SUBSCRIPTIONS

Yearly Rs. 10/-

H. Yearly Rs. 5/-

July

JUNE 1959

VOLUME II

NUMBER IV

Price Re. 1

MEDISCOPE

JOURNAL OF MEDICINE & SURGERY

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VOLUME 2 JULY '59 NUMBER IV

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BOOK SECTION

MEDISCOPE
Journal of Medicine & Surgery

VOL. II JULY 1959 No. IV

Modern Treatment of Leukaemia

By

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Treatment of Leukaemia has assumed great importance in view of its greater incidence in recent years and it is going to be a problem of greatest magnitude next, perhaps, to Carcinoma. It is admitted that the atomic explosions with their radiations are a fruitful cause of Leukaemia.

In the U.S.A. a special department has been established in the New York Memorial Centre for examination, study and treatment, of patients suffering from Leukaemia Hodgekin's disease and lymphosarcoma. This department is known as 'LYMPHOMA SERVICE'. Each case is discussed in a weekly meeting, and a line of treatment decided upon.

The chief development in this group of diseases has been the growing interest in chemotherapy and as such it is worth giving a review of the various groups of chemotherapeutics now in use.

1. *Alkylating substances.* Under this comes HN_2 (methyl-bis (B-chloro ethyl) amine. This is given intravenously in a dose of 0.4 mg. per kilo of body weight in single or divided doses. Its administration may be repeated after one month's interval.

Recently some other nitrogen mustard compounds have been prepared that are suitable for oral administration. The best known of these are "R48", B-naphthyl-bis-(B-chlorethyl) amine, and "R151", B-naphthyl-bis (B-chloropropyl) amine. They appear to be less effective than HN_2. But their use particularly "R151" along with urethane has been found to be encouraging. They have a synergistic effect when used together. This combination product is produced by Boots Pure Drug Co. in capsule form containing 25 mg. of R-151 and 0.5 g. urethane and the dose is 2-4 capsules a day.

2. TEM (Triethylenemelamine). This is given orally in a dose of 1.5-5 mgm-daily two or three times a week for one month or longer; the total dose being approximately 20-40 mg.

3. TEPA (Triethylenephosphonamide) This is administered intramuscularly in a dose of 0.2 mg, per kilo of body weight daily. The total dose is 1.5—2.5 mg/kg.

4. MYLERAN (GT-41). (1:4 dimethane sulphono - xybutane) - administered orally, the dose being 2.6 mg. daily. The total dose administered may be between 150-200 mg.

These substances act by inhibiting the proliferation of neoplastic cells and rapidly reproducing normal blood cells by interfering with the synthesis of nucleo-proteins. In some way they act similarly to irradiations. All these agents have a toxic action on the bone marrow and may cause anaemia, leucopaenia and thrombocytopaenia, which usually appear two or three weeks after the end of treatment. The dose indicated against each is an approximate one and has no absolute value, as the treatment should be continued until a positive result is obtained or given up for toxic manifestations.

B. Antimetabolites: This group includes:—

1. *Folic acid antegonists.* To this group belong AMINOPTERIN (4-aminopteroyl glutamic acid) given orally in 0.25 to 1 mg. dose daily for three to four weeks; and METHOPTERIN (4 : amino - N_{10}-methyl-pteroylglutamic acid) which is also given orally in 2.5 - 5 mg. dose daily for the same period.

Folic acid antagonists act by blocking the transformation of folic into folinic acid; during the synthesis of purines and pyrimidines, thus inhibiting cellular growth and multiplication. Toxic effects are anorexia, ulceration of the oral mucosa, diarrhoea and alteration of the bone marrow with megaloblastosis.

2. *Purine antagonists.* The principal compound in this group is 6-MERCAPTOPURINE or 6-MP which is administered orally in 1.5-2.5 mg. per kilo, doses daily for 3-8 weeks. This act by inhibiting the synthesis of nucleic acid, by interfering in the nucleotide nucleoside nucleic acid synthesis. Toxic effects of the compound is generally rare but in very high doses may produce anaemia leucopaenia and tyrombocytopaenia.

3. *Amino acid like substances, or amino acid antagonists*, the best known compound being *Azaserine*, which is given orally or intravenously in a dose of 2.5-10 mg./kg. daily for 1-5 weeks. This group of compounds act by presumably inhibiting the utilization of formates and glycine in purin synthesis. Nausea, vomiting, diarrhoea and rarely slight leucopaenia are the commonest toxic effects that may be produced by their administration.

4. *Antimitotics.* This group includes numerous therapeutic agents including urethane, Fowler's solution, Colcemid (desacetyl methylcolchicine) a derivative of colchicine. These inhibit cellular growth and reproduction by their toxic action. Prolonged administration of these drugs produces gastro - intestinal disturbances, alteration of the bone marrow, with anaemia, leukopaenia and thrombocytopaenia.

C. Steroid Hormone. This includes:

1. *Cortisone*:—This is administered orally or intramuscularly and the dose is 100 400 mg: daily.

2. *Hydrocortisone*:—This is given either orally or intravenously in a dose of 50-200 mg. a day.

3. *Prednison*—is given orally in a dose of 50-300 mgm, daily in divided dosage.

4. *Prednisolone*—The dose is the same as Prednisone. Usually the dose of prednisone and prednisolone is approximately one quarter or one-fifth that of Cortisone. But sometimes it is given in bigger doses.

5. ACTH. given either intramuscularly or intravenously in a dose of 60-200 mg. and 25-50 mg. respectively daily.

ACUTE LEUKAEMIA

Until lately the treatment of acute leukaemia has been disappointing particularly in children. Recently, however, the use of chemotherapy has been found to be effective in producing more or less long standing remission in a large percentage of young patients. Antimetabolites and steroid therapy have been the basis of such treatment. The steroid hormones produce rapid but short remissions, and antimetabolites act slowly over a period of 3–8 weeks before its beneficial effect is apparent but it gives longer and more complete remissions.

Whatever may be the condition of the patient and the acuteness of the symptoms; with high fever, and haemorrhagic diathesis, it is advisable to start the treatment with steroids wither Cortisone, ACTH or Prednisone or Prednisolone. Cortisone is given in a dosage of 100-200 mgm. daily; ACTH 25 mgs daily intramuscularly; and Prednisone or Prednisolone is given orally in 100 mg. dose daily. Statistics show marked improvement and remission of acute symptoms under this treatment in about 50-70% of young patients.

Once the acute stage has been controlled with steroid therapy the treatment should be continued with antimetabolites; and in this methopterin and 6-mercaptopurine (6-MP) have particularly been recommended.

If the patient does not exhibit any acute symptoms, the treatment may at once be started with anti-metabolites and the choice of the particular drug depends on the leucocytic count. If it is high, preference is given to 6-MP, and, if low, to methopterin. The treatment may have to be repeated.

Steroid followed by antimetabolites are the treatment of choice in any age group if the symptoms are acute. But steroids lose their effectiveness in older age group and this seems to be proportional to the age i.e., higher the age group the less effective it is.

Of the antimetabolites 6-MP is the therapy of choice and gives better results than methopterin.

This combined treatment with steroids and antimetabolites is much more effective in children than in adults: and although over 50% of infant patients benefit from the above treatment, improvement of the haematological and clinical picture lasting several months is seen only in about 25% of young persons and 10-15% in adults.

If the patient is resistant to above treatment folic acid antagonist may be useful.

Radiation therapy is not used in acute stage although exposure of the spleen to low doses of radiation say 25-50 r. according to the patient's age may lead to temporary improvement in a few cases. The daily examination of blood is important.

Blood transfusion may be required to counteract bleeding.

CHRONIC LEUKAEMIA.

Chronic myeloid leukaemia has a much better prognosis under modern treatment. It can be treated by radiology or by chemotherapy. Both give excellent results so far as clinical picture is concerned. Both produce fall in the circulating white cells. reduction of the immature cells, increase in haemoglobin percentage, reduction in the size of Liver and the Spleen and improvement in the general well-being. These improvements are more dramatic in the initial stage of the disease. But in spite of theencouraging results as enumerated above, the survival rate does not seem to be influenced by it and the survival period after diagnosis still remains between 3 and 4 years.

Radiological treatment is the most commonly used therapy in chronic myeloid leukaemia. This may be given in small doses as a "spray" over the entire body or repeatedly over the spleen. administered by a competent radiologist.

X-radiation aims at the destruction of the proliferating cells, and thereby to reduce the size of the enlarged, unfiltrated organs (Spleen and Liver). Destruction of abnormal cells in the bone marrow allows normal production of red cells and thus cure anaemia. The X-ray course is repeated when symptoms recur or the leucocyte count goes above 50,000. The nausea and malaise associated with X-radiation may be minimized by administration of Pyridoxine immediately before each exposure. Radiotherapy is contra-indicated when leucopaenia or severe thrombocytopaenia is present.

Radio-active phosphorus has been used successfully in chronic myeloid leukaemia. Doses of approximately 1/10 mc. per kg. are administered orally or by intravenous injection. It has been demonstrated that cells with accelerated metabolism, such as leukaemic cells, draw the greater part of the radioactive element administered.

The most commonly used chemotherapeutics are :—

1. Arsenic in the form of Fowler's solution in gradually increasing dosage from 5 to 20 minims daily.

2. Urethane administered orally in a dose of 3 gms. daily for approximately one month.

3. TEM (Triethylenemelamine). the dose being 2.5 to 5 mg. twice weekly until remission of the symptoms. After this a lower maintenance dose should be continued.

4. Myleran (GT-41), (1, 4 dimethylsulfonoxybutane) starting with a dose of 4 to 6 mg. daily; and reduced subsequently until a low maintenance dose is finally fixed.

5. 6-MP (6, mercaptopurine) administered orally in a dose of 2·5 mg./kg. body weight.

6. Colcemid (desacetylmethylcolchicine) can be given orally in a dose of 3 mg. daily, raising it gradually after three or four days to 7-10 mg. depending on the behaviour of the leucocytic count. The dose is maintained until the leucocyte count has fallen to 25,000 per c.m.m., when the drug is withheld for three or four days after which a maintenance dose of 3-5 mg. dailr is given.

HN_2 (Methyl-bis (B-chloroethyl)-amine, is considered as the last resort in cases re istant to all the treatment enumerated above.

The administration of Cortisone and better still Predenisone may be useful in those cases showing particular tendency to haemorrhage.

Most workers preferred these newer drugs in the treatment of chronic myeloid leukaemia but Kiebl has used mustine (Nitrogen Mustard) and Urethane in combination, believing them to be synergistic. His method has been to give 1.25 mg. of the first and 1.5 g. of the second as a single intravenous injection daily until the leucocyte count has fallen to 20,000 per c.m.m. According to him six or seven patients treated responded by satisfactory and complete remissions.

LYMPHATIC LEUKAEMIA

Although there are a multiplicity of drugs which will induce remission in chronic myeloid leukaemia, the position in chronic lymphatic leukaemia is far less satisfactory.

