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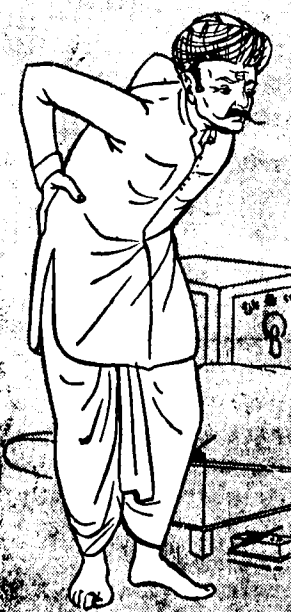
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MEDISCOPE

Journal of Medicine & Surgery

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MAY 1958

No. 3

Tropical Eosinophilia

By

Dr. R. SANKARAN, L.M.P.,
Tropical Eosinophilia, Asthma & Tuberculosis Clinic
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Tropical Eosinophilia is a definite clinical entity with persistent or recurrent cough resembling either the asthmatic or incipient tubercular, with massive increase of Eosinophils and a very high Leucocytosis with certain Lung and other systemic changes. It is a benign disease and not a symptom. Though the name gives an impression that it is peculiar to the Tropical Regions alone, from the reports published in the various medical journals we find that this disease is prevalent in all regions. The disease, though prevalent much earlier, was reckoned for the first time in 1938 by Saint Etienne as a distinct clinical entity. Subsequently in 1940, Dr. Frimodt Moller and Barton described the signs and symptoms of the disease associated with certain radiological changes in the lungs and blood findings of massive Eosinophilia and a very high Leucocytosis. They also established

that this disease is quite different from Leoffler's Syndrome and that it has nothing to do either with Bronchial Asthma or Pulmonary Tuberculosis. In 1943, Weingarten reported about 81 cases with massive eosinophilia and high leucocytosis with pulmonary symptoms in different communities of different ages ranging between 15 and 45, in both the sexes in the West Coast of India; because of his findings in the tropical region he named the disease as Tropical Eosinophilia. It has no bearing with the region, climate, atmospheric conditions, caste, community, sex and age; nor has it anything to do with the occupation or the social status of the patient; nor with the familial or constitutional peculiarity and allergic susceptibility, though some are of the view that the constitutional susceptibility has something to do with the disease.

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Aetiology. Aetiology is still obscure. Different theories are put up by different people according to the type of cases that they come across, such as Infective-endogenous and exogenous, chemical poisoning of slow ingestion and allergic; some even go to the extent of Endocrine disturbances as a result of which cortisone and similar endocrine preparations are being prescribed even today. In my own experience, after treating nearly 320 cases from the year 1943, I am of opinion that the aetiology is still obscure and nothing can be definitely said about it.

Physiology. Before we pass on to the various aspects of the disease, it will be better to have an idea about the Eosinophils and the Leucocytes. In a healthy individual the total number of Leucocytes per c. m. m. of blood is about 6500 to 10000, the minimum level being 6500 and the maximum being 10000. If either the minimum level is still diminished or the maximum level is exceeded, then it is a pathological condition. Of the Leucocytes, Polymorphos Eosinophils and Basophils have their origin mainly in the bone marrow, Lymphocytes and Monocytes in the Lymph glands and the Lymphatic tissues.

	Differential Count.	Absolute Count. Per cmm
Polymorphos	60 to 70%	1500-7000
Eosinophils	1 to 4%	150- 400 „
Basophils	0-25 to 0-5%	0- 100 „
Lymphocytes	20 to 35%	1500-2700 „
Monocytes	2 to 6%	350- 500 „

The functions of Eosinophils are mainly Phagocytic for bacterial and

certain chemical poisons and is also engaged in Protein Metabolism. They are increased in the following conditions: Thread-worm, Round-worm, and Hook-worm infections, in arsenical poisoning, in certain dermatological conditions, Periarthritis No. Josa, Hodgkins disease, Filariasis and Lymphosarcoma. But in the above conditions, very rarely the total goes beyond 13000 to 15000 per c. m. m. and the Eosinophils above 15 per cent.

Clinical Picture. The onset is more or less slow and insidious. The clinical picture of the disease is most characteristic excepting for minor variations. The symptoms of the disease are in the following order of importance as reported by the patients: (1) Cough (2) Low fever (3) Expiratory dyspnoea (4) Loss of weight (5) Pain in the chest and abdomen (6) Anorexia (7) Gastro-intestinal symptoms such as diarrhoea and vomiting and (8) Cutaneous manifestations.

Cough. The cough is the most distressing symptom for which the patient seeks relief. In the beginning it is dry, hacking and unproductive. Slowly it becomes paroxysmal and worse in the early hours of the morning between 2 A. M. and 6 A. M. Invariably, in the early stages, the day time is free from cough. The cough, especially in the children, ends in vomiting, whether the stomach is full or empty. If there is sputum, it is glary and tenacious, and later becomes thin and copious. The sputum is never offensive, purulent and rusty though very occasionally, that is one in hundred cases, there may be seen

in the end of the prolonged hacking cough a streak of blood.

Low Fever. In 50 per cent of the cases fever is present. In 25 per cent of the cases the disease sets in with slight fever for one or two days at the most one week. The rest of the 25 per cent of the cases are apyrexial. When pyrexia is present it is continuous and of the low type ranging from 99° to 100°. 5 F and persisting even after one or two injections of arsenic in the course of the treatment. Occasionally there is intermittent high fever, which is occasionally mistaken for malaria when the spleen is enlarged; but neither profuse perspiration nor remittance to the normal, as in the case of malaria, is seen. When there is consolidation of the lungs with fever, it is mistaken either for Pneumonia, Influenza, Bronchitis or Broncho-Pneumonia.

Expiratory Dyspnoea. As the disease advances the paroxysms of cough become more frequent followed by wheezing and expiratory dyspnoea. In the mornings after 6 A. M. the patient feels better. Hence invariably it is mistaken for Bronchial Asthma.

Loss of Weight. In the majority of cases there is a gradual loss of weight from the very beginning. When there is low temperature, cough and losses of weight, the patient is invariably alarmed thinking that it is Pulmonary Tuberculosis.

Pain in the Chest and Abdomen. Either substernal pain in the chest, which is very common or generalised pain in the chest and pain in the abdomen are the common complaints, especially in the adults. The pain is very rarely constrictive

in nature. The pain increases whenever there is a rise in temperature.

Anorexia. The patient has no taste for food. The patient is also apathetic to take any food; when there is this complaint the patient has a feeling that the chest and abdominal pain will increase, if food is taken.

Gastro-Intestinal Symptoms. About 15 to 20 per cent of the patients, especially in the case of children, there is vomiting and diarrhoea which does not abate with any other treatment excepting the arsenical treatment. In one of my cases an Executive Engineer of an Engineering Contractor Firm (construction) complained of gas in the stomach, loss of appetite, distaste for food with occasional diarrhoea, easily getting exhausted and occasional constricting pain in the substernal region in the abdomen. There was occasional cough. When all the other treatments failed, when he came to me, I had his blood examined and found that he had 54 per cent Eosinophilia and the total count of W. B. C. was 42000 per c. m. m. The skiagram of his lungs showed bilateral mottlings, but not of the Miliary Tuberculosis type. Sputum contained plenty of Eosinophils and a few pus cells.

Cutaneous Manifestations. Occasional papular eruptions are present. Out of my 320 cases, I had occasion to come across of this manifestation in only 15 patients. The eruptions resembled more or less papular urticarial lesions or papular tuberculoids and some were of the appearance of erythema nodosa. The lesions disappeared after a few injections of arsenic and never reappeared.

PHYSICAL SIGNS

(1) *Broncho-spasm.* In the majority of the cases of more than 2 months duration, Broncho-spasm and Hyper-resonance of the chest with prolonged expiration, with sibilant and sonorous ronchi all over the chest are the prominent signs wherein they are mistaken for Asthma. Emphysematous changes in the chest are seen in the patients suffering from this disease for a number of years.

(2) *Congestion and consolidation of the Lungs.* These signs are occasionally seen in about 5 per cent of the cases, when they are mistaken either for pneumonia, or Bronchopneumonia or acute Bronchitis.

(3) *Splenomegaly.* In about 40 per cent of the cases the spleen is slightly enlarged. It is soft and tender on palpation. With the arsenical treatment the spleen returns to the normal size, but the retrogression is very very slow when compared to the disappearance of the other symptoms of the disease.

(4) *Lymphadenopathy.* In about 30 per cent of the cases the cervical and axillary glands are found to be enlarged which are hard and discrete. Occasionally inguinal and mesenteric glands are also similarly found to be enlarged which also disappear after the treatment.

(5) *Pigmentation of the face.* In fair skinned people there is pigmentation of the face. The skin is dry and lustreless. In about 20 per cent of the cases *Butterfly-Pigmentation* of the face is seen. This is more clearly seen in the fair skinned people. The butterfly appearance of the pigmentation is as follows: the two most prominent parts of the cheek below the

eyes form the wings and the bridge of the nose the body.

In 2 of cases I found slightly depigmented round patches about the size of the pin head all over the body, especially more prominent on the back of the chest. The appearance was that of the patches that are seen after an attack of Chicken-pox immediately after the falling of the crusts. On careful enquiry, I was informed, that these patches appeared after 2 months from the onset of dry hacking cough and wheeze. These patches disappeared after the treatment.

LABORATORY INVESTIGATIONS

Examination of the Blood. According to my statistics, the examination of the blood—blood taken on an empty stomach in the early morning without any exertion by the patient and not taking any medicine at least 24 hours before—shows increased W. B. C. from 14000 to 64000 per c. m. m. and the Eosinophils from 15 to 84 per cent, the increase of Eosinophils always at the cost of the Polymorphos mainly. But the Leucocytosis and the increase of Eosinophils had no any definite proportional bearing either with the lungs symptoms or other symptoms of the disease. There is proportionate increase of Eosinophils in the absolute count also.

Examination of the Sputum. When the sputum is present and examined under the microscope, Eosinophils are found in clumps together with epithelial cells. In 95 per cent of the cases Charcoat-laden crystals and Crushmans spirals, which are present in the sputum of Asthmatic and chronic Bronchitic patients, are

absent. It is one of the factors to be taken into consideration in arriving at the diagnosis, especially when the eosinophilic count is between 13 and 18 per cent and the total W.B.C. is somewhere about 13000 to 16000 per c. m. m. In all cases it was negative for A. F. B. No spirochetes were seen. Occasionally staphylo and streptococci are seen. Pus cells are present in about 50 per cent cases of more than 1 year duration.

