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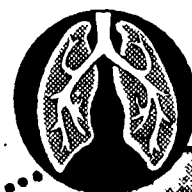
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No. I

Congenital Defect of the Skull

By

Dr. DHRUBA KUMAR SEN, M.S. (Cal.)

Visiting Surgeon, Nilratan Sircar Medical College Hospital, Calcutta.

INTRODUCTION: At birth, the membrane bones of the skull are separated by areas of fibrous tissue and over these areas the periosteal coverings of the bones become directly continuous with the underlying duramater. These are known as frontanelles. The anterolateral frontanelles (situated over the pterion) are practically closed at birth while those over the bregma and the lambda remain open till about the second year of life. Failure on the part of the frontanelles to close is noticed in cases of cranio-cleido-dysostosis. But in the absence of any other pathology such failure on the part of the frontanelles to close is extremely rare. The case under report is a healthy young man who presented with incomplete closure of either antero-lateral frontanelles.

CASE REPORT: R. S. 18, a college student was admitted to Nilratan Sircar Medical College Hospital on the 13th February, 1958. He complained of a

pulsating swelling on the right temporal region. History dated back at the time of his birth. He did not have any other neurological symptoms and only complained of a sensation of fainting while he applied pressure over the right temple. On clinical examination, a pretty big bony defect was detected over the right pterion (about 2 cm. x 5 cm). The pulsation of brain matter could be felt with the examining finger. Similar defect though of a smaller size (1 cm x 2 cm) was also detected over the left pterion. Ophthalmoscopic examination did not reveal any papilloedema. His temperature, pulse rate and blood-pressure were all within normal limits. The X-Ray of the skull revealed a big bony defect over the right pterion and a much smaller but similar defect over the opposite side, obviously caused by the failure of development of the squamo-temporal, parietal and the greater wing of the sphenoid and also a portion of the frontal bone. (Fig. 1). The

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ventriculography. He was sent back to us with a remark that ventriculogram did not reveal any underlying pathology. As has already been mentioned that the patient did not have any neurological symptoms, we decided to close the defect on the right pterion with bone-grafts from the iliac crest. He was operated by me on the 15th March, 1958 and at operation the dura was found to be covered only by the scalp and the temporal muscle. No adhesions could be seen between the bulging duramater and the overlying periostium. On further exploration, it was found that the defect extended towards the base of the skull involving the greater wing of the sphenoid and was ending up in the pterygoid fossa.

Over this region the defect was covered by the temporalis itself. The edges of the bones were very sharp and thin against which the duramater was wedged became adherent. By blunt dissection the duramater patient was sent over to the Neuro-surgical



(Fig. 1)



(Fig. 2)

Department of the S.S.K.M. Hospital for was freed from the margins and presently 3 pieces of bone grafts were raised from the right iliac crest and were conveniently placed to close the more superficial portion of the defect, while the lower part of the defect remained under the protective cover of the temporalis itself. The grafts were placed with the raw surface superficial and the smooth compact surface in contact with the dura, with the hope that the raw area of the grafts will receive their nourishment from the scalp while the duramater will remain in contact with the smooth compact layer of the bone and will form no adhesions. The wound was closed without drainage (Fig. 2.). Post-Operative recovery was uneventful and the patient was discharged on the 30th March, 1958.

Subsequent to Operation the patient did not have any sensation of fainting while he applied pressure over his temples. The patient attended the follow up clinic regularly over a period of 8 months and the result of bone grafting was perfect with complete symptomatic relief to the patient.

DISCUSSION: It is to be noted that no surgical interference was done to close to defect on the left side. The defect on the left side was small as compared with the right side and as such did not give rise to any symptoms. Hence we decided not to interfere with the same.

The defect on the right side was closed partially by bone grafts and as the deeper aspect of the defect was nicely covered by the temporalis we did not think it necessary to place another bone graft over that region. Moreover that part was not accessible from the lateral side of the face and application of external pressure would not reach that region.

We had a choice of three different materials for grafting viz. (a) Acrylic resin inlay, a plastic perspex like material, transparent and radio-translucent which should be fixed in position by suturing the edges to the pericranium. (b) Tantalum—a biologic.

ally inert metal which is shaped and fixed to the skull by tantalum screws and (c) Bone grafting—either from the iliac crest or rib or from the outer table of the skull.

We decided to close the defect with autogenous bone grafts taken from the iliac crest and did not interfere with the outer table of the thinned out skull over the region of the operation. Fixation, of acrylic resin inlay or tantalum would involve application of screws or sutures on the skull. The bone over the region of the defect was so thinned out that we did not think it suitable for applying screws or sutures.

Congenital defect of this type is of extremely rare occurrence particularly when all other frontanelles have showed perfect union. As has already been mentioned in the chapter of introduction of the article that large defects are seen in cases of cranio-cleido-dysostosis where the membrane bones

of the skull are mainly affected. Salmon (1944) reported presence of antero-lateral and postero lateral frontanelles in cases of cranio-cleido-dysostosis. But in our case, not only the portion of the bone developed from membrane was affected but the part of the sphenoid that was developed from the cartilagenous portion was also affected on the right side.

The case under report is very interesting indeed as the bony defects are not associated with any other pathology but present in an otherwise healthy normal individual and the author thought it worth reporting.

ACKNOWLEDGEMENT:— My thanks are due to Dr. A. K. Datta Gupta, Principal-Superintendent of the Nilratan Sircar Medical College Hospital for kindly permitting me to publish this case.

LITERATURE CITED:—

(1) Salmon, D.D. (1944) Radiology, 42, 391.

THE PROBLEM OF SYPHILIS TODAY:—The disease is by no means eradicated, and physicians and public health officials must be ever alert to the possibility that syphilis may still produce disability, financial ruin, dementia, and death.....Rein stated that syphilis is on the rise again. A Public Health Report estimated that 1,921,000 persons in the United States have syphilis requiring treatment. Many states and large cities find their venereal disease appropriations inadequate to permit an effective and progressive program. Of major concern is the fact that high rates for primary, secondary, and early latent syphilis are in the age group of 15 to 19, reaching a peak at the 20 to 24 year group. Rapidly declining rates over the past several years have prompted the optimistic demobilization of venereal disease control forces, reassignments of personnel and reduction of case findings, and reduction of diagnostic and treatment facilities, leaving many areas without means to discover cases or to combat sudden outbreaks. Rein.....stated that as physicians "we have the diagnostic tools and the therapeutic agents to control and even eradicate syphilis. But we are failing because of our own complacency about the disease, because of neglected educational programs, especially for the teenagers, and because of lack of interest or funds to find and treat the affected persons," — Herman, Beerman, M.D. The problem of Syphilis Today, A.M.A. Archives of Dermatology, August 1958, (via J.A.M.A. Decr 6, 1958)

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Examination of Urine

By

Dr. T. V. VENKATESAN, M.B.B.S., F.C.C.P. (USA), F.D.S. (Lond.) Madurai

The examination of urine is of great importance in the Diagnosis and progress in many diseases. Urine must be examined fresh. It is common to ask patients to bring their morning specimen of urine. This is erroneous since the best time to collect urine is 3 hours after a meal if pathological substances have to be examined.

Quantity of Urine: The normal quantity of urine with normal fluid intake and diet is about 1,500 to 2,000 cc. The urine passed during day is about 2 to 4 times greater than during night.

Polyuria: Causes:

(1) Physiological: (a) During cold season when sweating is impeded and (b) excessive intake of fluids.

(2) Pathological:

- (a) Diabetes mellitus
- (b) Diabetes insipidus
- (c) Chronic interstitial nephritis
- (d) Amyloid kidneys
- (e) Dietel's crisis in hydronephrosis
- (f) Hysteria
- (g) Diuretics
- (h) absorption of exudates as in anasarca.

Diminished quantity of urine occurs:

- (1) Acute nephritis
- (2) Sub acute nephritis
- (3) Cardiac kidney
- (4) Febrile states
- (5) In gastroenteritis.

Colour of Urine:

Normal urine is colourless or straw coloured, greenish yellow colour, occurs in jaundice. Red colour in haematuria. Brownish black in haemoglobinuria, Milky in chyluria.

Drugs can also produce change in colour a yellowish orange due to santalin.

Smell: Normal urine has ammoniacal smell. In diabetic ketoses, the urine has a pleasant odour.

Reaction of urine: Is tested by litmus paper—the blue litmus turning red in acid and vice versa in alkaline urine. Normal urine is slightly acid while in pyelitis and cystitis the urine is alkaline.

Specific gravity of urine: Is tested by a urinometer. If the quantity is not sufficient, add equal quantity of water and find the specific gravity and multiply by two the last two figures. Normal specific gravity varies between 1010 and 1015. A high specific gravity occurs in diabetes mellitus and low specific gravity occurs in diabetes insipidus. In cardiac failure, the specific gravity is high.

Transparency: Fresh urine is clear. Cloudiness may be caused by:

- (a) Urates in acid urine:
- (b) Phosphates in alkaline urine
- (c) Cells
- (d) Bacteria
- (e) Chyle
- (f) Sulphonamides
- (g) Mucus threads
- (h) Pus.

Examination for albumin:

(a) **Padry's test:** Fill 2/3 of test tube and boil the upper part. If clouding ensues, it may be due to phosphates or albumin. Add few drops of 10% acetic acid and heat. If clouding persists it is due to albumin.

(b) **Heller's test:** Take ½" Nitric acid in a test tube and add along the sides of the test tube Urine to form a junction between the two. If albumin is present a white ring is produced. An indefinite ring may be produced if urates are in excess and disappear on warming.

Significance of albumin in urine:

Albuminuria may occur without organic disease of kidney or with organic diseases of the kidney.

Without organic diseases of the kidney:

(1) Functional or orthostatic albuminuria:

In this condition the albuminuria occurs in erect posture and is absent in urine passed first in the morning. Similar albuminuria may occur after severe exercise.

(2) Febrile albuminuria is due to cloudy swelling of the kidney and subsides soon after temperature touches normal.

(3) In congestive heart failure, the albuminuria is due to venous congestion of the kidney. Hyaline casts may occur. Unlike the urine of nephritis the urine is loaded with urates.

(4) Albuminuria in pregnancy is toxic and disappears completely.

With organic diseases of the kidney:

(1) Nephritis. (2) Nephrosclerosis.

(3) Conditions associated with haematuria and pyuria.

Examination for albumoses and Bence-Jones protein:

Albumose: (Proteose) Urine is boiled with few drops of acetic acid and filtered while hot and then is tested with trichloroacetic acid and salicyl sulphonic acid. White precipitate is produced. Albumose occurs in suppurative conditions as empyema, in resolving pneumonia and during involution of uterus after delivery.