The treatment of chronic lymphatic leukaemia varies according to the state of the disease. In many slowly progressing cases, it is preferred to wait and to use the known therapy only at a later stage, and such therapy are not so very satisfactory.

Radiological treatment of the lymph nodes involved is the treatment of choice and the proper dose and frequency of exposure has to be worked out by experienced radiologists. Radio-active Phosphorus can also be used as in myeloid leukaemia.

It has been found that most of the usual chemotherapeutic agents have very little effect on chronic lymphatic leukaemia.

M. M. Wintrobe and his colleagues have found TEM (Triethylene melamin) the most effective, but most other workes in this field would agree that it leaves much to be desired. They point out the extreme sensitiveness to this drug of many patients with chronic lymphatic leukaemia and advocate an initial dose of 2.5 mg taken fasting and followed by an alkaline draught. Subsequent dosage depends on the patient's reaction. Alternatively it can be given in 1 to 1.5 mg. dosage twice a week, for long periods. always controlled by periodical examinations of the blood. until its toxic action, which is mainly manifested by anaemia and thrombocytopaenia, calls for an interruption of the treatment. In this case, the steroid hormones may be useful, as administration may be protracted, and does not give rise to rapid resistance : as in acute forms.

Galton and others reported satisfactory result with nitrogen Mustard. The average daily dose used by them was 0.1 to 0.2 mg. per kg. body weight by mouth; treatment was continued for three to six weeks Occasionally the drug had to be withhel. on account of neutropaenia or thrombocyd topaenia ; other toxic effects were rare and limited to dyspepsia.

The synergistic action of urethane and nitrogen mustard was first noted by Skipper. It was found by E. Keibl to be effective in chronic myeloid leukaemia as mentioned above and it appears too to be of value in myelomatosis. Inus and Rider claim benefit in patients with the disease given a combination of urethane and B naphthyl di (2-chloropropyl)-amine (R-1 1). This is prescribed in a capsule containing 25 mg. of R-151 and 0.5 gms of urethane; the dose being 2-4 capsules daily. Improvement was usually noted within three weeks of starting treatment.

The results are conflicting and in any case uncertain.

Great advences have been made recently in the treatment of leuka mias, but the progress still leaves much to be desired.

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Recent Advances in Treatment (C.N.S.)

By

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From a talk delivered at a symposium on Recent Advances in Treatment under the auspices of the Indian Medical Association, March 20th, 1959.

INTRODUCTION

The advent of a host of new pharmacological preparations, together with a better understanding of physiological mechanisms regulating normal nervous function and metabolism, have made it possible to treat various neurological diseases, formerly allowed to run their natural course.

In this short review, only a few of these will be considered.

CEREBROVASCULAR ACCIDENTS

Much has been learnt about the cerebral circulation in health and disease by the pioneer work of Kety ('949). This knowledge has allowed a more rational approach to the management of cerebrovascular catastrophes.

Thus the treatment of cerebral infarction resulting from cerebral thrombosis and embolism has been modified by the introduction of stellate ganglion block, the use of vasodilators and anticoagulants. Early surgical inter-ference in selected cases of cerebral haemorrhage has met with a certain degree of success.

1. Stellate Ganglion Block

Sympathetic control of the cerebral vasculature is a feeble one and blocking the sympathetics does not cause much dilatation of the blood vessels, as shown by cerebral blood flow studies (Schenkin, et al, 1951).

First used in 1925 by Leriche and Fontaine in France, on a case of traumatic hemiplegia, it has enjoyed a certain amount of worldwide popularity. If performed at all, it should be within the first 72 hours of the cerebral infarction. It is successful only in a certain proportion of cases, and not entirely free from the risk of sudden death.

2. Vasodilators

The nitrates increase the cerebral blood flow and the intracranial pressure (Murphy, 1954). Papaverine produces a diminution in the cerebrovascular resistance and hence improves the cerebral blood flow (Jayne et al, 1952). Nicotinic acid, though it does not increase the total cerebral blood flow, (Scheinberg and Jayne. 1952), produces a multiplication of the capillaries and enlargement of the arterioles in the brain of the monkey (Huertas and Forster, 1954). Histamine lowers the cerebrovascular resistance, but the cerebral blood flow is increased only when the mean arterial blood pressure is not decreased more than 12% (Shenkin, 1951). The sympatheticolytic drugs, like Priscol and Hydrazinophthalazine, have exactly the same effect as histamine on the cerebral blood flow (Murphy, 1954).

Aminophylline and caffeine, however, which increase the cerebrovascular resistance and decrease the cerebral blood flow, (Wechsler et al, 1950), should never be used in the management of strokes. This fact is not widely appreciated.

Carbon dioxide is the most potent and physiological of vasodilators and produces the most convincing increase in cerebral blood flow (Murphy, 1954). Inhalation of 5% carbon dioxide, for 5 minutes every hour, during the patient's waking period, is now extensively used in the treatment of strokes.

3. Anticoagulants

Once infarction has definitely sets in, great caution is required in the use of anticoagulant therapy. The main indications for the use of anticoagulant drugs are the following:—

(a) Basilar insufficieny, as suggested by vertigo, blurred vision and attacks of alternating hemiplegia

(b) Carotid insufficiency, which may be diagnosed by an arteriogram

(c) Repeated transient hemiplegic episodes, sometimes as many as 10 to 15 a day, the result of caroticovartebral insufficiency and a fluctuating blood pressure

(d) Embolisation from mitral stenosis or myocardial infarction.

All these constitute medical emergencies and should be promptly treated with Heparin, 5,000 units 6 hourly for the first 24 hours, combined with Tronexan or Dindevan. Later the coumarin preparations may be given alone, in doses sufficient to keep the blood prothrombin at about 40%.

PARKINSONISM

Advances in the treatment of this crippling affliction include the introduction of new drugs and the employment of surgical procedures aimed at interrupting the afferent and efferent fibres of the extra-pyramidal system.

1. Drugs

The solanaceous drugs are the oldest stand by in the treatment of Parkinsonism. Hyoscine hydrobromide and mixtures containing various proportions of the tinctures of hyoscyamus, belladonna and stramonium are still widely employed alone or in combination with the newer synthetic drugs.

Of the more recent synthetic preparations now in general use, trihexyphenydyl (Artane), benztropine sulphonate (Cogentin), procyclidin (Kemadrin) are the most useful. Combinations of these drugs, starting with small doses several times a day, appear to be of more value than the use of a single drug alone.

The antihistamines, diphenhydramine (Benadryl) and phenindiamine (Thephorin) are the most popular in this group of drugs. The one disadvantage of antihistaminic preparations is their tendency to produce drowsiness.

The above 3 groups of specific drugs all have more or less effect on the rigidity of Parkinsonism. As, however, these patients are disinclined to exert themselves, mental energisers, such as Ritalin and Dexedrine are used in varying combinations with the Meprobromates to alter the mood of the patient.

2. Surgical Procedures

The drug therapy of Parkinsonism has been very disappointing in the control of tremor. Moreover their continuous use is dependent on the co-operation of the patient and is fraught with side effects, drowsiness, giddiness, etc. For these reasons surgical procedures have been attempted form any years.

The 2 most popular techniques at the present time are the destruction of the globus pallidus by injection of alcohol (chemopallidectomy) or of the anterior limb of the internal capsule by electro-coagulation or open operation. Both procedures result in a transient paralysis accompanied by drowsiness and confusion for periods up to a week postoperatively. However, power later returns but tremor is permanently cost.

3. Rehabilitation Techniques

It is important for the doctor to shake the patient out of his mood of apathy and make him exercise regularly under the supervision of a trained physiotherapist.

Alternating movements, rhythmic movements and breathing exercises are simple procedures, which the patient can carry out at home.

Drugs by themselves are completely ineffective without physiotherapy.

METABOLIC DISORDERS

Only a few of the metabolic disturbances of the central nervous system are so far amenable to treatment, and this has become possible because of a better understanding of the physiological processes involved.

A) NEUROLOGICAL DISORDERS IN LIVER DISEASE

1. Hepatic Encephalopathy

Patients suffering from cirrhosis of the liver or from other more acute forms of liver failure, viz. infective hepatitis, or as a result of portocaval shunt operations, often show varying degrees of confusion, drowsiness or stupor, together with tremors and muscular rigidity, a syndrome which has come to be regarded as indicative of hepatic encephalopathy.

The general principles of treatment of hepatic coma are:—

(i) a low protein intake

(ii) the use of antibiotics, which are employed to cut down the intestinal becterial action and hence the liberation of nitrogenous products into the portal system and also to prevent bacterial invasion of and further damage to the liver

(iii) vitamin B complex, intravenously or intramuscularly

iv) correction of water and electrolyte disturbances

(v) adequate carbohydrate and fat intake

The above, (ii) to (v) may all be given as an intravenous drip, as advocated by Latner (1952), consisting of 10% dextrose in saline, to which is added thiamine, 50 mg., nicotinamide 150 mg., riboflavin 50 mg., and potassium chloride 0.5 gm. Latner (1952) also gives penicillin, 1,000,000 units and alpha-tocopherol 300 mg. intramuscularly daily.

(vi) glutamic acid, which was introduced by Walshe (1953) is supposed to mop up the surplus ammonia circulating in the blood in hepatic coma. It has, however, not proved to be entirely successfully in relieving hepatic coma.

2. Wilson's Disease

This hereditary and familial disorder of the nervous system, is characterised by a progressive dementia, hyperkinesis and rigidity, together with a peculiar pigmentation of the cornea. The essential biochemical defect is a lack of copper binding coeruloplasmin in the blood, which allows large amounts of copper to pass out in the urine and to be fixed in the tissues, including the central nervous system.

The treatment of this condition involves:—

(i) Diminishing copper absorption from the gut by giving Potassium Sulphide, 200 mgm three times a day by mouth.

(ii) Increasing copper excretion by means of dimercaprol (BAL) one ampoule twice a day for 7 days, every 3 months. Cummings gives 300 to 400 mg. of BAL once a week throughout the year.

(iii) Penicillamine (dimethylcystine) 300 mg. 5 times a day, two days in a week, considerably increases the urinary excretion of copper.

3. Phenylpyruvic Oligophrenia

First described by Folling (1934), this inborn inability to metabolise phenylalanine is due to the absence of the enzyme phenylalanine-hydroxylase in the liver cells. The phenylalanine, which cannot be metabolised in these patients, is excreted in the urine as phenylpyruvic acid, which can be detected by the addition of a freshly prepared 5% ferric chloride solution, which turns the urine a bluish-green colour.

As this condition is responsible for many cases of mental defect, it is worthwhile trying to prevent it by putting the child on a phenylalanine-free diet within two or three weeks of birth. Details of this diet have been worked out by Woolf (1958).

It may be as well to test the urine of all children at birth for phenylpyruvic acid. as this is the only way this disease can be diagnosed.

ACTH & CORTISONE IN NEUROLOGICAL DISORDERS

Steroid therapy indicated in two main types of neurological disease, primary affections of the central nervous system and the neurological complications of systemic disease.