E. S. R. In almost all cases it is very high ranging from 20 to 60 m. m. at the end of 1 hour (Westergren method. Normal 7 to 15 m. m. in the males and 12 to 17 m. m. in the females).

Cutaneous Test. In 15 cases of mine I tried intradermal injections of 2 or 3 drops of the patient's own serum in the fore-arm about one and half inches apart. The reaction was negative even after 48 hours. In 6 cases histamine sensitive test also proved negative.

Radiological Findings. In almost all the cases of more than 2 months duration, in the skiagram of the chest, diffused mottlings of the lungs, bilateral and symmetrical, more marked in the hilar regions are the common findings. The mottlings are coarse, unlike in the Miliary Tuberculosis and are of the size of half-split pea or big sago, usually called Sago-spots, with a central dense Zone and ill-defined blurred outline. In addition to these findings or occasionally independent of the above findings, linear markings of the lungs of increased density radiating from the hilum, fan shaped in appearance, are the common findings in almost in all the cases. The right side of the heart, especially the right auricle

is always enlarged unlike in tuberculosis where the heart is invariably pyreformed in shape.

DIFFERENTIAL DIAGNOSIS

(1) *From Asthma.* In Asthma high Leucocytosis, that is beyond 14000 per c. m. m. and high percentage of Eosinophils (i.e.) above 15 per cent is very rare. The dyspnoea and the paroxysm of cough are very severe but not restlessness, anxiety and suffocation as it is in Asthma. In this connection it will be worthwhile for us to know the description and the clinical picture of Asthma in the words of Hyde Salter, who himself was a sufferer. It runs as follows:

"With a face expressive of the intensest anxiety, unable to move, speak, or even make signs, the chest distended and fixed, the head thrown back between the elevated shoulders, the muscles of respiration rigid and tightened like chords, and tugging and straining for every breath that is drawn, the surfaces pallid or livid, cold and sweating—such are the signs of which the dreadful suffering manifests itself.....to be fairly imagined it should be witnessed—to be fairly appreciated it should be experienced."
(BEMP. Vol. II)

In Asthma there will not be Splenomegaly and Lymphadenopathy. The absence of familial history of allergy and rhinitis will be additional factors to come to a diagnosis.

(ii) *From Leoffler's Syndrome.* In Leoffler's Syndrome, the Leucocytosis and Eosinophilia are generally mild and transient without splenomegaly and with a tendency for spontaneous speedy recovery without any treatment are the points to be taken into consideration;

the lung shadows are also very fleeting and transient.

When the percentage of Eosinophils is between 13 and 18 per cent and the total count and W. B. C. is somewhere about 15000 to 16000 per c. m. m. it is very difficult to come to a diagnosis, in which case stools should be examined for Round-worms and Hook-worms, blood for Filaria, before we come to a definite diagnosis of Eosinophilic Lungs. The skin condition also should be taken into consideration.

(iii) *From Tuberculosis.* To differentiate from pulmonary tuberculosis, especially the Miliary Type, it will not be very difficult, if we remember that the Coarse Sago-spots or the Split-pea spots, dense in the centre and blurred and indefinite at the periphery. Invariably in tuberculosis the heart will be pyreformed in shape and in Eosinophilia the heart will be enlarged on the right side especially the right auricle. In pulmonary tuberculosis the sputum and or faeces on repeated examination will invariably reveal the presence of A. F. B. The Leucocytosis and percentage of Eosinophils will not be very high. On the other hand, the Lymphocytes and the Monocytes in the advanced stages of tuberculosis will be increased in numbers.

Definite diagnosis can be made on blood and sputum examination along with the skiagram of the chest. On clinical grounds alone it is very difficult to come to a diagnosis.

Treatment. Once the diagnosis is made the treatment is very easy. Arsenic is the treatment of choice for complete cure. Autohaemotherapy, Adrenaline

Antihistamines, Cortisone preparations, A. C. T. H., Ephedrine, Penicillin and the other anti-biotics are of no use. Sometimes any one of these may give some relief, that too for a short period - a few days or weeks; after sometime the disease may reappear with all its virulency and vengeance. Even in arsenical preparations, Pentavalent Arsenic seems to be more effective than the Triavalent. Even in the Pentavalent preparations intravenous route of administration seems to be more effective than the oral or intramuscular route. Of course all the precautions that are necessary in giving the arsenical preparations have to be taken. The urine for albumin, blood and casts, the eye, the oral cavity, the liver and the skin condition should be carefully examined before the arsenic treatment is commenced.

If the patient is very weak and anaemic before the arsenic treatment is given, the patient has to be treated for anaemia by giving Cyanocobalamin (B. P.) and Liver Extract. The number of injections depends upon the condition of the patient. If there is intractable diarrhoea that has to be treated according to the condition. Stomatitis has to be treated with Nicotnamide and plenty of Vitamin C. At least 500 mgm of Vit. C should be given in a day. If there is failing vision, of course arsenic treatment should not be given but only palliative treatment has to be given. If the patient is very weak, a good preparation of a whole protein food of very high biological value containing all the essential amino-acid and Vitamins should

be given along with plenty of Vitamin C. If scabies are present, it has to be treated first.

When the patient is fit for Arsenical treatment, in the case of patients of 14 years and above on an average 12 intravenous injections are given once in 4 days, 6 being calcium glucono-galactogluconate 10% 10 cc with Vitamin C 500 mgm (of dependable make) and the other 6 being the solution of Mapharside in calcium glucono galacto-gluconate 10 per cent 10 cc with Vitamin C, alternating with each other, commencing with Calcium glucono galacto gluconate 10 per cent 10cc with Vitamin C 500 mgm. Careful watch should be kept on the patient for any untoward reaction or intolerance of arsenic. The urine should be examined everyday the arsenic is being given. If there is albumin or renal casts, that condition has to be treated first before arsenic is given. It is absolutely necessary for the urine to be examined on every arsenic-day, otherwise serious complications will arise and arsenic treatment will become unpopular for no fault of the drug. In the meantime, some protective treatment must be given to relieve the patient of the distress. When there is reaction, the interval in between the arsenic injections should be increased and Vitamin C should be given in plenty by mouth. The patient should be sufficiently warned about the arsenic reaction and should be asked to have complete rest on the day of injection and to report to the doctor, for any intolerance, either mild or severe. In spite of all these precautions, if the

patient cannot tolerate arsenic, it has to be discontinued and some other palliative treatment such as autohaemotherapy or Diethylcarbamazine may be given. After finishing the 12 injections, the patient is put on Leucarson 2 grains and 200 mgms of Vitamin C immediately after the 2 principal meals for the first ten days. From the 11th to the 20th day the patient is put on plain Vitamin C 400 mgms only. Then from the 21st to the 30th day, once again Leucarson and Vitamin C are administered as on the first ten days. A week after finishing the administration of the Leucarson and Vitamin C, once again the blood has to be examined and if the total count has come down to 10000 per c.m.m. and the Eosinophils 10 per cent, all further treatment is stopped. If the total Leucocytes and the Eosinophils are above the limit mentioned above, it is better to give one or two Mapharside injections intravenously alternating with Calcium-Vit. C, the number of arsenic injections depending on the blood findings.

In the case of the patients between 12 and 14 years, it is better to give Acetylarsen with Vit. C 200 mgm intramuscularly once in 5 or 6 days, in all about 6 to 8 injections, the number of injections depending upon the findings of the blood.

While giving Acetylarsen injections all the precautions that are taken in the case of adults should be followed. In the case of children between 4 and 11 years of age, Child dose of Acetylarsen with Vit. C 200 mgm should be injected once in 6 days and in the case of patients between 11 and 12 years of age three-

fourth adult dose of Acetylarson with Vitamin C 200 mgm is given intramuscularly once in 5 or 6 days depending on the condition and the reaction. The number of injections has to be decided on the condition of the individual. For the patients between the ages 4 and 8, $\frac{1}{2}$ to $\frac{3}{4}$ grain of Leucarson with 200 mgms of Vit.C is given and for the patients between the ages 8 and 14, 1 grain of Leucarson with 200 mgm of Vitamin C is given, the mode of administration being the same as in the case of the adults. Here also, as in the case of adults, one week after the cessation of the arsenic oral treatment, blood should be examined and if necessary further arsenic treatment should be given depending upon the blood findings.

Diet: During and after the course of treatment the patient is advised easily digestible diet with plenty of green vegetables, fresh butter-milk and milk preparations. In the case of non-vegetarians lean meat, river fish, liver and bone soup may be taken. The patient is warned not to take preparations, made in oil, rooty vegetables and sweets; is also advised to abstain from oil bath at least for 3 to 4 years, if not more. In my experience I have found the day on which the patient takes the oil bath, in that night and subsequent days the paroxysmal cough once again appears with much intensity and these are flaring up of the Eosinophils. The patient is also advised some good protein preparation with Vitamins and minerals, Vitamin B Complex, Vitamin C and easily assimilable Calcium and nourishing tonics. OIL, OILY PREPARATIONS, SUGAR & JAGGERY PREPARATIONS, ROOTY VEGETABLES, MANGO & JACK FRUITS, OIL BATH AND SOUR BUTTER-MILK SHOULD BE COMPLETELY AVOIDED.

Six months after the cessation of the arsenic treatment, once again the blood should be examined and in the majority of cases the blood picture is normal if the patients have regularly followed the instructions. They are invariably free from further trouble, provided they follow the diet restrictions and no oil bath is taken.

Though some members of the medical profession are against arsenic treatment I am strongly of opinion, because of my experience, and that is also the opinion of some of the senior members of the profession, that if proper precautions are taken and the proper dosage of arsenic is given, it is absolutely harmless and there will not be any occasion to feel sorry for instituting arsenic treatment. On the other hand the patients will feel highly grateful for the medical man for saving him from this troublesome annoying malady. As for my own part, I have not felt sorry for giving Arsenic treatment even once, when I have treated 320 cases in the course of 14 years.

Thesis. The name is a misnomer. The Aetiology is obscure. It has nothing to do either with Bronchial Asthma or Pulmonary Tuberculosis or Loeffler's Syndrome, once as it was thought to be. Blood and sputum examination together with the skiagram of the chest is absolutely necessary to come to a definite diagnosis. Pentavalent arsenical preparations in combination with calcium and Vitamin C administered intravenously seems to be the treatment of choice in preference to the other arsenical preparations. Oil bath, food prepared in oil, sweets, farinaceous foods seem to aggravate the condition. Since the Eosinophils are always increased at the cost of Polymorphos, it requires some special investigation with regard to the conditions affecting the bone-marrow.

