacterium lepra has been found in the placenta and the umbilical cord³. If the infants are separated from their lepra parents immediately after the birth they do not suffer from the disease. This is very well seen in the 160 families who migrated to America but none of them suffered. Similarly, the segregation of patients in the Nauru islands in the Pacific confirms this. On the other hand, when children have suffered from leprosy they have transmitted the disease to their parents.

Leprosy does not confine itself to any geographical boundary. It is found in tropics, subtropical zones and frigid zones. So there is no special geographical distribution but it is apparently more common in tropical climates an account of the bad hygienic

conditions. There is certainly a definite relationship to the sanitary condition of the country. When the people live under better sanitary conditions the disease does not spread.

The exact mode of infection is still debated—either by the direct spread or by any other route, though the direct contagion is the most important.

Clinically, we get two types—the nodular when small diffuse nodules appear on different parts of the body and when the face is severely involved it gives a leonine appearance; the maculocutaneous, when patches of anæsthesia, pigmentary changes, bronzing of the skin or leucodermal patches, bullous eruptions in the course of a nerve, nerve thickening, sensory changes e.g., numbness, tingling, anæsthesia in the distribution of the nerves, and in very advanced cases paralysis and atrophy of the muscles may occur. Mixed forms of these two types may be met with.

With the use of Diasone, Promin, Promizole, and D.A.D.P.S. the leprosy patients are much benefited and in most cases they are completely cured. The old remedy—Chaulmoogra oil—is still of immense value, especially for the nerve cases.

We can do a lot for these unfortunate victims who were despised and looked upon with contempt by the social fabric, if we detect the disease early. By suitable propaganda—education of the public with the help of magic lantern slides, small film shows at cinema halls, folk tales, elementary talks to school and college boys about the disease, etc. much can be achieved to bring the cases early to the notice of the individual. Further, the lepra patients should never be condemned as this attitude makes them conceal their ailments for some time. They, in whom the disease has advanced, should not be allowed to earn their living by begging at the railway stations, public places, public streets and at the places of worship. Proper arrangements have to be provided for their segregation to leprosy homes and similar institutions. Unfortunately such institutions are very few in our country. Will the philanthropic people rise to the occasion and donate large sums of money and join hands with the Government to start such homes or hospitals? It is then and then only that such a scourge of mankind will be conquered.

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IDIOPATHIC HYPOPIESIS*

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IDIOPATHIC hypopiesis is important to the clinician from the point of view of differential diagnosis and rarity.

Definition.—Idiopathic hypopiesis may be defined as a condition of low blood pressure, in which both systolic and diastolic are low and for which no cause can be assigned and which has no pathological import but a physiological normality for the individual.

HISTORY.—It is fitting to traverse a little on the realm of history of medicine and quite interesting too to understand how the present day knowledge of blood pressure recording came to exist.

During the time of the Father of Medicine, Hippocrates, he enumerated the theory of four humours and said that they existed in equilibrium. But only at the time of Galen the experimental physiology found a footing and he proved that artery contained blood and not air as was held by the Alexandrian School. But his theory of circulation of blood led people into wrong roads for several centuries after him.

After Galen, medical science in Europe had restful sleep up to the 16th century when Von Hohenheim, better known as Paracelsus, became convinced that medicine could progress only if it was stripped off the confusing mass of Galenic theory and were re-clothed in the simple practical precepts of Hippocrates. With a spirit of revolt he began his teaching career by publicly burning the works of Galen and of Arabian physicians and by issuing printed pamphlets stating among other things that if he wished to prove anything he did not do so by quoting authorities but by observations, trial and experiment.

Notable advance in the theory of blood circulation was made by William Harvey in the year 1628. Previous notions about the theory of blood circulation were shattered and the proofs that the movement is in a circle were discovered by William Harvey to whom also belongs the credit of pointing out the methods by which almost every physiological problem must be studied. His discovery is the greatest after Galen.

Yet the discovery that the ends of the arteries are connected to the beginnings of veins by a definite system of small tubes we now call capillaries was made by Malpighi in the year 1661.

At the time of Harvey it was discovered that the blood pressure exerts certain amount of pressure on the arterial wall and it is ejected out of the heart with great force. The first to make an

advance on the method of demonstrating blood pressure was Rev. Stephen Hales, Vicar of Teddington in 1722.

The study of blood pressure was not satisfactory until the introduction by Carl Ludwig in 1847 of the Kymograph in which Poiseuill's hæmo-dynamo meter was combined with apparatus to record the oscillations of the mercury.

Rough and ready method of finding the blood pressure by an apparatus was found out by Riva Rocci. Martin's modification of Riva Rocci is the one we are using at present.

PHYSIOLOGY:—A dose of physiology would not be out of place as it gives a clear, cogent idea to appreciate the complex mechanism of the variation of the blood pressure. At the outset let me define blood pressure. Blood pressure is defined as the pressure exerted by the contained blood on the wall of the vessel. Hence it includes endocardial pressure, arterial pressure, capillary pressure and venous blood pressure. We are here concerned only with the arterial blood pressure.

It has now been definitely established that all the blood vessels in the body are under the control of the vaso-motor centres which lie in the floor of the 4th ventricle, a few millimetres above the calamus scriptorius of the medulla. There are also subsidiary centres in the spinal cord which is suggested by the fact that the recovery of blood pressure may occur even after the vasoconstrictor centre has been cut off by the section of the medulla.

There are receptive or sensory areas in the cardio aortic region and the carotid sinus. The variation of the arterial pressure in these areas causes a series of different vegetative reflex from which three are specially important; the reflex modification of the heart rhythm, the reflex modification of blood pressure by vasoconstriction or dilatation and after intense stimulation a reflex increase of Adrenalin secretion. The afferent fibres originating from the carotid sinus reach the central nervous system by way of the glossopharyngeal nerve. The afferent fibres originating from the cardio aortic region join the superior branches of the vagus. The carotid and aortic nerves form together the presso-receptor nerves.

Increase of blood pressure in these receptive areas is followed by bradycardia due to stimulation of the depressor and the inhibition of the cardio-accelerator nerves; the diminution of the blood pressure which follows is due to inhibition of the vaso-constrictor centre and therefore decrease of its continuously transmitted pressor impulses to the small arteries, and arterioles stimulation of the vasodilator centre and reflex inhibition of Adrenalin secretion.

Decrease of blood pressure causes an exactly opposite reflex; tachycardia, stimulation of the vaso-constrictor centre and of Adrenalin secretion and inhibition of the vasodilator centres. In this reflex mechanism, Adrenalin plays a secondary part and comes

* A paper read before the Tinnevely Branch of the Indian Medical Association at the meeting held on March 31, 1951.

into action only when a considerable change of blood pressure occurs.

A sufficiently strong variation of blood pressure in one vascular district stimulates the extremely sensitive receptive areas and an opposite reaction in another district will follow, thus re-establishing quickly the pressor balance.

It is through this auto-regulation that the body maintains a constant blood pressure. The tone of the vaso-motor centres is maintained by the chemical and hormonal stimuli from the diencephalon and the periphery, and the inhibition transmitted through the pressor-receptor nerves. According to Heymans and Bouckaert, carbon dioxide and hydrogen ions have a particularly great influence on this tone, though both of them and oxygen, influence the pressor-receptors too.

This neuro-vascular reaction takes place mainly in the splanchnic circulation, skin, muscle vessels, thyroid and coronary vessels.

It is to be noted that a decrease in the carotic pressure causes peripheral vaso-constriction but dilatation of the blood vessels of the brain in order to ensure an adequate blood supply. An important function of the carotid sinus is to guard the delicate brain tissues from the changes of blood pressure.

From the above mechanism of reflex regulation we may now summarize the factors which determine and vary the blood pressure magnitude:

- (1) The peripheral resistance.
- (2) The elasticity of the vessels.
- (3) The output of the heart.

The output of the heart in turn depends on (a) the efficiency of the heart and (b) the venous return. The blood pressure falls down severely if both the efficiency of the heart and the venous return are affected; but the smaller degrees of fall are compensated by the increase in the peripheral resistance and diminution in the capacity of the circulation, especially the veins and capillaries, amount of blood reaching the veins from the arteries, the respiratory movements and the compression of the veins by movements.

Having considered the physiology of blood pressure generally we are led to the conclusion that the blood pressure may be systolic and diastolic. The arterial pressure which synchronises with the systole of the heart is called systolic and the diastole of the heart is called diastolic and the difference between these, the pulse pressure. As such, the low blood pressure may affect the systolic or the diastolic or both the blood pressures.

There are two methods of noting the blood pressure. One is by the palpatory method and the other is the auditory method. To estimate the blood pressure accurately auditory method is the simple, quick and trustworthy one. In the healthy young adults

examined by this method in the sitting posture, the normal systolic pressure averages about 110 to 135 mm. Hg. and the diastolic pressure between 70 to 80 mm. Hg. in different individuals. Anything below these figures is low blood pressure.

Incidents, ætiology and pathology.—S. C. Robinson who in 1939 with M. Bruer analysed the range of normal blood pressure among 10,883 urban inhabitants now argues that hypotension is an ideal blood pressure level and not a disease. Higher limit for systolic hypotension is 110 m.m. Hg. and for the diastolic pressure 70 mm. has been selected by him. Foreign observers have found the incidence of hypotension among the population as 20%. Among Robinson's 10,883 cases of investigation he has found 25% as hypotensive systolic and 34% as hypotensive diastolic.

Age:—Since it is not a pathological hypotension no age is exempt. It is there for the individual from birth. Hypotensives maintain an even level of blood pressure. The blood pressure does not rise with advancing years in them.

Heredity:—Just as other characters it can also be inherited.

Sex:—Of the 10,883 cases examined by S. C. Robinson 7,478 are men and 3,405 women. Among 7,478 men, 42 and among 3,405 women 56 are hypotensives, thereby making a ratio of the incidence of hypotension as 1:3 between men and women.

As we have defined, idiopathic low tension is only a physiological entity; but there are so many pathological conditions in which low tension is a symptom due to the causes already enumerated. It is idiopathic because there is no cause for the sub-tension, and physiological due to not known constitutional changes. Yet, one must know the pathological conditions in which the hypotension is the prominent symptom from the point of view of differential diagnosis. The factors which effect the systolic low tension:

1. Lowered myocardial efficiency as in acute febrile conditions such as diphtheria, influenza, and coronary thrombosis.
2. Loss of peripheral resistance from relaxation of the arterioles as in kala-azar, T. B., cancer, shock and collapse, diabetic coma, acute febrile conditions as pneumonia, typhoid or diphtheria.
3. Lowered viscosity or volume of the blood as in anæmias from any cause or loss of fluids as in severe vomiting and purging.
4. Paralysis of the vaso-motor system as in:
 - (a) Bowel diseases as in cholera, bacillary dysentery, ptomaine poisoning, also algid malaria.
 - (b) Addison's disease, acromegaly.
 - (c) Vagal over action as in syncopal attacks, partial or total sympathectomy. Splanchnic stasis is frequently associated with lowered vasomotor tone.

Causes of low diastolic pressure.—High grade vaso-dilatation as in aortic regurgitation, arterio-venous aneurysm, patent

ducts arteriosis, thyrotoxicosis, neuro-circulatory asthenia including effort syndrome.

Symptomatology.—The symptomatology can be summarized in the saying, "not exactly ill and yet rarely well." They complain of early fatigue on physical and mental effort. They have certain amount of dizziness and a tendency to mental depression and headache. Coldness and pallor of extremities are very marked. Pulse is accelerated. I would like to classify postural hypotension under a different heading, lowered vaso-motor tone, though some authors describe it under idiopathic hypotension, since it is considered to be a disease of the sympathetic nervous system. It is argued by foreign authors that 3% of over 2,000 fit air pilots in England were found to be hypotensives and as such this hypotension is only a physiological normality.

DIAGNOSIS:—Fatigue, headache, dizziness, coldness and pallor combined with low blood pressure which is not very much affected by posture confirms the diagnosis.

DIFFERENTIAL DIAGNOSIS:—1. In all these cases of low blood pressure, the previous history is very essential.

2. Apart from the low blood pressure, were there any other symptoms such as anæmia, emaciation, or syncopal attack or fever, diarrhoea, dysentery or his previous ailments or any pain in the chest or abdomen or the symptoms are aggravated by effort should be considered. In addition to this, the condition of the heart arteries and kidneys should be ascertained.

Since you don't meet with fever in a case of low blood pressure, the conditions such as typhoid, influenza, pneumonia, diphtheria, kala-azar, malaria and black-water-fever can be weeded out.

Lack of diarrhoea and vomiting or dysentery fizzles out cholera, dysentery and diarrhoeas.

Absence of extreme emaciation, anæmia and pigmentation scores out Addison's disease.

Absence of signs and symptoms suggestive of cardiac disorders rules out cardiac inefficiency.

This leaves us with vaso-vagal attack and postural hypotension.

In postural hypotension the variation in blood pressure when he is lying horizontally and assumes an erect posture, is very great—the diastolic even goes to 30 or 20 mm. Hg. and systolic 50 mm. Hg. and they get giddiness, headache, pallor, blurring of vision, tremor of the hands and inability to understand directions.

In idiopathic blood pressure, the variation is only about 10 mm. or 20 mm. at the most. If the variation is greater than 20 mm. Hg., the patient gets the signs of diminution of the supply of blood to the brain and that is in postural hypotension.

When everything seems to go wrong

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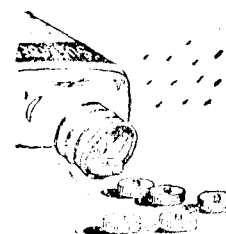
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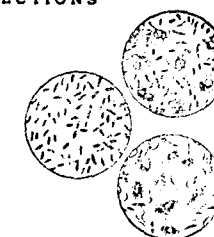


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Having eliminated the postural hypotension we are left with the vasovagal attack. Under this misleading title, Gowers described a recurrent paroxysmal symptom-complex with some or all the following components: a sensation of fullness in the epigastrium; precordial pain or discomfort, difficulty in breathing, a sense of impending death; a slowness of mental operations but without disturbance of consciousness, a sense of physical fatigue and coldness of face and extremities. These symptoms wax and wane gradually and may be present for 4 hours from the onset.

Gowers stated that he used the term "vasovagal" as a purely descriptive one, but without implying any theory of causation. Unfortunately those who have adopted his terminology have overlooked its lack of foundation. Further, the various descriptions of these attacks to be found in the literature are based almost wholly upon hearsay, the attacks themselves been but rarely observed and do not provide any evidence of vasovagal involvement. Thus the pulse is said to be accelerated, not decreased or irregular, while the facial pallor and coldness might equally be the result of local vasoconstriction or splanchnic dilatation. A further vagueness has been lent to the conception by the different senses in which it has been employed. Thus, Collier has used the term for attacks in which convulsions and loss of consciousness occurred, though Gowers expressly stated that consciousness was not disturbed. In short, the term has no precise meaning, no sound basis of observation and no proper place in the neurological terminology.

The alternative term recently suggested for the attacks described by Gowers namely "Periventricular epilepsy" must also lack value until we have some precise information as to the nature of the attacks themselves. Lewis has pointed out that the term vasovagal may rightly be applied to the common fainting or syncopal attacks, and it is better to restrict the term to these.

Having considered the vasovagal attack, in a case of idiopathic low blood pressure no loss of consciousness occurs; pulse is accelerated and symptoms do not cause any anxiety.

TREATMENT.—As it is a physiological condition no drug therapy is required; and a normal life of rest and work should be advised. An ample amount of nutritious and easily assimilable food and a periodic administration of general tonics are indicated. Yet the dictum in hypotensives "not exactly ill and yet rarely well" should be remembered.

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ARTERIO-VEINUS ANEURYSMS AS A NEURO SURGICAL PROBLEM*

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ARTERIO-VEINUS aneurysms of the brain are exactly like arterio-venous aneurysms situated elsewhere in the body except for differences of detail due to local environment. Whereas arterio-venous aneurysms in the general vascular system are of two types, congenital and acquired, those in the brain are solely of congenital derivation. The formation of an acquired arterio-venous fistula or aneurysms is dependent upon the contiguity of a large artery and vein. This vascular arrangement is common in the extremities and elsewhere but in the brain larger venous and arterial trunks run independently after the embryonic period. Arterio-venous aneurysms were first recognized and described by William Hunter in 1757. It was not until 1895 (138 years later) that Steinhil reported the first lesion of this type in the brain. About 93 cases have accumulated in the literature twelve by Dandy, another of equal number by Cushing and Bailey and the rest by Olivecrona. Arterio-venous aneurysms occur in about 1% of a series of brain tumour. They are located in any part of the brain, though the cerebral hemispheres are for more frequently involved than the cerebellum. They may be entirely within the brain or only in part. The variety of designation under which they have been reported, has been due to an imperfect understanding of their underlying character. That they are arterio-venous is easily demonstrated at operation. The red arterial blood can be seen pumping into and mixing with dark venous blood with each heart beat. Usually a thrill can be felt. If a large cortical vein is tied by mistake, it may rupture after being greatly distended by the increased arterial pressure within. Such an accident would be impossible if the pressure was venous.