Primary affections of the central nervous system

The injection of vaccines, sera or chemical substances, and ill-defined upper respiratory infections are often followed by an explosive allergic reaction in the central nervous system. Such a reaction may present as an encephalitis (acute haemorrhagic leucoencephalopathy), an acute disseminated encephalomyelitis, or a myelitis or a nerve root and peripheral nerve affection. The syndrome of Guillain-Barre, encephalo-myelo-radiculo-neuritis, is a good example of such a response.

In this whole group of disorders, cortisone has been used with varying degrees of success. It has to be given early and in large doses.

The post-exanthematous encephalo-myelitides are also a variant of the above group.

Chronic recurrent neuropathies, have been shown to remit with cortisone and relapse on reducing the dose of the drug, a certain maintenance dose being required ndefinitely. (Austin, 1958).

Cortisone has been used in various infeitions of the central nervous system. It is of value in adrenal failure associated with meningococcal septicaemia. In tuberculous meningitis it has been used systemically as well as intrathecally, in addition to the routine anti-tubercular line of treatment. The toxaemia of tetanus in certain fulminant cases has also been reduced by cortisone.

A few neurological diseases have been doubtfully influenced by cortisone. The patient with myasthenia gravis is often worse during treatment with ACTH, but in about 50% of the cases, a remission occur son cessation of the therapy. In disseminated sclerosis, cortisone is of no value. More recently the steroids have been tried in the early and late management of cerebrovascular accidents, in the early stages to localise the infarct and reduce oedema, and later on to improve the morale and facilitate rehabilitation,

Central nervous system involvement in systemic disease

In the collagen disorders, e.g. polymyositis, dermatomyositis, polyarteritis nodosa and lupus erythematosus, which involve the central nervous system, cortisone is the only treatment of any value.

Sarcoidosis is well-known to extend to the central nervous system. Cortisone has been found to be much more effective than the hitherto advocated treatment of this condition by calciferol,

The reticulosis, Hodgkin's disease & lymphatic leukaemia, which often cause deposits in the central nervous system, are ameliorated to some extent by cortisone therapy.

CONCLUSION

Only some of the diseases of the central nervous system to which newer therapies are applicable, have been considered here. It will be seen, how ever that more exactdiagnosis and biochemical understanding have opened new vistas and that perhaps we are on the threshold of an era when more and more neurological disorders will become amenable to effective treatment.

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Pediatric Dermatology

By

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Introduction:

The clinical manifestations of skin disorders in children often has certain special manifestations. Hence a resume of the pediatric dermatology is since qua non. It is a useful subject not only from the view point of the dermatologist but also from that of the pediatrician.

Care of the Children's Skin

New Born:

A very important problem is the care of the skin of the new born. The various steps are:

1. Removal of the excess of blood and vernix shortly after birth.
2. Restriction of the bathing to the minimum during hospital stay.
3. Cleaning the napkin area with cotton soaked in light mineral oil.
4. Dusting the intertriginous areas with non irritating powder as Talc.

Bacterial Infections of the skin in Children

I. Primary:

1. Impetigo: (a) Contagiosa
   (b) of New born
   (c) Dermatitis Exfoliativa
   (d) Furfuraceous
   (e) Bullous.
2. Superficial Follicullitis and ecthyma.
3. Furunculosis: carbuncle and Erysipelas.

II. Secondary Superimposed on:

1. Skin disease: Seborrhoea, psoriasis, Neurodermatitis.
2. Allergy: Infantile eczema.
3. Parasites: Pediculi, scabies, Ring worms.
4. Infection viral: Herpes simplex varicella and others.

1. Impetigo Contagiosa:

This is a streptococeal infection with vesicles which become pustular and crust formation takes place. The crusts contain an staphylococci. The individual lesions vary in size and various types of lesions may be noticed as erythema, macule, vesicle, pustule and crust formation.

2. Impetigo of the Now born:

This is a bullous impetigo of the newborn occurring about the 4th day of life and is a severe form of impetigo. The lesions rupture and form crusts. New bullae form and lesions spread. Severe constitutional symptoms may occur.

3. Dermatitis exfoliativa or Reiter's disease:

This is an exfoliative form or condition occurring at first on the face and spreads to the body. There is a tendency to form vesicles and bullae. In the bulla the epidermis may fleck off easily. The mucous membranes may be involved and severe constitutionel sympoms may occur. The disease is caused by strepto and staphylococi.

4. Follicullitis:

This is a staphylococcal infection of hair follicles and may involve scalp or body.

5. Furfuraceous impetigo:

The condition may be mistaken for Fungus skin. The lesions may be round or oval or irregularly shaped.

6. Ecthyma:

Streptococcal or staphylococcal infection may be responsible. A vesicle or

crust and later ulcer formation are the features.

7. Furuncle and boil:

This is an infection of the pilosebaceous follicle. Recurrent furuncles in children may be due to agammaglobulinemia.

8. Erysipelas:

This streptococcal infection produces severe constitutional symptoms with raised crimson red lesion and a well defined border.

Secondary bacterial infection may occur over primary disease skin.

Principles of treatment

1. Removal of crusts by:
   (a) Starch boric poultice.
   (b) 2% Soda sulphate
   (c) 2% Sodabicarb
   (d) Warm olive oil
2. Antibiotic ointment externally
3. Chemo therapeautics and antibiotics internally.

A combination of antibiotic and antinflammatory compound as prednisolone is useful externally in pyogenic dermatitis.

The various combinations found useful are:

(a) Prednisolone-Chloramphenicol ointment.

(b) Hydrocortisone and Neomycin

(c) A Combination of hydrocortisone, neomycin and polymyxin.

For systemic administration the following are found useful.

(a) Penicillin sulpha combination orally.
(b) Penicillin V orally.
(c) Penicillin Crystalline paranterally.
(d) Erythromycin orally.

Viral and Rickettsial disease:

Ural Diseases:

1. Warts.
2. Molluscum contagiosum
3. Herpes simplex
4. Herpes zoster
5. Vaccinia
6. Third disease.
7. Fourth disease
8. Erythema infectiosum (Fifth disease)
9. Roseola infantum (Sixth disease)

Rickettsial diseases

Viral diseases:

Warts or Verrucae:

Types: The common wart is called verruca Vulgaris and is seen on the fingers and hands. It varies in size from a pin's head to a pea. The colour is yellowish brown or brownish black.

2. *Filiform wart*: is threadlike pointed excrescence, pale pink in colour.

3. *Digitate warts*:—In this there are numerous finger like processes and the base is often constricted.

4. *Plantor wart*:—There is a warty growth on the plantar surface of foot and is often painful.

Treatment: There is no virucidal agent for warts. The treatment consists in destruction modality and psychotherapy.

The destructive measures are:

(a) Electro desiccation.
(b) Chemo surgery by the application of 10% Podophyllin in Tr. Benzoinco or 20% Formalin in Lanolin or Trichlor Acetic acid.

Psycho therapy: consits in the use of Tranquillisers.

Molluscum Contagiosum:

This is a viral infection of the skin producing a small sessile pedunculated umbilicated tumours. In molluscum contagiosum, squeezing with fingers tends to make the molluscum body prop out.

Treatment: consists in Broad spectrum Antibiotics and the simple shelling out of the molluscum body.

Herpes simplex: Is the most ubiquitious of all these viral diseases. The virus of

Herpes simplex produces various manifestations.

A. Skin:
   (i) Herpes simplex
   (ii) Eczema Herpeticum
   (iii) Tranmatic Herpes

B. Muco cutaneous junction:
   (i) Herpes Labialis
   (ii) Herpes Pigmentalis
   (iii) Vulvo vaginitis.

C. Mucous membrane:
   (i) Acute gingivo stomatitis.
   (ii) Recurrent stomatitis.

D. Central Nervous System:
   Meningo Encephatitis.

E. Conjunctivitis and Kerato conjunctivitis.

F. Generalised infection.

The virus produces multiloculated vesicles deep in epidermis and in the edge of the vesicles ballooning degeneration of the epidermal cells can be seen. The intranuclear inclusion bodies are known as Lipschutz bodies.

Treatment: (a) Broad spectrum Antibiotics internally,

(b) Zephiran (1 in 1,000) externally.

Herpeszoster Varicella

The virus producing the above two diseases are the same. Varicella may occur in contacts with zoster and vice versa. Zoster may occur primarily spontaneously or may be associated with same traumatic episode or triggered by such agents as leukemia and tumour and so forth. Zoster may occur in relation to spinal nerves or cranial nerves as V and VII; the latter producing zoster opthalmicus and Ramsay Hunt's Syndrome. The treatment consists in externally applying Calamine lotion and internally broad spectrum antibiotics. It has been found that postherpetic neuralgia can be prevented by the injection of posterior pituitary extract.

Vaccinia: In the course of vaccination against variola, elevation of temperature, malaise and generalised vaccinal eruption may occur. Dr. Kaposi has described a condition of disseminated herpes or Kaposi varicelliform dermatitis. This may occur due to contact with either vaccination or contact with either vaccinia or Herpes simplex.

Third disease: An infectious disease occurring in boys has been described by Dr. Gulhrie. This is characterised by fever lymphadenitis, conjuctivitis and a macular eruption on the trunk.

Duke's disease: This a mild contagious disease characterised by pharyngitis, lymphadenopathy and generalised erythematous eruption.

Erythema infectiosum or Fifth disease: This disease is characterised by endemicity and afebrile course, erythema and oedema occuring on the face in a butterfly pattern. The eruption lasts for 7 to 9 days. The above three conditions have selflimited course and the treatment is purely symptomatic.

Rickettsialpox: This condition is caused by Rickettsia Akhaelian and transmitted to human beings to the rodent mite. This is more common in the United States.

The features are 1. Primary lesion. 2. Lymphadenitis 3. A maculopapular eruption.

The disease responds to broad spectrum antibiotics.

Fungal infections of skin.

Tinea capitis or Ringworm of the scalp may be due to Microsporon or Trichophyton. The two important microsporon genera causing ringworm of the scalp are *(a)* Microspora audouni which is anthrophilic.

(b) Microsporon canis which is zoophilic.

Trichophyton also produces ringworm of the scalp.

CLINICAL TYPES OF RINGWORM OF SCALP

Type A.

Grey patch ringworm. There are several small areas of infection in which the hair is dull and broken off. There is no inflammatory response.

The Second type B.

There are several small spots and the borders show evidence of inflammation.

The Third Type C.

Is caused by virulent Fungus infection. The inflammatory reaction is severe. The lesion is indurated inflamed. The lesion is single and is a boggy swelling resembling an abscess. It is called kerion celsi. The fourth type is called Black dot ringworm and is caused by a Faviform fungus.

Diagnosis of the ringworm of the scalp is made by:

(a) Demonstration of the ringworm.

(b) The infected hairs when examined by U. V. R. passed through a wood filter will show a feeble greenish flourscence.

Therapy in Tinea capitis:

There are two main approaches in treatment:

(a) Fungicidal ointment combined with manual epilation of infected hairs.