Bence-Jone's Protein: Add a few drops of acetic acid and immerse in a water bath and heat. Urine becomes turbid at 40°, precipitate becomes at 60° and disappears at boiling point to reappear on cooling. This occurs in multiple myeloma.

Sugars in Urine: Boil 5 cc of Benedict's solution in a test tube and add 8 drops of urine. With trace of sugar the colour changes green, with moderate sugar to yellow and with larger amounts to brick red.

This may occur due to sugar or non-sugar substance. The sugar may be glucose

or other sugars. Lactose occurs in pregnancy and lactation pentosuria occurs due to ingestion of large quantities of fruits as grapes, plums and cherries. Fructosuria may occur in severe diabetes or after ingestion of fruits and may occur along with glucose in diabetes. Galactosuria is an inborn error of metabolism and occurs with liver and spleen enlargement, anaemia and albuminuria. Non-sugar reducing substances are salicylates, morphine, erythromycin, antipyrin, Pyramidon and synthetic oestrogen. Homogentisic acid is excreted in alkaptonuria and can reduce Benedict's solution but the urine when voided turns reddish brown colour. Yeast fermentation test helps to distinguish various sugars. The urine is boiled to destroy the gas forming organisms and cooled before adding yeast. Then add Benedict's solution and test. Yeast fermentation is positive only for glucose and fructose.

Test for Fructose: To 5 cc of Seliwanoff's reagent add 0.5 cc of urine and boil. It will produce a red colour.

Total for Lactose: Add 2 gm of lead acetate to 10 cc of Urine and boil. Lactose gives a red precipitate. This is called Rebner's test.

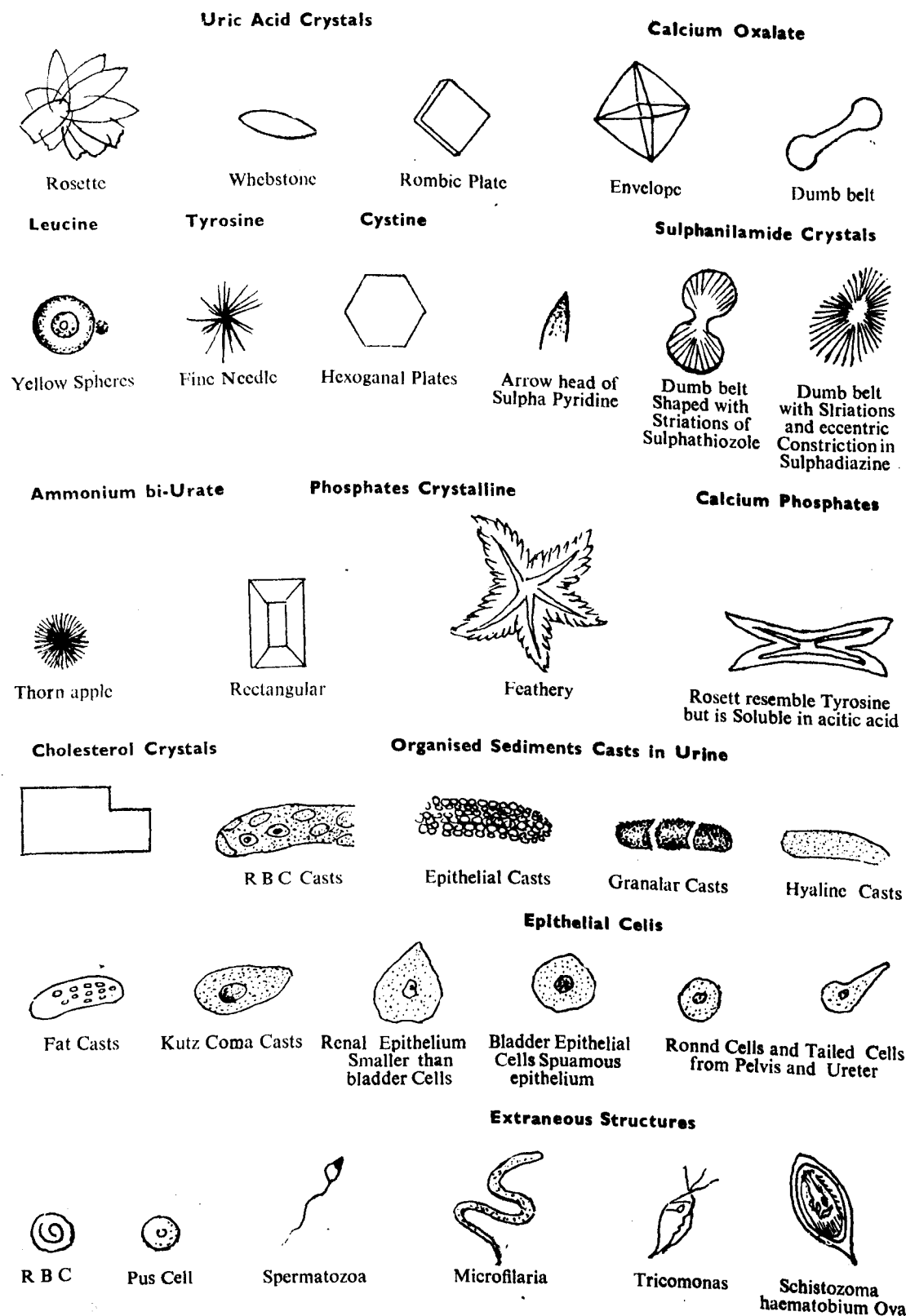
Acetone in Urine: Rothera's test:

Saturate 5 cc of urine with ammonium sulphate or ammonium chloride. Add few crystals of sodium nitroprusside and shake well. Pour along the sides, Liquor ammonia fortis. At the junction a permanganate colour is produced.

Bile Salt: Hay's test:

To urine in a conical glass sprinkle some flowers of sulphur. If sulphur sinks to bottom, bile salt is present.

Bile pigment: Of various tests mentioned the sensitive test is Harrison's spot test. 5 cc of 10% barium chloride and 10 cc of urine are mixed and the mixture is filtered. To this, add two drops of Fouchet's reagent to the filter paper. A positive result is shown by bluish green colour.



Urobilin in urine: To 10 cc of urine add 1/2 cc of Lugol's Iodine. Urobilongen is converted into urobilin. Next add equal amount of saturated alcoholic solution of zinc acetate or zinc chloride and filter. A greenish fluorescence indicates urobilin.

Blood or eahmoglobin in urine:

Urine in eahmaturia contains red cells which can be seen under the microscope of a cenrifuged specimen. Haemoglobin is best tested with a spectroscop.

Porphyrin in Urine: 5 cc of glacial acetic acid to 100 cc of Urine and shake and allow to stand for 2 days. A red precipitate is formed.

Melanin in Urine: To 5 cc of urine add 10% Ferric Chloride drop by drop a brownish black precipitate is formed.

Microscopic examination of urine:

The unorganised sediments are Uric acid oxalate, leucine, crystone and tyrosine. Sulphonamide crystals and fat globules are deposited in acid urine and ammonium bicarbonate and phosphates are deposited in alkaline urine.

RENAL FUNCTION TESTS:

- (1) Qualitative examination of urine for albumin.
- (2) Examination of centrifuged specimen of urine for casts.
- (3) Specific gravity of urine: The presence of a high specific gravity usually excludes severe renal damage.
- (5) **Fishberg's concentration test;**

The patient abstains from taking any fluids for 15 hours before the test. The bladder is emptied at 8 A.M. and the urine voided at 9, 10 and 11 A.M. is examined for specic-gravity. Normally the specific gravity of one of the specimens should exceed 1022.

(2) **Volhard's water eleimination test:** The patient is tested over night and after emptying bladder at 7 A.M. is given 1400 cc of water. The urine is voided at 8 A.M. and 11 A.M. The total volume of these 4 specimens should exceed 1200 cc and the specific gravity of one will be below 1005.

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(3) Normal persons show a day to night volume of the ratio of 3: 9 and the night urine specific gravity exceeding 1017.

OTHER RENAL FUNCTION TESTS:

Blood analysis:

(1) Normal NPN is 20 to 40 mgm. The NPN includes urea, uric acid, creatinine. This is increased in renal failure. Similar rises occur with creatinine.

(2) Normal creatinine is 1 to 2 mgm.

(3) **Phenosulphapthalein test (PSP)**

The bladder is emptied. The patient drinks about 200 cc of water and 6 mgm of PSP injection intravenously. The amount of dye excreted in urine is estimated. Normal persons excrete 40 to 60% in first hour and 60 to 85% during the second hour.

Van Sivke's urea clearance test:

This is defined as the volume of blood cleared of urine in one minute.

The patient has a normal diet. He is to empty bladder at 9 A.M. after which he drinks a glass of water. The bladder is emptied at 10 A.M., 11 A.M. and 12 Noon. The urine is estimated for urea and blood collected for urea between 10 A.M. and 11 A.M, and examined. The volume of urine per minute can be calculated.

V. Volume of urine per minute.

B. Blood urea in gm%

U. Urine urea in gm%

If U is greater than 2 the maximum clearance is

$$cm: \frac{V \times U}{B} \times 1.33.$$

If it is less than 2

$$cs = \frac{UXU}{B} \times 1.85$$

Normal result is 100% values below 75 is abnormal.

Urea concentration test of Maclean.

The patient is given a light diet on the previous day and next day he is given 15 gm of urea and specimens collected hourly for 3 specimens and examined. If the second and third specimen contains less than 2% it indicates renal inefficiency.

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"Cortisone in the Treatment of Septicemias"

By

THERESE BERTRAND-FONTAINE & PIERRE CHEYMOL

Ever since its introduction in human therapy in 1949 the conceptions concerning the properties of cortisone have evolved considerably. The main controversy initially ranged on its action against infectious processes, giving rise to all sorts of contradictions and equivocations.

As a matter of fact the anti-inflammatory products, cortisone and A.C.T.H. were initially considered by virtue thereof as likely to favour the onset and development of infections released by the break-down of "inflammatory barriers".

This stand was not only dogmatic at the time but ever since 1950, THORN, DUBOIS-FERRIERE and others reported observations testifying in fact to the development of infections severe in patients submitted to corticotherapy. When we analyse carefully the details of these observations it is quite evident that the imputation of certain untoward complications to cortico-therapy was somewhat biased and, on the other hand, we verify that the number of such complications is really minimal compared with the large number of patients that have taken and are taking cortisone without any drawbacks. However, there is no question of denying this risk of infection even though it may be much less frequent and less alarming than it has been alleged. It does exist and at times justifies a certain anxiety, precautions and a strict supervision over patients submitted to a corticosteroid treatment.

It was almost simultaneously that the initial observations evidencing a favourable action of corticotherapy in the infectious processes were published. In 1950, ROCHE

successfully employed A.C.T.H. in typhoid and in the same year FINLAND obtained remarkable results with the same hormone in 2 cases of severe pneumonia.