PATHOLOGY:—Arterio-venous aneurysms are composed of three parts:—(1) An arterial branch or branches—the inlet.

(2) Numerous venous tributaries—the outlet.

(3) An interposed bed of tortuous vessels, the so called angioma forming a tumour of varying size and taking the place of normal capillary bed. Occasionally an anomalous vessel, doubtless a pre-existing congenital channel established as direct communication between artery and vein instead of through a collection of vessels. Owing to the fact that venous tributaries are affected over such an extensive area, *i.e.*, until the arterial pressure is in large part dissipated in the veins, the total area of the brain affected by the aneurysms is usually quite large. The actual bed of the interposed vascular tumour, the so called angioma is also quite variable in size

* Specially contributed to THE ANTISEPTIC.

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and character. It may be composed of vessels which look like arteries and histologically have arterial walls, or the vessel may be in large part venous both in the gross appearance and histologically or they may be both arterial and venous, when venous, the channels are much larger and may even dilate into pouches. The walls of the vessels are angiomatic and calcified for which reason they are prone to rupture and the emanating veins are tremendously enlarged and frequently tortuous. The artery or arteries, supplying the aneurysms is larger than the corresponding artery on the opposite side. This dilatation extends backward in the arterial branch usually to the carotid artery and may even extend into the neck. Actual enlargement of the heart is not uncommon in systemic arterio-venous aneurysms. Not infrequently other congenital abnormalities of the cortical veins and convolution aneurysms of the scalp either entirely independent or in association with the intracranial aneurysms of similar type are not uncommon.

CLINICAL FEATURES:—Arterio-venous aneurysms are found in both sexes, though the greater number have been in males.

The principal symptoms are:—(1) Epilepsy usually but not always Jacksonian.

(2) Some degree of motor or sensory disturbance.

(3) Cerebral hæmorrhage.

The actual manifestations depend of course upon the exact location of the tumour but since most, by no means all, arise from the branches of the middle cerebral arteries and are located in the paracentral area, some grade of motor weakness is usually present. In the characteristic expression of this lesion Jacksonian epilepsy is present for many years, then a slight motor weakness develops and progresses very slowly or perhaps is increased rather suddenly, from time to time with partial improvement after such increase. The slowly progressive course is due to slow expansion of the angiomatic portion, the intermittent feature associated with the convulsions being a transient weakness of the part affected for several minutes or hours after convulsion, and is with fairly quick return to the previous level of function. This is however, by no means characteristic of this type of lesion but is common to many brain tumours. A large hæmorrhage may occur at any time either with or without exertion. From all reports in the literature, death resulted from cerebral hæmorrhage in 40 per cent. In some instances recovery follows for the time being; paralysis is usually a sequel. Other neurological symptoms are hemianopia, astereognosis, diplopia ankle clonus, positive Babinski and increased deep reflexes and loss of sight, taste, smell. Signs and symptoms of severe intracranial pressure occur only with cerebral hæmorrhage. Patients may or may not have headaches. Headaches may or may not be located on the side of recorded hæmorrhage. Both arose from the vertebral artery and

its branches. They presented clinical manifestations of cerebellar tumours together with intra-cranial pressure due to occlusion of the aqueduct of Sylvius. One of the most remarkable features about cerebral arterio-venous aneurysms is the late development of signs and symptoms. In nearly half of all cases there had not been any intra-cranial symptoms until after the thirtieth year and in one patient not until the forty eighth year. Since arterio-venous aneurysms are of congenital origin, the lesions had existed in a dormant state since birth. The delayed time of appearance of epilepsy might appear incredible were it not known that epilepsy may appear with equal tardiness from the other known cerebral lesions. That symptoms do not usually appear until early adult life is doubtless due to changes (thrombosis and calcification) that appear prematurely in the inherently defective walls. Although the lesions occupy a volume of the cranial chamber comparable to that of a large tumour, frequently as large as one's fist, this space has been compensated in the early months of life when adjustment can readily be made by enlargement of the head if necessary. In late years each thrombosed emissary vessel reduces the venous bed and throws an additional strain and causes slow progression of the tumour in size and correspondingly increased susceptibility to rupture. It is to be impressed that symptom and signs of arterio-venous aneurysms do occur in childhood. Indeed they must always be borne in mind as a possible source of early epilepsy and partial hemiplegia.

DIAGNOSIS.—(1) X-ray of skull. (2) Cerebral angiography.

(1) In most cases the X-ray is negative, but at times the wall of some part or parts of the lesion is sufficiently dense in deposited calcium to throw a sharply defined shadow.

(2) *Cerebral angiography*:—Performed after precutaneous injection of Umbradil (dildrast, perabrodil) into the carotid artery according to the technique described in my article July 1950, *Journal of I.M.A.*, page 355.

TREATMENT.—Cushing, Bailey and Dandy considered these lesions practically inoperable and recommended decompression and X-ray treatment. Bengstrand, Olive-Crona and Tonnis 1936 published a monograph on angiomatous malformations and tumours of the brain where they mention successful removal of arterio-venous aneurysms of brain in five cases. All the operations were performed by Olive-Crona, the first one in 1932. At a meeting of the Scandinavian Neurosurgeons in September 1946 Olive-Crona presented 43 cases in which he had performed radical excision of the lesion in 24 cases with 3 fatalities. In other cases various procedures were carried out, chiefly ligature of accessible arteries and the carotid artery. It was felt that ligature of the carotid artery might even be dangerous though it is the rule in cases of cerebral aneurysms which are also cause of cerebral hæmorrhage. Recently Trupp

and Sachs reported a series of cases of vascular tumours of the brain and spinal cord treated by electrocoagulation, with a very low coagulating current, stroking along the vessel walls. They think it is possible to shrink and obliterate them. The value of the procedure seems little uncertain though it is possible to coagulate the superficially situated vessels, but the main part of the lesion is usually deep situated and cannot be reached by the coagulation current. The number of reported cases of the successful removal of an arterio-venous aneurysms is very small. Penfield reported 2 cases, Pilcher 3, Dolt 7; from the roentgenogram of Pilcher cases it appears that at least one of the cases corresponds to the type of the lesion we call a sturge-weber's disease which is a well defined anatomic and clinical entity and should be differentiated from the arterio-venous aneurysms. Pilcher is of opinion that most patients with these require radical surgical removal. The following cases represent all the intracerebral arterio-venous removed from July 1946 to December 1948 in Neuro Surgical Clinic, Stockholm.

Case Summary.—Case I. Male student aged 19 years. In 1942 and 1945 the patient had attacks of subarachnoid bleeding, the last of which was followed by left sided hemiplegia. After this there was improvement, but a moderate left sided spastic hemiparesis especially of the leg still remained. He had occasional slight headache and was admitted to the Neuro-Surgical Clinic, July 1946.

Examination:—There was slight left sided central facial palsy. Moderate left sided hemiparesis was present, more pronounced in the leg, with slight atrophy, reflexes were increased and Babinski sign was positive on the left. Optic discs were swollen about 1 Dioptre.

Roentgenogram of the skull did not reveal any pathological changes in its structure of vascular channels. Posteriorly in the right parietal region, about 1 cm. from the mid-line, there was an irregular calcification of the size of a pea.

Angiography (*Fig. 1-a and 1-b, vide picture*):—The anterior cerebral artery was enlarged and tortuous and was the main feeding artery to an arterio-venous aneurysm of the size of walnut located in the parietal region near the mid-line. From the aneurysm there were some large draining vein to the superior longitudinal sinus. The calcification seen in roentgenogram was situated in the aneurysm.

Encephalography:—There was no dislocation of the ventricular system. The upper part of right lateral ventricle showed a slight local dilatation, behind which, at the level of the calcification, there was a slight depression of the upper wall of the ventricle. The left lateral ventricle showed no deformity.

Operation (July 16):—As a first step, the internal carotid artery in the neck was exposed and a loop put around it, thereby

making it possible to strangulate the artery if necessary. After opening the dura, some enlarged arterialized veins were found near the mid-line going to the superior longitudinal sinus. The medial surface of the hemisphere was carefully lifted away from the falx and the aneurysm was removed after ligating some larger tortuous operation, the patient was able to move his left leg, fist and toes. No blood transfusion was necessary.

Post-operative course:—The patient made a rapid recovery. The day after operation there was slight weakness and feeling of numbness in the left arm but these symptoms disappeared after a few days. In the left leg the weakness was perhaps a little more pronounced than before operation. There were no sensory disturbances. The patient left hospital on July 30. A control examination was done on September 6, 1946. There was pronounced improvement of the paresis and the patient was in good condition. Angiography showed that the arterio-venous aneurysm had been totally removed. The anterior cerebral artery of the left side was also filled with contrast. In November 1948 the patient was still in good condition and of full working capacity though on one occasion he had sudden feeling of numbness with twitching in the left arm which might have been an epileptic fit. No paresis followed.

COMMENT:—This case was the first in which an angiography was performed after the complete removal of an arterio-venous aneurysm. Some interesting facts regarding the circulation of the brain were observed. The dilated tortuous, anterior cerebral artery had regained its normal size and configuration. The peripheral part of the vascular tree were also better filled with contrast than on the first examination. The whole brain circulation had regained its normal appearance after removal of the vascular malformation. The clinical result of the operation must be considered as very good.

Case II.—Male worker, aged 31 years.

In 1937 the patient suffered a head injury with slight brain concussion. His present illness started in 1945, when during a bicycle race he fell and lost consciousness for a short time. This was followed by severe headache which continued for 3 weeks combined with vomiting and impaired vision. He gradually improved.

On February 2, 1947 he was brought to hospital in an unconscious state. On the same day he regained consciousness after some hours and complained of headache. He vomited and stiffness of the neck was observed. A lumbar puncture showed increased pressure and hæmorrhagic C.S.F. There was slight papilloedema, but no other neurological signs were found. He gradually improved. On March 24 he was admitted to the Neurological Clinic, Serafiene Lasarette and a few days later to the Neuro-Surgical Clinic.

Examination:—There was stiffness of neck. The eye grounds showed slight choking of discs. There was left sided homonymous hemianopia. Slight mental disturbances were noted.

Röntgenogram of skull was normal.

Angiography (Fig. 2):—There was a good contrast in the carotid artery and its intracranial branches. In the right parieto-occipital region there was an arterio-venous aneurysm of the size of a plum with two large feeding arteries coming from the vessels in the sylvian fissure. From the aneurysm there was a large draining vein to the superior longitudinal sinus.

Operation (April 1):—As a preliminary the carotid artery in the neck was exposed and a loop placed round the vessel to permit strangulation during operation if necessary. The aneurysm was located rather close to and under surface of the occipital lobe, about 2-3 cm., lateral to the midline there was also a rather large feeding artery coming from the posterior cerebral artery. The aneurysm reached the posterior horn of the lateral ventricle. This was opened, but there were not vascular connections between the aneurysm and the vessels of the choroid plexus. After the various vessels had been clamped, the aneurysm could be removed. A blood transfusion of 400 c.c. was given during the operation. After practically all big intracranial operations performed under local anaesthesia, the patient is left in position on the operation table for 3 hours; the loop around the carotid artery is usually removed immediately after operation, but in this case because of the patients prone position, it was done after moving the patient from the table to his bed. During this procedure the loop around the carotid artery fastened in something on the operating table and the artery was torn. Extreme severe bleeding occurred which, after some difficulty was arrested and the carotid artery was ligated. The patient's condition was critical for some moments till a blood transfusion had been given. Although altogether he was given 8 blood transfusions of 400 c.c. each, the post-operative course was then very good. He had no paresis and no sensory disturbances. The left sided homonymous hemianopia persisted. There were slight mental disturbances of the same degree as before operation; the patient left hospital in good condition on April 19. Since September 1947 he had had full working capacity in his ordinary work with improvement in mental symptoms and hemianopia.

COMMENT:—After complication of a lesion of the carotid artery, the practice of placing a loop around the carotid artery in applying vascular clamp during the operation was abandoned. The clinical result of the operation was very good.

Case III.—Male worker, aged 44 years, admitted to Neurosurgical Clinic, April 11, 1947.

Previous history:—Five years previously the patient had diplopia, slight pains in the left arm. During the past years he had sometimes complained of dizziness.

Present illness:—During the night of April 5, 1947 he awakened with severe headache and vomiting. He went to the toilet and was found there unconscious. He was immediately brought to Neurological Clinic.

Examination:—The patient was drowsy and able to answer, objective findings; slight left sided paralysis of the 6th nerve, possible diminished left corneal reflex, slight left sided ptosis, pronounced aphasia, homonymous hemianopia to the right and a right sided hemiparesis. The discs were not choked. There was no stiffness of the neck, no systolic bruit.

Röntgenogram of the skull was normal.

Angiography:—A left sided arterio-venous aneurysm of the size of a plum, was seen rather deeply situated in the middle of the sylvian fissure. The feeding arteries were not enlarged, but some dilated draining veins were found (*Fig. 3-a and b*). On admission the patient was rather drowsy. As he did not improve during the following days but became more somnolent, it was decided to operate.

Operation (April 16):—On opening the dura, the surface of the brain was covered by a subdural hæmatoma, 1–2 cm. in thickness. There was also an intracerebral hæmatoma of the size of a hen's egg in the sylvian fissure and the temporal lobe. After removing the hæmatomas with the sucker, some of the feeding arteries could be seen and ligated and the aneurysm could then be dissected free and removed. Immediately after the patient was able to move his right arm and leg, and also to answer. One blood transfusion of 400 c.c. was given at the end of the operation.

Subsequent course:—The first day after operation the aphasia was marked but then improved gradually. There was no paresis and sensory disturbances were absent. A right sided pleuropneumonia developed, but he improved and left hospital on May 13, 1947. Angiography showed that the lesion had been totally removed (*Fig. 3-c*). The aphasia had improved, but was still pronounced. In July he had an epileptic fit and was unconscious for some minutes. The last report was in August 1948. He was still rather aphasic and was not able to work.

COMMENT.—In this the development of a large subdural hæmatoma in connection with rupture of the arterio-venous aneurysm made an urgent operation necessary. An angiography was first made an urgent operation necessary and the lesion considered operable. The clinical end result in this case was not good. The large intracerebral hæmorrhage had destroyed too much brain tissue. The improvement of the hemiparesis occurred but the aphasic symptoms persisted and were too pronounced to render the patient capable of work.

Case IV.—Female housewife, aged 38 years.

In 1928 the patient had a head injury and was unconscious for 24 hours. During the years 1924 to 1930 she had attacks of headache once or twice a year, rather severe followed by vomiting, and each attack lasted for about 5 to 6 days. Since 1930 these attacks had become more frequent, once or twice a month, and were preceded by something which might be called a visual aura to the right in the visual field. During the last year her headache had been more continuous. Since 1941 there had been weakness and numbness in the left arm and leg. In 1942 epileptic fits occurred in the left side of the body with loss of consciousness. The patient was admitted to Neurological Clinic, Serafin Lasarette, June 6, 1947.

Examination:—Slight weakness of the left arm was found with impaired deep sensibility and astereognosis. There was no aphasia. A slight homonymous hemianopia in the upper part of the visual field to the right was found.

Röntgenogram of the skull was normal.

Encephalography.—On air injection after suboccipital puncture, no air got into the ventricular system.

Angiography after injection in the right carotid artery was normal. The patient was admitted to the neurosurgical clinic with suspected brain tumour. Ventriculography revealed a slight dislocation of the ventricular system to the right. The left temporal horn was dislocated upwards and there was an irregular indentation in the lateral wall of the lateral ventricle suggesting an expanding lesion in the posterior part of the left temporal lobe (*Fig. 4-d*).

Angiography in the left internal carotid artery revealed a large arterio-venous aneurysm, of the size of a hen's egg, in the left temporal lobe. The circulation of the blood through the aneurysm was very rapid and after a few seconds all the contrast had passed the aneurysm (*Fig. 4-a and b*).

Operation (July 18):—Dissection was performed close to the aneurysm which could be followed practically to the pole of the occipital lobe. Medially the aneurysm reached to the lateral ventricle, and the indentation seen in the ventriculogram corresponded to the large pathological vessels bulging into the lateral wall of the ventricle. There were vascular connections between the choroid plexus and the aneurysm, feeding arteries as well as draining veins. After ligating the vessels, the aneurysm was removed. Five blood transfusions, each 400 c.c. were given during the operation.

Post-operative course:—Immediately after the operation the patient was able to move the right arm and leg, and answer question. The next day she was a little drowsy, with aphasia and a right sided hemiparesis. Subsequently there was marked improvement and

when she left hospital on August 7 there was only slight sensoric aphasia right sided hemianopia.

Arteriography on July 31 showed that the aneurysm had been totally removed (*Fig. 4-c*).