(b) X-ray epilation of hairs.

The hairs of the scalp should be clipped short and the infected hairs be epilated One of the following Antifungal agents can be used:—

1. 20% Salicylic acid Ointment.
2. 3% Hydr. ammoniato ointment.
3. Undecylenic ointment,
4. Asterol (a benzyl Thiozol compound)
5. 2% Iodine in Benzol
6. Salicylicamide ointment.

The use of epilation by oral Thallium Acetate has been given up due to undesirable side effects When there is inflammation, treatment should be directed against secondary infection by a course of Broadspectrum antibiotics, before instituting the above treatment.

FAVUS

This is a chronic Fungus infection usually involving the scalp and occasionally the glabrous skin and nails. It is characterised by yellowish cup shaped crusts or scutula and a peculiar mousy odour. The disease may be confused with seborrhoeic Dermatitis, Psoriasis, Impetigo and Folliculitis Decalvans.

Treatment: The crusts are removed by warm olive oil and Fungicidal ointment are used.

Tinea circinata: Ringworms of the glabrous skin. The disease is characterised by a growing edge showing redness, oedema scaly and fine vesiculation. The lesions may be round or oval. The condition responds to chrysarobin ointment or 1% Cignolin or Derobin.

Tinea Cruris: Is caused mainly by Epidermophytosis and rarely the microsporons. The lesions are usually found on the thighs and nataluleft. The disease responds to Whitfields ointment; Chrysarobin Ointment or 1% Cignolin or Derobin Ointment.

In the management of the above two conditions, viz Tinea circinata and Tinea cruris, care must be taken that while using Chrysarobin or Derobin or Cignolin the hands are to be washed and eyes not to be touched by soiled hands.

Fungus Infection—Nails: This usually is due to trichophyton rubrum and is one of the most difficult conditions to eradicate.

Treatment: Consists in scraping the nails and applying a highly concentrated Lithium Bromide and nail lacquer.

Fungus infection of hannds & Feet:

Tinea pedis or *Fungus Infection of Feet*: may show three clinical manifestations:

(1) Acute vesiculo bullous type.

(2) Chronic Intertriginous lesions producing white sodden appearances between toes.

(3) Chronic Hyper keratotic lesions.

Tinea Manuum or Fungus:

Infections of hands may be:—

1. Vesiculo pustular type.
2. Interdiginous type.
3. Hyperkeratotic and squamous type.

Treatment: During vesicular and vesiculpustular type the hands and feet are to be immersed in 1 in 10,000 warm potassium bath for 15 minutes and after drying to be painted with 0.5% Aluminium Acetate or 0.25% Silver nitras.

For the Interdiginous type the toes or fingers kept separate by a wick of gauze and the lesions are painted with 2% Gentian Violet in water or 2% Benzol During the Hyperkeratotic stage the following powder is used during daily and the ointment, during night.

Powder:	R/	Thymol	1	part
		Boric acid	10	,,
		Zinc oxide	20	,,
		Talc	69	,,

Make into dusting powder.

Paint: *Castallani's paint*:

R/ Saturated Alcoholic solution of Basic Fuschin	10	parts.
5% Aqueous phenol	100	parts
Boric acid	1	parts
Acetone	5	parts
Resorcivol	10	parts

Dispense in amber coloured bottle and to be kept away from light.

The other ointment used is 20% Salicylic acid and 20% Benzoic acid in Toilet Lanoline.

Moniliasis. (Thrush)

This infection is caused by Candida Albicans which is a yeast like fungus. The infants are usually affected with oral candidiasis There are multiple white adherent pseudomembranous and are to be distinguished from Milk, candy. The other sites are vagina and nailplates, scrotum and natal clefts.

An acute inflammatory lesion may happen die to candidiasis producing Mohitial granuloma.

Nystatin tablets are given in milk or syrup and Mycostatin ointments used externally. Vaginal tablets are useful for local application.

Infantile Eczyme:

This is a perplexing and exasperating condition. The eczematoid dermatoses commonly occur in the cheek, chin and forehead. The centre area of the face is spared. The lesions may be acute, subacute or chronic inflammation of the skin with intense pruri is. The lesions are symmetrical and during the acute phase exudation may occur. Secondary lesions may take place over the lesions since the exudation offer suitable terrain for bacterial growth. The lesions may become generalised. Exacerbations and remissions may occur.

During childhood the atopic dermatitis assumes or prurigo or lichenoid forms. During the prurigo type there are large succulent elevated reddish brown papules with scratched central top. The lichenoid type consists of small reddish flat topped red to brown papules while tend to become confluent.

Leiner's Disease: Is a generalised inflammation of the skin with desqumation of the epidermis. Diarrhoea is common in Leiner's disease and is often serious. Secondary infection is frequent. There is often Low serum protein and oedema.

Principles of Treatment: of Eczema in Children:

Before instituting the treatment a detailed history is essential.

1. Do the parents or any of the Siblings are having Asthma or Eczema or Hay fever?
2. How old was the child when eczema began?
3. Any Allergy to any food.
4. Does the child sleep on a feather pillow or is there a cat, bird or a dog near about?
5. Does the baby keep the mother awake all the night?

The next procedure is to note if the conditions are acute. acute exudative, infected. subacute or chronic.

Treatment in General

1. Patient should be kept in a nursing home or hospital if the condition is generalised.
2. Regulation of diet.

Cow's milk is likely to produce sensitivity and then is to be changed to Goat's milk or allergilac or Lactodex or Nutramigen. Milk contains 4 proteins Lactalbumin, casein Lactoglobulin or opalasin. The last exists only in traces and similarly Lactoglobulin sensitivity is more due to casein.

EGG: White of egg should be kept out of diet.

Wheat: Sensitivity is important in many children since it produces steatorrhoea and Eczema, The Happy Tappy crackers are very good for children. It contains Tapioca flour, apple juice and dried milk.

Tomato juice should not be given to a child with atopic Dermatitis. It produces skin lesions with greater frequency, possibly due to the pigment it contains.

Orange juice:—Many mothers complain that their babies break out when it is given. It is a good content of Vitamin C, but this can be given as Multivitamin or Vitamin C drops. In orange juice there are three contents which produce Allergic Symptoms.

(1) The protein of juice.

(2) of seed and

(3) peel oil. The orange peel has been found to produce contact Dermatitis.

Emotional disturbances play an important part in causing atopic Dermatitis. Benactazyne is helpful in acting as an useful tranquilliser. Other tranquillisers are Miltowns and Equanil.

Secondary infection must be combated by Broadspectrum antibiotic or crystalline Penicillin, or Penicillin V tablets. Multivitamin drops are essential.

The Local treatment: For bath no soap should be used; for soap aggrevates the skin lesions Starch bath is the best. For Acute Exudative lesion, Burrow's solution (Liq. Aluminium Acetas) 1 teaspoonful to 1 pint of water or 1 in 10,000 potassium permanganas or camomile tea may be used. The last is produced by steeping 4 teaspoonfuls of camomile in a pint of water. During the acule stage without exudation Calamine Lotion is useful. If there is a severe itching add Liq. carb. detergens 1 dr. add to one ounce. During the Subacute stat-Zinc cream is a preparation of choice. During the chronic state I prefer to use swartz ointment.

This contains:

R/	Salicylic acid		gr 30
	Mercurochrome		gr 20
	Aqua	qs	
	Anhydrous Lanoline Petroleum	} ad each	oz 1

If there is severe inflammation and there is secondary infection a combination of Hydrocortisone or Prednisolone with neomycin or chloramphenicol is used externally:

Corticosteroids in the treatment of Eczema in infants and children:

These drugs are useful to get over acute state and cannot completely eradicate the skin lesions. The drugs are to be used, with great caution.

Prednisolone for childern may be given in 10 mgm tablets for a few days, 7.5 mgm for a few days and then 5 mgm daily and tailed of gradually.

Parasitic infections of Skin:

The common parasitic diseases of the skin are:

* 1. Scabies. (2) Pediculosis. (3) Insectbites.

Scabies is a commonest skin disease occurding in a family. If a doctor enters a house were scabies is present, he will see the whole family sitting round a table and scratching (fiddling) as if in symphony The scabetic lesions are found on the webs of fingers, ulnar border of the hands anterior axillary folds, buttocks and pubic region.

In the management of scabies certain general principles are essential. The first is to lay open the burrows by a preliminary application of soap and water and scrubbing with a brush. This greatly facilitates in exposing the lesions for the second stage in treatment. Clothes worn to be changed and sterilised in boiling water.

Articles that cannot be boiled as blankets etc. should be pressed with hot iron. After preliminary bath with hot water and and soap one of the following ointments can be used. (a) Sulphur ointment. (b) Eurax (c) 25 % Benzyl Benzoate Emulson.

Every member of the family should be treated. When there is secondary infection treat infection with penicillin and Sulpha drugs before using one of the Antiscabetic preparations.

Pediculus capitis: capitis, corporis and pubis are infestations with Lice. Pediculus capitis is more common in children. The parasites and eva can be treated effectively with DDT Lotion or Kwell Cream or Eurax Ointment. If there is severe infestation, the scalp hairs are to be removed.

Insectbites:

Many insectbites produce pain, Liquor Ammonic is applied externally.

ALOPECIA AREATA

This is a type of Baldness in which circumscribed, oval or round patches are produced on the scalp. The condition is treated with stimulating Hair Lotion. The following is a useful preparation:

Lactic acid	dr 6
Castor oil	dr 3
Spirit Myrcae	dr 6

Seborrhoea: The term seborrhoea is applied to over activity of the oilyglands producing excessive formation of sebaceous material and abnormal oiliness of the skin.

Seborrhoea of the scalp is treated by frequent washing and the application of the following Lotion:

Liq. amm Tinct. Cantharidine	}	aa 2·5%
Spit vini Aqua Distillas	}	aa 100 %

Shampooing of the scalp is a useful procedure for which one of the following can be used:

a. Sudermo. (b) Selsun (c) Sebix shampoo.

Acne Vulgaris: Is one of the common skin diseases occuring in girls and boys. There is seborrhoea oleosa preceding the outset of Acne Vulgaris. The causative organism is Bacillus Acne. Endocrinal imbalance and emotional instability contribute to the occurance.

The treatment consists in the application of "Pusey's Linament" especially when there are pustules.

Pusey's Linament:—

R/	Powdered Traga canth	dr 1
	Phenol & Glycerine mixed	m 10
	Zinc oxide and Calamine	oz 1
	Olive oil	oz 4
	Water qs	pint one
	Oil of Bergamot	m 20
	Ft. Linament.	

Oestrogens in small amount reduce the Sebaceous activity and hence is given in small amounts.

TUBERCULAR DISEASES OF SKIN:

Tubercular diseases of skin can be divided as:

(1) Primary Tubercular Complex
(2) Secondary Tuberculosis.

I. Primary Tubercular diseases of skin:

This is more apt to occur in children than adults. The lesion is a papule growing to form a plaque which often ulcerates. The histological picture is acute inflammatory reaction in which numerous tubercle bacilli can be isolated.