We were possibly the first in 1952 to cure with cortisone certain staphylococcal septicemias resistant to all antibiotics. Ever since the trials have multiplied and there is presently no infectious disease in which corticotherapy has not been tried with varying results. As a matter of fact success has of course not been uniform and the most spectacular and significant results are obtained precisely in septicemic processes and in the severe forms of microbial disease; on the other hand, in the infections with a chronic tendency, in viral or parasitic diseases the results obtained are mediocre, nil and at times unfavourable.

It must be admitted today that adreno-cortical hormone therapy has conquered a rightful place in anti-infectious therapy particularly when a septicemic process is present. However, the hormone treatment undoubtedly of an unquestionable value, remains an empirical treatment, the theoretical bases of which are the least dogmatic and not easily grasped, so that in the mind of the observers the spectacular success is paradoxical in view of the mystery in which it is shrouded. It is from this double perspective, practical and theoretical, that we shall consider this question.

1. Results Obtained with Corticotherapy in Septicemias

1. Typhoid and Paratyphoid Fevers

In the wake of the early protagonists of this treatment, ROCHE, SMADEL and

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WOODWARD, numerous authors tried the combination cortisone-chloramphenicol in typhoid fever; a particular mention must be made of the trials by DURAND and RENOUX and the report by BENHAMOU to the Algiers Congress in 1955 which, being supplemented by the thesis of student BONTHOUX, constitutes a precious formulation of this therapy.

The administration of cortisone to a patient affected with typhoid fever enables to get rid of the fever and to ensure the disappearance of the distension within 6 to 24 hours, quite uniformly, so long as sufficient doses are administered. This apyrexia is definitive provided the treatment is continued uninterruptedly for several days and without going below a minimum active dose varying according to the subjects. This last important point in the conduct of the treatment has been observed by numerous authors and we ourselves have been able to verify its validity in a particularly typical case.

Therefore, in the common forms of typhoid fever the combination of corticotherapy and chloromycetin quickly controls the fever and the distension and ensures a less painful evolution of the disease; on the other hand, according to several authors, it practically led to the disappearance of certain complications which remained frequent and severe with chloromycetin, namely myocarditis and collapse. With regard to the relapses their frequency is reduced when the treatment is correctly instituted. The malignant or prolonged forms find their prognosis transformed by corticotherapy in which the latter is recognised as a definite indication.

From the biological point of view an important fact should be observed in so far as, save very rare exceptions the blood-cultures and tissue-cultures are not modified by the cortisone treatment. With regard to the serum diagnosis, with the exception of DURAND and RENOUX, no author

has pointed out any qualitative or quantitative modification under the influence of cortisone. We shall see further the interpretation that these facts suggest.

2. Meningococcemias

Almost all the observations concern the Water-house-Friderichsen syndrome, an affection which due to its very physiopathology should have benefited from corticotherapy.

After the first communication by NELSON and GOLDSTEIN in 1951, the observations of success due to cortisone multiplied the mselves and there is unanimous agreement now concerning the usefulness of this therapy.

The sudden abruptness of this disease is well-known: hormone therapy should therefore be instituted at an early stage and in massive doses because the majority of the failures recorded are due to a delay in the institution of treatment. The latter should be considered as an emergency treatment and in the most severe cases it should be conducted just as the reanimation in traumatic shock, cortisone being then administered by the quickest route: the intravenous infusion.

Under these conditions we obtained veritable resurrections and the major interest of cortisone will be to give the antibiotics the time to act.

It is unquestionable that the early and systematic employment of cortisone has transformed the prognosis of this terrible affection. Without going into physiopathological discussions we must draw attention on 2 points:

—On the one hand, that D.O.C.A. and the total adrenal extracts are ineffective in the majority of cases.

—On the other hand, that successful cures have been obtained with A.C.T.H., which may appear surprising in these patients functionally deprived of adrenal secretions.

3. Staphylococcemias

Out of all the common germs the staphylococcus is the one germ that is most

resistant to the range of antibiotics instituted against it, thus being insensitive to them. This particular resistance explains both the modification in the clinical picture of septicemias, the protracted forms of which go on multiplying, and the difficulties of a treatment based on the verification of a sensitivity to antibiotics which at times exists partially in vitro and is not at all present in the patient with the result that septicemias due to staphylococci practically resistant to all antibiotics have become more frequent.

It is in 2 similar cases after all therapeutic resources were exhausted that we were led in 1952 to employ cortisone with a success much beyond our expectations. In the inability of mentioning the details of these observations we shall merely recall that they were cases of extremely acute septicemias resistant to all antibiotics instituted massively on the basis of data from antibiograms, presenting multiple suppured localisations and the prognosis of which seemed very much doubtful. It is against this background of despair that corticotherapy was instituted with spectacular effects: drop in temperature and a quick improvement in the general condition.

Several times cortisone was sought to be discontinued, the patient being always under antibiotics and every time the fever resumed in intensity and the disquieting symptoms re-appeared; normalcy was again restored with a reinstitution of cortisone. In the second observation at the time of a relapse cortisone was administered without any combined antibiotic and cure was ensured nonetheless.

Ever since we have observed other similar cases that have only confirmed these results, numerous publications concerning which illustrate its worth (NEBOUT and FORESTIER, TERRASSE, LUBETZKI, MOZER, BENHAMOU, etc.). From these overall investigations we may draw certain conclusions:

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—From the theoretical point of view we shall particularly bear in mind the fact that cortisone has enabled to cure septicemias due to staphylococci practically resistant to all antibiotics. It never caused any relapse nor any extension of the infectious process and, on the contrary, in the majority of our observations its favourable action was demonstrated several times in the absence of all antibiotic treatment. We should therefore realise that there is no question of a hypothetical potentialisation of antibiotics since the latter had proved inactive and the addition of cortisone never modified the data from successive antibiograms.

For the same reason we cannot support the theory so often advanced about the anti-inflammatory action of cortisone which, liberating the germs from their hide-outs, would permit their destruction by the antibiotics. Now the facts are: the action of cortisone was identical whether combined with antibiotics or not. Under these conditions we were compelled to recognise an anti-infectious action in favour of cortisone. There is of course no direct antibiotic action on the staphylococcus but an indirect action being exerted on the background.

—From the practical point of view the results obtained permit us to state that the institution of corticotherapy in combination with antibiotics seems justified in all severe forms of staphylococcemias resistant to antibiotics. It is established that in such cases it is the only agent capable of modifying favourably a fatal evolution.

4. Brucellosis

In the wake of SPINK and HALL numerous authors, particularly DURAND and RENOUX have employed A.C.T.H. and Cortisone in the treatment of brucellosis.

It is in the acute septicemic forms that corticotherapy has recorded its finest successes. On the other hand, in the chronic forms such a spectacular efficacy has not

been established. We must recognise a certain similarity with the action exerted by the corticosteroids in typhoid fever. A defervescence and on improvement in the general condition are quickly obtained and here again the fact that cortisone does not act directly on the germ but on the background medium is confirmed, revealing itself frequently incapable of rendering the blood-cultures negative. The antibiotic treatment is therefore the basis of the therapeutic action.

5. Other Septicemic Processes

In 1955, WORMS & Coll. reported the observation of a septicemia due to *B. Coli* cured by the combination of cortisone and antibiotics. They believe that in this case the action of cortisone responds to two mechanisms;

—Antitoxic action, acting on the manifestations of the malignant syndrome.

—Anti-inflammatory and fibrinolytic action, rendering the microbial focus accessible to antibiotics.

MORIN and Coll reported in 1953 2 cases of endocarditis due to enterococci cured with antibiotics, in one of which cortisone was employed and brought an improvement is equally reported by BENHAMOU in a case of Osler's disease. In Italy, BONCOMPAGNI & Coll. point out certain successes obtained with A.C.T.H. and cortisone combined with antibiotics in 2 cases of slow septicemias. Finally we may point out the successes obtained by DONZELOT and KAUFMANN in infectious endocarditis with negative blood-cultures.

II. Mode of Action of Corticotherapy in Septicemic Processes

We restricted ourselves in the first chapter to the enunciation of the results obtained by employing cortisone in septicemias. They represent a clinical trial which is sufficient to convince of the value of this therapy but it sheds hardly any light on its

mode of action. Such an ignorance proves to be not only annoying from the scientific point of view but particularly disquieting from the practical point of view and one would like to know what renders these hormones pro-infectious in certain cases (of course very rare), and anti-infectious in others. We had recourse to the laboratory for these answers which we could not obtain clinically.

A. Experimental Facts

We shall formulate here merely the broad conclusions that follow from the analysis of a large number of investigations. Contrary to what is observed clinically the vast majority of the authors investigating the action of cortisone on experimental infections report an aggravation. Numerous investigators even found a spontaneous development of infection due to saprophytic germs in animals submitted to the action of cortisone or A.C.T.H. But if we take the trouble of analysing the details thereof, it appears that these trials were carried out without taking into account either the animal species or the daily and total doses administered or the time of institution of corticotherapy in relation to the infection. Now these are very important factors. As a matter of fact:

(1) The various animal species react in quite different ways to corticosteroids.

(2) The doses usually employed in the course of the trial are 10 to 100 times higher than those employed clinically.

(3) The date of institution of corticotherapy in relation to the supervening of the infection is equally an important factor if we recall that clinically the cases of superinfection under cortisone are much more numerous than the cases of aggravation of an infectious process existing prior to corticotherapy.

Under these circumstances we feel convinced that the contradiction established between the clinical data and those from animal trial results to a large extent from a

superficial examination of the question. In proof of our assertion we have the fact that the very few authors who cared to ensure in the laboratory the same conditions of administration of the hormone as in human clinic have precisely obtained similar results as those verified clinically (ROBINSON, VAN CAUWENBERGE). These results may be summed up as follows:

(1) The aggravating action of cortisone manifests itself in 2 cases:

(A) When strong doses of cortisone are administered.

(B) When small doses are administered for several days before the microbial inoculation.

(2) The favourable action of cortisone exerts itself when it is administered in average doses after the microbial inoculation.

Such are the overall results and in order to explain them we investigated the influence of cortisone on the different mechanisms involved in the course of the infectious process.

Action of Cortisone on Microbial Germs

A fact proved both clinically as well as in the laboratory, cortisone has no direct action on the microbial germs. At the same time nothing warrants the claim that it exerts any influence on the efficacy of the antibiotics.

Anti-inflammatory Action of Cortisone

After RAGAN, all authors who investigated this question are agreed in recognising that cortisone exerts an anti-inflammatory action which is constant, irrespective of the animal species investigated and probably of the doses employed, even though here again this last factor has not been sufficiently investigated. We must however, observe that this unanimity of results concerns only the overall inflammatory phenomena and under this designation we group quite different reactions; the physiopathological significance of which is variable and the behaviour of which even under the influence of cortisone is variable.