At examination on Aug. 31, 1948 there was marked improvement. Only a slight amnesic aphasia could be observed. There was no right sided hemiparesis. The left sided hemiparesis was of the same degree as before operation. There had been some slight attacks of petitmal. She had full working capacity.

COMMENT.—That this formidable lesion in the left temporal lobe was responsible for the Jacksonian fits in the left side of body is, of course questionable. If this was the case, and the future might give an answer to this question, it is very remarkable. The clinical result of the operation was very good.

Case V.—Male, clerk, aged 36 years. In 1932 and 1934 he had head injuries with loss of consciousness.

Present illness:—Started in 1942 with sudden attack of unconsciousness for 7–8 minutes. Similar attacks occurred 5 to 6 times during the following years. They were unaccompanied by epileptic fits, and there was no paresis afterwards. Since 1946, 2 to 3 times a month he had twitching in the left side of the face with a feeling of numbness and paræsthesia also noted in the left arm. For the last few months headache of varying degrees had been present. During the past years he had slight mental disturbances. He was admitted to the Neurological Clinic, March 31, 1947.

Examination:—Neurological findings aside from partial 3rd nerve paralysis on the left side, were negative. On auscultation over the right maxillary sinus a short bruit was heard synchronous with the pulse.

Röntgenogram of skull and encephalography normal.

Angiography:—There was good contrast in the sylvian vessel but none in the anterior cerebral artery. In the posterior part of the right frontal lobe, about 1 cm. above the sylvian fissure an arterio-venous aneurysm of the size of a plum with some dilated feeding arteries from the sylvian vessels and a very large vein going to the superior longitudinal sinus. In the middle of the temporal lobe there was another smaller arterio-venous aneurysm disclosed (*Fig. 5-a and b*) as the symptoms were very slight it was decided not to operate and he was discharged from the hospital. However as the epileptic fits increased in frequency the patient after a few months became very anxious about his condition and wanted to take the risk of operation.

Operation (August 1947):—The brain surface was very vascular with enlarged tortuous arteries. One of the arteries was

followed down into a brain sulcus, where the aneurysm could be grasped with forceps and lifted out after the feeding arteries and draining vein had been ligated with silver clips. The same procedure was then carried out with the other aneurysm. There were several large vessels between the two aneurysms. Some of these were extirpated but others had to be left alone. These vessels, were very fragile and it was difficult to stop the bleeding from them.

Post operative course:—During the first few days there was a paresis of the left hand with numbness. This however rapidly improved and when the patient left hospital on September 17 there was only a very slight weakness and numbness left, more especially in the thumb.

Angiography:—September 15 showed that both aneurysms had been removed but that between them there were still some pathological vessels (*Fig. 5-c*). The patient started to work in January 1948, he still had some headache but not to the same degree as before operation. He had 3 attacks of epileptic fits, Jacksonian type, localized to the left leg and arm. He was a little restless and nervous.

Angiography:—September 27, 1948 showed that the widened pathological vessels between the two aneurysms had disappeared (*Fig. 5-d*).

COMMENT.—An unusual case in which two aneurysms were removed at one stage. An arteriogram 1 year after operation revealed the interesting facts that the dilated pathological vessels between the two lesions, observed immediately after the operation, had disappeared.

Case VI.—Female, house wife aged 30 years.

In 1946 and 1947 the patient had attacks of sub-arachnoidal hæmorrhage with complete recovery. On Jan. 8, 1948 another attack occurred and she was admitted to the Neurological Clinic.

Examination:—The patient complained of severe headache. There was no bruit. Neurological findings were pronounced stiffness in the neck, and homonymous hemianopia on the left side. After some days the hemianopia disappeared. Angiography revealed an arterio-venous aneurysm of the size of a mandarin, in the right parietal lobe. Most of the feeding arteries came from the sylvian fissure. There were also some from the anterior cerebral artery. There were widened draining veins to the superior longitudinal sinus. The aneurysm reached rather deeply into the brain tissue and the deepest point was only 2 cm. from the midline (*Fig. 6-a and b*). The patient was admitted to the Neurosurgical Clinic on Feb. 2, 1948 where an operation was performed on the same day. One of the feeding arteries was followed into the depth of the brain tissue where the aneurysm was found surrounded by sclerotic discoloured

brain tissue indicating that hæmorrhages had occurred. The dissection of the aneurysm was therefore rather difficult as only after ligating the feeding arteries around the aneurysm, and the draining vein, the aneurysm could be removed.

Post-operative course:—The patient made a rapid recovery. There was no post-operative paresis and she was able to leave Hospital on Feb. 17. The day before her leaving an angiography had been performed which showed that the arterio-venous aneurysm had been totally removed. The feeding arteries were of normal size (*Fig. 6-c and d.*) On control examination on May 4 the patient was in a very good condition and of full working capacity. There were no subjective symptoms, no paresis and the visual field was normal. The clinical result of the operation was excellent.

Case VII.—Male, farm labourer, aged 34 years.

In 1924 the patient had a head injury. Since 1925 he had epileptic fits, with numbness in the right hand and arm, and right side of neck, combined with loss of consciousness for 5 minutes. There was no paresis afterwards. Slight mental symptoms were noted. He had no headache.

Examination:—Neurological findings were normal. Roentgenogram of skull and encephalography were normal.

Angiography:—In the left parietal lobe an arteriovenous aneurysm of the size of a walnut was found. Particularly one of the feeding arteries from the sylvian vessels was enlarged and tortuous. One widened draining vein to superior longitudinal sinus was seen. There was no contrast in the anterior cerebral artery (*Fig. 7-a and b*).

Operation (April):—The larger feeding artery was ligated close to the aneurysm which could then be dissected free from the surrounding brain tissue and removed after ligation of the draining vein.

Post-operative course:—The day following the operation there was no paresis, but this gradually developed in the right side, together with aphasia, and the patient had repeated Jacksonian fits. These symptoms disappeared and when the patient left the Hospital on April 24, there was only slight weakness in the right arm, more pronounced weakness in the right foot and slight hyperæsthesia, of the right foot and leg. Reflexes on the right side increased, Babinski sign negative bilaterally.

Angiography on April 17 showed that aneurysm had been totally removed. The dilatation of the feeding artery had diminished (*Fig. 7-c*). On examination in October 1948 the hemiparesis had completely disappeared. The patient had epileptic fits of the same type as before, but more frequent.



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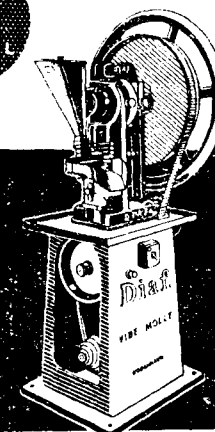
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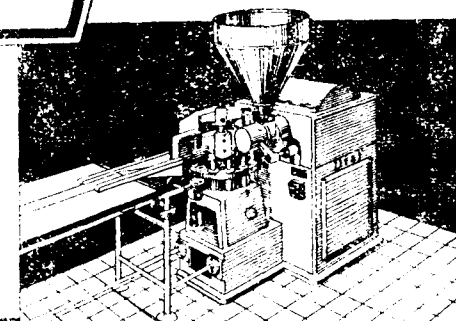
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COMMENT:—The hemiparesis and aphasia that appeared some days after operation were probably due to local oedema. The angiography done half an year after operation showed that brain circulation had regained its normal appearance from a clinical point of view the frequency of the epileptic fits had increased. In spite of such a lesion with this localization even if the clinical symptoms are very slight, should be removed. A rupture of this aneurysm might have given an intracerebral hæmorrhage with destruction of brain tissue causing an irreparable hemiplegia. The patient is full of working capacity.

Case VIII.—Patient, male, aged forty. Since 1932 this patient had Jacksonian fits in the right side of the body starting in the right foot. The frequency of the fits had increased during the last years. The first year he lost consciousness in connection with the fits, but not during the later years. After the attacks he had a feeling of numbness in the right arm and leg and the next day he became somnolent. He was admitted to the Neuro-surgical Clinic on April 9.

Examination:—Neurological findings were pronounced right sided hemiparesis with total paralysis of the arm and bilateral choked discs. There was no aphasia.

Röntgenogram of the skull was normal.

Angiography:—After injection into the left internal carotid artery, in the parietal lobe an arterio-venous aneurysm of the size of a mandrin was found. The lesion was localized near the midline and received its arterial supply both from the veins to the superior longitudinal sinus. (*Fig. 8-a*). An angiography was also performed after injection into the right internal carotid artery. There was contrast in both anterior cerebral arteries, and the aneurysm was clearly seen in this examination (*Fig. 8-b*).

Encephalography after sub-occipital puncture revealed a slight dilatation of both lateral ventricles, a little more pronounced on the right side. The upper wall of the left lateral ventricle was a little depressed corresponding to the localization of the aneurysm.

Operation (April 22):—On the medial surface of the hemisphere some feeding arteries from the anterior cerebral artery were ligated. The dissection of the aneurysm was rather difficult, and was complicated by a lesion of one of the enlarged draining veins. After removal of the aneurysm there was some bleeding from dilated vessels surrounding the aneurysm. The only possible method of checking that bleeding was to put a piece of spongistan on the bleeding surface and leave it in place. On turning down the flap a lesion of the superior longitudinal sinus had occurred so it was necessary to suture it and cover the sinus with some pieces of spongistan. Eleven blood transfusion of 400 cc. each were given during the operation.

Post-operative course:—The first day there was a right sided hemiplegia, but in a few days the movements of the leg returned. There was no aphasia.

Angiography (On May 14) showed that the aneurysm had been removed. Dilatation of the anterior cerebral artery persisted partially. The enlargement of the feeding arteries from the middle cerebral artery had diminished. The patient left hospital on May 21 (*Fig. 8-c*). On examination on August 24, a pronounced improvement was seen. He was able to walk around with a stick and move the right arm slightly. He had two small epileptic fits with twitchings in the right leg without loss of consciousness. In February 1949 he was able to walk around without a stick; but the right arm was still rather paretic.

Angiography showed that the pathological vessels had disappeared and the anterior cerebral artery had regained its normal size (*Fig. 8-d*).

COMMENT.—A post-operative study of the brain circulation showed that vessels of abnormal size and configuration regaining their normal appearance after the removal of an arterio-venous aneurysm.

Case IX.—Female, aged 52, house wife. For 1½ years before admission the patient had observed that the left part of the visual field sometimes disappeared for a few seconds. In the summer of 1947 she had pain in the occipital region, with stiffness on the neck. After a week she was all right again. There was no other symptoms. On March 16, 1948 she had new attack of headache, slight dizziness and subjective left sided homonymous hemianopia. After a few days the headache increased, continued with vomiting and stiffness in the neck. She was admitted to the neurological clinic where left sided homonymous hemianopia was observed and a lumbar puncture revealed a sanguineous C.S.F.

Röntgenogram on both sides of the skull showed rather large vascular markings, but no other changes in its structure and no distinction. The foramen spinosum was rather larger but of the same size on both sides.

Angiography was performed on several occasions in the carotid arteries of both sides. On the right side, close to the mid-line in the occipital region, there was a small aneurysm and a little more laterally a vascular saccular malformation of the size of a pea was seen; from this region there were some dilated draining veins. The feeding arteries to this vascular malformation appeared to be two enlarged tortuous branches from the external carotid artery, probably the posterior branch of the middle meningeal artery, one on each side (*Fig. 9-a and b*). The lesion was considered to be an arterio-venous aneurysm in the right occipital lobe.

Encephalography:—There was no dislocation or deformation of ventricular system. In the right occipital lobe near the mid-line

corresponding to the localization of the arterio-venous aneurysm there was signs of local atrophy.

Operation (July 19):—The middle meningeal artery was dilated. No abnormal connections between the dura and the brain were seen. An incision was made 2-3 cm. laterally from the midline where the brain tissue was a little softer than usual. A cystic formation of the size of a walnut, was found communicating with the ventricular system. In the medial wall of that cyst there was brownish green, rather dense formation of the size of a walnut which looked like an organised hæmatoma. From the middle of this formation no enlarged feeding arteries could be seen. It was very easy to remove the whole formation. Microscopical examination revealed an arterio-venous aneurysm with large vessels of arterial type with very thick walls surrounded by an organized hæmatoma.

Post-operative course:—Good recovery. No paresis, but a left sided hemianopia was still present when the patient left hospital on Aug. 5.

Angiography on August 2 showed that the vascular malformation had been removed (*Fig. 9-c*).

COMMENT:—The angiographic study in this case was rather difficult and it was not so easy to come to a conclusion regarding the localization of this lesion. A thrombotization and organization of the aneurysm had occurred. The clinical result was excellent and the patient is of full working capacity.

Case X.—Female, aged 20, house wife. In 1940 a sudden attack of right sided hemiplegia occurred. The patient was admitted to hospital where a lumbar puncture revealed a subarachnoidal hæmorrhage. There was marked improvement during the next month and after 6 months she was alright again. Since 1943 she had attacks of paræsthesia in the right arm and since Mar. 1944 epileptic fits of Jacksonian type, starting in the right arm with loss of consciousness. These attacks had occurred about 3 times a year, but during the last year this had become more frequent. She was admitted to the Neurosurgical Clinic on Nov. 10, 1948.

Examination:—The eye grounds were normal and disturbance of deep sensibility of the right foot was found. Reflex is increased and Babinski sign was positive on the right side. Röntgenogram of the skull was normal.

Encephalography revealed a light dilatation of the left lateral ventricle.

Angiography:—Above the posterior part of the sylvian fissure an arterio-venous aneurysm of the size of a mandarin was seen. The lesion reached down rather deep, the deepest part being only ½ cm. from the midline. There were enlarged feeding arteries mainly from the sylvian vessels but also some feeding vessels from the

anterior cerebral artery. Widened draining veins went to the superior longitudinal sinus (*Fig. 10-a, b*).

Operation (Nov. 17):—Some dilated vessels were seen on the surface, but the aneurysm was rather deeply situated close to the wall of the lateral ventricle with vascular connection with the choroid plexus. Posteriorly the aneurysm was limited by a cavity which had been formed by previous hæmorrhage. Because of the deep localization of the aneurysm and the vascular connections with the choroid plexus the removal of the lesion was rather difficult. At the end of the operation there was a pronounced motor aphasia and complete hemiplegia.

Post-operative course:—During the days following the operation there was gradual improvement of the hemiplegia and when the patient left hospital on December 10 she was able to walk around but there was still a paresis of the hand and fingers though improved. On discharge from hospital no aphasic disturbances could be observed.

Angiography on (Dec. 7) showed that the aneurysm had been removed and that the size of the feeding arteries was now normal (*Fig. 10-c and d*).

COMMENT:—The size and localization of this lesion made an attempt at removal rather hazardous. On the other hand rupture of the aneurysm might have caused irreparable hemiplegia and aphasia. The ultimate result could not as yet be judged; but the post-operative course speaks in favour of a good result.

Summary.—The patients with these lesions die of hæmorrhage or are completely incapacitated and that the mental deterioration which is a common feature in case of inveterate lesion also strengthens the argument in favour of removal of the lesion. During the last few years the indication for operation have been widened and even large aneurysms in the left hemisphere have been successfully removed. The poor prognosis for epileptic cases with an inveterate lesion speaks in favour of an early operation in those cases where epilepsy is the only symptom. Pilcher recommends radical surgical removal of these lesions but adds that nevertheless, the surgeon must be able to recognize the limits of such possibilities and must have the courage to withdraw when faced with lesions beyond these limitations. The clinical result of these operations has been very good in 7 patients and all are of full working capacity (Cases 1, 2, 4, 5, 6, 7, and 9). In 2 cases an improvement had occurred when the symptoms before and after the operation were compared; but both were invalids, one with pronounced aphasia and the other with hemiparesis. The most interesting thing in this series was the angiographic study of the brain circulation and the appearance of brain vessels before and after removal of the lesion. In most of the cases there was a very rapid return of vessels to the normal, usually within 2-3 weeks, which before the operation were enlarged and

Arterio-venous Aneurysms as a Neurosurgical Problem

Dr. R. S. Seagat

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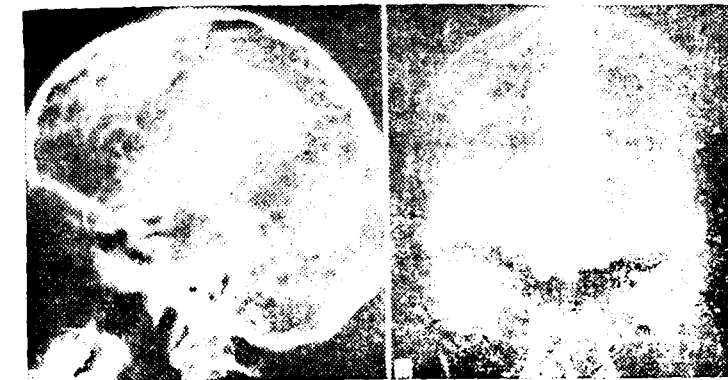


Fig 1-a

Fig 1-b

Fig 1-a, 1-b:—Note the difference in size and appearance of the ant. cerebral artery on the angiograms performed six weeks after the operation. [Vide page 999]



Fig 1-c

Fig 1-c:—Six weeks after the operation.