Trachoma and Achromycin *

By

DR. J. M. PAHWA, M.B.B.S. (LKO), D.O. Z.O. (Vienna) M.S., (Luck) Eye Hospital, Sitapur

TRACHOMA. A chronic contagious disease of human conjunctiva and cornea, is perhaps the most terrible and tenacious scourge of mankind. It is a greatest single cause of blindness and economical loss in our country. During our ophthalmic survey of 1408 school children of NES Block Hargoon (Sitapur) we found that 57.5% of them were suffering from trachoma in the following stages:—

Stage 1...16. 9% }
Stage 2...54. 2% } (Maccallan's Type)

Stage 3...22. 3% }
Stage 4... 6. 6% } (Maccallan's Type)

From the survey, we came to the conclusion that younger the patient, more active was the infection. In high school elderly children, the disease had passed on to the chronic stage or some of the cases had gone on to spontaneous resolution. During our complete survey in that Block we found the following figures.

Age.	Trachoma	Predominant stages	Associated conjunctivitis
0 to 6 Years	60%	1st and 2nd Stage	68%
6 to 15 Years	68%	3rd and 2nd Stage	23%
Above 15 Years	90%	4th and 3rd Stage	17%

It is sufficiently clear from the above figures that in infants and children trachoma was in the active stages, moreover in adults and mothers it was found to be comparatively quiet and little infective with no inclusions. It is possible that these infants contract trachoma from young children in the family, though the possibility from the mother cannot finally be ruled out.

The onset is often preceded by acute conjunctivitis while in other cases the onset is insidious. I think it all depends upon the amount and virulence of virus and the condition of the conjunctival epithelium. The more massive the former and healthier the latter, the

greater will be the tendency of the onset to be acute. The associated conjunctivitis was mostly staphylococcal and occasionally of Koch-Waks type.

During our survey we also found that trachoma was much less in Eastern districts (Gorakhpore and Azamgarh etc) though living conditions and personal hygienic factors were in no way different from those prevailing in Western districts where trachoma is in abundance.

We are launching a research scheme to solve this problem with the help of the Geological department etc. (Temperature, humidity, Silica content of land, fly index, living and economic conditions of the persons and infectivity of virus etc.).

*Read during Trachoma Seminar for May, 1957, held in Eye Hospital, Sitapur.

It is almost certain that trachoma is a virus disease. Several workers have even demonstrated the presence of trachoma virus in the follicles by electronic microscopic examination. The trachoma virus is said to be allied to Rickettsia bodies (chlamydozocale group). The inclusion bodies which consist of initial body of Lindner (basophilic) or elementary granules of small size (Eosinophilic) indeed 2 phases of one infective unit are strictly limited to the superficial epithelial cells obviously because the nutritive condition over there are most favourable for the growth and multiplication of its virus which never takes place outside the cell.

Clinically it manifests itself into four typical stages (Maccallan) but I should like to point out that the disease very often starts or even may be worse in the lower lid, so its examination is very essential. Herbert's pits in the lower limbus are not rarity during routine examination. Upper fornix must be carefully seen with the help of a lid hook to achieve complete eversion. Following clinical signs are of diagnostic value:—

1. Follicles behind the tarsal conjunctiva.
2. Limbal follicles and Herbert's pits.
3. Infiltration and vascularisation of the upper half of the cornea. Look for it by a loupe or slit lamp.
4. Thickening and hypertrophy of the tarsus and sinuous lid border.
5. Inclusion bodies in the scrapings from the epithelium of conjunctiva or cornea. These are readily found in early

cases, so that only positive results are reliable. Negative results are uncertain.

6. Scar formation on the upper tarsal conjunctiva in the form of Arlt's line or star-shaped scar.

7. Degeneration of Follicles. Trachoma is the only disease in which follicles undergo degeneration.

Combination of Pannus with any of these signs is pathognomic.

TREATMENT: During the last decade the fight against trachoma has been greatly intensified.

Chemotherapy and recent anti-biotics have changed its gloomy outlook. A day should not be far off when trachoma will no longer remain unconquered and feared of as was once pneumonia in pre-sulphonamide and pre-penicillin days. Physical, biological and other destructive methods used previously in its treatment have absolutely no place in a modern clinic, but silver and copper have their indications still. former in its acute stage with some discharge and latter in chronic Follicular and Scarring stage.

Due to wide prevalence of blindness resulting from trachoma, a search for an effective therapeutic agent is constantly going on. The present study has been undertaken with Achromycin (Lederle) which is a crystalline broad spectrum anti-biotic. It is more stable than Aureomycein but it is almost similar in its spectrum of anti-bacterial activity though it differs slightly in its structure. Experimentally it has been proved beyond doubt that it reaches the aqueous and anterior part of the eye in effective concentration after local drops

and sub-conjunctival injections etc. Leopold has shown in rabbits that corneal bath or eye drops in concentration of 25mg per cc produce very effective corneal levels for 4 hours. Aqueous levels are adequate upto 2 hours in the intact and upto 4 hours in the inflamed eye. Sub-conjunctival injections in concentration upto 5mg per cc are safe, while eye drops in concentration upto 25 mg per cc are innocuous and intra-corneal injections in concentration greater than 1 mg per cc are dangerous.

The anti-bacterial spectrum of Achromycin includes a majority of eye pathogens. Good results have been met with in bacterial conjunctivitis caused by *E. Coli*, *H. Influenza*, *Staphylococcus Aureus*, *Micro-coccal Pyogenes*, *Streptococcal Hemolyticus* and others. Though we have tried this drug in many ocular conditions but its study has been particularly made towards trachoma by

local therapy. Our results have indeed been very favourable in cases of Blepharitis, particularly of Staphylococcal ulcerative type which was resistant to many therapeutic agents.

We divided our patients into 2 groups for purposes of study and evaluation of results:—

Group 1:— Local drops of 1% oily Achromycin solution twice daily.

Group 2:— Local drops as in group 1 but with sulphadiazine tablets by mouth (2 tabs. 3 times a day for 7 days followed by a similar course after a gap of 7 days).

We soon realised that the results with the second group were more favourable; so later on we have made our study with this combined therapy in 50 cases of trachoma. Table 1 given below gives the figures of patients treated in various stages and the results of this with combined therapy.

Table 1.

Stage of Trachoma.	Total No. of cases.	Inclusions Present.	Cured.	Improved.	Not improved.
1.	6	5—80.00%	6—100.00%	Nil	Nil
2.	30	18—60.00%	21—70.00%	6—20.00%	3—10.00%
3.	14	2—14.30%	3—21.40%	5—36.70%	6—42.90%

Cases cured were confirmed by cortisone tests i.e., instillation of 0.5% cortisone into the conjunctival sac 3 times a day for 5 days showed no relapse of

symptoms and scrapings were negative.

Table 2 shows the figure of number of cases cured 30 cases (or 60% in week's with this combined therapy:—

Table. 2

Stage of trachoma.	TREATMENT GIVEN FOR WEEKS.						
	3	4	5	6	7	8	more than 8
1.	4	1	-	1	-	-	-
2.	2	5	5	6	3	-	-
3.	-	-	-	1	-	1	1

Follicles disappear much earlier than papillae which behave quite indifferently, earliest they disappear was within 25 days but majority of cases took even more

than 30 days and in several cases they did not disappear at all even after 42 days.

Following is the response table of these patients:—

1. Symptoms disappeared :—	3—7 days	= 4.5 days average.
2. Corneal ulcer healed :—	4—10 days	= 6.9 „ „
3. Pannus disappeared :—	11—17 days	= 13.1 „ „
4. Follicles disappeared :—	15—25 days	= 18.7 „ „
5. Papillae disappeared :—	25—42 days	

Three cases of trachoma in second stage which were not responding or showing delayed response to Aureomycein showed marked improvement to Achromycein.

Toxic reactions to local Achromycein eye drops and ointment used in the study were rare. No reaction was observed in any case. We all know that essential cure i. e., eradication of causative agents precedes clinical cure particularly in chronic cases where inflammation is proliferative in character. The drug has been particularly found to be effective in trachomatous keratitis where pannus retrogresses rather quickly. Secondary infection associated with it always clears up within short time.

SUMMARY:

Achromycein (Ledrlé) is a useful drug in the treatment of ocular infection. It is found to be effective particularly in Staphylococcal infection leading to

Blephritis with or without dermatitis of lids.

2. It has no irritating effects on the eye and maintains its efficacy for a longer time than many other antibiotics.

3. It is effective in combating the trachoma virus and practically every case of acute trachoma can be cured in 2 to 4 weeks time. In chronic cases the results have not been so encouraging, and moreover it requires prolonged treatment.

4. It is found to be effective in 60% cases of trachoma.

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“Injured Elbow”

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(Continued from April '58 Issue)

3. *Reflex vasospasm.* Injury to the wall of the artery or thrombosis within the arteries sets up reflex vasoconstriction which affects not only the vessel itself, but all the arteries of the limb including those upon which the collateral circulation depends.

Volkman's Ischaemic Contracture: This is one of the dreaded complications of fractures around the elbow.

In 1881 Volkman wrote “For good many years, I have observed that paralysis and contractures of the extremities, which one sees after the use of tightly applied bandages, are not as people had previously thought, due to paralysis of nerves through pressure but to a sudden extensive disintegration of contractile tissues. The paralysis and contractures must be accepted as purely myogenic. They occur as a result of prolonged deprivation of arterial blood. The frequent, simultaneous and often very marked stasis seems only to accelerate the occurrence of paralysis.

Little has been advised or changed in his summary of the etiology and pathology of Ischaemic contracture.

This complication most frequently follow supracondylar fracture. Backward

displacement of the small fragment stretches the artery across the fracture site causing contusion laceration or complete severance.

Early Clinical Diagnosis: Earliest signs usually appear within 4-6 hours after the injury or treatment of fracture. A few cases have been reported in which the onset occurred after 48 hours; and others after one week.

The characteristic symptoms and signs may be summarised as Pain, Pallor, Paralysis and Pulselessness. Pain is variable. The color of the hand may be cyanotic or pallid, in the initial stages. The former suggests a venous and the latter an arterial type of onset. Paralysis is relatively late in onset. Pulselessness is invariable, insensitivity of the fingers and immobility in a slightly flexed position are late signs. Radial pulse is unreliable as a danger signal. Its absence is not an indication for interference, nor its presence is a guarantee that ischaemia will not develop. If the capillary circulation is good and there is no pain of paralysis, Volkman's will not develop.