II. *Reinfection Tuberculosis*; This is usually mono organic. The skin is affected. The other organs are free. The infection may be localised or generalised.

A. *Localised infection*:— Depending on the Virulence of the organism and the resistance of the host. The lesion may show either a slight eascation as in Lupus Vulgaris, moderate as in tuberculosa verracosa cutis and considerable amount as in scrofuloderma and Tuberculosis cutis orificialis.

1. *Lupus Vulgaris*:— Any part of the body may be affected but face is the commonest site. The lesions show peripheral spread and the centre shows spontaneous healing. The peripheral areas show pinhead sized deep seated nodules. If the lesions are pressed with a glass side or a diascope, the erythema is obliterated and the so called apple jelly nodule becomes apparent. The lupus nodule shows a positive probe test. A tooth pick is gently pressed into nodule and will often stay in the lesion.

Histopathology : caseation necrosis is slight. There are several typical tubercules.

2. *Tuberculosis verrucosa cutis*: This represents an infection of immune skin with virulent bacilli.

The lesions are verrucous hyperkeratotic areas. Surrounded by inflammatory border, crusts may be intermingled.

Histopathology: The epidermis shows acanthosis (increased thickness of stratum malphigi), hyperkerarosis (thickness of hornylayers) and papillamatoses (Tumour like proliferation of skin) Beneath the epidermis there is an inflammatory infiltrate containing polymorphs lymphocytes and tubercule. Tubercle bacille are more numorous than lupus vulgaris and hence occasionally seen.

3. *Scrofuloderma (Tuberculosis cutis colliquativa)*

The underlying tubercular disease of lymph gland or bones come to surface.

Histopathology: There is necroses of the epidermus and dermis and tuberculoid granulation tissue.

4. *Tubercolosis cutis orificialis* are ulcers occuring aground the mucous membrances.

Generalised lesions: These are called Tuberculoids. The lesions are due to haematogenous disseminate infection. The lesions occur in patients with high immunity. The baccilli are rapidly destroyed in the skin.

Types are:

1. *Micropapular tuberculoid*: (Rosacea like tuberculoid of Levandowsky) These lesions are small slightly indurated papules occuring on the face. There is diffuse erythema of the face.

Histopathology: These are islands of epitheloid cells with admixture of lymphiocytes and few or no giant cells. Caseation is absent.

2. *Papulonecrotic Tuberculoid*: The initial lesions are papules which become nectrotic and eventually breakdown. The lesions occur on the face, cheek and ears.

Histopathology: There is a central area of necrosis surrounded by a some inflammation which is largely nonspecific. It may show a moderate amount of caseation necrosis. The walls of the vessels may be invaded by inflammatory cells.

3. *Lichenscrofulosorum*: The lesions occur in showers. There are red small papules with a tendency to grouping.

Histopathology: The lesions show pure epitheloid tubercles occasional giant cells and no caseation.

4. *Erethema induratum*: The lesions are chronic recurrent eruptions occuring on the calves of girls. They are red indurated painless. Ulceration occurs producing a punched out appearance.

Histopathology: During the early phase histological changes are limited to lower dermis and the subcutis. The infiltrate is tuberculoid in type. There are epitheloid cells giant cells and occasionally in tubercle arrangement. The infiltrate occurs between fat cells and replaces them (Proliferation atrophy of fat) caseation necroses is nearly always present. The blood vessels show proliferative changes and infiltration with round cells.

Treatment of Tubercular diseases of the skin.

The most effective antitubercular drug is Isoniazid 150 mgm daily doses in children. A combination of streptomycin and isoniazid is the best method of treatment.

The former method of calciferol is good but in prolonged and large doses it is likely to produce, headache, indigestion, anorexia thirst, polyuria, constipation and loss of weight, photo-phobia and coma.

LEPROSY

In recent years it has been shown that children and not adults are susceptible to infection.

Certain proportion of healthy contacts harbour leprosy bacilli in the so called closed cases. Bacilli can be recovered by concentration method. All types of leprosy show involvement of Nerves and the alteration of sensation depend on the amount of Neural involvement. The lepromin test is only a measure of a potential tissue immunity.

Clinically the lesions may be (a) Macular (b) Infiltrated. (c) Polyneuritic.

Mucular—1. There is a hypopigmented patch. The edge is seen. The patch shows thermal anaesthesia. The peripheral nerves show evidence of thickening. This is simp le Neural. Trophic changes are seen as Trophic dermatitis or ulcer.

2. *Tuberculoid macule* The edge is definitely raised. The peripheral Nerve show thickening subcutaneous Nerves show thickening.

3. The lepromatous macule is flush with the surface and shows no clear edge. The outline is hazy or indefinite.

The Lepromin test is positive in Tuberculoid type. Amacule associated with a slightly positive lepromin reaction, it is difficult to state if it is going to develop into tuberculoid or lepromatous case.

Infiltrated lesion: may be classified as

(a) Tuberculoid (Lepromin positive.)
(b) Lepromatous (Lepromin Negative.)
(c) Dimorphous (Lepromin weakly positive.)

Where clinically there is doubt the histopathological section is helpful. There is no free subepidermal zone in tuberculoid cases. There are epitheloid cells, giant cells. The nerves are invaded grossly. The bacilli are absent or few.

In lepromatous type there is a free subepidermal zone. There is no evidence of focalisation. Macrophages which show commencing vacuolisation is seen called Virchow cells. The nerves are not invaded but there is perineural invovelment. The baccilli are in large numbers showing "fish in stream" appearance. The dimorphous type shows both features. It is essential to recognise the dimorphous type since the tissue are in an unstable immunological condition and hence violent reactions will occur with sulphone therapy.

Polyneuritic type; This type shows no lesion. The condition may be called neuritis anaesthetical. The deep reflexes are brisk. There is thickening of the nerves.

Treatment: 1. Sulphone D. D. S:— 25 mgm twice a week and increased to 100 mgm twice weekly in a fortnight for children.

2. Sulphaterone: Dose: ½ cc I.M. twice weekly and increasedly ½ cc till 1½ cc I. M. twice weekly is given.

Reactions: Lepro natous Leprosy patients may show erythema Nodosum Leprosorum. When this condition occurs, sulphone therapy may be continued cautiously or suspended altogether. In lepromatous type the sulphones prepare the bacilli for phagocytoses and hence inadequate dosage or discontinuance will produce pockets of bacilli which cause recurrence. Fever and arthritis may occur in leprosy and are treated with prednisolone and Sulfomin injections.

Tuberculoid Leprosy

In this condition, the sulphones stimulate leprabacillus and as a result a favourable tissue response is obtained. Resolution occurs.

Since suiphones interfere with absorption of iron, a combination of iron and B 1 is administered during sulphone therapy.

—:o:—

Metastatic Corneal Abscess

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Presence of yellow puriform deposit placed deeply in the cornea has recently been observed in a number of cases and has been described in the literature by various authors under various names (Fuchs, 1915 Bryn 1924, from Axenfield Clinic, Enroth 1927 Hippel 1895-1918, Hancock and Bietti 1895-1908)

In this clinical study we found deep infiltration of cornea to a varying degree from involvement of a small sector to complete opacification of the cornea by the yellow puriform substance and termed it as metastatic corneal abscess. Usually there is a single large sharply defined central deposit separated from the limbus by a clear or only faint opaque zone. Usually but one eye is affected and is associated with hypopyon iritis which indeed probably constitutes its starting point. The abscess may be due to syphilis (Fuchs) tuberculosis (Hancock and Bietti) pyogenic infection or trauma Our series showed pyorrhoea alveolaris and small pox to be the cause (Agarwal 1959). In any event the immediate cause must be a toxin derived from inflammed iris and acting on cornea from behind.

The infection may be introduced either from without through perforating injury or may be metastatic. There are however no essential pathologic differences in the two conditions. The corneal periphery owing to its better nutrition remains clear while the infiltration gathers round the defenceless centre. In syphilitic cases spirochaetes penetrate and wander through tissues in a short time without necessarily causing injury of all the tissues through which they pass thus marginal zone of cornea would remain relatively clear. In tuberculosis cases it was thought that the lesion was associated with tubercle and presumed that there was metastatic deposition of tubercle bacilli in the eye and keratitis was secondary to or coincident with iritis.

Case 1, A boy of one year was admitted to the children ward of this hospital and was diagnosed tuberculous meningitis. The boy suffered from cholera six months and from diphtheria three months preceding tuberculous meningitis. Four weeks after admission into the hospital the baby develops central puriform infiltration in the left cornea. There was no congestion of the eye and no ulceration of the cornea nor there was any hypopyon.

Case 2. A boy three years of age had small pox. He developed corneal ulceration of the left eye two weeks after postulation. The left cornea showed deep purulent infiltration of the whole cornea only leaving its priphery clear. The cornea showed central staining with fluorescein and there was no hypopyon.

Case 3. A lady 30 years of age came with history of conjunctivitis in the left eye for eight days. She had hypopyon for lase five days and was under treatment for thh same elsewhere. The lower three fourtt cornea was completely opaque and anterior chamber looked full of pus. There was faint central staining. The tension was normal and perception and projection of light present in all quadrants. She had her four upper and four lower teeth out because of pyorrhoea alveolaris.

Treatment.

In all our cases we have followed the following line of treatment besides the usual local treatment and cleaning of the eyes.

1. Penicillin 100,000 dissolved in 0.3 c. c. of mydricaine was given subconjectivally daily.
2. 3 mgms of Rondase (Evans Hyalurronidase) added with 1/4 c.c. of procaine in 1% strength was given by the retrobulbar route daiiy until disappearance of the corneal exudate.
3. Priscol 1 c.c. was given retrubulbarly in cases where intra-ocular tension was high along with Diamox tablets.

Penicillin was given to treat acuteness of the disease and corneal ulceration and conjunctivitis. Hyaluronidase was given as the main treatment for hypopyon ulcer and corneal infiltration. In the first case we supplemented our treatment with local hydrocortisone drops in 1% strength. The infiltration cleared with two injections of Rondase retrobulbarly but the baby did not show any sign of appreciation of light. He was put on nicotinic acid tablets by mouth on 100 mgm dose daily. While the eye was dilated and showed optic atrophy when the corneal infiltration had cleared there was sluggish reaction of the pupil at the end of two weeks treatment. The optic atrophy did not show any change in colour. It was difficult to judge the extent of recovery of vision. (Mathur, Gupta and Vyas).

The second case showed complete clearance of the infiltration from his left eye by the end of the second week hyaluronidase was given on alternate days in the second week).

Out of our series of three cases upto date it was only the last case which did not show satisfactory result. The hypopyon and corneal exudates disappeared within the first week but cornea perforated in the upper part and there occured iris prolapse. Anterior staphyloma formed as a result of weakening of the corneal stroma consequent on sloughing away of the anterior layers.

Discussion.