The same is the case with vascular phenomena of inflammation, the mechanism of which is being investigated by MENKIN for the last 20 years, without having been able to discover any definitive structure or to elucidate the point of impact of cortisone action.

Action of Cortisone on Phagocytosis

We find it impossible to draw a conclusion on this particular point; the investigators are of two divergent opinions: those that believe that cortisone reduces the activity of hystiocytes and of the reticulo-endothelial system and those who, on the contrary, attribute a stimulating action to it. It even happens that both the stands are successively taken by the same author (J. CRABBE).

Action on the Antibodies and the Allergic Phenomena

Here again and for the same reason we shall refrain from drawing a conclusion: all possible hypotheses have been upheld and even though there is a majority of investigators who opine in favour of an inhibitory action of cortisone on the formation of antibodies the fact is not proved, while on the other hand, the majority of clinical observations (mainly in typhoid fever) deny such an action.

With regard to the allergic phenomena the position is not clear either and the most common cases are those that evidence an inhibition, under the influence of cortisone, of phenomena of cutaneous allergy but there again it may be wondered if the anti-inflammatory action of cortisone plays a substantial role. As a matter of fact it must be admitted that multiple mechanisms intervene in the allergic phenomena but their complexity has not been fully grasped on a biological analysis.

Anti-pyretic Action of Cortisone

The antipyretic action of cortisone is the most commonly evidenced both in experimental infections as well as in infectious

disease in humans. However, it is not uniform and particularly it does not explain the successes obtained as demonstrated by BENHAMOU. The least that can be said is that cortisone does not behave as an ordinary antipyretic agent.

Influence of Cortisone on Microbial Toxins

We have here a few coherent results that may be summed up thus:

- (1) Cortisone has no action on exotoxins.
- (2) Cortisone exerts a protective action against certain endotoxins. The fact has been established by several investigators and particularly in a series of remarkable researches CHEDID and BOYER. Could it be, as these authors think, due to a select-carbohydrate lipidic antigen? It is possible but we must observe that the clinical results do not at all evidence such a specificity.
- (3) This action is indirectly exerted by an increase in the resistance of the patient. As a matter of fact cortisone is incapable of neutralising the pathogenic action of the toxin *in vitro* as well as *in vivo*. It protects the animal by acting locally, and the fact has been verified by CHEDID and BOYER on animals under parabiosis; and it is otherwise closer to the absence of action cortisone on microbial agents.

- (4) This action is variable according to the animal species considered.

B.-Physio-Pathological Interpretation

Can we draw any conclusions on the mode of action of cortisone in infections from this dissimilar set of clinical and experimental data?

We may recall first foremost that this hormone has no antibacterial action, neither *in vitro* nor *in vivo* (typhoid fever).

Several explanations have been offered:

- (1) Cortisone acting by hormonal substitution.

This theory has been mainly upheld by FRIDERICHSEN in order to explain the successes obtained in the syndrome bea-

ring his name. It, however, has to face serious difficulties: as a matter of fact if nobody contests the reality of adrenal insufficiency in purpura fulminans, it is nonetheless evident that it does not present itself as a pure and acute adrenal insufficiency corresponding to an Addisonian onset. We know for instance that the majority of such cases are not improved either with D.O.A.C. or with total adrenal extracts. NELSON and GOLDSTEIN underlined this fact; they believe that the hormonal modifications mainly affect the production of 11-oxy-and 17-ketosteroids, whereas the hydro-mineral balance is not modified. Under these conditions we may undoubtedly admit that the cure obtained after administration of glucocorticoids responds to a hormonal action but we must realise also that it is a very particular hormonal action of a high specificity, which largely exceeds the conceptions of FRIDERICHSEN.

This latter hypothesis being admitted there remains to explain why A.C.T.H. seems as active as cortisone when the hemorrhagic necrosis of the adrenals should deprive it of its receptor. There remains also to explain the cures obtained with cortisone or A.C.T.H. in other septicemias not involving an evident impairment of the adrenocortical function.

- (2) Cortisone acting by inhibition of inflammatory phenomena.

This hypothesis based on the very therapeutic properties of the hormone was the first to be advanced and its validity remains considerable in the eyes of certain authors who believe in the so called commonsense explanations. As a matter of fact this theory presents the advantage of conciliating two antagonistic points: should we believe in a pro-infectious action of cortisone, it is due to a sudden explosion of latent infections that no longer respect the inflammatory barriers any more; in the contrary case the favourable action of cortisone is explained by the destruction of microbial

hideouts permitting the attack of the germs by the antibiotics in a full-fledged campaign.

It is quite probable that this mode of action intervenes when cortisone is combined with antibiotics that prove to be active on the germ in question but it should be admitted that it explains nothing in cases where cortisone was employed alone or in combination with inactive antibiotics as we have been able to verify in staphylococcemias that we cured, thanks to cortisone.

Recently WORMS has become a protagonist of this theory invoking the anti-inflammatory action of cortisone. He restricts its indications in septicemias only to the cases where this mode of action may intervene, i.e. when the resistance of the germ *in vivo* contrasts with its sensitivity *in vitro*, which according to the author signifies the presence of a septic focus that its inflammatory nature renders insensitive against the penetration of antibiotics.

Carrying this dogmatic attitude to the extreme he goes to the extent of considering the resistance to all antibiotic *in vitro* as a contra-indication to corticotherapy; it is, however, in similar cases that cortisone permitted us to save patients almost on their death-beds.

- (3) Cortisone acting by suppression of toxic phenomena:

It is probable that the anti-toxic action of cortisone established by trail, intervenes to a large extent in its anti-infectious action; however, we ignore the exact import assumed by the toxic phenomena in microbial infections. Investigations by REILLY have drawn our attention since long on the importance of toxic impregnation of the organism in the course of typhoid fever. Recently the demonstration of the mechanism of collapse with chloromycetin has denoted that the liberation on a trial basis of typhus endotoxins was capable of reproducing certain clinical manifestations of the disease. Could we not invoke such a mechanism in the case of germs usually free

from endotoxins? With regard to the staphylococcus in particular HIGGINBOTHAM and DOUGHERTY attribute the nocivity of the germ to a bacterial substance liberated following the digestion of bacteria by the reticulo-endothelial system. We believe that such a mechanism intervenes in the majority of septicemic processes and that probably it explains at least partially the favourable results obtained with cortisone in these affections.

- (4) Influence of cortisone on the allergic reactions of the infectious disease:

We may recall preliminarily that the overall picture observed in typhoid fevers treated with cortisone is as follows:

Cortisone leads to the disappearance of all symptoms of the disease before chloramphenicol has had the time to kill the germs responsible. We have, therefore, envisaged thus in a quasi-experimental way the separation between the two elements of the infectious disease: the microbial specific factor and the non-specific reaction of the organism. If we want to unravel this paradox of a disease which has clinically disappeared but persists bacteriologically, we are forced to admit that the infectious disease in so far as its clinical expression is concerned, is entirely constituted by the reactions of the organism to the microbial agent.

Right from our earliest observations concerning staphylococcemias we were struck by similar findings, namely that things happened as if while suppressing the symptoms we equally suppressed the disease.

Ever since we are justified in wondering if the so called defence reactions against the bacterial aggression do not exceed their aim until constituting the basic characteristics of the disease. In other words is not every infectious disease already a self-sustained disease? It would be better understood when cortisone, opposing itself to the hyperergic phenomena wipes

out to some extent the clinical manifestations without attacking the microbial germ which under these circumstances should not be considered as the causal agent but as the factor inducing the onset of the disease.

It is probable that each of these hypotheses has a partial validity while it is probable that neither is entirely valid and finally it appears that the problem of cortisone infection is intimately connected, on the one hand, with the properties of the hormone and, on the other, hand, with certain physiopathological mechanisms not yet fully elucidated. We are therefore not dealing with a strictly definitive action limited only to toxins possessing a nite entity but with a problem connected with some other problems raised by the infectious disease and endocrinology in general and as such its solution is largely dependent on a widening of our horizons of knowledge in these spheres.

III. Therapeutic Indications

The ignorance in which we find ourselves with regard to the mechanism of action of the corticoid hormones in infections should not lead us to discard a drug, the efficacy of which has been proven. What would happen to the major clinical therapies if always a profound knowledge of their mode of action was insisted upon *a priori*? As a matter of fact the indication of corticotherapy in septicemias corresponds to a certain empiricism and from the practical point of view an analysis of the clinical cases published is certainly more fruitful on the whole than the theoretical considerations drawn from experimentation.

1. Indications

We may recall in the first place that an intense and selective antibiotic treatment should be continued throughout the duration of the infection. Under these conditions recourse to cortisone is justified in severe

septicemias whenever an unfavourable evolution may be feared, after failure of a suitable antibiotic treatment. The nature of the germ responsible and the notions of sensitivity or resistance *in vitro* matter not so much in establishing the indications as the severity of the septicemia.

However, there are two particular cases:

—The Waterhouse-Friderichsen's syndrome where cortisone treatment is a must in all cases and should generally be considered as an emergency treatment.

—The typhoid fever: in the malignant or prolonged forms the corticotherapy is equally called for. With regard to the usual forms it appears that the systematic employment of cortisone may be envisaged hereafter. It appears as a matter of fact to incur no risk and to present the advantage of ensuring for the patient a less painful evolution of his disease and possibly of preventing a secondary aggravation.

2. Contra-indications

Among the classical contra-indications a single one is formal: gastro-duodenal ulcers. The others are relative requiring special precautions in each case; nevertheless we should hesitate to prescribe cortisone in elderly patients, those with hereditary affections, diabetics, those with cardiac or renal insufficiency as well as in subjects presenting a psychic syndrome.

3.-Modalities of the Treatment

A) Choice of the Hormone

Cortisone is always preferable to A.C.T. H. because it acts more quickly, irrespective of the functional condition of the adrenal gland. Hydrocortisone seems to us permit a more versatile administration than cortisone; it permits smaller doses, the sodium chloride retention is smaller and it seems to be endowed with an efficacy equal, if not slightly superior to cortisone.

With regard to Prednisone (Metacortand-racin) we have no experience of it in infectious diseases.

B) Routes of administration

All the routes are permissible, their choice depending on the quickness of the desired action. We may merely recall that cortisone is as active orally as parenterally.

(C) Dosage

It is difficult to give precise indications concerning the dosage required since they are variable according to the cases. However, the majority of the authors are agreed in recommending strong doses at the beginning and then progressively diminishing; there exists of course a minimum active dose and the daily dosage may at times have to be increased in case of resumption of the symptoms of acuteness.