Treatment of established deformity: Once established treatment of the deformity is highly unsatisfactory.

Robert Jone's Method: With the wrist flexed, straight spatula splints are applied to each finger; a full length splint is then applied to the forearm and hand and the wrist is gradually dorsiflexed.

Another method consists of supporting the wrist in a splint in a very slightly dorsiflexed position and apply elastic traction loops to each individual finger. Improvement may be expected in 2 to 3 years. Residual deformity is then treated by lengthening the tendons or shortening the forearm bones.

Max Page (1923) advocated muscle-slide of the forearm muscles from the medial condyle of the humerus.

Pathological changes in the Muscles: At the onset the muscles are fulum colored, indurated, and tense with the fascial sheaths marked extravasation of blood and serum about the fibres. yet a true haematoma does not form. Within 48 hours great number of polymorphs accumulate about the fibres adding an inflammatory aspect to the picture. Even at the early stage many of the muscle fibres are necrotic showing fragmentation and disappearance of nuclei.

Organisation of the exudate begins within 4-5 days and well established within ten days. The number of destroyed fibres varies with the severity of the original lesion.

Histologically the connective tissue forms bundles and bands which remify throughout the muscle belly and replace the dead muscle fibres. The persistence of some living fibres explains why the muscle may regain some of its functions.

Nerve lesions associated with Volkmans. In the mild cases the deformity may be due to muscle retraction alone. In the more severe once, it is due to continuation of muscle retraction of forearm flexors and flaccid paralysis of the hand muscle, the latter resulting from peripheral nerve involvement.

The early subjective symptoms are numbness, severe pain and hyperaesthesia of the hand may be ascribed to compression of the nerves between indurated muscle bellies in addition to the fact that the amount of blood of the entire part has been diminished.

As the exudate within and between the fascial sheaths of the muscles become organised, the nerves are caught in the fibrotic process and are virtually strangled. This so called secondary involvement of nerve trunks is permanent and severe. They are sclerotic and reduced in size.

The median nerve is the most frequently involved, whether the contracture follows an elbow or forearm fracture. The ulnar nerve may be affected as well but the severity of involvement is less than that seen in the median nerve. Lesions of the radial nerve are rare and are seen only in the early stages.

Characteristic of uncomplicated Volkmans is the fact that when the red point in the fibrotic process has been reached the muscle retain some degree of contractility. This indicates that the nerve supply to the flexor muscle is intact.

Volkmans must be distinguished from that due to median nerve injury at the elbow due to fracture. All the branches

to the flexors of the wrist arise below the elbow joint. Because of this anatomical arrangement and because some function persists in the flexor muscles of the forearm, the clinical picture of Volkmans cannot be simulated by direct injury to the median nerve at the elbow.

In irritative nerve lesions, extensive fibrosis develops in the muscle bellies and joint capsules, resulting in severe contractions. In the classical Volkmans the changes within the muscle bellies occur within a few days from the onset of the symptoms; in nerve lesion the fibrotic changes take much longer to mature. Also the dense phlegmonous induration, palpable in the flexor bellies which is characteristic of Volkmans is not seen in uncomplicated nerve lesions.

Treatment of circulatory disturbances: If there is evidence of circulatory embarrassment and threatened ischaemia, all bandages are removed from the elbow and forearm; plaster if applied should be bivalved. The angulation of the elbow is reduced and ice bags applied to the part. Satisfactory position of the fragments and elevation of the forearm are maintained by keeping the limb in Thomas arm splint and applying skin traction. Alternately the extended limb is suspended from an upright fixed to the bed post. Loss of position of the fragments is insignificant compared to the permanent disability following unrelieved ischaemia.

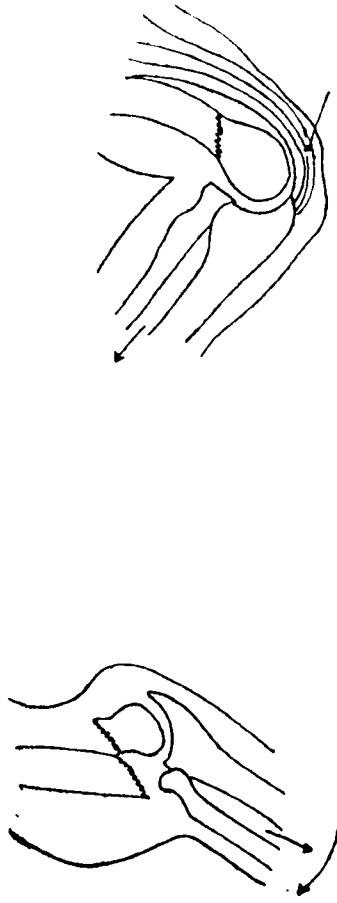
Intravenous papavarine and nicotinic acid and sympatholytic drugs like

Tetra-ethyl ammonium bromide can be tried to prevent reflex spasm and to produce vasodilatation of the collateral vessels.

Blocking the sympathetic ganglia also may be tried. If the symptoms are well advanced or do not subside with conservative measures, no time should be lost before exploring the cubital fossa and volar aspect of the forearm. The tough fascia enclosing the flexor muscles of the forearm is slit to permit the extension of edematous muscles and haematoma and thus relieve tension. The brachial artery is then exposed and the damaged segment excised to relieve vasospasm and reflex involvement of the intimate vasculature of the muscles. Delay is disastrous. Within 3-4 hours irreversible changes here take place. The all too frequent claw hand means inadequate or delayed treatment.

Rupture of Brachial artery following Supracondylar Fracture: Only half a dozen cases are reported in the literature.

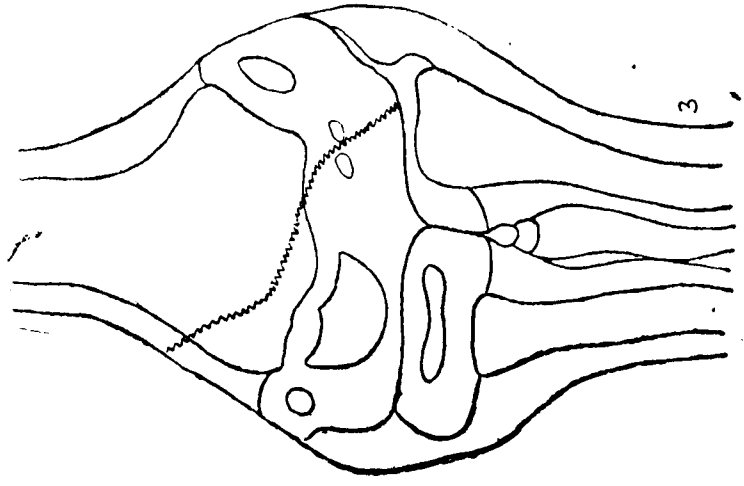
Rupture of the brachial artery in association with supracondylar fracture or dislocation of the elbow should be suspected whenever there is compound wound of the antecubital space. In the cases of closed injuries, this complication must be suspected whenever there is swelling and echymosis about the elbow together with obliteration of radial and ulnar pulse. Accurate distinction between traumatic vasospasm, arterial contusion or laceration or division of the artery can be achieved only by surgical exploration.



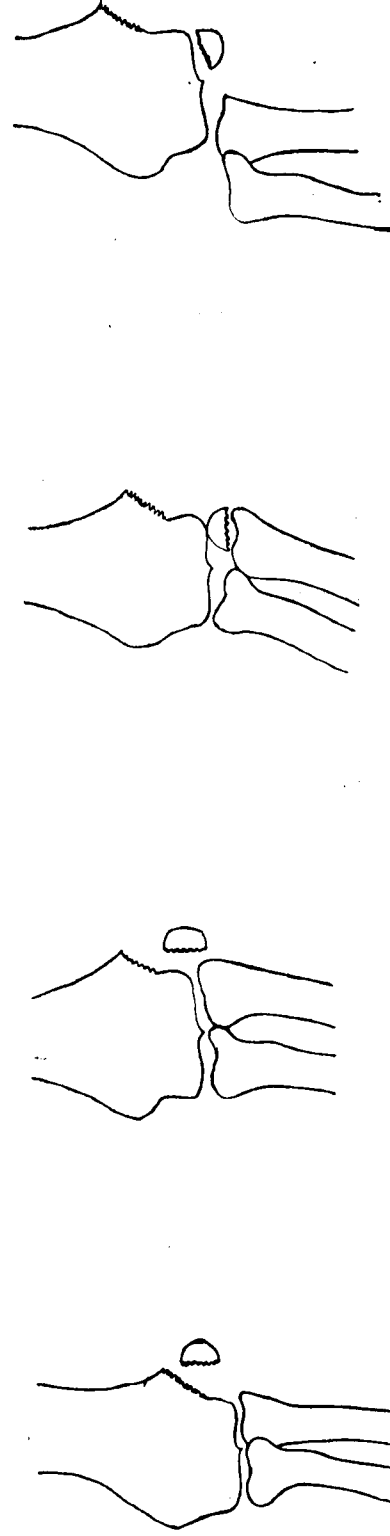
In a Supracondylar fracture the small fragment is usually displaced backwards, after traction this is corrected by flexing the joint.