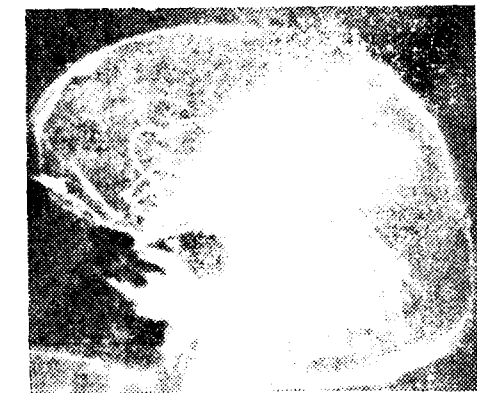


Fig 2

Fig 2:—Arterio-venous aneurysm in the right parieto-occipital region

[Vide page 1001]

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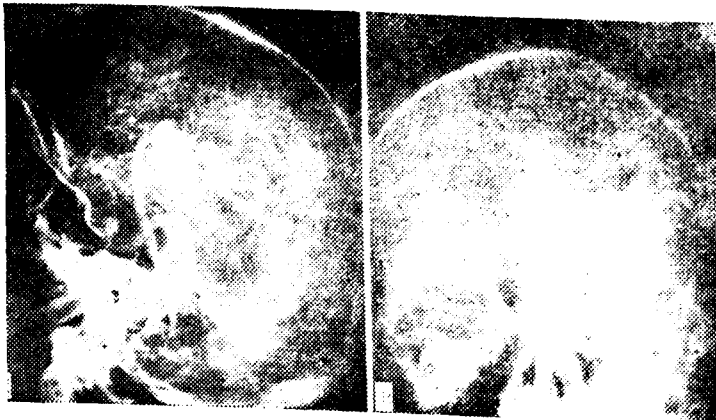


Fig 3-a

Fig 3-b

Fig 3-a, 3-b:—Left sided arterio-venous aneurysm deeply situated in the middle of the sylvian fissure. [Vide page 1002]



Fig 3-c

[Vide page 1002]

Arterio-venous Aneurysms as a Neurosurgical Problem

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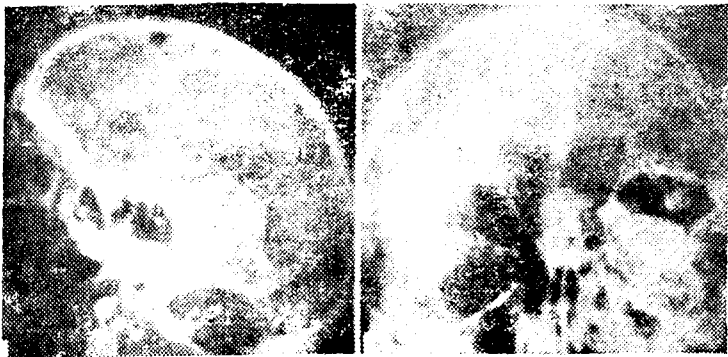


Fig 4-a

Fig 4-b

Fig 4-a, 4-b, 4-c:—Note the difference in contrast filling of the anterior cerebral artery in A and C. [Vide page 1003]

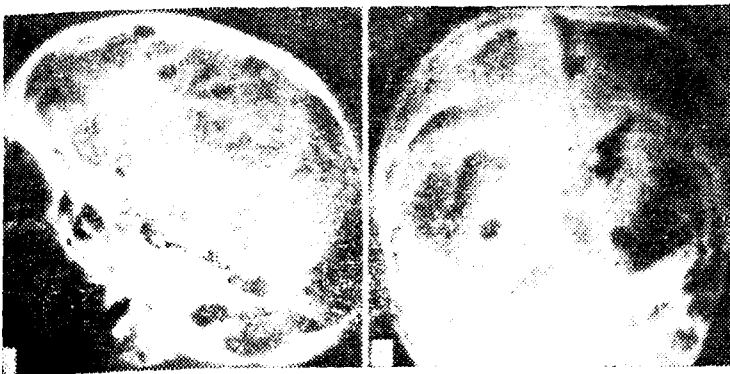


Fig 4-c

Fig 4-d

[Vide page 1004]

[Vide page 1003]

Fig 4-d:—The indentation in the lateral wall of the left lateral ventricle caused by the bulging vessels of the aneurysm.

Arterio-venous Aneurysms as a Neurosurgical Problem
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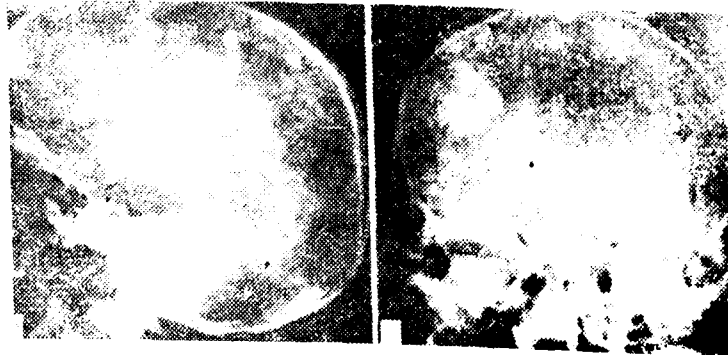


Fig 5-a

Fig 5-b

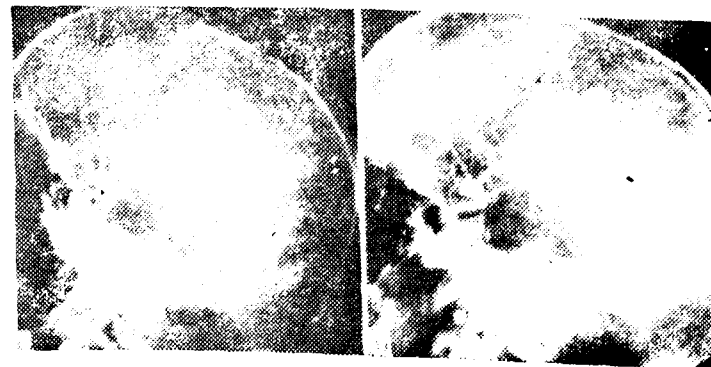


Fig 5-c

Fig 5-d

Fig 5-a, 5-b, 5-c, 5-d:—The contrast filling of the ant. cerebral artery which is practically lacking in the first examination (a) is clearly seen on the angiograms two weeks after operation (c).

In the latter some pathological vessels between the aneurysms are still evident, but on angiogram one year after the operation (d) these vessels have a normal appearance. [Vide page 1004 and 1005.]

Fig 6-a
[Vide page 1005]Fig 6-b
[Vide page 1006]

Fig 6-a, 6-b: Arterio-venous aneurysm in the right parietal lobe

Arterio-venous Aneurysms as a Neurosurgical Problem
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Fig 6-b

Fig 6-d

[Vide page 1005]

[Vide page 1006]

Fig 6-c, 6-d:—The feeding arteries were now of normal size.

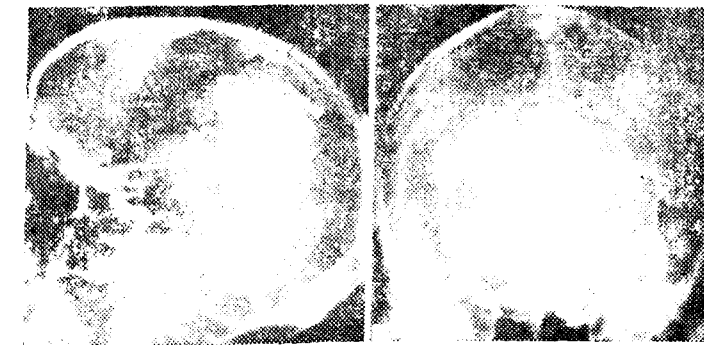


Fig 7-a

Fig 7-b

Fig 7-a, 7-b:—Arterio-venous aneurysm in the left parietal lobe
[Vide page 1006]



Fig 7-c

Fig 7-d

Fig 7-c, 7-d:—The main feeding artery three weeks after the operation is still dilated (c) but on an angiogram (d) 6 months later the artery has regained its normal size and appearance.

[Vide page 1006]

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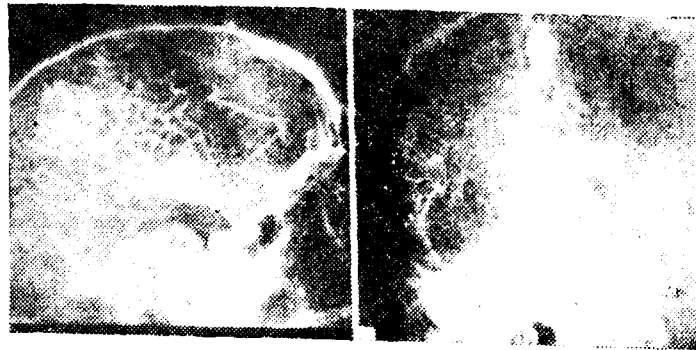


Fig 8-a

Fig 8-b

[Vide page 1007]



Fig 8-c

Fig 8-d

[Vide page 1008]

Fig 8-a, 8-b:—Arterio-venous aneurysm in the left parietal lobe (a) and is also seen after injection in the right carotid artery (b). On an angiogram 3 weeks after the operation there are still some dilated vessels (c) the anterior cerebral artery is enlarged, but nine months later the pathological vessels have disappeared and the anterior cerebral artery is of normal size (d).

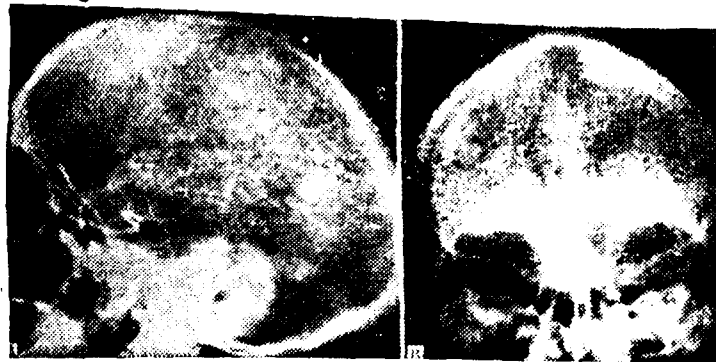


Fig 9-a

Fig 9-b

Fig 9-a, 9-b, 9-c:—Of the whole aneurysm only a small saccular malformation is filled with contrast. A thrombotization of the rest of the aneurysm had occurred.

[Vide page 1008]

Arterio-venous Aneurysms as a Neurosurgical Problem
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Fig 9-c

[Vide page 1009]

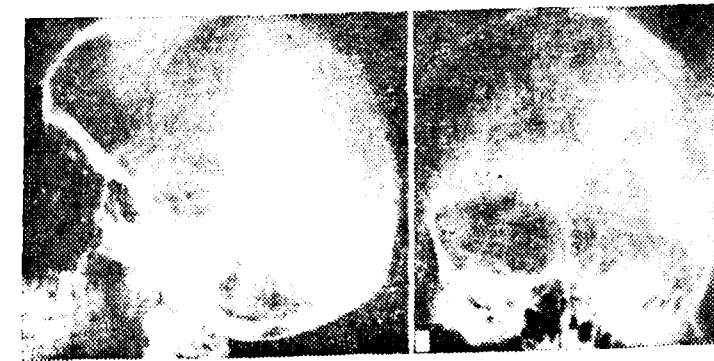


Fig 10-a

Fig 10-b

Fig 10-a, 10-b:—In the frontal picture (b) the connection between the anterior choroidal artery and the aneurysm is clearly seen (a) arterio-venous aneurysm above the posterior part of the sylvian fissure.

[Vide page 1010]

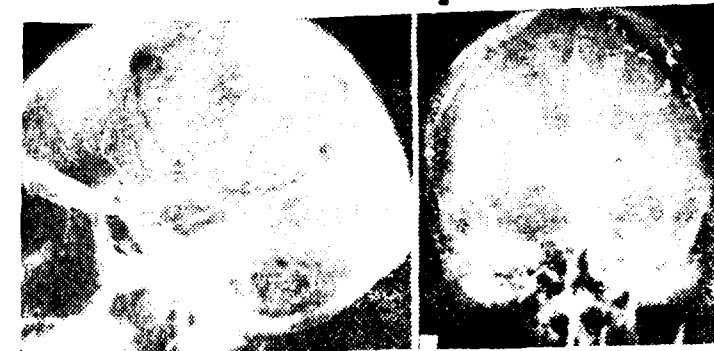


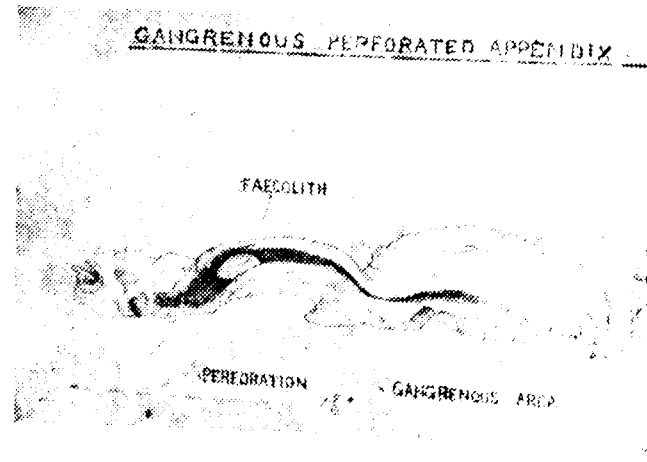
Fig 10-c

Fig 10-d

Fig 10-c, 10-d:—The size of the feeding artery was now normal.

[Vide page 1010]

Acute Appendicitis with Atypical Clinical Features
D. J. Reddy



[Vide page 1015]

Appendix laid open: Shows the fecolith and couple of perforations.

Decapitation
S. C. Mathur



[Vide page 1016]

tortuous. In other cases pathological vessels were still visible in the angiogram after that time, but a few months later they regained their normal appearance (Cases 4, 7, 8). From angiograms performed before operation it was evident that the contrast filling of vessels not belonging to the group of vessels that went to aneurysm was very poor. This might indicate poor nutrition and anoxemia of these parts of the brain which might cause brain atrophy and be responsible for the mental symptoms in inveterate cases. After the removal of the aneurysm the contrast filling of these parts of the brain was good and clearly seen. (Case 1, 4, 5).

Acknowledgements.—The Illustrations produced in this article are by the courtesy of the Neurosurgical Clinic, Serafiene Lasarette, Stockholm.

Thanks in particular are due to Miss Champa Jagan Nath for typing and arranging photographs.

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Cases and Comments

ACUTE APPENDICITIS WITH ATYPICAL CLINICAL FEATURES

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THE history of recognition of vermiform appendix and the various morbid processes to which it may be subject to, dates back to 1524. Berengario da carpi of Bologna was the first to spot appendix in 1524. Nineteen years later Andreas Vesalius described the anatomy of this organ in his book—*Defabrica*. In 1711 Heister was demonstrating dissection of the body in the Public theatre at Altdroff. He noticed then pus being discharged by the accidental breakage of the appendix. Villarny suggested in 1824 that colic in the right iliac fossa may be due to an inflamed appendix. Three years later Melier gave full accounts of appendicitis and apprised the profession with the fatal issues of the condition. Credit goes to Reginald Fitz who in 1886 grouped inflammatory conditions of appendix under appendicitis and also described the fatal complication of perforation. Three years later Charles McBurney described the point of maximum tenderness on the cutis in appendicitis which every student of medicine now remembers as "McBurney's point". McBurney also has to his credit improvisation of the incision for surgical approach to the appendix (1894).

One out of every seven hundred of the population is said to suffer from appendicitis. Appendicectomy is the commonest and life saving surgical operations performed—specially as an emergency. Appendicitis is said to be rare in India. This is due to the high roughage diet of the Indian. McCarrison is said to have met with no case in the hill tribes in North West India for 9 years. In Asiatics and Africans appendicitis is rare unless they take to European food. In this respect the wild animals compare well with them while animals kept in captivity in zoological gardens appendicitis is frequently being reported. It is apparent from what is said above that dietary habits, lack of physical exercises, and modes of life which restrict the natural tendencies to frequent and complete defaecation favour incidence of appendicitis and so more common in the urban population. Appendicitis is said to be considerably less in the non-meat-eating orients. 20.7% of 123 cases quoted by Kini *et al* have occurred in vegetarians. It must be realised that many cases of appendicitis pass off unrecognized owing to lack of medical attention in the several rural areas of our country.