By way of illustration a dosage of 300mg. of cortisone or 100 to 150 mg. of hydrocortisone in the first few days appears to have been fully satisfactory.

(D) Duration of Treatment

Even though certain authors follow the technique of a "lightning treatment", we believe on the contrary, just as KINSELL and JAHN that at least a week's treatment is called for in order to avoid a relapse. In certain cases even this treatment should be followed up for several weeks and it should involve no major risks if care is taken not to interrupt it abruptly and to continue the antibiotics for several days after the discontinuance of cortisone.

(E) Supervision over the treatment

It corresponds to the usual features: a salt-free diet, a daily recording of the blood pressure, supervision over change in weight, edemas, sodium and potassium contents in the blood and a preventive administration if need be of potassium chloride in small doses.

Under these conditions we consider that corticotherapy does not subject the patient to any risk. On the contrary, combined with antibiotics it constitutes a very potent therapeutic weapon which has permitted the restoration to normalcy of desperate conditions. The verification of such spectacular successes seems to justify the forecasts made in the remarkable article by MOZER as follows: "We should not rule out that finally infectious diseases should be one of the most evident indications for cortisone therapy, among all those suggested so far". We may also not rule out the possibility of a revision of a substantial number of our notions concerning infectious diseases on the basis of the results obtained with corticotherapy.

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GLEANINGS FROM THE MEDICAL PRESS

**ORAL TREATMENT OF DIABETES:
TRIAL OF PHENYLETHYL-DIGUAN-
IDE (D.B.I.)** G. H. Hall M. F. Crowly and
A. Bloom, *Brit. M. J.* 2: 71-74 (July 12)
1958 (London) (via *J. A.M.A.* Nov. 1, 1958)

In the course of study to determine the effectiveness of phenylethyldiguanide (D.B.I.) as a hypoglycemic agent in diabetes of all grades of severity, to gauge its side-effects, and to compare its mode of action with that of insulin and tolbutamide, 40 diabetic patients were admitted to the wards. The subjects ranged in age from 18 to 78 years, with a mean average of 57 years. A significant fall in blood sugar levels was observed in 24 of the 40 patients in response to a single 150-mg dose of D.B.I. The response began to be apparent at 4 hours and reached its maximum at 6. Although there was no significant change in the blood sugar levels during the period of dietary restriction alone, administration of D.B.I. led to a progressive fall in the mean blood sugars, the lowest level being attained by the 6th day of treatment. The urinary output of sugar reflected the blood sugar levels, and there was no evidence of an altered renal threshold. The 5 patients whose life depended on the administration of insulin developed ketosis very rapidly when insulin was withdrawn; their condition was not alleviated by the administration of D.B.I. although there was a small fall in the blood sugar level. In the remaining 35 patients only occasional positive "acetests" were recorded prior to treatment. After the administration of D.B.I. 11 of the 35 exhibited intermittent acetoneuria of greater frequency and concentration than prior to treatment; although normoglycemia and aglycosuria prevailed.

No effect of D.B.I. could be demonstrated on either plasma amino-acid nitrogen or serum inorganic phosphorous values. An inverse relationship was found to exist regarding the severity of diabetes and the response to D.B.I. patients with milder or moderately severe diabetes showing a fall in blood sugar level in response to the administration of D.B.I. Of the 14 newly diagnosed diabetics, 12 exhibited a good hypoglycemic response to D.B.I. and 5 of these did not have a relapse when D.B.I. was withdrawn. Of the patients with diabetes of long standing, 17 showed some hypoglycemic response; but none could subsequently be managed by diet alone, the impression being that D.B.I. was more effective in diabetes of recent onset. No fewer than 26 of the 40 patients developed gastrointestinal symptoms while receiving the drug; these symptoms abated upon discontinuance of the therapy. In 2 patients in whom tolbutamide had been employed unsuccessfully, D.B.I. was effective in lowering the blood sugar level. In 3 other patients in whom D.B.I. had produced normoglycemia and in whom treatment was discontinued because of nausea and vomiting, tolbutamide was slower and less effective in reducing the blood sugar value. It was impossible as previously planned in 25 of the 40 patients, to administer D.B.I. for 6 weeks because of unpleasant side-effects or because an adequate fall in blood sugar level had not been realized. Six of the remaining 15 patients are still receiving D.B.I. and their condition is satisfactorily controlled.

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**BRONCHIAL ASTHMA AND CHRONIC-
BRONCHITIS TREATED WITH HYD-
ROCORTISONE ACETATE INHALA-
TIONS:** W. H. Helm and F. Heyworth,
Brit. M.J. 2:765-768 (Sept. 27) 1958
(London) via *J.A.M.A.* Dec. 20, 1958.

Twenty-five male and 28 female patients; between the ages of 15 and 61 years, with

asthma or wheezy bronchitis of at least one year's duration, whose condition had been severe enough to necessitate periods of work, were hospitalised and treated with antibiotics and routine antispasmodic drugs before receiving hydrocortisone inhalations. The patients inhaled fine hydrocortisone acetate powder from a "pabracort" inhaler, the powder being in capsules containing 15 mg of hydrocortisone acetate and 75 mg of lactose powder, both being ground fine to a mean particle size of 5. The initial dose varied between 1 capsule 3 times a day and 1 capsule daily, given as half a capsule twice daily, according to the severity of the wheezing at the beginning of treatment. The dose was reduced to the minimum maintenance necessary as soon as improvement occurred. The usual maintenance dose was half a capsule twice daily. Treatment was stopped in patients with recurrent asthma, once the attack had been relieved for a week, and they were provided with capsules and instructed to restart at the onset of symptoms in a dose of between 1 and 3 capsules daily according to the severity of the attack. Patients who had failed to make any definite improvement during 2 weeks' treatment with inhalations were assessed as failures and the treatment was stopped.

Of the 53 patients, 43 (81%) improved on hydrocortisone inhalations, whereas 10 (19%) were not improved. Of the 43 patients who improved, 13 (30%) relapsed after periods of 1 to 11 months, whereas 30 (70%) maintained the improvement for periods varying between 2 months and 2 years. Best results were obtained in patients with recurrent acute asthma without significant bronchial infection, whereas the results in patients with wheezy bronchitis were disappointing. There were no definite complications, but one man had mild dyspepsia for a few weeks which may have been due to the hydrocortisone. Several patients put on weight, which was probably due partly to

improved health and partly to the effects of hydrocortisone absorption.

The inhalations of fine-particle hydrocortisone acetate powder thus were found to be an effective form of treatment in many patients with bronchial asthma. There is a strong tendency to relapse if the treatment has to be maintained, but prolonged benefit may continue if the inhalations can be used intermittently for recurrent attacks of asthma. Although the treatment may be successful in status asthmaticus, it is probably less effective in this condition than intravenous or oral administration of corticosteroid drugs, and its use is not recommended if the patient is dangerously ill. A considerable portion of the "inhaled" hydrocortisone is probably absorbed, but the dosage is low compared with that used in oral therapy with a corticosteroid and the chance of toxic effect is, therefore, much less. The optimum dosage is uncertain, but it probably varies from patient to patient. Little or no benefit is likely to be obtained from this form of treatment in patients with established chronic bronchitis.

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**PENICILLIN IN THE TREATMENT OF
STREPTOCOCCAL INFECTIONS: A
COMPARISON OF EFFECTIVENESS
OF FIVE DIFFERENT ORAL AND
ONE PARENTERAL FORM.** B.B.
Breese and F.A. Disney. *New England
J. Med.* 259: 57-62 (July 10) 1958 (Boston) via *J. A. M. A.*

The authors treated 611 children, between 1 and 17 years of age, who had infections due to beta-hemolytic streptococci, with 1 form of parenterally administered penicillin and different preparations given orally in order to determine their effectiveness against streptococcal infections. Of the 611 patients, 122 received a total dose of 600,000 units of benzathine penicillin G 237 were given a total dose of 8 million units of phenoxymethyl penicillin (Penicillin V) 114 received a total dose of 8 million units

of buffered potassium penicillin G 66 were given a total dose of 7.5 million units of buffered potassium penicillin G and probenecid and 72 received and 5 million units of buffered potassium penicillin G and probenecid suspension. These children were observed for 2 months after the institution of therapy. No patient was included in the study who did not have a throat culture at 11 days after the onset of therapy. Acute tonsillitis or pharyngitis was diagnosed in almost 70% of the patients 19% were carriers, 5.4% had cervical adenitis and 4.7% scarlet fever. The remaining patients had infections such as otitis media, sinusitis and cellulitis. Uniformly good immediate results were obtained with all the preparations tested.

The ultimate effectiveness of these drugs, as judged by the cure rate at the end of 2 months, the relapse rate within 25 days and the proportion of strongly positive cultures obtained from patients with relapses, indicated that a single intramuscular injection 600,000 units of benzathine penicillin G was superior to the preparations given orally. The parenterally administered penicillin preparation may have the additional value of preventing some subsequent unrelated streptococcal infection. Among the orally administered drugs no single

preparation was significantly superior to the others. The choice of which one to prescribe is incumbent on the physician. The skin reactions which were observed after probenecid had been used for more than a week were harmful, although not serious. Parenteral administration of penicillin is cheaper than the administration of oral preparations and is not associated with the problems encountered with the latter. Benzathine penicillin G given in a single dose of 600,000 units intramuscularly is the authors' first choice in the treatment of streptococcal infections in children. Its chief disadvantage lies in the painful local reactions frequently observed and the psychological trauma engendered by the use of a needle.

Therapeutically, but not prophylactically, the oral preparations used were almost as effective as the one administered parenterally. The choice of which oral drug to select should be based on factors other than effectiveness since there is no evidence to indicate the therapeutic superiority of any one product. Since cost is often the most important factor, among the oral preparations tested, buffered potassium penicillin G in a dosage of 800,000 units daily for 10 days is the first choice for oral treatment of streptococcal infections in children.

Notes & News

LEDERLE - CYANAMID INTERNATIONAL CONFERENCE

About 40 pharmaceutical executives from many countries of the Far East, South East Asia, Middle East, Europe and Australia, engaged in the organization of the worldwide distribution of important drugs and medicines of the famous research organization, Lederle Laboratories, a division of Cyanamid International, New York, assembled in New Delhi last week and went through a five day conference which concluded on 14th March 1959.

ANTIBIOTICS PRICES REDUCED

Among other important deliberations during the meeting, it was announced by a spokesman for Lederle-India that effective immediately the prices of the Lederle broad-spectrum antibiotics AUREOMYCIN and ACHROMYCIN were being reduced in India. Such reductions were made possible as a result of the recent opening of a new wing to Lederle-India's Bulsar Plant, providing additional manufacturing facilities and more space, thereby permitting increased output of these products. This reduction,

the spokesman added, is intended to make these life-saving drugs available to the population of India at more economic prices, thereby enabling MORE physicians to treat MORE patients with these valuable drugs.