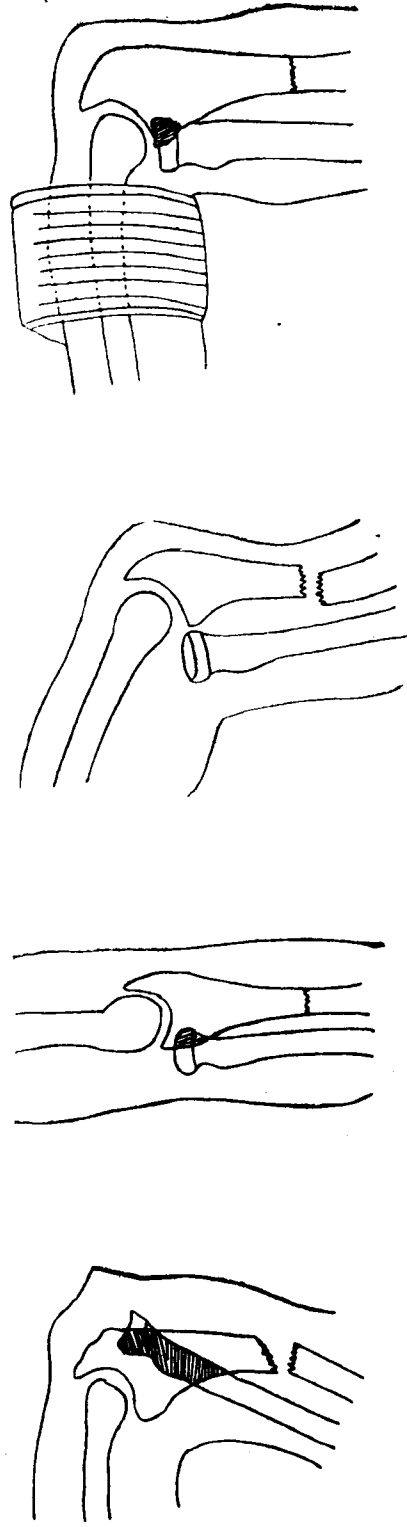
Occasionally there is forward displacement of the lower fragment, after traction the joint must be extended.



Line of fracture in separation of lateral condylar Epiphysis.



The four degrees of displacement of the epiphysis of the internal epicondyle.



Flexion Type.

Extension type Monteggia Fracture dislocation.

Once the condition is recognised Treatment is simple. Any artery in the upper extremity can be ligated and divided with relative impunity because of the presence of superabundant collateral circulation. Unless a satisfactory closure of the severed vessel can be performed simple ligation and division of the lacerated artery is the procedure of choice.

Nerve Injuries in Supracondylar Fracture: Nerve injuries complicating supracondylar fractures may be classified chronologically into two groups. (A) Primary nerve injuries sustained at the time of the bony injury by ischaemia, compression, contusion, traction or laceration of the nerve. (B) Secondary nerve injuries developing some weeks or months as a result of friction.

Primary Nerve Injuries: Any one of the important nerves crossing the elbow joint may be involved. They may be affected in the following ways.

1. *Ischaemia:* Loss of blood supply for 30 minutes causes temporary paralysis of two sensory and motor nerve endings. Ischaemia for a longer time may cause wallerian degeneration.

Ischaemic lesion is a lesion in continuity and hence recovers spontaneously within a few months.

2. *Compression Nerve Lesions:* The nerves may be contused by a dislocated or fractured bone. In about 15% of supracondylar fractures the median or ulnar nerves are contused by the lower margin of the main fragment of the humerus. The ulnar nerve is particularly liable to injury, when the distal

fragment is displaced forwards and outwards. Recovery usually complete within a few weeks or at the most within three months. No treatment is required other than reduction of the fracture. The elbow should be immobilised in extension after manipulative reduction of the fracture.

3. *Traction Nerve Injuries:* Ulnar nerve because of the peculiarity of its position is particularly susceptible for this.

Watson Jones in 1930 after analysing 5000 cases of elbow injuries, has come to the conclusion that primary ulnar palsy of elbow injuries has exactly the same pathogenesis as the delayed ulnar palsy, associated with cubitus valgus in ununited supracondylar fracture. Owing to the fixation of the nerve in the supracondylar groove, with its roof of fibrous tissue, the carrying outward of forearm into valgus position produces a direct traction on the nerve trunk: if on the other hand, the forearm is carried backwards and outwards, the roof of the supracondylar groove is torn and the nerve escaping backwards from its groove gains free mobility.

On the other hand, if the forearm bones are displaced outwards, the nerve is stretched over the back of the lower end of the humerus. Supracondylar fractures with forward displacement is an unusual fracture but when it occurs there is high incidence of ulnar paralysis.

Traction nerve injuries like those due to contusion are lesions in continuity and spontaneous restoration of function takes place.

Laceration of the nerve: Median Nerve: The median nerve paralysis in supracondylar fractures is usually due to a simple contusion. But occasionally the nerve may be severed over the sharp lower margin of the proximal fragment. Suspicion of laceration is aroused when there is marked displacement, the sharpness of the proximal fragment, completeness of paralysis, or the delay in recovery. If there is no recovery at the end of three months the nerve should be explored.

Secondary complications: These are myositis ossificans and secondary nerve lesions.

Myositis Ossificans: This is one of the easily avoidable complication. The presence of myositis shows mismanagement of the case often due to ignorance on the part of the patients.

It consists of formation of masses of new bone around the joint. The title Myositis Ossificans is a misnomer, as there is neither myositis nor ossificans in the muscles. It is more appropriately called "Traumatic Subperiosteal Ossification".

Pathogenesis: It was once thought that as a result of injury, osteoblasts escaped from the bone into the muscles, and lay down bone there. This view is no longer popular. It is now accepted that new bone formation occurs only within the limits of the displaced periosteum and the so called myositis ossificans is nothing more than ossification of subperiosteal haematoma. If displacement of periosteum is prevented and extensive haematoma are not allowed to occur this complication is never seen.

This complication is most often seen after the injuries around the elbow. The condition is more frequent after dislocations than in fractures because muscle and capsule insertions into the periosteum are invariably avulsed in a dislocation.

The two causes of ossification of the subperiosteal haematoma are 1. Delay in reducing the fracture or dislocation because the longer the periosteum is displaced the more advanced is the ossification of the haematoma. 2. Passive stretching and forcible vigorous movements soon after the reduction of the fracture, before the periosteum is firmly reattached to the bone.

It is the passive stretching and non-active exercises that redispaces the periosteum.

Treatment of myositis ossificans: Myositis is an entirely preventable condition. The most important thing is to stop the massage, force movements and manipulations. The task of the surgeon is not over after reduction of the fracture or dislocation. It must be his fundamental duty to instruct the parents that under no circumstances, massage be used. There is nothing that massage can do which active movements cannot. On the other hand, massage increases the stiffness, muscle spasm and invariably ends in myositis.

In an established case of myositis, the patient is advised to do active movements. No force is allowed at any time. The futility of carrying buckets of water or bags of sand, a hanging from the overhead beam, in the hope

SUPRA - CONDYLAR FRACTURES



Mrs. Naik. Supracondylar fracture before treatment.



Xray appearance more than 6 weeks after treatment by traction in extension. There is evidence of good calus formation.



Range of elbow movements nearly 7 months after injury.

that the joint will straighten out must be explained to the patient. The patient must regain movements by his own effort.

Absolute immobilisation of the elbow in a P.O.P. cast is advised by some. But this is not sound in principle as complete immobility allows consolidation of adhesions which had already occurred and permanent stiffness may then result.

As time goes on the patient gradually gains movements, and the mass of bone gets reduced in size.

There is no indication for operation until spontaneous recovery of movements as shown by the actual measurements ceased. Any bone spur that may be obstructing the movements may then be removed. But ultimate prognosis even after the operation is uncertain as the joint stiffness may be due to already existing adhesions.

Secondary Nerve Complications: These nerve complications occur a few weeks or months after the original injury. They are due to various causes, the chief among which are friction, compression and traction as a result of deformity.

1. Friction Nerve Complications: A fracture or dislocation causes roughening of the floor of the post-condylar groove and friction of the nerve on the roughened surface leads to neuritis. This is aggravated by massage and forced manipulations.

2. Compression: The nerve may get caught up in the callus, or pressed upon by scar tissue. Neurolysis may lead to spontaneous regeneration of the nerve.

3. Deformity: Malunited supracondylar fractures with cubitus valgus deformity leads to stretching of the ulnar nerve and gradually produces a late ulnar palsy. This is treated by anterior transposition of the nerve.

Flexion Contracture of the Elbow resulting from Supracondylar Fracture: Flexion contracture of the elbow resulting from fractures in that region are due to one of the following conditions:

1. Irregularity of joint surfaces with loss of cartilage or fibrous adhesions.
2. Malunited fractures with bony projections or excessive callus forming a bony obstacle.
3. Fibrous thickening and contractures of the capsular ligament causing loss of extensibility.

Following the fracture, functional recovery is complete at the end of one year and whatever limitation of movement is present at the end of that period is likely to be permanent.

Diagnosis of causes of Flexion Contracture:

Clinical Examination: In Bony Block Movement is limited abruptly when the capsule is contracted; the stoppage is of the less sudden and more elastic type. When limitation is present in both flexion and extension, it is a sign of intra articular damage.

X'Rays: Narrowing of the joint space or irregularity of the articular surfaces point to intra-articular involvement. If the flexion deformity is diagnosed to be due to contracture of the capsule capsulectomy is done.



SUPRACONDYLAR T-SHAPED FRACTURE

Simhachalam-Supracondylar T-shaped fracture before treatment.



Appearance 2 week after application of traction in extension in Thomas arm splint. A. P. and lateral views.



Xray taken nearly 7 months after injury. There is good union of fracture; the projecting spur of bone is getting absorbed.



Range of movements 3 months after treatment.



Tourniquet applied-Elbow exposed between the brachioradialis and brachialis be a vertical incision, radial nerve, retracted aside and the contracted capsule, along with any projecting piece of bone excised. The elbow should not be stretched all atonce as it may cause circulatory disturbance and damage to the nerve by forcible stretching. The elbow is immobilised at about 120° for a week or two and later extension gained by active movements.

The vertical incision crossing the elbow may cause some scar and contracture later on. Hence, the elbow can be exposed by an incision on the medial side of the elbow, and joint exposed by stripping the muscles in front of the elbow.

SUPRACONDYLAR T—SHAPED FRACTURES OR INTERCONDYLAR FRACTURES OF THE HUMERUS:

T shaped fractures occur more often in adults than in children. They are caused by a fall on the outstretched hand or the point of the elbow, the olecranon being forced upwards as a wedge between the Humeral condyles, piercing them apart and displacing them upwards and forwards. Displacement is considerable and the fracture is unstable.

Most brizarre deformities follow this type of fracture because of the complex muscle attachments around the elbow. Contraction of the triceps posteriorly and biceps and brachial anteriorly is the chief cause of the deformity. In addition the attachments of pronators and flexors on the trochlea and of supinators and extensors on the capitulum tends to exaggerate this deformity.