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Appendicitis is most common in the age group 20 to 30. Although appendix is a tiny little organ it is subject to acute, sub-acute and fibrosing types of inflammation with or without obstruction and is also the site of tumour formation. Acute inflammation of the appendix may be catarrhal or suppurative and may be associated with gangrene and/or perforation. Perforation often supervenes in an inflamed and obstructed appendix. Exacerbation of an acute attack is not uncommon in a case of subacute appendicitis.

Inflamed appendices removed at operation are often found to contain thread, whip and round-worms and orange pips or faecoliths. It is wise to rule out amœbiasis of appendix by a search for the amœba in the faeces as Clark reported 50% incidence of amœbic ulceration of appendix associated with intestinal amœbiasis.

Acute abdomen forms a significant percentage of surgical admissions and it was 4.3% in Kini's unit. Deaths from untreated cases of acute abdomen are not wanting. Out of 60 autopsies of deaths due to natural causes five died of acute abdomen in 1945-1947 in Burma. They were of perforated gangrenous appendicitis, intra-abdominal rupture of amœbic hepatic abscess, acute pancreatitis, perforated duodenal ulcer and hæmorrhage in an extra-hepatic bile duct carcinoma.

That acute suppurative appendicitis forms one of the acute abdominal conditions is well known to medical practitioners. Symptoms of acute appendicitis in the majority of cases are typical and marked by pain starting round about the umbilicus, localising around the right iliac fossa in 24 hours and associated with vomiting, temperature of 100°F. and maximum tenderness over the McBurney's point. Occasionally the symptoms are atypical and are far from being stereotyped. There are few abdominal conditions that can present as many different clinical pictures as acute appendicitis. Out of 198 recorded cases of Kini *et al* in one case pain was localised to the left hypochondrium. This is not surprising if one recalls to the mind that the appendix is variable in size, mobility and location. The table below indicates the percentage incidence of location of appendix met either at operation or autopsy.

Author	Number observed	Retro-colic %	Pre-ileal %	Para-colic %	Ectopic %	Pelvic %	Splenic %
P. C. Wakely	5000	59	4	4	0.6	24.5	7.6
Kini <i>et al</i>	32	71.9	3.1	3.1	—	19	3
D. J. Reddy	39	76.9	—	—	—	18	5

Prognosis in appendicitis is conditioned by the availability of immediate surgical aid. Mortality is less than 1% if operated before 12 hours of onset and mounts upto 11-18% when delayed till 36 hours. (Bailey, 1932). In over 80% peritonitis was the

cause of death. Christopher and Jennings reported 5% mortality for acute appendicitis while Halcomb reported 5 deaths in 153 cases, three of which were due to perforation and one was gangrenous. Mortality rate for appendicitis in King George Hospital for 1948 as recorded by Kini *et al* is 11% and significant. This is because 7 out of 16 cases were treated by conservative method. Perforation is one of the fatal complications of appendicitis but if recognised early and tackled by surgery supplemented by liberal use of Sulphonamides and antibiotics there may be no deaths at all. This is well substantiated by the fall in mortality figures from 3178 to 1774 for the years 1935 and 1945 respectively recorded by the Registrar-General for England and Wales.

Case report.—Major P.T.A., aged 28 years, was admitted in the Civil Hospital Tongyi (Burma) on 20-2-'47 for pain over the upper half of abdomen. The pain started at 9 a.m. on 19-2-'47. Vomited frequently. Pain was continuous and colicky in nature. On examination he was found to be restless. Abdomen distended to a slight extent. Temperature 99°F. Pulse: 100 per minute.

At 3-30 p.m. on 20-2-'47 the pain was severe over lower and left half of abdomen. He was evacuated to 49 Indian General Hospital, (80 miles being covered by an ambulance car).

Examination at 49 Indian General Hospital at 23.00 hours on 21-2-'47 revealed dry and furred tongue, distended abdomen and marked tenderness over the left iliac fossa. Left flank was dull to percussion and shifting dullness not elicitable.

Rectal examination: Tenderness over left iliac fossa+.

Enema:—Flatus only passed. W.B.C. count:—16,400 per cm.m. with 87% polymorphs; 12% lymphocytes and 1% monocyte. Urine contained albumin and granular casts. Pulse: 148 per min. Blood pressure 140/100 mm. of Hg.

Preoperative diagnosis.—“Diverticulitis with perforation.” Laparotomy was done at 03.10 hours on 22-2-'47. Pus was seen welling out of left paracolic gutter. Abdomen was closed by inserting suprapubic and left paracolic drainage tubes. Caecostomy was done. Evidences of peripheral circulatory failure were manifest. Patient was toxic and cyanotic. Temperature rose upto 106°F. Expired at 21.35 hours on 23-2-'47.

Autopsy findings.—The greater omentum was spread out. The paracolic gutters and pelvic cavity were filled with brownish turbid, thick pus. Coils of small intestine were glued to each other by flimsy flakes of fibrin. Well buried under the last few inches of the ileum and adherent to the retroperitoneal tissues was seen the much stretched out and elongated appendix. It was pointing towards the spleen and nearly approached the mid-line of the body. It was $7\frac{5}{8}$ inches in length. At the tip and an inch behind from the tip of the appendix were seen a couple of yawning type

of perforations with ragged edges. This area of the appendix was gangrenous. The path of the probe was obstructed by the presence of a well formed faecolith of the size of a peanut (*vide picture*). Coils of descending colon were stuck to the parietes by flakes of fibrin.

Autopsy diagnosis.—“Gangrenous perforated appendix.”

Summary and conclusions.—1. The dangers of acute appendicitis are discussed. The necessity for the physicians to realise fully that serious infection of appendix occurs without the so called “typical picture” is stressed. It pays well to picture in mind all the possible locations of appendix when one is confronted with atypical cases of appendicitis.

2. Acute appendicitis with localising signs and symptoms on the left half of the abdomen may be mistakenly diagnosed as “Diverticulitis”. In all these cases the appendix will be found lying in and around the pelvis and extending across the middle line. Operation is urgent in all these cases.

3. Early recognition and immediate surgical interference under the help of Sulphonamides or Antibiotics are the only certain methods to reduce the high mortality rate for acute appendicitis.

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Vitamin C Ascorbic Acid for Food Preservation

In addition to its role in animal and plant nutrition, the chemical properties of ascorbic acid have been useful in retarding colour and flavour changes in food products caused by oxidation and enzyme action. In a paper presented to the Food and Agriculture Division of the American Chemical Society Dr. Baurneind described the process. In addition to uniting with the oxygen of the air ascorbic acid can serve as a primary substrate or is involved indirectly in the activity of peroxidase etc. The ascorbic acid treatment of frozen cut fruit is now an accredited procedure in commercial freezing.

The development of the oxidative off-flavour in milk, and other dairy products can be retarded by adding ascorbic acid (Vitamin C) crystals; while treatment with small quantities of ascorbic acid is not a panacea for all troubles and deteriorative changes in food products it is certainly a method by which such undesirable changes can be delayed.—From *Food Manufac.*, London, Nov. 1950.

DECAPITATION

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DECAPITATION is a term applied to the method by which the neck of the foetus is cut to separate the head from the rest of the body.

It is to be performed in any of the following three types of cases :—

- (1) Impacted shoulder presentation when the foetus is dead and other methods of removing it fail.
- (2) Locked-twins.
- (3) Double monsters.

Case Report.—A female, named Dattu, aged 35 years, Patel by caste, was brought to the Hospital at 8 p.m. on 12th October '51 from a distance of 4 miles for delivery, as every attempt made by the quick dai who was attending her at her house had failed.

Previous history.—It was the seventh delivery and all her previous labours were normal. V. D. denied.

Present history.—It was not possible to gather the record as to when the membrane ruptured, and how long she had been in labour but it was reported that the hand was prolapsed for the last 6 hours.

Physical examination.—The patient was placed in lithotomy position for examination. The swollen hand of the foetus was protruding out of vagina. On examining internally by introducing the hand, the head and the breech were found lying in left and right iliac-fossa respectively. The head was lower than the breech in the womb and the back of the foetus was facing anteriorly. The child was dead. The local examination resulted into the conclusion that it was a case of shoulder presentation with dorso-anterior-lie.

N A D. on general examination.

TREATMENT.—The bladder and rectum were evacuated by means of catheter and soap water enema respectively. The parts were cleaned. The patient having been anesthetized, was placed in lithotomy position. Attempts were made to dislodge the presenting parts and to perform the internal version, converting into a foot presentation, but it was not possible. Efforts were made to deliver the child by evolution but it was a failure and now the decapitation remained the only remedy.

A clove hitch was tied around the wrist of the foetus which was prolapsing and the mid-wife was asked to apply traction on it, to bring down the neck of the child as much as possible. The left hand was introduced into the birth canal and the neck of the child was caught between the thumb and the index fingers. The decapitation hook was then introduced guided by the fingers of the left hand and the neck of the child was caught in the hook. Gentle traction was applied to the handle of the hook and it was moved to and fro, which divided the neck. The hook with serrated edge is preferred but it was not available and a sharp-edged hook being ready was employed.

The body of the child was removed by applying traction over prolapsed arm, the cord was cut and the child was separated. The hand was then introduced again and the head of the foetus was caught by introducing two fingers in his mouth and removed presenting partly face and neck. The placenta was gently separated from the wall of the uterus and removed by hand. There was not much P.P.H. the parts were cleaned and dressed and the patient was placed in Fowler's bed. She was injected four lacs units of Penicillin as a prophylaxis against puerperal sepsis and was given Ergot Quinine mixture. Penicillin was stopped from the next day as there were no signs of sepsis. The involution of the uterus was normal. As nothing was abnormal she was discharged from the hospital on the 6th day on the request of her relatives.

The photo of the foetus extracted, is appended here. (*Vide picture*).

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BACILLUS COLI INFECTION*

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1. The importance of this subject lies in the fact, that although manifestations of *B. coli* infection in human body are fairly common, they are easily overlooked and unsuspected and mostly mistaken for other common diseases like malaria, fevers of enteric group, worms, and tuberculosis in different parts of the body, particularly that of the intestines. This common error in diagnosis at an early stage, makes the illness a protracted one, with different clinical pictures at different stages of the illness, and making the case more complicated. Ultimately if *B. coli* infection is suspected, to confirm it, laboratory cultural methods become essential, which are easily not available, or the disease has to be confirmed and effectively treated or eliminated by an effective therapeutic test. Thus it is worthwhile to make an attempt to evaluate certain clinical facts, which can enable a practitioner, particularly one in the mofus-il, to differentiate this pathological condition from other common diseases for which it is easily mistaken.

2. The colon bacillus is absent in the colon of a newly born infant. It gets entrance into the system after birth, along with water, milk and other ingesta, and it thrives and continues its existence, in the colon of a human being, as a saprophyte, i.e. a non-pathological organism. Under certain favourable conditions it becomes pathological, migrates into other parts of the body, particularly organs of genito-urinary system and gives rise to different diseases, namely pyelitis cystitis, pyo-nephrosis, bacilluria, and bacteraemia. Other diseases which can be caused by the colon bacillus are appendicitis, cholangitis, cholecystitis, peritonitis and so on. In one particular case, more than one of the above pathological conditions, can be and are commonly present at one and the same time, and present a clinical picture, which is not constant and therefore likely to confuse the attending physician. It is believed that the bacillus can reach the kidney by three possible routes namely :—(1) through the blood-stream, (2) by ascending infection along the urinary tract, particularly in girls the urethra being short, and (3) directly from the ascending colon to the right kidney, both being adjacent. All these three methods of transmission of the bacilli appear to be possible, different routes being responsible in different cases.

3. The most common clinical condition met with is pyelitis. The patient is generally a girl. Boys are also affected. There is a sudden rise of temperature preceded by chill and there are other constitutional symptoms. In majority of cases, this is mistaken for malaria and treated as such, but with little success. The type of temperature in *B. coli* infection is not always constant. It may be intermittent and even remittant. Not properly diagnosed and treated at an early stage, this pyelitis may be superadded by cystitis, colitis, bacilluria etc. and the clinical picture may become more complicated and difficult to interpret. Secondary infection at this stage can give rise to symptoms of lung affections, and make the doctors job more difficult. Cases at this stage are mistaken for typhoid or paratyphoid A or B, and if the illness gradually assumed a more chronic aspect, it is mistaken for tuberculosis, mostly of the intestinal tract. Unless the *B. coli* infection as a possibility is kept in mind, the mistake is common, even at the hands of an experienced doctor.

4. **DIFFERENTIAL DIAGNOSIS.**—(a) If malaria is suspected, either blood examination for M P, or a therapeutic test by one or two Quinine injections along with exhibition of other suitable anti-malarial drugs, will enable to eliminate it.

* This article is based on a clinical talk, which the writer gave, in a recently held Annual Meeting of the Saurashtra Branch of the Indian Medical Association.

(b) If a fever of enteric group is suspected, points in its favour are, clear remittent type of temperature, with a slow pulse, typical typhoid tongue, presence of diarrhoea with characteristic foul smelling pea-soup stools, confused or delirious mental condition and presence of definite prostration. Widal's test may be done if possible.

(c) If the illness is a protracted one and is suspected to be one of a tuberculous nature, particularly in view of a rise in temperature for only a few hours in the afternoon or evening, always look out for presence of wasting, and absence of brightness in the patient's outlook. A tuberculous patient is usually wasted and more gloomy. E.S.R. and other laboratory methods may be availed of, if possible.

(d) Presence of history, allergic symptoms or a dose of Santonin will help to eliminate worms.

(e) Points suggesting B. coli infections are:—Pulse compared to temperature is not slow, the difference between morning and evening temperature is usually more than two degrees, there is constipation as a rule, mental condition is very clear irrespective of the length of the illness, and prostration is absent. If cystitis is present there is the classical symptom of false sensation of urination. Presence of colitis will cause meteorism and be easily mistaken for meteorism of a fever of enteric group. The B. coli organism is a very resistant germ, liable to cause relapses inspite of treatment. Urine in B. coli infection is acid, yellowish, opalescent, contains pus or shreds of pus and in a typical case has got characteristic fishy smell, which if once smelt, is never forgotten. Microscopically it contains pus cells, epithelial cells and swarms of bacteria. For determining actual presence of B. coli group organisms, culture is necessary. It should be remembered, that malaria, enteric or tuberculous cases are never mistaken for B. coli infection, but it is always the B. coli cases, which are mistaken for other diseases.

5. TREATMENT.—(a) Alkaline mixture, with large doses of Potassium Citrate even in children is indicated.

(b) Suitable doses of Sulphasuxidine Sulphathalidine or Irgafen tablets be given with the above mixture. This may not effect complete cure in all cases.

(c) Chloromycetin is reported to be an effective bacteriostatic agent in this infection. It may become the drug of choice in infections caused by organisms of the coli-typhoid group.

(d) Mandelic acid preparations for treatment of this infection, is now out of date and also do not help much.

(e) Bacillus coli Vaccine, (Stock-Vaccine) plain or mixed prepared by a reliable firm gives very good and prompt results, in chronic cases not yielding to other lines of treatment. The writer has found B. I. Co's preparation to be a very effective one.

(f) In still resistant cases, Autovaccines are indicated.

(g) Treatment must be continued for a pretty long time, even after apparent clinical cure as otherwise there is likelihood of a relapse.

INFANTILE HEPATIC CIRRHOSIS

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Disease. *Infantile cirrhosis of liver.*—It is a disease in which degeneration of the hepatic cells occurs in association with fibrosis spreading from the portal spaces to enclose various numbers of lobules.

Aetiology and pathogenesis.—This malady encroaches upon the life usually in infancy and early childhood, between the ages ranging from six months to three and four years. It is more common in boys than in girls. Its occurrence is definitely more among the orthodox vegetarians (Hindus) than among the non-vegetarian community. Sometimes it is found in the several members of the same family (may be due to common dietetic error or hereditary).

In the recent past years the aetiology of the disease has greatly been revised on the basis of the laborious laboratory researches. The old presumption of alcohol as an aetiological factor has now been replaced by the most potent fact, that it is due to the deficiency of some of the essential amino-acids and vitamins in the diet.

Toxæmias produced by acute infective fevers, gastro-intestinal upsets, as well as a subnormal state of health injure the hepatic cells and weaken its detoxicating functions. All these are pre-disposing factors and expose the liver for subsequent pathological changes.

In the absence of high grade proteins and vitamins in the diet the valuable tissue-proteins and stores are encroached upon and are consumed to fulfil the needs of the body, viz., development and repairs of the wear and tear of the body which is going on very calmly inside every cell of our system.

This protein depletion of the hepatic cells exposes it to the mechanism of the fatty infiltration of the liver (and it is the interim period when we feel soft fatty liver below the right costal margins).

The fatty infiltration in due course of time is replaced by the connective tissue hyperplasia and fibrosis following atrophy of the liver cells.