Some of the countries represented at this conference were: Greece, Egypt, Iran, Iraq, Jordan, Lebanon, Syria, Turkey, Australia, Burma, Ceylon, Hong Kong, India, Indonesia, Japan, New Zealand, Pakistan, Philippines, Singapore, Thailand and Vietnam. From the United States, Messrs. R. Roland, N. G. Tamblin, N. A. d'Entremont and M. Roca, representing Cyanamid-Lederle management were participating. Among other subjects, the conference reviewed some of the recent medical research developments in the field of drugs and pharmaceuticals in relation to their current application in the control of and treatment of diseases, particularly prevailing in the countries represented by the delegates. This conference, the first of its kind and magnitude to be organized by Lederle in India is one of a series of similar meetings being held in March in Europe and Latin America. Products of Lederle research such as HETRAZAN and AUREOMYCIN, with the discovery of which the eminent Indian Scientist, the late Dr. Yellapragada Subba Row was connected, are already finding increasing application in the Health Programs for control of endemic diseases in India.

SURGICAL REQUISITES TO BE PRODUCED AT INDIAN PLANT

Many of the vitally needed surgical requisites and allied products over which in the past years, so much foreign exchange has had to be utilised, will now be manufactured by Johnson & Johnson, in their new plant now fast nearing completion on a 24-acre site at Mulund, Bombay.

This unique factory, designed and constructed with a view to ensure production under exacting hygienic conditions, is the

result of Indo-American co-operation. It brings together the well-known firm of Johnson & Johnson and the Indian industrialist Mr. K. M. Premchand.

Johnson & Johnson entered the field of surgical dressings as far back as 1873 and is today the largest manufacturer of its kind in the world, producing a total of more than 1,200 different related products. Among these are such important products as sutures and surgical dressings, pressure sensitive tapes, medical plasters, gynecic therapeutic products, etc. Wellknown consumer products include Band-aid adhesive dressings, Tek Toothbrush, Johnson's Baby Powder and other baby products, Johnson's Prickly Heat Powder, amongst others.

The manufacture in India of these products will help considerably in lessening the strain on foreign exchange, which in the matter of imports of medicinal and pharmaceutical products amounted to more than sixteen crores of rupees in 1957 despite import restriction.

That the need for surgical dressings and allied products continues to grow is evident from the targets of the Second Five Year Plan. For instance, in 1950-51 the number of doctors stood at 59,000, in 1955-56 at 70,000 and the target for 1960-61 is 82,000. This is also the case with the number of hospital beds: in 1950-51 the number was 113,000, in 1955-56 it was 125,000 and the target for 1960-61 stands at 155,000.

Among many unique features is the method adopted for chemical sterilisation of sutures, which is being employed on an industrial scale for the first time anywhere in the world.

Always striving to develop new products and improve upon existing ones, Johnson & Johnson place maximum emphasis upon research. The plant at Mulund will have an extremely up-to-date laboratory where a constant check will be kept on the quality of materials and on the finished products.

(Continued on page 23)

PRACTITIONERS' FORUM...

QUESTIONS & ANSWERS

Etiology of Infantile Cirrhosis of the Liver

To the Editor: What are the modern views regarding the etiology of INFANTILE CIRRHOSIS OF LIVER, and what are the essential investigations to be done to come to a definite diagnosis? Discuss the present day therapy of the same with special reference to the role of corticosteroids.

—Dr. C. V. Krishna Rao, M.B.B.S., Madras

ANSWERS:

The Aetiology of infantile cirrhosis of the liver has not yet become quite clear in spite of all the work that has been done on this subject. Certain conclusions can however be drawn on the results of work done till now.

Hereditary:

Evidence in favour of hereditary transmission of the disease appears to be conclusive. It is quite common to find several cases of the disease occurring in the same family.

Social Status:

The vast majority of the cases occur in middle class families more often among vegetarians than among non-vegetarians. It hardly ever occurs among the very poor classes. In this connection it is interesting to note that protein deficiency syndromes like kwashiorkor is quite common among the very poor. It is now certain that cases of kwashiorkor never go on to infantile cirrhosis of the liver. Thus it is safe to conclude that protein deficiency is not the most important cause of infantile cirrhosis.

Role of infections:

Almost all cases exhibit a tendency to suffer frequently from various infections especially respiratory infections. This would suggest some kind of inability of the body to develop immunity against infection.

Liver Biopsy:

Needle-Biopsies of the liver have shown the presence of necrosis and diffuse fibrosis. There is absence of the bile duct obstruction and signs of biliary stasis. The liver responds to many types of injuries by under-going necrosis. Hence Biopsy does not carry us very far in determining the aetiology of the condition except to show that there is injury to the liver. Another significant finding is a negative one, namely the absence of giant cells which are very prominent in viral hepatitis. There is a steady deterioration in the functions of the liver as determined by bio-chemical tests there by confirming the fact that there is some adverse influence acting constantly on the liver.

So far we have dealt with unequivocal findings with respect to the aetiology of infantile cirrhosis. A number of investigations with inconclusive findings have been made with regard to various factors supposed to have a bearing on this disease. Among these, the important ones were concerning the role of breast milk, castor oil, primary infection of the liver and diet. None of the above have so far been proved to bear definite aetiological relationship to this disease.

From the various facts mentioned above certain conclusions seem obvious. The

familial nature of the disease indicates that probably in many of these cases there is an inherited inherent defect of the liver. There is also a tendency to develop infections easily most probably due to inability to form immune bodies. Repeated infections can produce progressive damage to the liver. Protein deficiency plays a secondary role, in that deficiency of essential amino acids, makes the liver even more vulnerable to injury. It is doubtful whether some of the cases actually develop viral hepatitis at some stage of the disease and thus further aggravate the condition.

To summarise, the aetiology of infantile cirrhosis is three fold.

Firstly there is the inherited weakness of the liver, secondly there is the tendency to develop infections and thirdly the reaction of the very to noxious influences resulting in necrosis of the liver cells. In addition there is the secondary effect of deficiency of lipo-trophic substances which normally protect the liver from injury to a certain extent.

Investigations for diagnosis of the condition are based on various liver function tests. The most important ones are, estimation of total protein, albumin and globulin in the serum and serum-electrophoresis. The former shows reduction in total protein and reversal of albumin globulin ratio. The latter usually reveals an increase in the gamma globulin fraction.

The final diagnosis however rests on liver biopsies. Needle biopses usually yield sufficient data. The usual findings are diffuse fibrosis, intense necrosis, Histiocytic reaction and absence of giant cells. There is also absence of bile duct obstruction, biliary stasis or evidence of infection of bile ducts.

Treatment:

Arising out of the aetiological factors so far as we know, it would be rational to consider the three fold nature of the causation of this disease. The first point namely weakness of the liver would indicate the necessity of giving plenty of first class proteins and vitamins necessary for the protection of the liver. Secondly all precautions should be taken to prevent infections and treat them as and when they occur. Thirdly one should try and prevent as far as possible, the necrotic reaction of the liver to noxious influence. This last can be carried out by the administration of prednisolone or similar hormones.

Since repeated infection is the most troublesome part of this disease, a great deal of attention has been paid to this aspect of treatment. Repeated small blood transfusions have been given with the object of raising the level of immunebodies in the patients blood. Gamma globulin injections have also been tried with a similar object in view.

(Continued from page 21)

From the production lines of this well-planned and hygienic unit, will come such products as sutures and surgical requisites, surgical dressings, orthopedic plaster- of paris bandages, adhesive plasters, pressuresensitive tapes, and the famous range of Johnson & Johnson's baby products, and other products for home use.

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Book Section

ANNOUNCEMENT

We are proud to announce that the 'Mediscope' dedicated to serve the Indian Medical Profession, has ventured to publish 'Text Books' for Medical Students & Doctors, thus affording our regular subscribers to possess 'Text Books' without paying specially for them.

The text books written by eminent authorities—Professors, Lecturers and Specialists—in Medicine and Surgery have graciously come forward to have their text books published in the 'Mediscope' serially, each author taking about eight to sixteen pages in each issue until the Text Book is completed.

From Feb. '59 issue the 'Mediscope' has commenced the valuable work of Dr. R. J. Maneksha, F.R.C.S. (Eng), on "PLASTIC SURGERY FOR MEDICAL STUDENTS & DOCTORS". From this issue (April '59) we are publishing, concurrently, the text book on "RETINAL DETACHMENT & ITS MODERN TREATMENT" by Dr. J. M. Pahwa, M.B., B.S., D.O., Z.O. (Vienna) M.S., Senior Ophthalmic Surgeon, Eye Hospital, Sitapur. Ofcourse the 'Text Books' will be in addition to the usual features of Articles, Case Reports, Gleanings, Notes & News etc.

As a few of our regular readers have observed, that the 'Book Section' not only affords an opportunity to possess text books, it also gives a very great incentive to the eminent Medicos of our country to come forward to write 'Text Books'.

Writing of a 'Text Book' suitable for the Universities is the hardest job, requiring the full time attention of an author at the sacrifice of his practice, not to mention of the financial risk.

Having risked time and finance none can be sure of the attitude of the Universities to recognise the merit of such costly works and prescribe them to the Medical Students.

Although the talent is not lacking in our country to write Text Books suitable to our Universities, many an authority on Medicine & Surgery, under whose coaching the Indian Medical Profession has today grown to a lakh and odd strong, have not ventured rightly, to indulge in the highly speculative art of writing text books. Consequently this great Nation still depends on the resources of our brethern abroad to feed us with text books. It certainly does not stand in good stead of our National Prestige.

To fill up the lacuna in the field of Medical Text Books by our Indian Authors, the Mediscope has boldly ventured to publish Text Books absorbing the financial strain of the authors, thus giving them an impetus to contribute their precious knowledge and wide experience in the field of Medicine & Surgery for the benefit of our Indian Medical Profession.

We fervently hope that the Indian Medical Profession will appreciate our aim—laudable indeed, and make our efforts asuccess.

—Editor.

BOOK SECTION

1. PLASTIC SURGERY FOR MEDICAL STUDENTS & DOCTORS

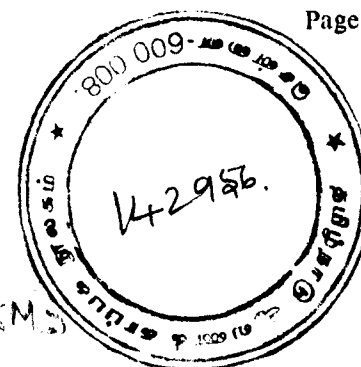
By R. J. MANEKSHA, F.R.C.S. (Eng.)