Treatment: It is a difficult fracture to treat and the diversity of views on the subject is an indication of the poor quality of results often obtained. In the words of Watson Jones "No injury of the elbow gives rise to more difficulty in treatment. It may be impossible to secure perfect alignment by manipulation and traction. Operative reduction is very difficult. In involves free dissection of the triceps and the capsule of the joint and there is usually no stability of the fragments, so that reduction can only be obtained by plates and screws. Adhesions of the tricep, contraction of the capsule and a vascular necrosis contribute to add to adhesion formation and permanent stiffness. Despite the perfect anatomical reposition which may have been secured, the final range of movement is less satisfactory than after manipulation. Operative reduction is not justifiable by results. Two alternatives are available, 1. To secure best possible apposition by manipulation and traction. 2. In grossly committed fractures with irreparable joint injury to perform an immediate arthroplasty".

Manipulative Reduction: Manipulative reduction followed by plaster application is too often followed by joint stiffness. Hence it is not recommended.

3. Rudolph Scott (1936) advocated the following method. The medial and lateral condyles are exposed and one prong of ice-tong is inserted into each. The arm is then introduced in a Thomas arm splint and traction applied by tying the end of the splint. By means of forward pull on the forearm,

the angulation deformity is corrected. This position is maintained for six weeks, following which the tong and splint are removed and active movements commenced.

But this method cannot be recommended as it too often causes stiffness and infection. Besides, it cannot be used in the presence of soft tissue injury and marked comminution. In the words of Watson Jones, "continuous skeletal traction by a pin is mentioned only to be condemned. It combines the worst features of all treatments. It is uncomfortable, unwise and unnecessary."

East-Wood (1937) treated 92 cases of T shaped fractures with satisfactory results by a method first recommended by H. O. Thomas and later popularised by Sir Robert Jones.

Under general anaesthesia the displaced fragments are manipulated into a better position, by means of strong tractions which the operator applied by gripping the forearm in the upper 1/3. At the same time the fracture is compressed laterally in an attempt to close the gap between the two condyles. The wrist is then slung from the neck in a collar and cuff, the angle between the forearm and humerus being less than 60. The success of treatment depends on gradual traction by the force of gravity than on the preliminary reduction, which can be frequently omitted without endangering the end result. Movements of hand and fingers are encouraged from the first day but the shoulder is not moved during the first fortnight, after this shoulder movements are permitted. At the same

time, gentle active movements of the elbow from the position in which it is found towards the position of full flexion are permitted provided these movements are painless. Failure to obtain satisfactory range of movement may be due to

(a) Gross distinction of bone including the head of the radius and the surrounding soft tissues; (b) Fixation of the elbow at force apart the fragments of the lower end of humerus; (c) Too long a period of immobilisation after bony union has begun.

This method of treatment, otherwise called "Bag of bones technique" is very simple and gives better results than operative reduction. It is particularly useful in the elderly as considerable range of movement may be obtained. The joint is however unstable and much power is lost. Besides, the tendency to forward tilting of the lower fragments is shown by the frequent limitation of the last 30-40 degrees of extension. It is not a method to be adopted generally and it should not be used particularly in the younger patient.

Mervyn Evans (1953) advocates operative reduction of the fracture. The T shaped fracture is converted into a supra-condylar fracture by accurate reduction and fixation. Any residual displacement between the two condyles and shaft is corrected by subsequent manipulation.

Technique: A vertical incision is made on either side of the elbow joint. The flexor and extensor muscles are slipped from the surfaces of the humerus by the injury and little further slipping is neces-

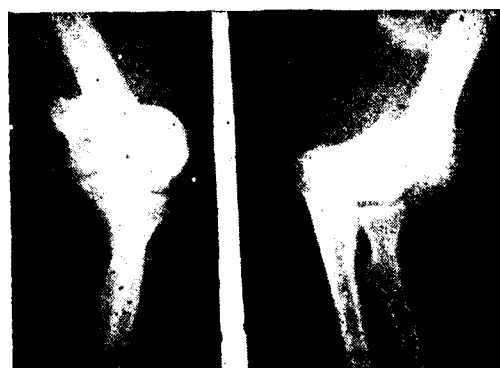
sary. They can be retracted easily without further damage. The joint is as a rule found widely open. The ulnar nerve should be exposed early in the dissection and transposed anteriorly. The index finger in the joint hooks the far condyle back into position against the near one. The break between the two is sharp and clear cut and it is not difficult to secure apposition. While this position is maintained with fingers, at 7/64" drill is inserted through the cancellous core of the two condyles and its position checked by x-ray. If the position is satisfactory, the drill is replaced by a screw. The united fragments are then manipulated under direct vision into as good a position with the shaft as possible and the wound closed. External is by Plaster of Paris bandage in moderate flexion for 3 weeks, after which active movements are started as soon as the stability of the supra-condylar element of the fracture allows.

This technique, if at all must be reserved for adolescents and young adults. Mervyn Evans who treated a number of cases on these lines, feels that screw fixation is reasonable in that it restores joint alignment, while the supracondylar element is dealt with by manipulation. Watson Jones, while he says "that the worst treatment of comminuted intercondylar fractures of the humerus is extensive operative reduction with internal fixation by triradiate plates and many screws such operations causing serious stiffness of the joint, by stripping the blood supply of the partly detached fragments and causing adhesion of muscle to bone" give limited acceptance of

the method adopted by Mervyn Evans and adds "This technique offers the hope of a successful compromise between the dangers of operative reduction and the difficulties of manipulative reduction."

What is then the best type of treatment of this most complicated fracture? The following method is being adopted in the Orthopaedics Department of this Hospital and the method is commendable not only because it is very simple, and the ultimate results are very gratifying.

The limb is supported in a Thomas arm splint and skin traction applied by means of strips of elastoplast on either side of the forearm. The condylar fragments are compressed between the two palms, so as to mould the fragments into the best possible position. A pad of wool may be placed behind the olecranon process, to correct the posterior displacement. The patient is made to do active movements of the wrist and fingers all the time. The skin traction is continued for about 2 weeks, at the end of which the elbow is tested for evidence of any callus formation. If callus is not formed, further period of traction is necessary. The skin traction is discontinued when callus is formed and a posterior plaster slab applied with the elbow flexed to the maximum extent that is possible. A week after, the slab is removed and the patient advised to do active movements of the elbow. During the intervals, the limb may be supported in a sling, to prevent any pain and muscle spasm. It will be very surprising to see the amount of movement regained



Somayajulu-Xray taken soon after fracture T-shaped fracture with much splaying of fragments.



Xray of the elbow 2 months after the fracture. There is evidence of good union.



A. P. & lateral view of the elbow a week after treatment by traction in extension. Displacement of the fragments is corrected to a large extent.



at the end of as early as 6 weeks by this very simple method of treatment.

Even in very badly comminuted fractures of the elbow the above method can be employed, and surprisingly good function may be gained. It is in this type of cases that the surgeon may be tempted to do an immediate arthroplasty. This is a thing not to be recommended. Even when there is severe comminution and gross displacement it is better to gain the best position that can be achieved by conservative treatment on the lines mentioned above. If the recovery of function is satisfactory, well and good. If the joint remains stiff, and painful, arthroplasty can be done and the result of late operation much better. Besides, arthroplasty caused flail and useless joint. For the reason, it is better that the fracture be treated conservatively in the beginning and to do an arthroplasty or arthrodesis later on, depending upon the age and occupation of the patient. Arthroplasty is to be preferred in the old and in those doing a sedentary type of work, whereas arthrodesis is to be preferred in the young and those doing heavy manual labour.

CONCLUSION :

1. The standard method of treatment of supra-condylar fractures should be by closed manipulation and immobilisation in a plaster slab and collar and cuff. The elbow should be in as much flexion as the swelling allow, provided radial pulse is retained. The optimum is about 30 above the right angle. The manipulation may be repeated but should not be delayed later than a week.

2. In the presence of severe edema, the limb is elevated and suspended; as soon as the edema subsides, the fracture is reduced by manipulation.

3. When there is evidence of circulatory embarrassment, all bandages are removed, the elbow is extended and conservative measures like sympathetic block (to relieve reflex vasospasm) intravenous papaverine etc. tried. If there is no improvement within-3-4 hours, the fascia in front of the elbow and forearm incised and brachial artery explored.

4. T shaped supracondylar fractures and grossly comminuted fractures are treated by skin traction in extension, in a Thomas arm splint. Any painful and stiff joint that may occur, is later treated by arthrodesis or arthroplasty.

5. Malunited supracondylar fractures are treated by supracondylar osteotomy to correct the deformity.

6. Compound supracondylar fractures are treated like any other compound fracture. Primary attention must be given for prevention of sepsis and the management of fracture must be subordinated to this important consideration.

7. Massage and forced manipulations are absolutely prohibited and have no place in the management of these fractures, as they are in others.

ANALYSIS OF LATERAL CONDYLAR FRACTURES

Out of 130 cases there were 10 cases of lateral condyle fracture, an incidence of 7.7%.

In Blunt's series it constituted 18.5% of all fractures around the elbow. But

according to Philip Wilson it forms only 2.5% of elbow fractures.

Age incidence:—The highest age at which the fracture occurred in this series was 10 years and the lowest was 4. This is in accordance with the fact that the fracture can occur only between 5-15 years.

Sex:—Of the 10 cases, 8 were males and 2 females.

Mechanism of injury:—All the patients gave history of fall on the outstretched hand.

Displacement:—2 cases did not show any displacement. Both were treated conservatively by immobilisation in plaster. 8 cases showed displacement of the fragment being rotated either a horizontal, vertical, axis, or more commonly on both.

Complications:—In one case the elbow dislocated posteriorly following open reduction. The dislocation was reduced under anesthesia and elbow put in plaster. This patient developed a stiff elbow, and he could not recover movement of more than 10° even at the end of 6 months. Probably there was damage to the capsule and soft tissues, and also destruction of articular cartilage. The ultimate result could not be traced in 5 cases. The remaining 4 cases regained a functionally good limb.

There was no incidence of ulnar palsy in any of the cases.

FRACTURES OF THE LATERAL CONDYLE

This is the next commonest injury of the elbow, after supra-condylar fracture. In Blount's series, it constituted 18.5% of elbow fractures. Massart and Cabout (1936) estimated that this fracture con-

stitutes 1/3 of all fractures of lower end of humerus but this figure is rather high. In Philip Wilson's series, it constituted 2.5% of fractures and dislocations around the elbow and 6% of injuries of the lower end of humerus and only 0.2% of total number of fractures and dislocations. In our series it formed 7.7% of fractures around the elbow.

The fracture occurs between 5-15 years.