Emphasis was laid by old writers on syphyilis as the main causative factor of this disease (Fuchs, 1915, Seefelder 1910, Meller 1917, Bryn 1924). It was reported that anti-syphilitic treatment was curative if it was started early in the diseases but proved ineffective if employed late. Tooker (1939) a case of ring abscess of cornea as a result of metastatic septic endophthalmitis resulting from focal sepsis. Fiegenbaum and Kornbleuth (1946) described a ease of posterior ring abscess of cornea of non-traumatic origin as a complication of Behcet's disease. Seale (1931) reported a case of diabetes with carbuncles developing interstitial corneal degeneration. He explained that the high tension and blocked angle aided by toxin laden aqueous so affected the adjacent tissues to cause their destruction. This explanation holds good for our last case where the extent of hypopyon and corneal infiltration, the age and duration of disease were adverse factors because as soon as the exudation of corneas disappeared the anterior layers sloughed off and perforation and staphyloma resulted. Following uncommon features were noted in our cases:

1. All the patients showed involvement of left eye.
2. Presence of hypopyon only in one case.
3. Small-pox as a causative factor in one case
4. Tuberculous meningitis as a causative factor in another case. Since the start of our present study we have successfully treated these cases of metastatic abscess with or without hypopyon formation with the above outlined treatment. Out of three cases reported so far two got well within a short time and we think that if we would have got the last case a little earlier we could have saved this case also.

Summary

Three cases of metastatic corneal abscess resulting from pyorrhoea alveolaris, small pox and tuberculous meningitis affecting only the left eye have described. Hyaluronidase by retrobulbar route was given as the main treatment along with other routine treatment. Two cases got well and the third developed anterior staphyloma.

Acknowledgement

I am thanful to Col. B.L. Taneja Medical Superintendent of Irwin Hospital for allowing me to publish this case series. My thanks are also due to Dr. N S. Jain in charge of the Ophthalmic Department but for whose ungrudging help use of this new drug in entirely a rare disease would have been hardly possible.

—:o:—

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Notes & News

GRANTS FOR DEVELOPING INDIGENOUS MEDICINE

A sum of Rs. 53,13,712 has been sanctioned so far durin g this plan period by the Union Ministry of Health for the development of indigenous systems of medicine including homoeopathy, reads an announcement dated May 13. The total provision in the second Five-Year Plan for this purpose is Rs. 100 lakhs. Financial assistance is given to private organizations and State Governments for research in indigenous systems and for upgrading teaching institutions.

Dusing 1958-59, Rs. 20.55 lakhs were also advanced to various State Governments as the Union Government's matching co ntribution to the expenditure incurred by them on the establishment upgrading of teaching institutions in indigenous systems of medicine, including homoeopathy. For the current financial year, a provision of about Rs. 30 lakhs has been made for grants to State Governments and Private organizations for research and upgrading.

The institutions to whom grants are given are engaged either in research work or in teaching of indigenous systems of medicine. Research is generally confined to clinical trials of various Ayurvedic, Unani and Homoeopathic drugs and medicines. To some institutions grants have been sanctioned for plantation of medicinal herbs used in Ayurvedic and Unani treatment. To others grants have been given for publishing works on Ayurveda and Unani systems of medicine.

During the first Five-Year Plan, the Union Government spent a sum of Rs. 12,82,710 against the provision of Rs. 37.50 lakhs. The response during the first Five-Year Plan was not encouraging as most of the State Governments had not made sufficient provision in their own Plan schemes for promotion of indigenous systems of medicine.

DEMOGRAPHIC ADVISORY COMMITTEE

The Ministry of Health has constituted a Demographic Advisory Committee to advise on research and studies in inter-relationship between economic, social and population changes on the reproductive pattern, attitudes and motivation affecting the size of the family.

The chairman of the committee is Dr. V.K.R.V. Rao, Vice Chancellor of Delhi University. The members are: Prof. P.C. Mahalanobis. F.R.S.. Indian Statistical Institute' Calcutta; Shri P. Thomas, M.P.; and Shri Ashok Mitra, I.C.S., Registrar General, India. Lt. Col. B.L. Raina, Director, Family Planning, is Member Secretary. The committee can co-opt additional members for *ad hoc* purposes. The members of the committee will hold office for a period of three years.

The committee will advise on training and research in demography keeping in view that these will be of assistance to Government in taking economic and social measures for programmes of national reconstruction.

The committee will also coordinate demographic research schemes receiving financial assistance from the Government of India, review the progress made at the Centres getting financial aid from the Ministry of Health, and examine and recommend proposals submitted to the Ministry of Health for financial help for demographic research.

The Union Governmet has made a provision of Rs. 30 lakhs in the second plan for demographic training and research. A Demographic Training and Research Centre has deen established in Bombay in collaboration with Sir Dorabji Tata Trust and the United Nations. Three demographic centres have also been set up at the Delhi school of Economics, Delhi University; Indian Statisticals, Institute, Calcutta; and the Department of Statistics, Government of Kerala, Trivandrum.

COMMITTEE ON MEDICAL EDUCATION

The Union Ministry of Health has constituted a committee to assess the present facilities for post-graduate medical education in the country, says an announcement dated May 9. Dr. B. C. Roy, Chief Minister of West Bengal, is chairman of the committee.

The terms of reference of the committee broadly are (i) assessment (or evaluation) of the existing facilities for post-graduate medical education in the country; and (ii) formulation of recommendations for the future plan of development of post-graduate medical education.

HEALTH EDUCATION BUREAUX

Answering a question in the Rajya Sabha on May 4, Shri D. P. Karmarkar, Minister of Health, said that the Government of India have formulated a scheme for the establishment of ten Health Education Bureaux in the States with Central assistance during the second Five-year plan period and have circulated it to the State Governments.

The Minister added that in order to encourage the State Governments to establish these Bureaux, the expenditure of Rs. 99,540 recurring and Rs. 10,000 non-recurring would be shared by the Union Government with the State Governments on the approved pattern.

For the purpose of establishing greater co-ordination between the Central and State Health Education Bureaux, it was proposed to hold periodical meetings, conferences, seminars etc. of health educators.

TRAINING OF NEPALESE NURSES

In a written reply to a question in the Lok Sadha on May 1, Shri D. P. Karmarkar, Minister of Health, stated that eleven Nepalese nurses had come for training in the midwifery course at Irwin Hospital New Delhi. Their expenses would be borne by the World Health Organization.

PROGRESS OF GOITRE CONTROL SCHEMES

Two field survey units of the Union Ministry of Health are now engaged in de-limiting the areas of Goitre endemicity, says an announcement of May 26. One team is now in the Mandi district in Himachal Pradesh after finishing its work in the Bilaspur district. The other team has started surveys in the Siang Frontier Division of N.E.F.A.

The Health Ministry's scheme for the control of goitre proposes to cover a population of 87.5 lakhs during the Second Five-Year Plan which has an allocation of Rs. 18 lakhs for the purpose. The Scheme envisages the estimation of the magnitude of the problem. Supply of iodised salt to inhabitants of areas affected with endemic goitre and assessment of results of iodine prophylaxis.

Only iodised salt will be made available in the selected endemic areas in the States of Punjab, Himachal Pradesh. U. P., Bihar, Assam and NEFA in the first instance for consumption at the same price as ordinary salt. The cost of iodisation will be met by the Union Government.

The UNICEF has agreed to provide one iodisation plant capable of processing 70 tons of salt per day sufficient for a population of 2.75 million. The plant is expected to arrive by September, 1959, and will be installed at Sambhar Lake. The UNICEF is also giving two vehicles on loan for the two survey teams. W.H.O's assistance will be in the shape of technical advice and guidance.

Endemic goitre associated with cretinism, deaf-mutism and various other grades of physical and mental deterioration is prevalent in some regions of India. The southern slopes of the Himalayas covering a distance of over 1,500 miles and consisting of the northern parts of Kashmir, Punjab, Himachal Pradesh, U. P., Bihar; West Bengal, Assam, NEFA and Manipur are probably the most classical areas of endemic goitre. Starting from Gilgit and Chitral in Kashmir, the goitre belt covers the Kangra valley in Punjab, the Himalayan tracts of Tehri, Garhwal, Almora, Naini Tal, the Sub-Himalayan regions of Bahraich, Gonda, Basti and Gorakhpur in U. P.; the Champaran, Darbhanga and Purnea districts of Bihar; the Darjeeling and Cooch-Behar districts of West Bengal; terminating in the Naga and Lushai hills Assam. In the north; the belt embraces the States of Nepal, Sikkim, and Bhutan. Apart from a small portion of the tribes of Ranchi Plateau, goitre is rare elsewhere in the country. About 9 million persons are affected with goitre in India.

HEALTH REPORT FOR MARCH

Births and deaths registered in towns with a population of 30,000 and over during March, 1959, were respectively 27 and 11 per thousand of population as against 25 and 10 during the month of February, 1959.

According to the available reports the country remained free from plague. Reports of cholera and small-pox, however, continued to come in from some parts of the country.

Cholera: Based on preliminary data for the third week of April, 1959, i.e. week ended April 25, mild incidence of cholera was reported from the Puri district of Orissa State, and Bankura, Burdwan and Calcutta city in West Bengal. Mild incidence was also reported from the Ujjain district of Madhya Pradesh.

Small-pox: Heavy incidence was reported from the districts of Kotah and Sikar and considerable incidence was reported from the Jhunjhunu district of Rajasthan. Considerable incidence continued to be reported from the Madras city. Mild incidence was reported from the districts of Anantapur, Cuddapah, East Godavari, West Godavari, Guntur, Hyderabad, Krishna, Nellore, Srikakulam, Visakhapatnam, and Kurnool in Andhra Pradesh; Cachar district in Assam; Monghyr district in Bihar; Kottayam district in Kerala; the districts of Ahmednagar, East Khandesh, Greater Bombay, Amreli, Poona, Satara South, Sholapur, and Nagpur in Bombay State; Chingleput, Coimbatore, North Arcot, South Arcot, Tanjore and Salem districts in Madras State; Bellary, Belgaum, Chickmagalur, Coorg, Dharwar Kilar, Mandya, Shimoga South Kanara, Bangalore, Chitaldurg, Mysore and Hassan districts in Mysore State; Cuttack Dhenkanal, Kalahandi, Keenjhar and Sambalpur districts in Orissa; Ferozpur and Ludhiana districts in Punjab; Nagpur district in Rajasthan; Allahabad, Azamgarh, Bareilly, Dehra Dun, Deoria, Kanpur, Lucknow, Faizabad, Garhwal, Hardoi, Jhansi and Shahjahanpur districts in Uttar Pradesh; Birbhum, Calcutta city, Hooghly, Murishadabad and Midnapur districts in West Bengal; and Karikal and Pondichery.

Gastro-enteritis: During the week ended April 18, 208 cases and 24 deaths were reported from a few districts in Uttar Pradesh.

Kyasanur Forest Disease: Eight attacks with no deaths were reported from Shimoga district in Mysore State.

Influenza: During the second week of April, 26 cases were reported from Poona city as against 75 cases and one death during week ended April 11, 1959. Reports of sporadic cases were also received from Assam, Mysore and Punjab.