Thus the precious cellular structure of the liver, which has got so many functions to perform for the maintenance of life, is preceded by the cheap connective and fibrous tissues. (The liver is shrunken and hard to feel:—"a Hob-nail-liver"). The contraction of the fibrous tissue leads to the obstruction to the intrahepatic branches of the portal vein and a rise of pressure in its tributaries. This results in the dilatation in the superficial tributaries, which go to form the compensatory collateral circulation in the different group of veins.

Symptoms and signs (early).—The child grows pale and dull. The body is felt warm with no temperature and the patient perspires on the head and temples while asleep and kicks off the bed clothes. He becomes more irritable and restless later on. Complexion in this disease affords a very striking feature. In the early cases the patient is of waxy pale colour and as the disease advances the complexion changes muddy or ashy depending on the intensity of the hepatic-toxæmia. The conjunctiva remain whitish or anæmic but later on may be jaundiced due to liver damage. Appetite is poor; but sometimes it is voracious too. Digestive system is usually deranged. There is constipation with occasional passage of pale clay coloured stools. In the late cases the diarrhoea may be present with offensive stools. In all the early cases liver is enlarged to varying sizes which is soft and firm. Splenomegaly is also present in most of the patients.

Symptoms and signs (late).—There is distension of abdomen. The prominence of the superficial abdominal veins indicates the attempt of establishment of the

collateral circulation. Symptoms of gastritis are present, viz., nausea, vomiting, dyspepsia, flatulence, etc. Subsequent minute erosion in stomach may give rise to hæmorrhage. The blood may come out with vomiting or may be passed as melæna. Epistaxis are also common in late cases.

Temperature is usually raised. Urine is scanty and high coloured with deposits of urates. There may be albuminuria in bad cases. The liver is usually hard, irregular and shrunken.

Ascites and anasarca:—Ascites is present in most of the cases in the late stages to a varying amount. Sometimes the bulky ascites produces embarrassment on the respiratory and circulatory systems. Ascites is the result of the portal congestion and obliteration to the venous return as well as hypoproteinaemia. Normally the protoplasm of the body cells keeps up the osmotic tension *in situ*. But whenever the cell suffers protein deficiency it loses its integrity of maintaining such tension with the result that the water leaks out in the adjacent loose areolar tissues giving rise to collection of fluid therein. In some cases it may be secondary due to chronic peritonitis. Puffiness of the face and œdema of the feet is also common.

There is generalised muscular atrophy and wasting. Anæmia of the different ætiology may be present along with the liver damages.

In far advanced hepatic-insufficiency symptoms such as nausea, vomiting, depression, debility and coma may be seen.

DIAGNOSIS.—It is usually easy to arrive at by typical signs and symptoms with liver enlargement.

PROGNOSIS.—In all the liver cases the prognosis should be given very cautiously. It is favourable in early cases without ascites. In advanced cases with gross hepatic insufficiency; ascites; with persistent temperature and hæmorrhage, the out-look is grave. Patient usually dies of complications.

COMPLICATIONS.—Ascites, circulatory failure, hæmorrhage, diarrhœa and infectious fever like pneumonia and bronchopneumonia, etc.

TREATMENT.—This is a game of patience, and the patient requires an intensive, expensive and lengthy treatment.

It should be aimed at to furnish sufficient quantity of various amino-acids, vitamins and minerals. A high caloric diet containing a maximum amount of proteins and carbohydrates but very poor in fats is advised. Such diet is said to mobilise the lipoids of the liver, repairs liver-damages and restores hepatic functions. Amino-acids as Methionine and Choline in prevention and treatment of the hepatic-lesions have proved their values. They appear to be the most beneficial in cases with no ascites. Vitamin "B" Complex and Vitamin "C" prescribed orally and some potent liver extract parenterally should be treatment. If possible, proteins like meat, eggs, livers, etc. from the animal source should also be considered in older children. Skimmed milk, fruit juices, glucose, etc. should be given as a routine.

In malarious cases Quinine should be administered intermittently. Complications should be watched and treated accordingly as they arise.

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INFANTILE BERI BERI

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I PUBLISHED some observations on Infantile and Puerperal Beri beri in the Antiseptic (1946). I have been meeting with cases now and then and feel that the disease is more common than is detected. Many cases of gastro-enteritis or broncho-pneumonia proving fatal, seem to be secondary to infantile beri beri, and the root cause, if not discovered early, a fatal result is certain to follow. I report here below an acute cardiac type of infantile beri beri complicated with bronchopneumonia and gastro-enteritis.

A male infant, aged 3 months, was brought in a railway train over a distance of about 350 miles on the event of transfer of his father to a new place. The infant was the third child for his mother, the two older ones being boys aged 6 and 3 years respectively. The mother aged about 23 years is apparently healthy and is said to have suffered from diarrhœa and glossitis in her second pregnancy and had œdema of legs while the present infant was in utero. Three doctors appear to have been consulted while there was œdema of legs but they told that it was of no special significance and could be left alone although the previous history is suggestive of at least a part of B complex deficiency (Riboflavin and Nicotinic Acid). The mother however is fairly anæmic and somewhat puffed up all over but does not complain of any signs or symptoms of neuritis or cardiac distress. She delivered uneventfully and was suckling the infant. The child appeared to be thriving well, till he was three months' old. Soon after the journey referred to above the child appeared to have a cold and the next day slight fever. The usual home remedies were tried for two days and as the child was developing bronchitis and could not swallow a mixture, powders of Sulphathiazole available, in appropriate doses were given. The child began to cry paroxysmally. From the next day it was vomiting occasionally and was having greenish mucoid stools with bits of curds and tenesmus. I happened to be in the same house and the child being related I had to take up the responsibility of treating him. Infantile beri beri suggested itself to me and when the history was probed into it transpired that the child was having greenish mucoid stools for 4 or 5 days prior to the present illness. Almost in a few hours the child began to show further signs and symptoms and three tablets (3 mgr.) of Aneurin (Berin) was given by mouth. The child was slightly better from crying for a time, but as paroxysmal attacks of crying continued, Thiamin or B₁ 5 mgr. was given parenterally. In spite of it the child began to develop threatening signs and symptoms of an acute cardiac type by leaps and bounds. There were the characteristic sign of paroxysmal whining, moaning and crying as if the child was having a colic, extreme restlessness, occasional vomiting, dyspnœa dysphagia, aphonia ptosis of the eye lids and intense cyanosis. Eyes were sunken and the child became almost moribund. There were fits of coughing for several minutes at a stretch and for a whole day it was thought the child would collapse any time. On 21-8-'51 The child's heart was very much enlarged to the right the heart sounds very rapid, almost uncountable, 200 or more per min. with a typical tic-tac rhythm, the interval between first and second sounds could not be made out. Both lungs were full of rales and rhonchi and there were areas of bronchial breathing and patches of impaired resonance. As the child's condition was very serious from cardiac distress the first consideration was to relieve it. On 21-8-'51 at 8 a.m. 25 mgm. of B₁ was injected to start with followed 2 hours later by 50 mgm. Cyanosis was not relieved and the child did not have motion or urine, abdomen distended, child stopped crying and appeared drowsy. Amyl Nitras inhalations and a full tablet of Nitro-glycerine 1/100 of a grain were given by mouth, and injections of B₁ 50 mgm. repeated at 12 noon and again at 3 p.m. A mixture of Bromide 2 grs. Digitalis 2 mins, with 5 mgrs. of B₁ by mouth was given *ad infinitum* every hour. Hot fomentations and a glycerine enema were added to the treatment.

The child had a motion and passed urine at 4 p.m. At 8 p.m. the child's condition was better, and he began to cry when being injected which was absent before he had motion and urine and began to take feeds. The heart sounds were less rapid. Penicillin 50,000 units was then given in aqueous solution with 50 mgrs. of B₁ again parenterally. 1/200 grain of Nitroglycerine was also repeated by mouth. The child slept well at night and till 5 a.m. the following morning.

22-8-'51.—Penicillin 50,000 units and B₁ 50 mgrs. were repeated separately at 6 a.m., while B₁ by mouth and the mixture of Bromide and Digitalis continued every 2 hours. Till 12 noon, the child appeared quiet and started whining and crying again. 50 mgrs. of B₁ was repeated by injection at 12-30 p.m. and the child stopped crying in a few minutes and was relieved of cyanosis. This improvement lasted for 3 hours and at 3-30 p.m. the child again became restless, cyanosed, and whining. 50,000 units of Penicillin, half a tablet of Digitalin, and 50 mgr. of B₁ were repeated by injection. The child became quiet in less than 10 minutes. At 6-30 p.m. three hours after the previous injection, the child began to relapse into a condition of restlessness, crying and cyanosis. It was then thought that the dose of Thiamin or B₁ parenterally 50,000 units were given. Aureomycin was got by this time for the lung condition and a capsule of 250 mgrs. was given in honey. Amyl Nitras inhalations repeated. The child as usual began to get relief in a few minutes but was occasionally crying at night in spite of mixture and B₁ by mouth repeated thrice. The child fell asleep at 2 a.m.

23-8-'51.—Slept quietly for 6 hours since 2 a.m. face slightly cyanosed, tossed head from side to side. Aureomycin 1/2 capsule was repeated every 6 hours, and 50 mgrs. of B₁ injected at 8-30 a.m. and B₁ repeated by mouth every 4 hours with mixture Bromide and Digitalis. 1-30 p.m. pulse countable 118 per minute respirations 44 heart sounds regular pause between 1st and 2nd sounds made out. Lungs rales and rhonchi though slightly cyanosed, the child was sleeping quiet. The effect of B₁ lasted for 11 hours. At 7-30 p.m. B₁ 50 mgm. repeated by injection. Slept quietly the whole night.

24-8-'51.—Temperature normal. Pulse 116. Respirations 32. Child quiet and playing. B₁ 25 mgrs. given by injection at 8 a.m. and repeated again at 8 p.m. with a mixture of Iodide, Squill, Digitalis, and B₁ by mouth every four hours. 25-8-'51.—Pulse 112. Respiration 32. Temperature normal. Heart sounds regular and no tic-tac rhythm, lungs clearing. General condition satisfactory. Injections of B₁ was stopped, B₁ 30 mgrs. continued by mouth in 5 mg. doses. 26-8-'51.—Continued to improve no injections of B₁. B₁ by mouth 25 mgrs. in divided doses. Lungs almost clear.

27-8-'51.—Child was tried on a few spoons of diluted cow's milk with glucose for want of mother's milk. An hour later, he started crying. Abdomen became distended and the face began to get cyanosed 50 mgrs. of B₁ was injected to be on the safe side and the abdomen fomented. The child passed some wind and was relieved.

28-8-'51.—20 mgrs. B₁ was continued by mouth and the cow's milk changed to semilac (dried milk powder). Lungs clear.

29-8-'51.—Child slept well and playing while awake.

The following conclusions can be drawn from the case cited above. It is stated by How that "In most acute cases of beri beri (cardiac) one injection of 5 mgrs. Aneurin followed by 3 mgrs. by mouth sufficed"—Manson page 459 (1950). It is also however stated that in moribund cases immediate intravenous injection of 50 mgrs. of Aneurin repeated two or three times in 24 hours is necessary. In the present case what is found enough in most acute cases appeared to go nowhere, and intravenous medication for an infant of 3 months is neither easy nor feasible, for a general practitioner under ordinary home conditions. The only course appeared therefore was to give heroic doses of B₁ intra-muscularly or subcutaneously. On an average 250 mgrs. of B₁ was given by injection or orally

for three days of which nearly 200 mgrs. was given by injection. 50 mgrs. by injection repeated 3 hourly during critical period and thereafter thrice, twice or once according to the necessity appear to be best. A larger dose than 50 mgrs. at one injection did not appear to have kept up the effect longer as was seen in this case when 100 mgrs. was given once. All the typical symptoms and signs as dysphagia, difficulty for swallowing, were present. The child used to take several minutes to swallow even a small spoon of medicine or milk during the worst period; there was absolute loss of voice, aphonia, followed by days of paroxysmal crying, marked drooping of eyelids, ptosis, and extreme restlessness. Breathlessness, sighing and gasping threatened the child several times. The gastro-intestinal signs and symptoms in the form of vomiting and greenish mucoid stools, I have found as a regular feature in the cirrars more as a rule rather as a separate type. It is however not unusual to meet with a combination of 2 or 3 of the types described *viz.* gastro-intestinal, aphonic, and cardiac as is seen in the present case. I consider the child lucky in having recovered.

A CASE OF CONGENITAL VAGINAL ATRESIA

M. D. ADATIA, M.D., F.C.P.S., B.Sc., M.G.O.,
Jai Hind Estates, Bhuleswar, Bombay.

THE condition is commonly of congenital origin. The external genitals and the lower one-third of the vagina develop from urogenital sinus, while the upper two-thirds of the vagina develop from the lowermost part of the müllerian ducts. In the case presented here there was a lack of canalisation of the upper two thirds of the vagina. The incidence is reported to be 1 in 5000.

Clinical history:—In the middle of December 1948, a young girl, (Mrs. K.P.M.) was brought to me for consultation by her parents. She was seventeen years of age and had been married for last two years. Lately her husband had been demanding a divorce from her on the ground of dyspareunia. Her parents were worried and brought over the girl to me for examination and treatment, if possible. Till the age of fifteen when she got married she never menstruated. During the two years of her married life she had little blood spottings for five to six times and she took these as her menstrual discharges. The intercourse had never been successful and satisfactory and thus aroused much bitterness between the couple.

Few days prior to the day on which she was brought for consultation a forcible attempt at intercourse was followed by retention of urine for 24 hours.

She had been, then, taken to a nearby practitioner who catheterised her and, incidentally, detected an absence of the vagina.

Physical examination:—The girl looked quite healthy, had a good feminine physique and secondary sex characters seemed to have been normally developed.

She had one extra rudimentary finger on the left hand.

Her weight was 95 lbs.; her pulse 84 per minute; blood pressure 110/70 mms. of mercury. On external examination her genitals were found to be quite normal.

On bimanual examination the vagina was represented by a shallow depression about 3 cms. in depth and 3 cms. in width. On speculum examination a small pin hole was detected in the centre of the vaginal depression. A small thin probe could be passed inside this hole for about half an inch. A small knob like structure could be felt per rectum which simulated uterus.

Investigations:—Her blood and urine, were examined but no abnormality was found. Kahn test was negative.

DIAGNOSIS:—The condition was diagnosed as vaginal atresia.

TREATMENT:—An attempt was made on 29-12-48 to remedy the defect by operative procedure. The patient was kept in lithotomy position and the operation was carried out under spinal anaesthesia.

The small pin-hole opening in the blunt cloacal depression was dilated, and the edges of the orifice were dissected out and undermined. These edges were incised at 4 places in 3, 6, 9 and 12 o'clock position and the orifice was now made sufficiently large.

It was easier now to dissect out the space between the bladder and the rectum. This space was purposely dissected in an oblique direction little to the left side so as to have less bleeding and, to prevent the pressure of the bladder and rectum to obliterate the newly constructed space.

A dilator No. 18 and a sound were kept in the rectum and bladder respectively. The dissection was carried further until the peritoneum was reached.

By means of index fingers of both hands the cavity was stretched laterally so as to make it large enough to take the mould. The cavity was packed with gauze to prevent oozing of blood.

The mould selected for the purpose was of light wood and of the size 12 cms. by 7 cms. The mould was painted with liquid paraffin and was covered with a rubber condom. The covered mould was dipped in saline and was, then, placed in the newly constructed space from which the packings were now removed. Particular care was taken to see that the mould was completely behind the perineum and it was fitting firmly in the cavity.

An incision was then made outwards on each side into the labia. Both the inner edges of this incision were sutured together on the mould with interrupted catgut sutures so as not to allow the mould to slip out.

The two outer edges of the incision were similarly brought together in such a way that the two tiers of sutures make an operculum. A small hole was left under the meatus to allow of drainage.

Post-operative management:—Local discomfort was not much. The discharge was profuse and offensive for the first few days but later on became odourless. The patient was given liquid paraffin daily and was catheterised every eight hours. Penicillin and Streptomycin were given intramuscularly for first 6 days.

The mould was taken out after 3 weeks by removing the sutures on the labial skin. The mould was pulled out after fixing the screw.

The patient was then, advised to put glass rest by the daytime and oestrogenic colpon bougies (1000 units) for the night time.

The epithelium from the four flaps was seen to be proliferating well and it was gradually covering the walls of the vaginal space. At the end of six weeks the whole space was observed to be satisfactorily covered with the epithelium.