Mechanism of Injury:—In direct trauma plays the major role, and the mechanism is generally adduction of the forearm on the humerus, with the elbow incompletely extended, thus causing strain on the lateral ligament and avulsing the condyle. This accounts for the tear in the overlying muscular aponeurosis and the rotary displacement of the fragment outward as a result of muscle pull. It is probable that in cases with incomplete displacement and absence of rotary deformity the fractures are produced by a force transmitted directly upward through the radius to the capitulum from a fall on the outstretched hand.

McLair believes that muscle effort alone could not cause the fracture with the typical displacement. He believes that the elbow is dislocated posterolaterally and carries the fragment with it. If the dislocation is incomplete the fragment moves downwards, laterally and backwards. If the dislocation marked, the fragment is pulled backwards. The dislocated joint surfaces fall forwards approximately into their normal position and reduce spontaneously, and the fragment lies anterior to the humerus. Clinically no dislocation appears to be present.

Pathology:—The injury consists of a fracture starting at the lateral side of the lower end of humerus at a point from about 1/4" to 1/2" above the epiphyseal line of the capitulum and extending obliquely downward and inward to the region of trochlea where it breaks through the articular cartilage into the elbow joint, thus separating a triangular fragment with a diameter of from 1" to 1½" and consisting of capitular epiphysis and a fragment of metaphysis. Through apart of its course the line of fracture may correspond with the epiphysal line but invariably a thin plaque of bone is detached from the metaphysis, chiefly from the lateral border and posterior space.

If the normal shape of the epiphysis of the capitulum is recalled, two degrees of displacement can be recognised: (1) The muscular aponeurosis overlying the fragment may be torn incompletely and the condyle is displaced laterally but not rotated and its fractured surface remains more or less in apposition with the fractured surface of its bed. (2) The muscular aponeurosis is torn completely, and the fragment displaced and rotated not only on its long axis but also on the horizontal axis, so that the articular cartilage of the fragment lies in apposition to the fractured surface of its bed, and the inner part of the epiphysis on the outside.

The displacement is due to contraction of the long extensor muscles of the wrist and fingers which take their origin by means of a common tendon from the lateral condyle of humerus. At operation, these long extensor muscles are

found to be stripped away from the lateral surface of the humerus but retaining their attachment to the condyle. There is generally a transverse tear in the muscular aponeurosis, directly overlying the condyle. The lateral ligament of the elbow retains its attachment to the condylar fragment and provides a hinge upon which the fragment rotates. The degree of rotary displacement is probably governed by the width of the tear in the muscular aponeurosis, since the ability of the muscles to pull the lateral border of the condylar fragment downward depends on the extent to which they are freed from the adjacent muscles. When the displacement is of the incomplete type, it is probable that the overlying muscular aponeurosis has remained in tact. In such cases, the condylar fragment is displaced laterally and somewhat posteriorly but is not rotated upon its horizontal axis. Another element commonly present in the displacement is axial torsion, as a result of which the lateral margin of the fragment is carried posteriorly and the medial border is brought forward.

The essential point to be emphasised in a case of rotary displacement is that the fractured surface of the condylar fragment is carried away from any contact with its opposing surface. Under these conditions bony union is impossible and pseudoarthrosis invariably results. Sir Ashley Cooper and later on Massart Cabout assumed though wrongly that interruption of the blood supply to the condylar fragment accounted for the frequency of non-union.

(To be Continued).

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DR. B. RAMA RAU, B.Sc., M.B.B.S.

Editor

GLEANINGS FROM THE MEDICAL PRESS



LEPROSY AND TUBERCULOSIS—(*Arquivos Mineiros de Leprologia*, vol. 16, 1956, via (Sao Paulo), (Via) J. A. M. A. Oct. 26, 1957).

According to Dr. R. D. Azulay there is an immunological inter-relationship between leprosy and tuberculosis. BCG vaccination can change the lepromin reaction in man or animal. This vaccination helps lepers who show clinical, bacteriological and histopathological cure by converting the lepromin test from negative to positive, thus indicating an increase of resistance to the leprotic infection. BCG vaccination confers a partial immunity to Mycobacterium lepræmuri infection in the white rat. By an injection of lepromin in guinea-pigs, it is not possible to produce a sensitivity to a second inoculation of antigen. Dr. Azulay's results agree with those of N. S. Campos and J. Rcsenberg.



COBALT 60 TELE THERAPY IN CANCER:— (Gilbert H. Fletcher M. D. JAMA May 18, 1957).

Cobalt 60 teletherapy is essentially a form of super voltage radiation. It offers the possibility of increasing the dosage of radiation to tumours because of better skin tolerance and a slight

increase in tolerance of the deeper structures. It must be used with due regard to the possible later appearance of subcutaneous and deep muscle fibrosis and other signs of cumulative damage and the curability of a tumor is one of its fundamental biological properties.

Cobalt 60 teletherapy is not indicated in a number of conditions e. g. lymphomas in areas of superficial lymph nodes. Its use is elective in a number of other conditions, e. g. mediastinal and retroperitoneal lymphomas.

Its use is imperative in a considerable number of conditions, including cancer of the oro-pharynx and urinary bladder and late cancer of the Cervix and Vagina. It has technical advantages that need to be explored further but an objective and unemotional approach is necessary.



INFLUENZA EPIDEMIC IN SINGAPORE CHILDREN: (Clinical impressions) (Swee, Y. O., and Dourado, H.; B. M. J. 2 : 1523-1525, 1957).

In May 1957 severe cases of Influenza were admitted, into the wards. 250 cases of influenza diagnosed clinically were discussed. *History*—Often the family was affected with milder form of the disease. Clinical manifestations: *age group*: 1 to 5 years age most affected. *Sex*: No discrepancy. *Symptoms*: Sudden onset of fever, cough and fits. Vomiting common in younger children and occurred soon after taking feeds. *Physical examination*: Temperature 100° to 105° Flushing of skin; throat: red and granular; lungs usually clear;

abdomen: no abnormality; central nervous system: drowsiness was a common feature. *Investigation:* Total WBC often normal. Chest radiograph often normal or showed increased hilar markings; *Courses:* fever 3 to 5 days and convalescence rapid in uncomplicated cases. *Pulmonary complications:* Lung abscess, unilateral or bilateral empyema. *Neurological complications:* Tremors and hemiplegia.

Prognosis & Treatment: Majority of cases did well. No specific drug was found to be effective. Treatment was mainly symptomatic. Sulphonamides and antibiotics were used to prevent secondary infections. Cortisone was used in cases of circulatory collapse.

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TREATMENT OF BELL'S PALSY:—
(L. B. W. Jongkees, *Neurology*, 7: 697-702, Oct. 1957, Minneapolis, via J. A. M. A., Jan. '58.)

The author reports the case of a woman with a total paralysis of 12 days' duration of the facial nerve. She had distressing pains deep in the ear, which itself proved to be normal. At operation a swollen, fiercely red facial nerve was found. The swelling was limited to the vertical segment inside the facial canal. The chorda tympani was also swollen and red. A small piece of the chorda tympani was examined microscopically; there was no sign of inflammation, the nerve fibres were degenerated, the myelin sheaths were strongly swollen, and the axis cylinders showed sections of different dimensions. There was no trace of

inflammation, but degeneration only, provoked by compression and oedema, was revealed by the histological examination. These findings support the concept that Bell's palsy is the consequence of an arteriolar spasm of the nutrient vessels of the facial nerve. Conservative treatment should be directed against the arteriolar spasm and must, therefore, pursue vasodilatation of the feeding arteries. It should be started as soon as possible, but a large number of patients may never recover from Bell's palsy if treated by conservative means only.

The function of the paralyzed nerve was restored by an operative decompression of the diseased nerve in all 10 patients in whom this procedure was carried out within 3 months after the onset of the disease. Four of 5 patients with more than 3-months' duration of the palsy were improved by the operation, but a paresis remained. In 1 patient no improvement followed the decompression performed 4 months after the onset. No disturbance of lacrimal secretion which could not be explained by a concomitant conjunctivitis or a lagophthalmos was observed in any of the patients. Treated conservatively, the patient who shows no trace of improvement and total electrical degeneration after more than 6 weeks has an extremely small chance to recover. The duration of the palsy is of primary importance for the prognosis. A patient with a total Bell's palsy who shows no improvement after 2 months should be subjected to

decompression treatment. Nerves showing an increasing paresis or a recurrence also should be decompressed. An operation may be indicated much earlier in patients complaining of severe pain deep in the ear and loss of taste, especially when the electrical reactions of nerves or muscles show degeneration.

★

THROMBOSIS OF THE DEEP LEG VEINS DUE TO PROLONGED SITTING:—John Homans, M.D. *The New England Journal of Medicine*, Jan. 28, 1954, via J.A.M.A. Dec. 1957, Page 2162).

The prolonged sitting position occasions a degree of dependency of stasis that may result in the rapid development of a quiet type of thrombosis in the deep veins of calf. From this a propagation of clot and pulmonary embolism may immediately follow.....The rhythmic forward, pulsatile motion of the foot, often noticed when one knee is crossed over the other, shows that the popliteal artery of the crossed leg is likely to be compressed in this position and warns that it may be a cause of venous stasis as well. Such matters are important enough to suggest the advisability of making movements of the toes, feet and lower legs when one is sitting for long periods and of getting up and exercising when opportunity offers. The right leg seems more susceptible than the left. There is evidence that persons over fifty years of age should have this particularly in mind that, physicians should be alert to recognise the significance of lameness after airplane flights, automobile trips and

other occasions of prolonged seated position.

★

MECONIUM ILEUS:—B. Seidenberg, M.D. and E. S. Hurwitt, M.D. *The Surgical Clinics of North America* Dec. 1956, via J.A.M.A. Dec. 21, 1957, Page 2073)

The term "meconium ileus" a mechanical small bowel obstruction in the newborn, secondary to alterations in the physical and chemical properties of the meconium. These alterations are essentially due to quantitative and qualitative changes in the pancreatic secretions, resulting in a tarry meconium that becomes increasingly more viscid in the distal and nutritional requirements, the ready availability of broad-spectrum antibiotics for long-term administration, and suggestive surgical approach, the survival has been obtained in many of these infants. According to present day concepts, meconium ileus is one of the manifestations of a generalised disease of the exocrine glands, especially of the mucus-secreting variety. In 1905 Lands-teiner first described meconium ileus and the relationship of pancreatic disease in the newborn to the grossly abnormal meconium. A complete pancreatic lesion may rarely be caused by a congenital stenosis of the pancreatic ducts, as described by Kornblith and Otani and again by Hurwitt and Arnheim. In 1938 Anderson described fibrocystic disease of the pancreas and contended that meconium ileus was merely an early manifestation of this condition. Farber examined 18 infants who died of meco-

mium ileus and found in each instance an obstructive lesion in the pancreas characterised by inspissation and secretions, dilation of ducts, atrophy of acini, and fibrosis of the pancreas. He demonstrated the relationship between the defective enzyme production in patients with this pancreatic lesion and the altered physical state of meconium in those with meconium ileus. Buchanan and Raport concluded that the meconium in meconium ileus is an abnormal secretory product of the intestinal and gastric glands; that its chemical reaction is more like one of protein than of the carbohydrate of normal infants, and that its abnormal viscosity is due its higher mucoprotein content. "It appears that the abnormality in meconium ileus is a result of the production of an excessive amount of abnormal mucoprotein which is not digested because of pancreatic achylia" according to Shwachman et al.