Note: Epidemic means 100 cases or more per week per million of population.

Considerable incidence means 30-50 cases per week per million of population. Mild incidence means less than 30 cases under week per million of populati

BOOK SECTION-I

(Contd.)

PLASTIC SURGERY

FOR

MEDICAL STUDENTS

AND

DOCTORS

BY

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

SPECIALIST IN PLASTIC SURGERY

What is the best way to find out the root cause of the trouble?

A detailed report of the accident is necessary to find out the root cause of the trouble. The following condition should be investigated to find out the cause of the accident:

1. Whether the machine is at fault and a protective guard could be added so as to make the accident impossible to occur.

2. Whether any personal factor mentioned above is responsible.

3. The unsafe act by the worker should be thoroughly investigated, accidents very often happen to the left hand if the person is not accustomed to do the work with it because the left hand is not sufficiently trained for that type of work.

The main implements causing injuries to the hand are circular saws, power presses, and buzz planes. Of the total compensation given to the worker 20% or more is for hand injuries. This suggests that the highest standard of primary treatment is essential for good ultimate results and thus a decrease in the compensation necessary to the worker. The Australian Government realised that it was in their own interest that cases of hand injuries should receive the best of treatment from the start till the hand was cured to its maximum extent so that the compensation would be minimised for the injured party.

Principles In First Aid Treatment. What is the main rule to be followed?

It should be made a rule in every factory that every case of injury be it major or minor should be immediately reported to the medical man in charge because it is noticed that very often when the injury is a minor one the worker fails to report himself with ultimate deformity of the hand. Every

cut or abrasion on the hand must be cleaned and kept covered till it has completely healed up. The task of cleaning and keeping the hand covered is of a medical man and not the worker himself. Most infective complications occur from smaller injuries and not from major ones and this is because the above principle is not followed properly.

How should major injuries be handled ?

When a major injury occurs to the hand it is a catastrophy to the worker. There should be a systematic way of handling these types of accidents. The medical man should control the haemorrhage by either a pressure dressing or a tourniquet which should not be kept at a time for more than one hour, anti-shock measures should be instituted immediately and after adequate splintage the patient should be transferred to a hospital where operative treatment can be undertaken. Cotton wool dipped in strong antiseptic like tincture iodine or tincture benzoin adds to the difficulty at the time of operation. A mild antiseptic on a gauze piece is the ideal first aid dressing.

What kind of treatment should be carried out at the hospital ?

All cases of hand injuries should be treated after admission by a comptent person and should not be relegated to the medical student or the houseman working under inadequate facilities. Repeated examination of the injured part should be avoided. If the factory medical officer has forwarded detailed notes of the accident there is no need for the wound to be opened repeatedly as this produces more trauma to the delicate structures of the hand.

What are the principles of treatment ?

The principles of treatment are clean well, stitch well, heal well. Good anaesthesia is absolutely necessary for good surgery, either local or general anaesthesia is given.

The surgeon himself applies the tourniquet and procedes in an orderly fashion to deal with the wound. The part is first thoroughly cleaned with Cetavlon solution, first the surrounding area and then the damaged part. Any foreign body visible is picked up. Next the part is made as clean as possible by removing all non viable tissue. It is a mistake to close the wound over dead tissues because there is bound to be septic complications. If it is not possible to close the wound, flap of skin and soft tissue is transferred from the surrounding area and any raw area left after this transfer is skin grafted.

How is this type of surgery called ?

This type of surgery is called traumatic but it should be Atraumatic. It is the continued responsibility of the operating surgeon to follow the case till it is perfectly healed.

How many types of hand injuries are there ?

There are two types of hand injuries (1) the tidy type of injury where the cut is a clean cut, the nerves and muscles are very often divided but fracture of the bones is uncommon. Most of the household type of injuries are of this type. There is rarely devitalisation of tissue. In these types of cases it is advisable to do a primary repair of the muscle tendons and nerves and completely stitch up the wound without fear of any complication. (2) *The untidy injuries*. In these types of wounds there is extensive laceration of tissue. Muscle tendons and nerves are injured but rarely divided. Fractures of bones are common. Devitalised tissues is a common occurrence. The treatment is to remove all the devitalised part, make the wound as clean as possible and leave the repair of the nerves and tendons to a later date. Fractures of bone are splinted at the same time. The wound is dressed by the surgeon by firm pressured

dressing which is maintained throughout the period of healing. The tourniquet is then removed and the part kept elevated for the first 24 hours.

What is the line of post opetative care ?

Early healing rather than early movement is the immediate object of primary treatment. " Wounds should be healed before movements are commenced "-Rank. All the sutures should not be removed at the same time but those where there is less tension on the skin should be removed on the 7th or 8th day. Pressured dressings are maintained throughout the period of healing. If one finger is injured the remaining fingers should not be bandaged. All compensation claims should be explained to the patient to avoid any misunderstanding which might prevent the further rehabilitation of the injured hand. Active exercise at work is more inducive to restoration of function than working on a steriotype gadget to restore mobility of the part.

CHAPTER X

BURNS

* * *

How Could burns be prevented?

The prevention of burns is a nationwide problem. It is the duty of the state to educate the masses with proper precautions to avoid burns, accidental burns whilst cooking on a gas stove has lead to innumerable fatalities in each town of India. Keeping the stove on the ground whilst cooking invites the sari to catch fire—a very dangerous occupation. Stoves should be kept on a platform or table. Saries should be worn in such a manner that no ends are liable to come in contact with the flame. Very often the sari catches fire from behind and before the person is aware it is completely on fire—severe and generally fatal burns are thus encountered. Nylon burns very quickly. Burns in children are common during the Divali Season. Children should be taught the proper way of handling crackers. Dangerous crackers—"atom bomb" and the like should be prohibited by the government. More burns occur in Divali season in children than in the whole year. Burns of the hand is common in children because of their inherent tendency to touch and feel every object—burns of the palms and fingers leads to crippling contractures and useless hands—with loss of manpower.

How are burns classified ?

The physiopathology of burns has undergone much change since the last few years. Burns are now classified more simply as:

(1) Superficial skin loss

(2) Total skin loss.

In the first variety the ultimate healing is without scar tissue formation and hence does not require skin replacement. In the second type the ultimate healing is with scar replacement of a granulating wound. It is necessary for any person treating total skin loss to understand this very important fact that the best thing for such a case is early skin replacement and not months of septic dressing to the inconvenience of the patient and elaborate waste of energy on his part.

What is a Scar ?

Scar by itself is an interesting entity—hard and indurated in the beginning it tends to soften down with years, painful and angry in the early stages becomes less so later on. A scar is always smaller than the area it has replaced—hence when a scar is excised to the normal tissue the area is often more than twice the size of the scar—this has proved often the pitfall for the beginner. Due to stretch on the surrounding normal tissue ectropian of the eyelids, lips, rolling up of alar margins is common deformity in a Burns Case. Scar is the result of second intention healing.

What are the different stages of burns? What physiological changes occur ?

I. A patient receiving sudden burns ofte ⊐ suffers from Primary Shock. It is a vaso-vagal or of neurogenic origin. Usually the patient survives it and then enters the post shock period.

II. During this time he is likely to suffer from the following;

(A) Fluid loss from the burnt area—there is of about 250 c.c. for every 10% of body burns in 24 hours. This produces haemoconcentration but it is necessary to realise that in burns over 20% there is also marked destruction of the R.B.Cs. Body fluids are distributed in 2 compartments-the intra and the extracellular part. The main portion is intracellular and forms 50% of Body Weight as shown in the figure. The extracellular component is made up of Interstitial fluid which is about 15% of the Body Weight whilst the other component of blood plasma forms only 5% of the body weight. Loss or fluid from the interstitial part due to oedema accounts for 2.8 Liters in 48 hours for every 10 burns. The normal daily water loss is made from the total amount of urine passed i.e., 1000 cc., about 1200 cc. through the lungs and about 100 cc. in the stools. The normal person usually soakes in about 1200 cc. of water and about 700 cc. in food. If he is unable to drink or eat this amount is extra to the usual daily loss. Vomitting might add to the amount of fluid loss. All these points are to be considered where fluid replacement is to be calculated.

FIGURE 1

"Rules of Nine" for assessing the extent of the burned areas in relation to the whole body surface.

(WALLACE, 1951).

Head and neck		= 9 %
Front of trunk	2 9%	= 18 %
Back of trunk	2 9%	= 18 %
Whole upper limb		= 9 %
Whole lower limb	2 9%	= 18 %
Genitalia		= 1 %
Palm of hand and fingers		= 1 %

Body tries to conserve its sodium content which is mostly present in the extracellular component. The pottasium is more in the intracellular part as shown in the figure. In the early stages the loss of sodium is compensated by the shift of pottasium. Then in order to preserve the sodium content - the kidney excretes more of pottasium salt and conserves the sodium element. The urine also excretes 17 keto steroids.

If the patient is passing about 30 cc. of urine per hour he is well compensated as regards his fluid balance. For this reason it is advisable to insert a catheter in a burn tcase and chait the hourly output of urine. If the fluid balance is notmaintained, patient goes in for oliguria and shock is pre-eminent.

(B) The second important phase in the post shock period is the onset of Toxaemia. This is due to the liberation of Histamine like substance from the burnt tissue. Local wound sepsis if it has occurred adds to the toxaemia.

(C) Secondary shock with failure of periplural circulation - thls occurs due to not restoring the fluid balance and complete failure of the capillary tone. It usually occurs after 72 hours of burning.

III. The third important phase in burns is the stage of Granulating wound surface. This phase could only occur if early skin grafting is not done. During this period there is excessive loss of body proteins and loss of weight healing of the granulating surface could only take place by surrounding skin and not from the deeper surface as there is complete skin loss. Painful dressings for months on end are being done at many places when an early skin graft would solve the problem in 8-10 days. It is the duty of all well informed general practitioners to requst the attending surgeon to do early grafting - if such facilities are not available then patient

should be transferred to any hospital where this is possible. If ultimate healing is allowed without grafting, it leads to the last phase of burns, i.e., the phase of contracturs - all the surrounding tissue producing hideous deformities which are most marked on the flexor surfaces like the neck, armpit and elbow joint. Removal of the scar tissue always reveals a defect far in excess to the size of the scar.

What are the important points to be considered in treatment ?

(1) When a person is burnt in the faily where the doctor is attending it is the doctors decision whether that patient should be treated at home or shifted to the hospital. The decision will be made on many factors like the previous experience of the doctor, the amount of daily care and nursing the patient will require and also on the extent of the burns. It is a safe rule to shift the patient to hospital if he has a burns of more than 15 to 20% of his body surface burnt.

(2) The rule of 9. (Fig. I) Dr. Wallace has distributed the whole body into ratios of 9 and the chart is self explanatory. The doctor should be able to approximately judge the total amount of burnt surface and hence be able to calculate the amount of fluids the patient will require.

(3) In the early stages it is difficult to decide if the burns are superficial or deep. After 10 days if the part is not healed up then they are full skin loss burn and require early coverage.