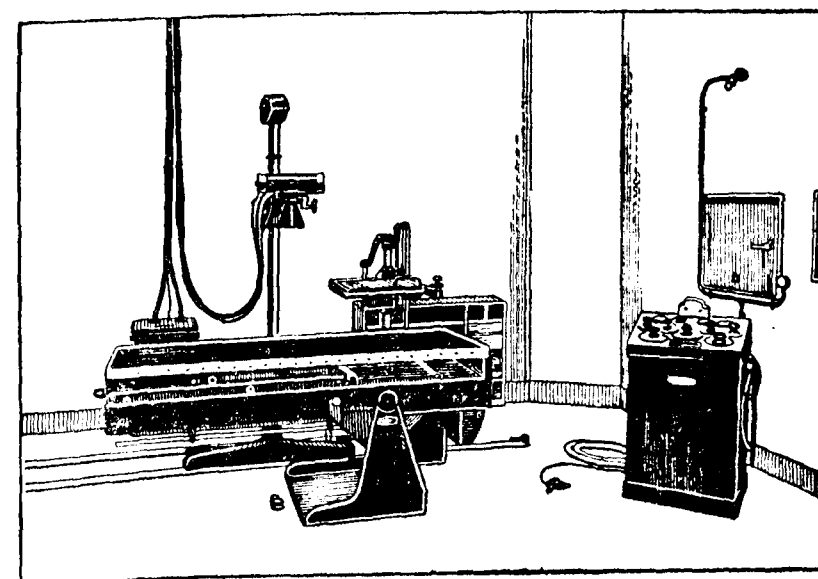
At this stage a bimanual examination was done and it showed a small knob like mass at the top representing a rudimentary uterus. The ovaries were easily palpable on each side. An attempt was made to pass a thin probe in the mass, but cervical os could not be traced deeper down.

Follow-up.—About two years after the operation the patient was brought in for examination by her husband, who, it seemed, had abandoned the idea of divorce. The woman was getting scanty but regular menstrual periods. Vaginal examination showed an undersized uterus, with a small conical cervix. The sound could be passed through os only up to $\frac{3}{4}$ of an inch. Both husband and wife showed an eagerness to have a child. Tubal insufflation test was done but the results were negative. The patient is still under observation.

Discussion.—Wharton L. R. has described and advocated this simple construction of vagina, and has reported satisfactory results. Skin grafting was not carried out, but the four flaps of the depression, showed very satisfactory growth of squamous epithelium extending all over the newly constructed space within a period of only six weeks. Insertion of colpon bougies and administration of Anti-biotics helped the epithelialization. Detection of a small cervix and rudimentary uterus, keeps the couple hopeful for conception.

Summary:—A case of congenital atresia of the upper two thirds of the vagina is reported and satisfactory construction of vagina was carried out without skin grafting.

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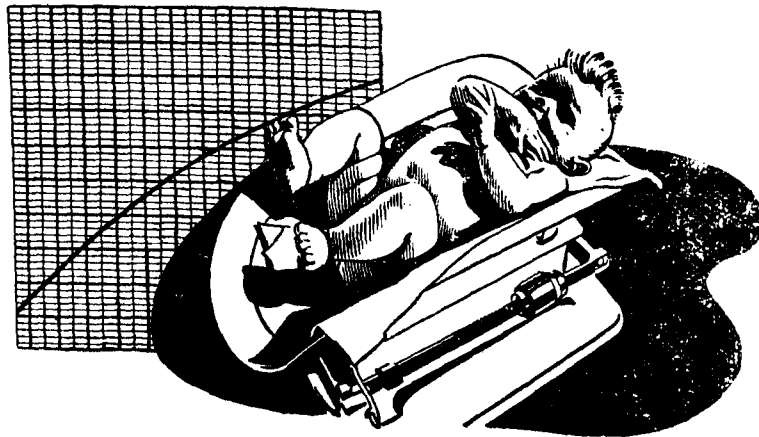
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ESTD. 1904.

A Monthly Journal of Medicine and Surgery

Published on the 16th of every month.

Editors: U. RAMA RAU & U. KRISHNA RAU, M.B., B.S.

Asst. Editor: U. VASUDEVA RAU, M.B., B.S.

Editorial & Publishing Office:

"RAMARAU BUILDINGS," 323-24, Thambu Chetty St., Madras-1.

Annual Subscription: Rs. 7-8-0: Foreign—Rs. 10—Post Paid.

Vol. 48

DECEMBER, 1951

No. 12

EXTENSION OF REFRESHER COURSES

As a result of intensive research carried on in every country, medical science is advancing by leaps and bounds. What is considered up-to-date knowledge today becomes out of date tomorrow as a result of some new discovery. If therefore the members of the profession are to keep themselves abreast of the times and to make that knowledge useful to the public they must be posted with up-to-date developments. Medical journals help considerably in disseminating knowledge of the latest developments in medical science. But those who can afford to go in for the costly medical journals are limited, and these are practically confined to large cities. The members of the profession who are confined to rural and urban areas have very little scope to expand and improve their knowledge. It was why refresher courses have been instituted to brush up their knowledge and to enable them to get themselves posted with the latest developments. But unfortunately these are limited and few and far between that they cannot be considered to fulfil the dire need. Besides the number of those who can take advantage of this course at a time is considerably limited that many have to be left out of the reckoning, not to speak of the inconvenience and difficulty likely to be felt by those who, though anxious to take advantage of it, have to deny themselves this privilege as a result of the difficulties likely to arise in getting themselves relieved at least temporarily from their posts of duty owing to official exigencies. To the extent facilities for brushing up their knowledge have to be denied to the members of the profession on these and other grounds to that extent not only they but also the public become losers. It is therefore necessary that something

should be done, and that too without delay to afford the members of the profession the opportunity to expand their knowledge.

The Hon'ble Shri A. B. SHETTY, Minister for Agriculture drew pointed attention to this deficiency in the present arrangement and suggested the desirability of extending refresher courses to district centres so that more of our medical men may avail themselves of such facilities. The needs for such an extension had long been evident and it is well that at least now its need had been recognised by persons in authority. Let us hope that the Hon'ble Minister would persuade his colleagues in the cabinet, and especially the Hon'ble the Minister for Public Health and Medical Relief, to take steps to give effect to his suggestion at an early date. The head quarters are fairly well-equipped at present and are likely to become better equipped in the future as a result of the many plans now decided upon and are under contemplation both from the point of view of personnel and also in regard to equipment. Further the headquarters hospitals being generally full with patients suffering from various types of diseases will afford excellent material to obtain practical knowledge. It would also not be inconvenient to the medical practitioner to make a sojourn to the District Headquarters Hospital for a while to brush up their knowledge not only by the courses of lectures delivered but also by coming into contact with their brethren in the profession and exchanging ideas and discussing various problems arising there from. We are sure that if such courses are arranged in District Headquarters the profession will only be too glad and willing to avail themselves of this opportunity. We trust that the suggestion of the Hon'ble the Minister for Agriculture will be taken up by the Government of Madras in right earnest and acted upon.

There is one other suggestion of the Hon'ble the Minister which deserves attention. He advised members of the profession to read new books and journals to improve their knowledge. Even the most well-placed member of the profession will find it difficult in these hard days to go in for books and journals as has been suggested by the Hon'ble the Minister. The Indian Medical Association is the accredited organisation of doctors practising the Western system of medicine and it has branch organisation practically in every District Headquarter not to speak of the branches in other places as well. Almost all those who practise the Allopathic system are members of this organisation. Each of them has a library of its own. If only the Government will come forward to make grants to these organisations for the development of their library, it would serve the purpose of all the members of the profession who can make use of these books and journals to improve their knowledge. Such a grant need not be taxing to the exchequer as an annual grant of a fixed sum will go a great way to develop these libraries.

And the effect of it would be marvellous indeed. We request the Government to give this matter their earnest attention and set apart a lump sum for such a grant in the budget for the next year.

CONSTRUCTIVE MEDICINE

It is pleasing to note that the Government of Madras have begun to give serious consideration to the problem of rural medical relief. Addressing the annual meeting of the South Kanara Medical Association on November 17, the Hon'ble Shri A. B. SHETTY, Minister for Agriculture and ex-minister for Public Health and Medical Relief devoted a considerable portion of his address to this problem. While he made no mention to the objection raised by his successor in office and the ex-minister for Medical Relief to making it obligatory on the part of medical graduates to work in rural areas for some time on the ground that it would violate the fundamental right guaranteed in the constitution to do business anywhere they pleased, it appears clear from his speech that he is anxious to make medical officers serve compulsorily in rural areas for a term of years. This means that he intends to send government medical officers working in cities and urban areas to rural parts to serve the public with the experience they have gained by working in crowded hospitals and getting into contact with a variety of diseases of civilisation. Whether this is the accepted policy of the Government also should be made clear without any delay.

The main difficulty that faces the Government at the present moment is the lack of personnel to strengthen city and Headquarter Hospitals which require additional staff to meet the growing needs of the public, and to man the hospitals in rural areas. Many of the institutions started in rural areas for the purpose of affording relief to the rural population have had to be closed down for lack of personnel. We do not think that it would be the object of the Hon'ble Minister to depute personnel from city and urban hospitals to rural areas to man the institutions that have been closed down or to run institutions that the Government may intend to establish. Even assuming it is so, the city and urban hospitals would be depleted to that extent of the staff they have at the present moment. It therefore follows that if the present shortage is to be met, it can only be made possible by starting more Medical Colleges and increasing the output of medical graduates. The Hon'ble Minister appears to be under the impression that with the establishment of a Medical College at Madura and the development of the Medical College at Guntur to a full-blown institution, the problem of lack of personnel would be solved. If it is so, no one will be more gratified than ourselves.

But unfortunately the position is not so simple and easy as the Hon'ble Minister envisages. We have times without number pointed

introduce two syringe bulbs full of warm tap water as his irrigation. The patient then holds a pad against the stoma and walks about the room for five minutes. Thereafter, fluid and stool are evacuated into a large kidney basin held beneath the colostomy. Once the time of the irrigation is set, it must be adhered to daily. The nurse in attendance must do no more than hand the syringe to the patient. Some individuals prefer to introduce the fluid sitting up; others are unable to retain it except in the prone position during the early training period. Only clear water may be returned for the first twenty irrigations although most patients have results within the first five. It is emphasized that there should be no stool except with the daily enema. For each evacuation other than that with the irrigation the giving of 4 cc. or more of paregoric is prescribed.

A third talk places the future management in the patient's hands. As he gains confidence, he is ordered to reduce the size of his dressings until eventually only an eye patch is used to cover the colostomy orifice, the paucity of the patch being a factor in his psychologic training. Many failures must be anticipated before complete confidence is obtained. However, by the time the patient is ready for discharge from the hospital he will have some success with his control of the colostomy opening and be quite proud of it.

Upon discharge, together with bulb

syringe and kidney basin the patient is given a prescription for paregoric and warned to observe what foods produce diarrhea. These can be checked by dietary experimentation and should be excluded from his diet. In our experience, corn, bananas, cabbage, prunes and beer have been the most frequent offenders.

The surgeon does well to have colostomized patients return for follow-up visits at the same time. Those having control of gas will teach the partially controlled. More important, the latter will be inspired to search harder for the food, drinks, tobacco or even medication that causes flatus in their individual case. Some patients, alert to the sensation of peristaltic rushes, learn to fold their arms across their abdomen so that the anterior aspect of their left forearm inconspicuously causes pressure over the eye-patch-covered abdominal anus. By such methods many cases are reduced to only an occasional innocuous slip.

A well instructed colostomy patient is not retiring and looks with disdain upon anyone wearing a colostomy bag or appliance. He takes pride in mingling with society secure in the knowledge that his secret of an abdominal anus can be known to none save his physician and intimate friends.

Paul Meecray, Jr., M.D. and Herbert Bowman, M.D., Editorial—*The American Journal of Surgery*, October 1950.

NEWS AND NOTES

Dr. Vaman D. Sathaye, General Secretary of the All-India Medical Licentiate's Association writes:

"The 38th Annual Conference of the All-India Medical Licentiate's Association will be held at Meerut (U.P.) on the 24th, 25th and 26th December 1951, under the presidency of Dr. Manoharlal Kapur of Jhansi."

NOTICE

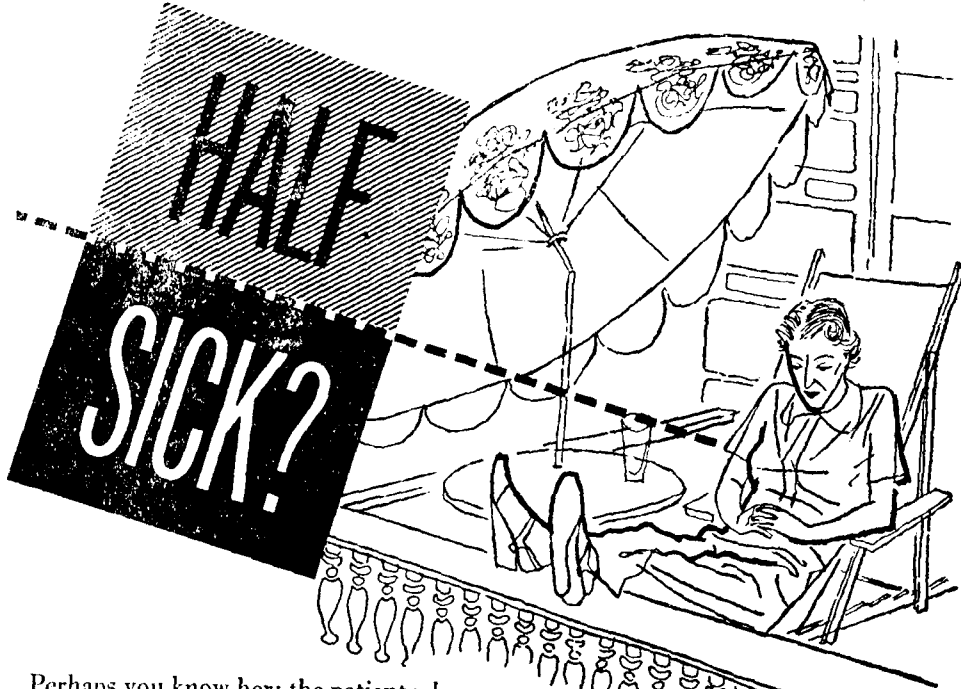
The Madras Medical Register for 1951 will be printed early in January 1952. All Registered Medical Practitioners are requested to intimate any change in address to the Registrar, Madras Medical Council, 81, Mount Road, Madras-6, before 31st December, 1951. The 1950 Register was published in Fort St. George Gazette on 1st May 1951.

30th Nov., 1951.

K. K. SHENAI,
Registrar, Madras Medical Council.

CORRIGENDUM

Dr. K. S. Gaswala's article published in the November '51 issue of the 'Antiseptic.' In line 38 on page 933 the word "Mercury injections" is incorrect, it should be "Mercury inunctions."