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THE INFLUENCE OF MATERNAL IRON DEFICIENCY ON THE NEWBORN: T. R. C. Sisson and C. J. Lund, A.M.A J. Dis. Child. 94: 525, 1957

To determine the influence of maternal iron deficiency on the newborn infant, eighteen anaemic women and an equal number of nonanaemic women and their babies were selected for comparative studies. Although the serum iron of the newborn reflected the iron supply of the mother, it was not directly related to the maternal serum iron value. The offsprings of the nonanaemic mothers had an average of 111 ug. per 100

cc. while the infants of the anaemic mothers had an average of 78 ug. per 100 cc. The mean circulating haemoglobin mass of 14.00 gm per kilogram was observed in the babies born of anaemic mothers while the newborn infants of nonanaemic mothers had a mass of 16.5 gm per kilogram. It was observed that the mean red cell volume to be 35.0 cc. & 40.00 cc. per kilogram in the infants of anaemic and nonanaemic groups respectively. Nonanaemic mothers rarely gave birth to an infant with evidence of iron deficiency anaemia, but the infants of anaemic mothers frequently shared the iron deficiency anaemia. It may tend to relieve the newborn baby of this deficiency, if the mother is treated effectively with iron.

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ASPIRIN & CORTISONE FOR RHEUMATOID ARTHRITIS (B. M. J. 5023 847, 1957).

In the treatment of early rheumatoid arthritis, aspirin seems to prove effective for long term therapy atleast during the initial stages of the disease than Cortisone, which seems to be somewhat more probable to such serious complications of therapy.

While 30 patients were treated with Cortisone initially, 31 patients were given Aspirin to the total of 61 patients having arthritis between 3 & 9 months. One fourth had complete remission in each group and the next one fourth continued to have very active disease; but the balance number of cases had slight symptoms.

In case of Cortisone group, the mean haemoglobin was 0.5% higher and mean sedimentation rate was 8 mm. higher than the Aspirin treated group. The two groups did not differ significantly in respect of Roentgenographic findings.

Five patients of the Aspirin group and one from Cortisone group had been lost to follow up at the four years of treatment.

Four patients from the Aspirin group within four years were switched over to another Drug; 7 patients from Cortisone group were transferred to Aspirin, while 2 to other drugs during the period. Complications of therapy were not observed in Aspirin group of patients, while it were infrequent among Cortisone group, It appears that Aspirin is preferable in most cases.

★

TREATMENT OF INFLUENZA WITH CORTISONE AND ANTIBIOTICS.— J. A. M. A., 165 : 12, Nov. 23, 1957. (via) Antiseptic, Vol. 55 No. 5 Page 388).

Dr. R. Cruz-Coke and co-worker^s reported to the Medical Society of Santiago on the pandemics of influenza which recently swept over Chile causing many deaths. The case fatality rate was not high, but because of the large number of persons involved the toll was great. Pulmonary complications were the main causes of death. They developed in persons whose general condition was poor and who did not respond to antibiotics. During July and August 1957, they treated 365 hospitalized patients with influenza complicated by pneumonopathies. The death-rate in this selected group of patients was 21.6

per cent and 50 per cent in those with bronchopneumonia. As soon as these patients were admitted to the hospital, they were given 800,000 units of penicillin; 1 g. of streptomycin and 2 g of tetracycline daily. All the patients with severe pulmonary complications, died notwithstanding the fact that in many cases the complications occurred when the patient was already hospitalized. Because of this high case fatality rate, it was decided to add 100 mg. of cortisone or 20 mg. of prednisone to the routine treatment. The dosage was gradually reduced after 5 to 10 days of combined treatment.

The results of the combined treatment were excellent. The general condition of the patient rapidly improved, the fever abated, the pulse become slower, and the dyspnoea rapidly disappeared. Viral hepatitis, which was a complication in two of the author's seven patients, followed a favourable course with cardiac insufficiency; and bronchopulmonary complications of influenza were given cardiotonics and were restricted as to use of salt in the diet, in addition to the combined treatment. The cortisone or prednisone had no influence on the retention of liquids by the tissues. In one patient who had diabetes, cardiac insufficiency, and severe complications of influenza, the combined treatment did not affect the metabolism of carbohydrates. The combined treatment was of great benefit to a patient with active bilateral pulmonary tuberculosis when he became ill with influenza. This patient received antibiotics, cortisone, and isoniazid.

Notes & News

CENTRAL HEALTH SERVICE SCHEME

In reply to a question in the Rajya Sabha on April 28, Shri D. P. Karmarkar, Union Minister for Health, said that the Government of India proposed to include all the doctors working in the contributory Health Service Scheme for Government servants in the Central Health Service Scheme.

HEALTH MINISTER'S WELFARE FUND

Answering questions in the Rajya Sabha on April 28 on the Health Minister's Welfare Fund and Discretionary Grant in 1957, Shri D. P. Karmarkar, Minister for Health, said that the T. B. Association of India was given a grant of Rs. 1,43,000 from the Welfare Fund in 1957.

The Minister laid on the Table a statement giving the names of 108 institutions which were given help totalling Rs. 5 lakhs from the Health Minister's Discretionary Grant during the year 1957-58.

U.S. AID FOR HEALTH PROJECTS

Assistance amounting to \$ 1,18,80,596.22 had been received from the Government of the U. S. A. for health projects in India, said Shri D. P. Karmarkar, Minister for Health, in reply to a question in the Lok Sabha on April 26.

The assistance was in respect of medical colleges and allied institutions in India, the orientation training projects, the national water supply and sanitation

programme, the national malaria control programme and the national filaria control programme.

INDIA'S GIFT OF VACCINES TO PAKISTAN

To meet the outbreak of epidemics in East Pakistan, the Government of India have made a gift of one lakh doses of cholera vaccine and one lakh doses of Smallpox vaccine to the Government of Pakistan. These have been despatched to East Pakistan by air.

PENICILLIN SALES AT PIMPRI

The State-owned penicillin factory at Pimpri set up a new record in the sales of the drug in March.

The total gross sales during the month were Rs. 27.65 lakhs – the highest for any single month so far.

The previous record sales were in January 1958, when they amounted to Rs. 26.91 lakhs. The total gross sales during the whole of 1957-58 amounted to over Rs. 1.8 crores as against Rs. 57.8 lakhs during 1956-57. This sharp increase in sales was mainly due to the substantial increase in the production of penicillin.

The total production of penicillin, certified and passed, during 1957-58 was 21.43 million mega units as against 9.9 million mega units in 1956-57 and 0.62 million mega units in 1955-56.

The Pimpri factory has been set up by the Central Government with the assistance of the United Nations Children's Emergency Fund and the United Nations Technical Assistance Administration. It went into production in August 1955.

To meet the increased requirements of penicillin in the country, the installed capacity of the plant is being expanded by about 60%. With this expansion, which is expected to be completed this year, the annual production is expected to go up to 26 to 28 million mega units.

DRUG IMPORTS

The value of drugs imported into India during the month of February was Rs. 1,00,14,017. The number of samples of drugs examined from imported consignments was 796.

EQUIPMENT FOR MEDICAL COLLEGES

Shri D. P. Karmarkar, Minister for Health, said in a written answer to a question in the Lok Sabha on April 26 that the first Five Year Plan provided Rs. 71.5 lakhs for the supply of equipment to medical colleges and research institutions and a provision of Rs. 100 lakhs had been made in the second Plan. The Government of India also sanctioned the payment of cash grants amounting to Rs. 6,71,876 to medical colleges and research institutions for equipment during 1957-58.

NATIONAL FORMULARY COMMITTEE

The National Formulary Committee held its final meeting in New Delhi on April 25 and 26 and approved the National Formulary of India.

The Formulary was drafted with a view to bringing about a simplified system of treatment in all hospitals and Government institutions, which would result in a considerable saving in the nation's drug bill.

The Committee was appointed by the Government in November 1956 under

the chairmanship of Dr. B. B. Yodh, with Shri S. K. Borkar as Member-Secretary.

INDIGENOUS SYSTEMS OF MEDICINE

1. A provision of Rs. 100 lakhs has been made in the second Five Year Plan of the Union Government for the development of indigenous systems of medicine, including the homoeopathic system.

2. In the State Plans for the development of indigenous systems of medicine, a provision of Rs. 521.83 lakhs has also been made. Of this, a sum of Rs. 221.49 lakhs has been earmarked for the improvement of existing colleges and the establishment of new ones.

3. Central assistance will be given during the Plan period on the basis (i) to meet the recurring cost of maintenance of research beds in selected institutions for approved research schemes at the rate of Rs. 2,000 per bed per annum (ii) for *ad hoc* research schemes on merit, and (iii) for improving the existing teaching institutions in the States.

4. On the recommendations of the three Advisory Committees on Ayurveda, Unani and Homoeopathy, a sum of Rs. 2,93,500 has been sanctioned so far to various institutions in different State Governments during 1957-58 for the development of indigenous systems of medicine, including homoeopathy.

5. At the meeting of ayurvedic experts convened by the Planning Commission on June 21 and 22, 1957, at Bombay, it was recommended that a Central Council of Ayurvedic Research should be established.

MAY '58

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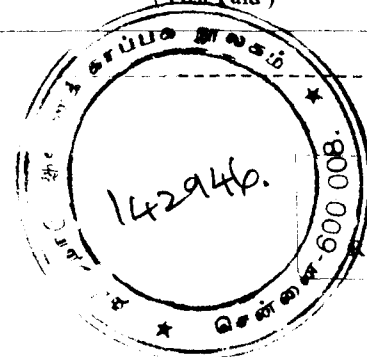
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