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2. RETINAL DETACHMENT AND ITS MODERN TREATMENT

By J. M. PAHWA, M.S.S.S., D.O.,
Z.O. (Vienna) M.S. (Luck)

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N 59.211

Hare-lip and Cleft-palate

What are the usual precautions after this type of operations?

In an infant, breast feeding is not possible for a few days so the child is usually fed with a teaspoon. When the operation is done before the age of two years, the hands are tied to wooden splints so that the child does not touch the operated part.

Is any dressing applied after the operation?

The operated part is usually kept covered for 24 to 48 hours and is then left open. Some surgeons prefer to apply the Logan's Bow which is supposed to avoid tension on the suture line when the child cries.

When are the stitches removed?

The stitches are removed usually on the fifth day.

CLEFT-PALATE.

★ ★ ★

What is Cleft-palate?

Cleft-palate is the deformity where the palate is divided since birth. Like the hare-lip it may be a complete or an incomplete cleft. It may occur alone or with a hare-lip deformity.

What is the effect on a person suffering from cleft palate deformity?

Due to the escape of air from the mouth to the nose, the person speaks with a nasal voice which is very often inaudible. There is difficulty in eating and very often there is leakage of fluid through the nose.

Why is speech defective in cleft palate cases?

When a normal person speaks the muscular soft palate is thrown backwards and it touches the part of the pharynx

Plastic Surgery for Medical Students & Doctors

thus dividing the nose and the mouth into two distinct cavities. In a person with a cleft palate this closure is not possible and there is an escape of air through the nasal cavity. Speech is also defective if the tongue does not move properly, also if there is a defect of the upper lip like hare-lip, and when the general intelligence of the person is low.

What is the treatment of cleft palate?

The ideal treatment is to close the cleft by Plastic Surgery. If this is not possible when the gap is too big or due to previous operations there is damage to the tissue then an artificial partition (obturator) is fitted by the dentist.



At what age is operation advisable?

Operation is advisable before the age of two i.e., before the child develops defective speech habits. Operation can always be done at a later age but if bad speech has already developed it is very difficult to improve this by operation alone.

Hare-lip and Cleft-palate

Is speech training necessary?

No case of cleft palate is treated completely without speech training which plays a very important part in the improvement of the patient's speech. Wrong speech habits that have been formed have to be corrected by the speech therapist.

What are the complications of the operation?

Separation of the stitched flaps may occur. Trying to repair a very wide cleft usually ends in failure. Flaps stitched under tension usually give way.

What is the treatment of a failed case?

Usually an obturator is put in when a gap persists after operation. Repair by extrapalatal tissue is the other alternative.

When does the wound heal up?

The wound usually heals up within a week's time.

Are there any special precautions?

The child is instructed not to touch the palate with the tongue. Mouth is kept clean with antiseptic gargles and lozenges.

When can the child be permitted to eat?

The child is allowed to eat soft food from the day after the operation.

How long does the person stay in the hospital?

The person stays in the hospital for a week.

If the gap after the first operation is a small one, can it be repaired by surgery again?

Yes, after three to six months.

CHAPTER VI

Nose

★ ★ ★

HUMP—NOSE

SADDLE—NOSE

FOREHEAD OR INDIAN RHINOPLASTY

★ ★ ★

What is the importance of the nose in plastic surgery?

The nose the most prominent part of the face and the centre of observation. The nose also lends character to the face. A person's character does not depend upon his nose as noses can be so easily altered nowadays by means of plastic surgery!

Which is the most common plastic operation on the face?

On an average the most common plastic operation on the face is that performed on the nose.

How does a patient approach a plastic surgeon?

Patients know that their nose is defective and the plastic surgeon has not to make the diagnosis in these type of cases. Patients also suffer from difficulty in breathing when there is deviated septum or saddle nose defect. Psychological cases are refused operation.

What are the improvements after Plastic Surgery on the nose?

The majority of the patients derive a benefit from the improved breathing as well as from the advantages due to improved appearance.

Nose

What is the structure of the nose?

The nose consists of a bony part in the upper half and a cartilaginous lower part. On the outside it is covered by skin and on the inside by mucous membrane. The top part of the nose is the bridge and the lower part the nostrils. Both the nostrils are separated by the columella. Each compartment of the nose is separated by the septum of the nose.

What are the ideal lines of the nose?

The bridge of the nose should be in line with the chin and when seen from the side, the ideal nose projects about 30 degrees from a line joining the forehead to the chin. The columella joins the lip at an angle (columello-lip) and the ideal angle is about 90 to 100 degrees. The nostril should be triangular in shape when seen from below. A very wide nostril gives a negroid type of appearance to the face.

What is the ideal size of the nose?

The nose should be equal to the length of the forehead or equal to the size of the ear. Usually it varies from 2½ to 3½ inches from the bridge to the tip of the nose.

What is the shape of the nose in an infant?

In an infant the bridge of the nose is not developed. But as the child grows this gradually rises up to normal.

Many uninformed mothers are often worried about their child's nose at this age.

What are the types of defective noses that require plastic surgery?

Surgery of the nose can be done defects present since birth or acquired defects caused by accidents, diseases, and

growths. The usual type of deformities that respond to nasalplasty are (a) protrubance of the central ridge, called 'humped nose' or 'hooked nose' (b) depression of the bridge and ridge of the nose called 'saddle nose' (c) twisted nose and (d) deformities of the tip of the nose or nostrils, (e) deformities of the columella.

Are there any types of noses difficult to correct?

Noses that have very thick skin are difficult to correct and only some improvement is possible but may not satisfy the patient and hence they must be warned before operation.

What is the ideal age for performing a nasalplasty?

The ideal age is from 17 to 18 years of age. Prior to this age the cartilage and bones of the nose are not fully developed.

Can a patient get an idea before operation of what his nose and face will look like after operation?

It is possible to give an approximate idea only.

What is the pre-operative care of the patient?

All cases are first properly examined. Photographs in different angles of the nose as well as a plaster of paris cast is usually prepared by the surgeon. The patient is usually admitted one evening previous to the day of operation and nose cavity is properly cleansed. The patient should not be suffering from a cold in the nose.

What type of anaesthesia is generally used?

Many surgeons prefer to do the nose operation under local anaesthesia whilst other surgeons prefer general anaesthesia. Hypotensive anaesthesia is preferable if facilities are

are available for doing this method. It gives the surgeon a bloodless field of operation.

Where is the usual incision in nose operations?

The cut is generally made inside the nostril and no scar is seen on the the outside. Sometime the columella is divided when it joins the lip.

What is the reatment of ta humped nose?

The hump is removed and the nose is narrowed and the tip of the nose is elevated.

What are the usual steps in a rhinoplasty for hump nose?

Intranasal incision is used at all times by the majority of surgeons. It starts laterally between the upper and lower alar cartilages and extends medially over the septal area—the columella is next incised from the lower part of the septal cartilage. The incision is ideally made with Joseph's knife which is then advanced over the upper lateral cartilage to separate the periostium over the bony nasal bridge. Next the periosteal elevator is substituted to uncover the bone laterally. The whole of the dorsum of the nose is visualised by passing the qufrichts nasal retractor. With angled scissors medial a triangular wedge of upper lateral cartilage and mucosa is excised. Next with No. 15 or 11 Bard Parker knife the lowest part of the lump is cut for about $\frac{1}{2}$ an inch. In this notch the MacIndoe's nasal chisel is introduced and the whole nasal hump is separated by side to side movement of the chisel. No hammering is necessary. The sides of the chisel being blunt the skin is not damaged. The curved and the straight nasal rasp (Maltz) are next used to give the correct curvature at the bridge and smother the bony mar-

gins. Any finer trimming of the septal cartilage is done with No. 11 blade knife. At some centres the hump is shaved or even cut away with Bone cutting forceps (Eckhoff).

Through the lateral part of the original incision or through a separate incision on each side the periostium over the most lateral part of the bridge of the nose is separated and with an angled Joseph's nasal saw the bone is divided right upto the inner side of the eye. This part is sometimes done with chisel and hammer. The medial side of nasal support is then fractured with straight chisel and hammer. The fractured bone is then pushed medially on both sides with manual pressure thus narrowing the nasal bridge.

After removing the hump and narrowing the bridge the tip and alar cartilages will be out of proportion to the reduced size of the upper half of the nose and now this lower part is next tackled. This is the most delicate part of the operation on the tip of the nose. At this stage the columello-lip angle can be altered to the desired angle by cutting away the lower part of the septum with knife. A prominent nasal spine is taken away with bone punch.

Exposure of the lower alar cartilage—this is best done by the eversion technique without using a second marginal incision. With small curved scissors the skin is separated from the lateral and medial curves of the lower lateral cartilage and is pulled out by sharp skin hook. The required amount of the upper margin of this cartilage is excised with scissors.

Excision on both sides must be equal. On replacing the remaining cartilage the tip becomes narrow and nostrils take up the required shape. The lower margin of the alar cartilage is not cut as sometimes a fallen nostril is likely. Some surgeons use a second marginal incision and remove the

Nose

cartilage. When major part of the cartilage requires removal the lining mucosa must be preserved to avoid any pinch nose appearance.

If the nostrils are still very wide they can be further narrowed by removing a triangular wedge from the lateral side of the alar base. This means a small incision on the skin which is in the natural fold of the nose and does not show except from very near.

This completes the operation, the nose is reviewed from all sides by using a small mirror so that the surgeon has not to shift his position. Any small adjustment is then made till the desired shape is obtained. The operated cavity is flushed out with saline.

The upper lateral cartilage flap is then stitched to septal mucosa with catgut. The columella is stitched to the septum. The nose is lightly plugged with ribbon gauze.

How soon after surgery does one resume normal breathing through the nose?

After five to six days when the nasal packs are removed.

How long does the swelling take to subside completely?

The post operative swelling completely disappears in about three months. The nose is usually massaged from the fifteenth day onwards of the operation.

Is an operated nose more susceptible to injury than a non-operated nose?

No. Unless the injury occurs during the first month after operation.

SADDLE NOSE DEFECT AND ITS TREATMENT

★ ★ ★

What is "Saddle" nose?

"Caving in" or saddle nose deformity is the most common of all nasal abnormalities. Here the dorsal line of the nose is depressed. It is very disfiguring and since it is often thought by the lay person as a stigma of specific infection, its correction is the most sought after. Its correction should not be denied unless on psychological causes inherent in the patient.

What is the supporting frame work of the nose?

The supporting framework of the nose has the septum in the midline which forms the main strut and on it depends, the dorsal line of the nose, its height and angle. The upper one third of the nose has the two nasal bones and the frontal processes of the maxillae forming the bony bridge of the nose, the middle third has the upper lateral and the lower third the lower lateral cartilages.