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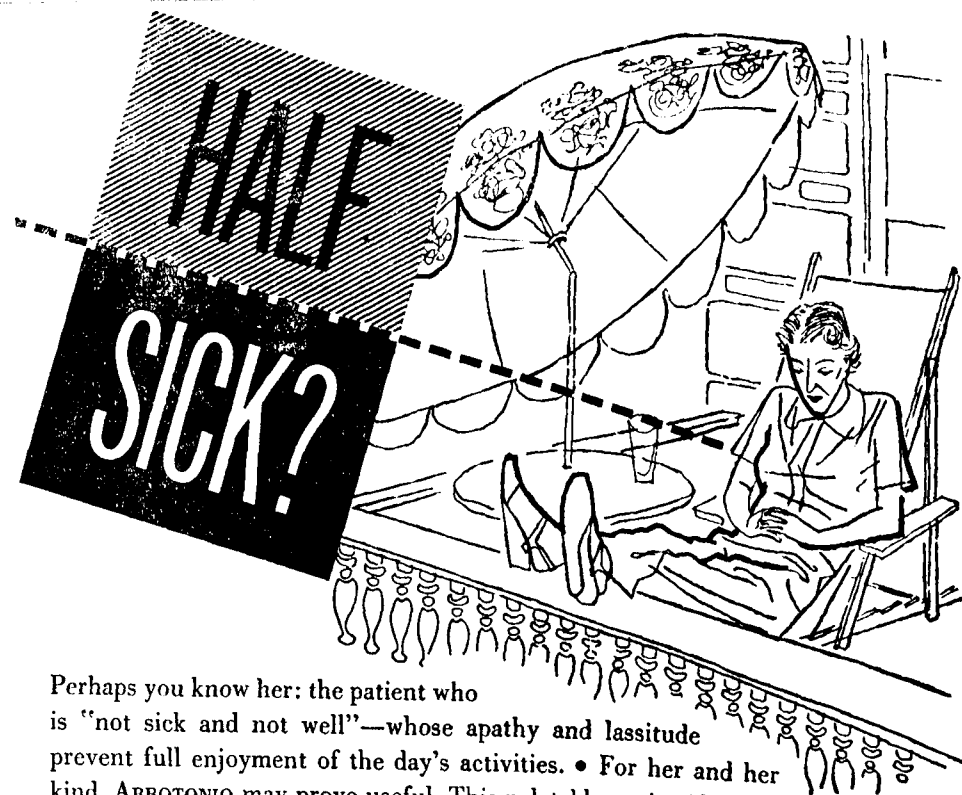
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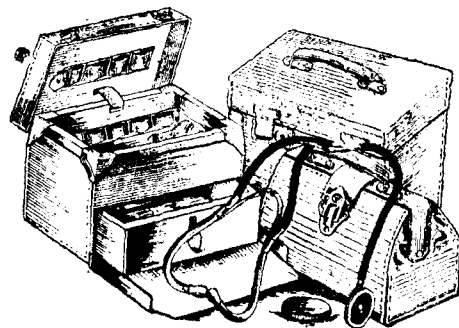
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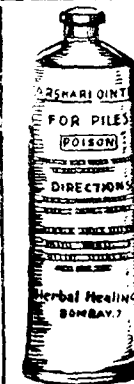
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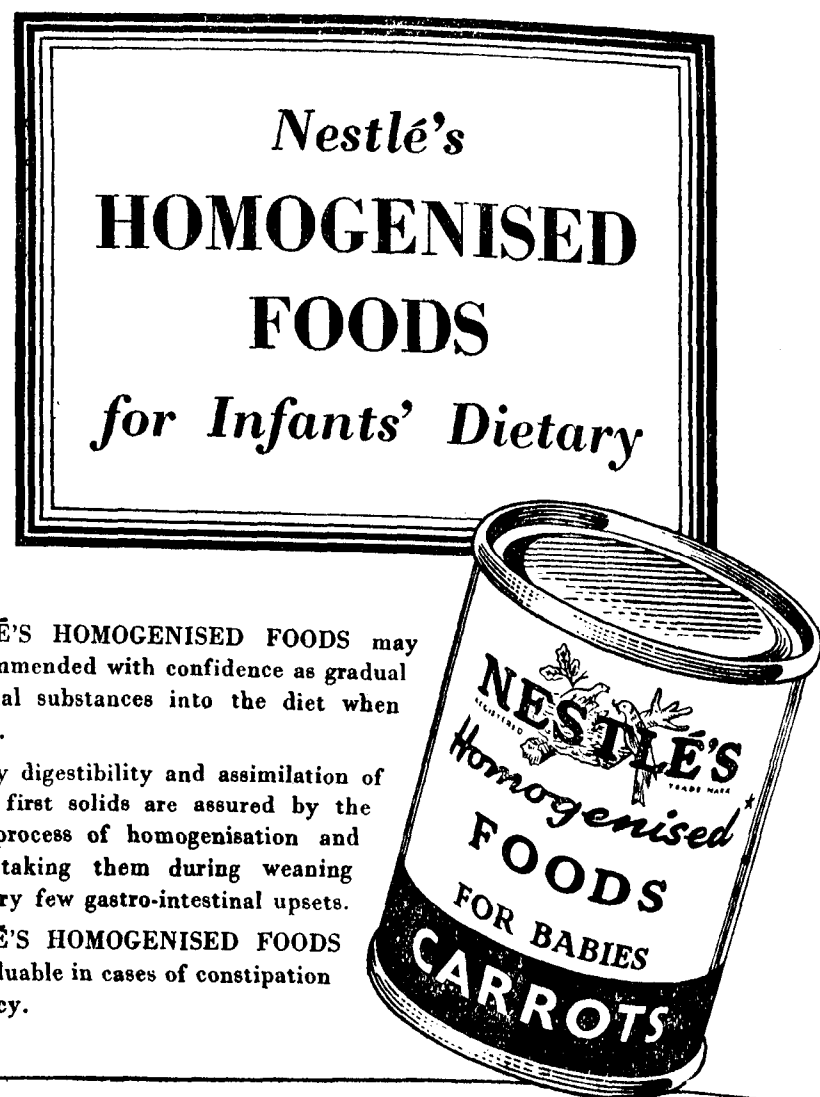
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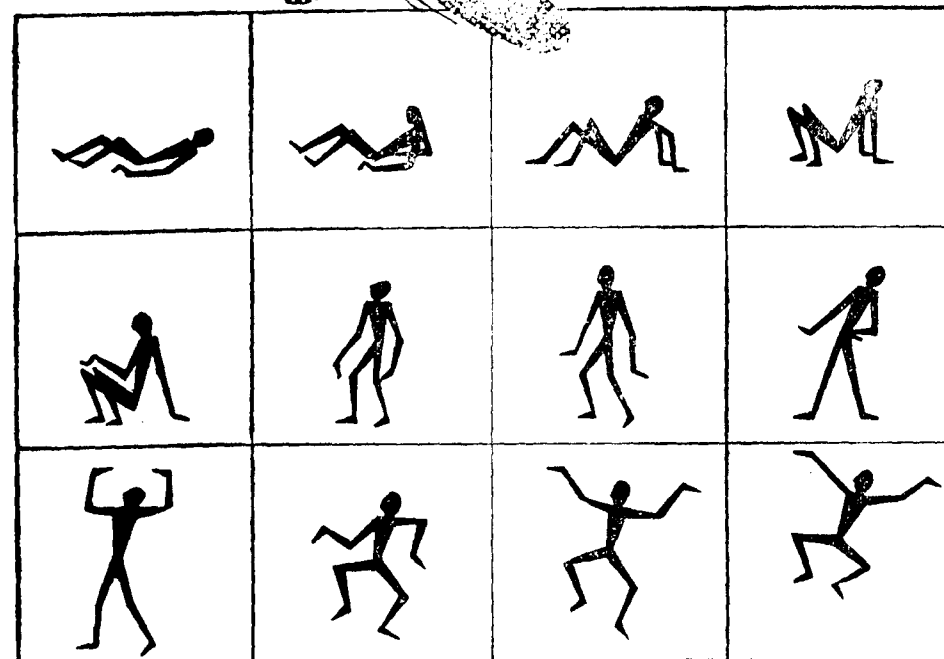
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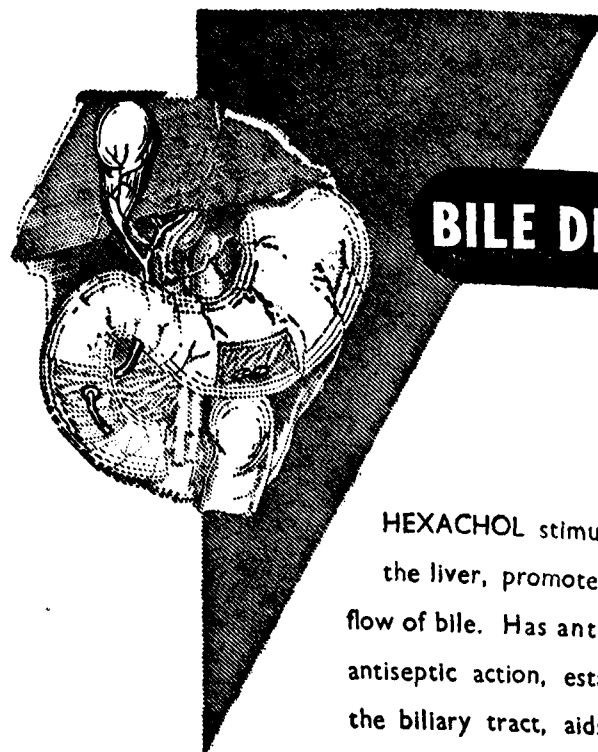


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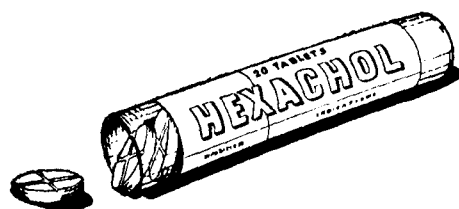
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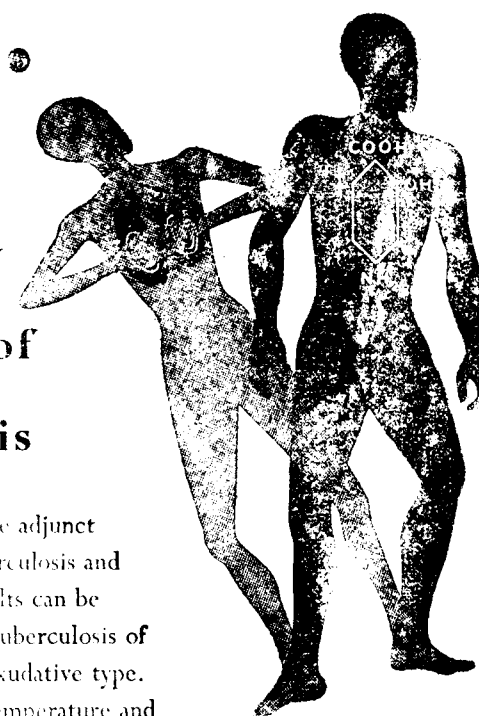
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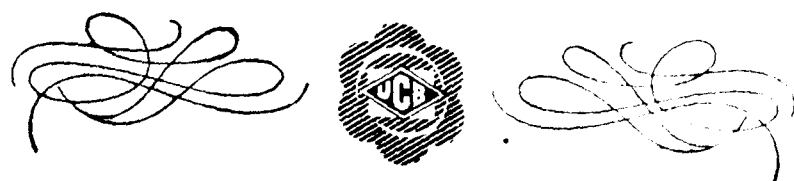
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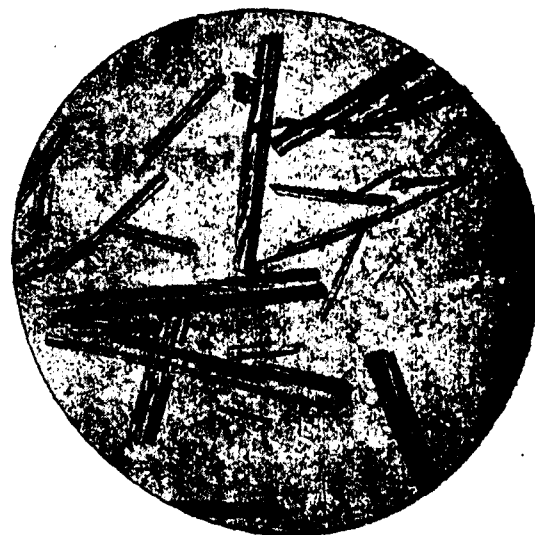
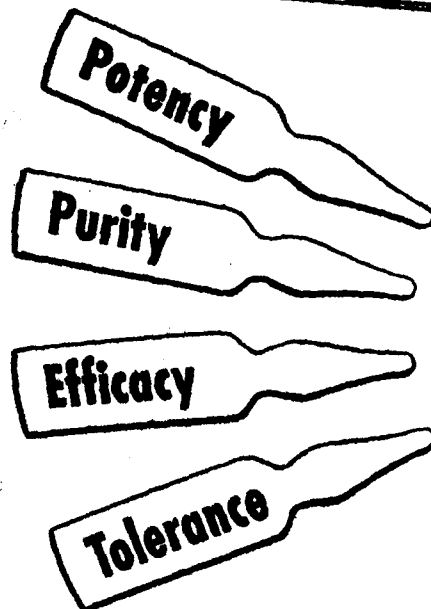
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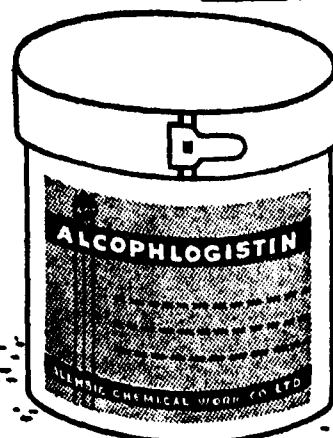
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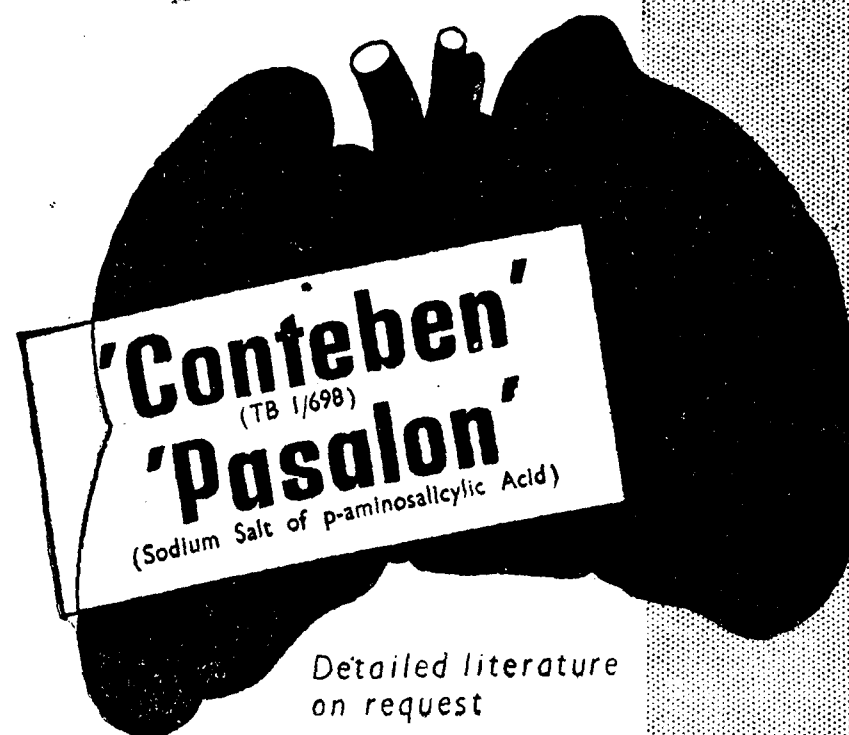


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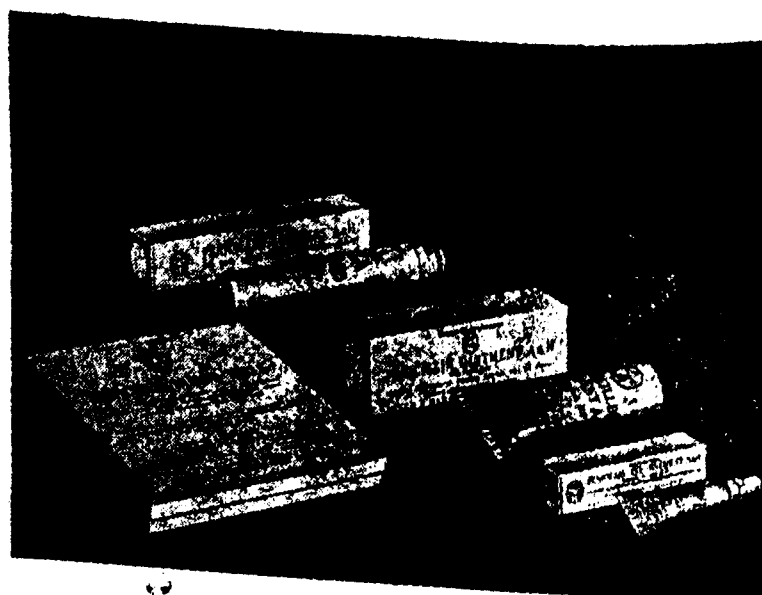
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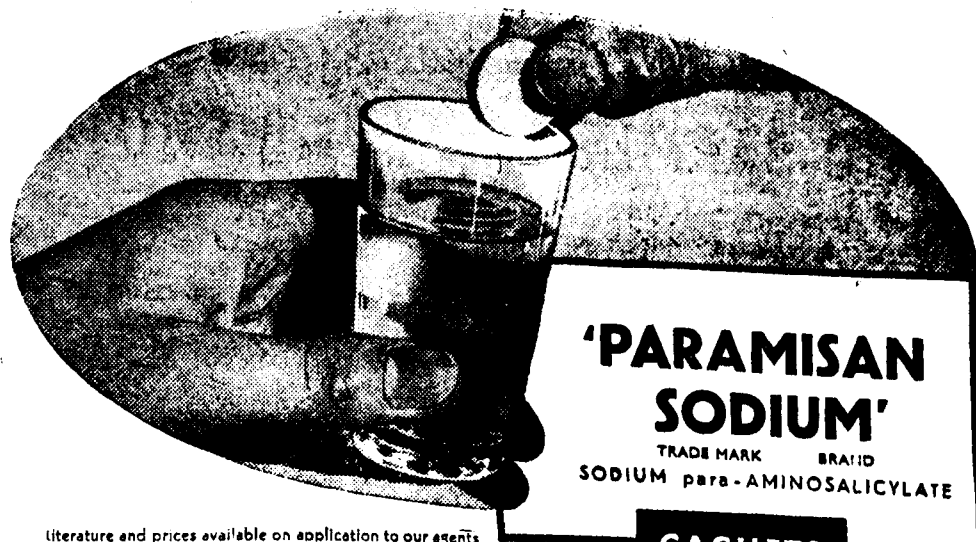
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Editorial & Publishing Office: 323-24, Thambu Chetty Street, Madras 1.

London Office: 24-27, High Holborn W. C. 1

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January to December, 1951

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