(4) Principles in fluid replacemxnt—a fairly simple rule is to give 2.8 liters of fluid in 48 hours for every 10% burns. Evans has formulated a more definite plan thus:—

1 cc. ot Colloid × wt. of patient in Kgm × % of Burns plus same amount of electrolyte solution + 2000 cc. of Glucose in Water. This is the total amount necessary per day. Of the total Colloids half is given in the form of Blood. Half the total amount calculated is given in first 8 hours and the remaining in the rest of the day.

The total daily loss of fluids amounts to -

1200 cc. through lungs.
1000 cc. through kidneys.
1200 cc. water intake.
700 cc. in food.

Total 4100 cc.

The last two factors are variable depending upon whether the patient can take food and drink orally. The intake and output chart should be maintained and if the patient is passing 30 cc. of urine per hour he is within safe limits of fluid balance. If urine output falls below 25 cc. per hour he requires more fluids. In an unconscious patient a catheter should be retained in the urinary bladder and output per hour charted. For every 1 cc: of saline give 2 cc. of 5% Glucose in water.

After giving half the amount of Colloids as Blood give the remaining half in the form of plasma or plasma expanders like P.V.P. or Dextran. Dextran is a polymer of glucose it is used as 6% solution in saline.

(5) Potassium Loss—Body tries to preserve the sodium iron but at the expense of the potassium iron which is excreted through the kidneys. Hence the potassium level in blood falls. It is necessary to replace it and any of the following measures could be adopted -

If the patient can take fluids orally give 3 qrs of Potassium Chloride or phosphate in Fruit Juice—4 times a day (I.V. give 2% Kcl. in 5% Glucose in water).

(6) Sodium loss—this is replaced by giving orally

1.5 gms. of Sodium Bicarb.
3 gm. of Sodium Chloride.
with a litre of water.

It could be sweetened with glucose and patient told to take sips of it regularly. To replace the sodium chloride and potassium irons Hartman's solution is given intravenously. This is useful if the patient is unconscious

(7) Protein Loss—When the patient has large granulating areas of raw skin there is a marked depletion of serum proteins. This loss must be stopped by early skin replacement. Proteins metabolism should be maintained by high protein diet and repeated transfusions of blood and plasma.

(8) Sedatives—the present day tendency is to avoid strong sedative in burns. It is believed and it is true that if the fluid balance is well restored the patient does not complain of much pain hence sedatives are not necessary.

II. The Local Treatment of Burns.

When is the local treatment of burns undertaken? The local treatment of burns has often to wait for a few hours till a severely burnt patient is treated medically so as to restore the disturbed metabolic condition. Only when the patient has been treated for shock and his condition is out of danger should the surgeon think of the local care of the burnt area. Till such time it is advisable to cover up the burnt part with a sterile sheet.

What are the aims of local treatment ? The local treatment aims at the following important points :-

(1) To secure aseptic healing of the part whenever possible and as early as possible.

(2) To prevent the occurance of resistant organisms in the burnt area. The dangerous pathogens are :

(i) Strep. pyogenes.
(ii) Stap. aurous.
(iii) Ps. pyocyaneus.

Strep. pyogenes is usually sensitive to local and systemic penicillin.

Stap. aurous is sensitive to the tetracyclines but soon develops resistance to them while Ps. pyocyaneus could be prevented by local application of polymyxin E. There is unfortunately no ideal local application which has a prophylactic value against all the three organisms.

(3) Safeguard against cross infection by dressing all cases in a special room by a trained team of surgeons and assistants.

What is the closed method of local treatment of burns ?

After the burnt area is cleaned by irrigation with saline and gentle sponging with mild antiseptic, the loose pieces of burnt skin are debridded and all the blisters are evacuated.

Tropical use of broad spectrum antibiotic reduces the incidence of septecaemia. The pressure dressing is then applied to cover well beyond the burnt area.

The dressings are maintained for 14 days and when opened it will be noticed that all superficial burns should

BOOK SECTION-2

(Contd.)

Retinal Detachment

AND

Its Modern Treatment

BY

Dr. J. M. PAHWA

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When once the retina becomes perforated without forming any attachment to the choroid around the *perforation* or in other words, if the vitreal space begins to communicate with the subretinal space, the retina will become detached primarily by mechanical means. Once more the rotatory movements of the eye play an important part, and subvitreal liquid enters by the law of inertia under the retina.

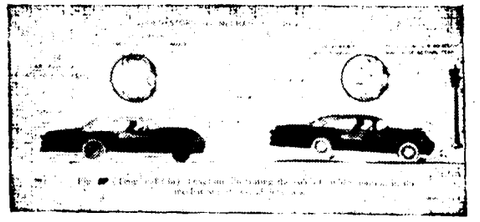


Fig. 17. Diagram illustrating the role of sudden motion in the mechanism of retinal detachment, (Teng & Chi).

By the same law if there is liquid beneath the retina and eye continues to rotate, this liquid undermines the retina more and more and the detachment becomes larger. It is because of this that Lindner's prescription of staenopic glasses (Loch-Brille) is of paramount importance in every case of retinal detachment.

In addition to this mechanical cause, physicochemical factor also exercises some influence. Normally we know that choroid has a powerful absorbent action. This is one of the factors which keeps the retina in place in the living eye (suction of the choroid). If a retinal tear develops, the absorbing action of the choroid causes a negative pressure and attracts liquid from inside the eye under the retina, where it will be absorbed. hence there develops a larger production of the liquid from the ciliary body in order to replace the loss and to keep the pressure of the eye normal. Increased production of liquid by the ciliary body, changes its chemical composition and this changed liquid

leads to further contraction of detached vitreous. As it adheres in the anterior part of the eye, it pulls the retina into the eye and thus the bullous retinal detachment develops. This peculiar appearance of the recent retinal detachment was the reason why the theory of exudation remained for so long as the leading theory. These fresh bullous detachments faltten out in time because the detached vitreous in retinal detachment becomes more and more liquified.

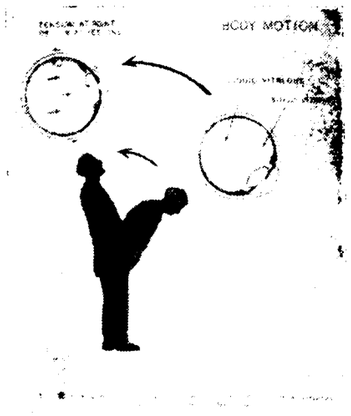


Fig. 18. Diagram illustrating the role of body movements in the movements in the mechanism of retinal detachment

Cause of secondary tears :—

As the retina becomes detached owing to the rotatory movement of the eye, further shrinking of the detached vitreous often leads to the production of secondary tears At some places the retina may be adherent to the choroid and if the retinal detachment spreads-the retina will rupture at those places.

Arruga's conception of traction theory.

He thinks that detachment of the retina is an accident due almost always to a disease of this membrane, though at the moment of the detachment the retina may play a passiv, role and act only as a result of its retractibility. He thinks that as a result of previous inflammation (chorioretinitis) or degenerative processes (atrophy and cystoid degeneration),

the retina becomes adherent to the vitreous in certain places. At the same time it becomes thinner, weaker and retractile, a property found in all tissues following inflammation or degenerative process. Then movements of the eyes, sudden jerks, blows on the head and other slight injuries may make the adhesion of the vitreous produce a rent in the retina (Fig 12).

The retractile tendency of the sclerosed retina makes the torn edges of this membrance curve inwardly. At this point, the motion of the eye forces the vitreous in the retina enlarging the rent and allowing the vitreous to pass behind the retina,

3. Weve's theory of Retinal Cysts :—

In 1934, Weve reported a group of cases in which retinal cysts had separated the retina from the choroid and lead finally to the laceration and detachment. Weve concluded that this aetiologic factor is by no means rare and leads regularly to detachment.

Weve's materials consisted of six cases each of which had one or more retinal cyst in a position which led to the assumption of a casual connection between the cyst and tear. The cysts were situated in all cases below and temporally and in the extreme periphery. This solitary cyst formation in the retina can explain the occurrence of spontaneous tears at the ora-serrata in a number of cases of retinal detachment.

Cyst formation in the retina with detachment was also reported by Kurz and Jancke.

4. Theory of distension.

It means that stretching of the outer coats of the eye is the cause of detachment of the retina. It was first suggested

by V. Graefe (1857) because of high incidence of detachment in myopes, as he thought that the retina was not so extensible as the two outer coats. He presumed that in the elongation of the eye in axial myopia, it became separated *assuming* the *chord* of the arc which it cannot describe. This theory was further elaborated by schweigger (1889), Hanssen (1919) Fuchs (1937) but does not withstand criticism because during youth, when the myopic eye is stretched the most, the retina has less tendency to become detached. Moreover retina is as extensible as the choroid, perhaps *more* so is seen by the fact that it usually remains intact when a contusion produces a choroidal tear and in the extreme rarity of detachment in posterior *staphylomata*

It seems clear that there is no such thing as a specific "Myopic detachment" but mypoia acts only as a predisposing factor, the predisposition lies in the associated myopic degeneration and not in the distension. It is probable that the only occasion when distension acts as the main aetiological factor is in cases of violent and sudden distension due to trauma.

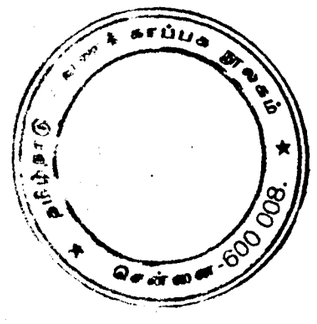
5. Theory of Exudation and Hypotony :

These cannot in any way explain the pathogenesis of Idiopathic retinal detachment, the former explains well cases of Exudative detachments (Coat's disease, orbital cellulitis, albuminuric retinitis, angiomatosis of the retina, sympathetic ophthalmia, and acute uveitis etc., and the latter where there is sudden hypotony due to loss of vitreous etc.

6.-Macdonald suggested that hydrodynamic phenomenon may play some role in detachment of the retina specially in persons who do heavy weight lifting. The transmitted intracranial pressure obstructs the central retinal vein and the excess fluid from the choroidal exudate and the central retinal artery collects in the retina and the intra-retinal space, with resulting detachment.

7. Kruckmann considered the role of muscular action (particularly two obliques) in the development of detachment incases of indirect trauma. Bartles also drew *attention* to the role of superior oblique muscle in explaining the prevalance of retinal holes in the superior temporal quadrants.

8. Mr. Agarwal no doubt agrees that primary event in the aetiology of retinal detachment is the degeneration of vitreous but he thinks that it is due to the action of hyaluronidase produced during this process which acts on the congenitally weak spots (Granular patch of Teng & Katzin) resulting in hole formation and detachment,



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Published by M. Umapathi at 57, Burkit Road, Madras 17 and Printed by Krishna Srinivasan at Industries Press, 4, Motilal Street Madras 17 Editor : Dr. B Rama Rau B.Sc. M.B., B.S

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