How is saddle nose formed and how is it classified?

Loss of any of these above mentioned supporting structures may lead to saddle nose deformity. Dr. Joseph has divided saddle nose defects into three types.

I. Simple type—with normal length of the nose. It may be with or without loss of tip support. This is the most common variety and is due to the following causes:—

1. *Congenital*—It is either hereditary or familial.

(a) Congenital retrusion of the nasal elements.

Nose

(b) Familial—Negroid type of nose.

(c) Congenital defect accompanying cleft lip and palate defects.

2. *Acquired*:—

(a) Trauma—Injury to the nose in young age is very common. Squashed in type of fracture is the result of direct blow or fall on the nose. If not corrected it results in a typical saddle nose defect. There is excessive width of the nose at the bridge.

Operative trauma i.e., during septal resection is responsible when dorsal support is lost. It is always advisable to preserve cartilage and bone in the dorsal part of the nose or the present day tendency is to do a conservative corrective resection of the septum with minimal removal of the cartilage itself.

(b) *By Infection*—Suppuration of septum due to abscess and osteomyelitis of the bones is not rare in children. Specific infection either of congenital or acquired tertiary stage destroys not only the supporting structure but also the mucosal lining thus very often producing nasal defect of the type II and III of Joseph now to be described.

II. Complex saddle nose with normal length—Here although the nose is of normal length and the tip is in normal position there is not only loss of supporting tissue but also destruction of the overlying skin. The three very common lesions producing this condition besides a severe trauma to the nose are produced by Lupus and Tropical Sore. Lupus is not common in our country as in the West but Tropical Sore on the nose is seen in the Punjab as also in the Middle East. Leprosy is endemic in India.

III. Saddle nose with shortening—The whole nose is shortened and the tip of the nose is pushed in and upwards. The dorsal line of the nose is very short. This gives a typical “dished in” appearance of the nose. Its main causes are Syphilis and Leprosy. A different type of the classification of saddle noses is useful and simple.

I. At the nasal bridge—1) Depressed fractures in young age. 2) Smash in type of injury at any age.

II. At midsection—1) Old fractures. 2) Excessive removal of septum or bony hump in rhinoplasty. 3) Specific infection. 4) Excessive prominence of the nasal bridge or tip of the nose with saddle in midsection. 5) After cleft palate operation in some cases due to failure of bony development.

III. At the Tip of the Nose—1) Excessive removal of septal support. 2) Lupus infection.

IV. Total—A complete smash in type of injury with fracture of the maxillae and total retrusion of the nose with “dished in” appearance.

What is the treatment of Saddle Nose?

The primary consideration is to find out the cause of the defect and to judge the amount of tissue loss i.e., the supporting as well as the lining and covering. Lost tissue must be replaced by similar tissue.

A. *Fracture* :—Here there is no loss of tissue but generally a displacement at the bridge of the nose with widening. The treatment is to refracture the bones and lever them again into position—maintenance of the bones in the reset position should be by adequate splintage. For unstable fractures wiring and external splintage is necessary. If the bridge line fails to come up in spite of this method, extra support may be inserted later on.

Retinal Detachment

AND

Its Modern Treatment

BY

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Introduction & History

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The term "detachment of the retina" is a misnomer because in the first place retina as a whole is not involved, but a cleavage occurs between the two primitive retinal layers, the pigment epithelium remaining in position attached to the membrane of Bruch, while the inner (neural) layers become separated from it. (Fig 1) Moreover, apart from attachments at the nerve head and the ora serrata, the two layers are never attached, between the two the potential space of the optic vesicle is bridged across only by the processes of the pigment cells. The retina, therefore is never, strictly speaking, detached, but its inner layer becomes displaced from the outer, allowing the potential space of the primary optic vesicle to become an actual one; (Fig 2) it would therefore be more accurate to speak of separation of the retina. To change a term, however, so convenient and which has gained the rights of possession by long usage, would be mere pedantry, and therefore also the term "sub-retinal space" and "sub-retinal fluid" will be employed where "inter-retinal" would be the more correct appellation.

Detachment of retina in its wider meaning includes any lifting up of the retina by a pathologic condition underneath it such as tumour, blood, pus or serum. In its narrower meaning and in a clinical sense it stands for an independent primary disease of the retina which is characterised by certain degenerative changes in it by the formation of tears and holes in the retina and by the accumulation of fluid behind it.

Retinal Detachment and its Modern Treatment

So retinal detachment is not a pathological entity, but rather an anatomical accident which may result from many pathological processes. According as to whether these underlying causes are obvious or obscure, it has been the custom to classify detachments as (1) secondary, or as (2) idiopathic (spontaneous). Such a classification has the merit of differentiating between the two completely different view points from which a detachment is considered from the clinical aspect. In the first case the detachment is merely an incident in a clinical picture dominated by a neoplasm, gross inflammation, or some other pathological feature of primary significance, and the clinical importance of the detachment is frequently the fact that its presence may obscure the diagnosis; in the second, the detachment itself is the essential catastrophe, the causal lesion often being so slight as to elude detection and the presence of a retinal detachment becomes the essential clinical feature. Our knowledge of the aetiology of retinal detachment is still by no means perfect; it seems probable, however, that the difference between the two classes may be of degree rather than of kind and that all detachments are secondary, the so-called idiopathic cases being dependent on inflammatory or degenerative changes so slight as to be recognised only with difficulty or sometimes not at all; it is the latter type of retinal detachment alone that will be dealt with in this book, while secondary detachments will only be discussed somewhat briefly.

It is befitting before we proceed further to look back to the earliest recognition of detachment of the retina in the history of ophthalmology. The important books in morbid anatomy by Morgagni (1740) and De Karzowitz (1781) don't mention it. Earlier writers have mentioned animal's eyes in which it occurred and the possibility of its occurrence in man. It is mentioned by St. Yves (1722) but we find that he

Introduction and History

confused it with "Mouches Volantes" so it was not until much later that reliable histological observations were made by Ware (1805) and Wardrop (1818) and they referred to the condition as "Hydrops Subchoroidalis" to distinguish it from detachment of the choroid "Hydrops Subsclerotalis" which was in these days considered to be much more common.

It was not seen in situ until expert observers like Chelius (1839) J. Sichel (1841) and Desmarres (1847) saw through the dilated pupil a white and at times a vascularised membrane. It was this appearance that Beer (1817) originally referred to as "Amaurotic Cat's eye". Invention of Ophthalmoscope was necessary before it could be studied clinically or any idea gained of its frequency. The early descriptions given by Coccius, Van Tricht, Arlt (1853) and A. Von-Graefe (1855) were fairly accurate and it appears that Von-Graefe fully proved the truth of the statements made by him when first seeing the fundus with an ophthalmoscope "Helmholtz has unfolded to us a new world". Because of their onset Von-Graefe considered most detachments of the retina due to subretinal haemorrhage. Stellwag (1856) as a result of anatomical investigation was able to disprove this idea. Arlt (1853) originated the theory of choroidal effusion as the causative factor and 5 years later Muller showed that the organisation and contraction of fibrous tissue in the vitreous was the cause in certain eyes. When Magnus (1883) was studying the various causes of complete blindness, he considered that 0.7% were due to retinal detachment.

The whole story is occupied by the records of a long series of hypotheses to account for the occurrence of the so called Idiopathic type of detachment and of as long a series of attempts to bring about its reposition, all of which proved

Retinal Detachment and Its Modern Treatment

abortive until comparatively recent years, when after much patient research, Jules Gonin of Lausanne (1924-28) emphasized the importance of retinal tears in the aetiology of the condition and the possibility of its cure by their closure. However the first year's of the results published by Gonin were received with skepticism, until 1929 at the International Congress of Ophthalmology in Amsterdam. His principal followers Amsler, Arruga, Weve and Lindner later on universally adopted this and improved his method. The service which he thus rendered to ophthalmology should not be assessed alone in terms of the therapeutic revolution which he inaugurated but also in the light of the stimulus which his work gave to the intensive and productive research into the aetiology pathology and the treatment of this condition which has appeared with in last three to four decades and is still continuing.

In some of the old cases with vitreous changes etc. shortening of eye-ball by complete or perforating method (Mueller, Lindner) Lamellar method (Shapland & Paufigue) Reefing (Weve) Scleral buckling (Custodis & Schepens) or Scleral outfolding (Everett & Casterviejo) and vitreous implantation (Schaffer) have changed the gloomy out look. Recently Photo-coagulation method of Mayer Schwickerath used either alone or in combination with diathermy and scleral resection have opened up a new therapeutic era in Ophthalmology. In Lancet 1929, it was mentioned that "there are few Ophthalmic surgeons who can't count on the fingers of one hand the cases of cures of retinal detachment within their own present experience." But now to-day 70-90% of these cases are curable and I think with all these improvements and in this age of satellites and space travels-a-day will not be farther off when the results may be as good as that of cataract extraction.

Introduction and History

Before ending this chapter I will like to mention some historical facts which Prof. Vogt placed before the International Ophthalmic Congress held in Madrid in April 1953.

Who was the first to see a retinal tear?

Coccius in (1858). Next was Von-graefe who regarded the tear as part of healing process. Gritel Hasen in 1871 and Schweigger in 1875 advanced the opposite view.

Who recognised for the first time that the tear was the cause for detachment?

De Wecker of Paris in 1870. Then Leber in 1882 popularised the mode of its development. According to him every actual detachment, which can be recognised with the ophthalmoscope is caused by a retinal tear.

Who was the first to select the region of retinal tear and the place of beginning of the detachment as the site for operation?

Schoeler of Berlin in 1889. He injected Tr. of Iodine between the detached vitreous and the retina in the neighbourhood of the tear and in the place of beginning of the detachment, in other words in the pre-retinal space.

Martin, de Wecker (1881) used Ignipuncture, but Galezowski of Paris (1902) used this to close a retinal tear in the operative treatment of retinal detachments.

PRACTITIONERS' FORUM

Dear Doctor,

If you have some specific problems relating to some of the cases you have in hand, kindly send us your questions giving the necessary details.

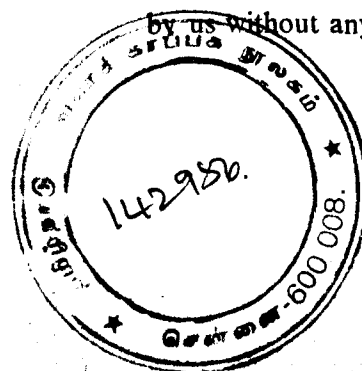
The questions will be answered by eminent Specialists on the subject. The question and answer will be published in the columns of 'Mediscope'.

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Very truly Yours,
DR. B. RAMA RAU, B.Sc., M.B.B.S.
Editor

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