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Editor: Dr. P. R. Venkappaya.

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No. 4

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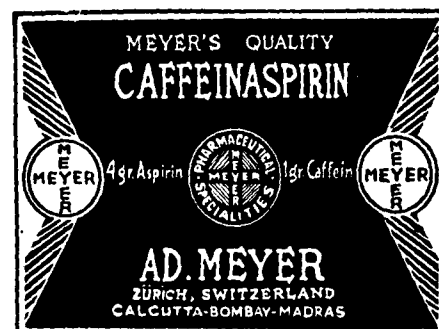
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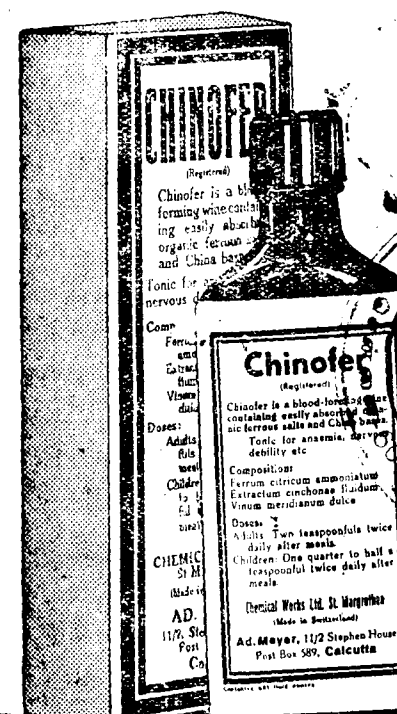
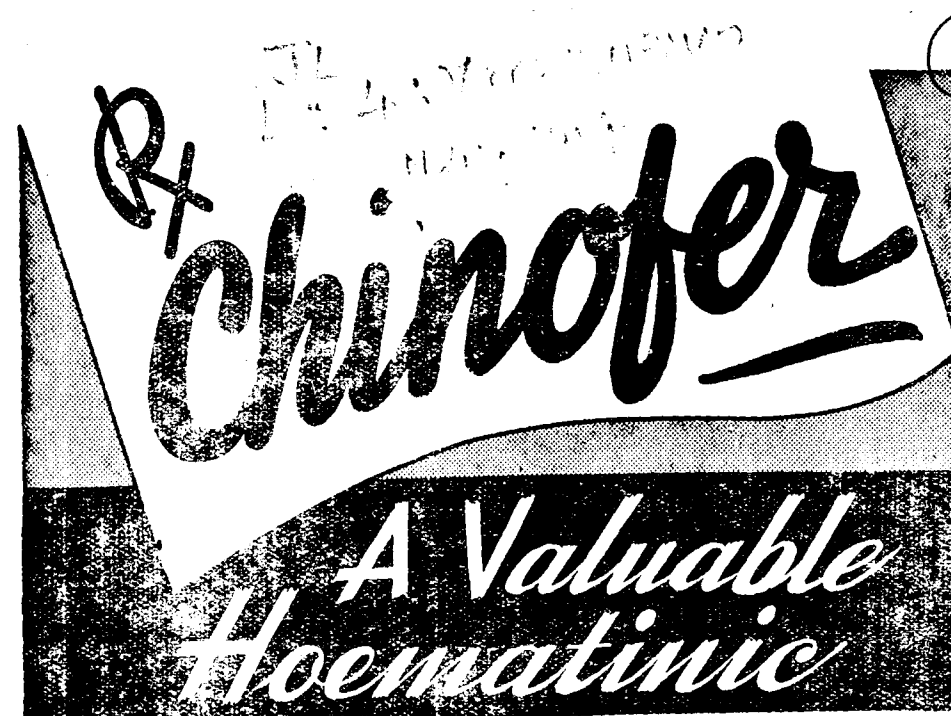
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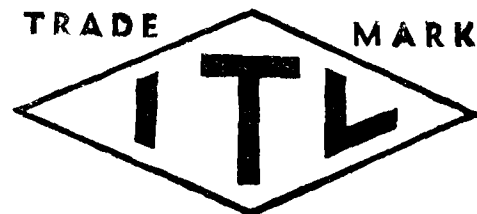
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MEDICO-SURGICAL SUGGESTIONS

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"CARBUNCLE AND ITS TREATMENT."*

BY DR. S. K. GUPTA, B.D. SC., B.O. (PB).

LABORATORY CLINIC, (ANALYST).

S. Krishna Laboratories (Regd). Railway Road, Pasrur. (Pb).

Definition:—Carbuncle is inflammation of the *skin* and *cellular tissue* causing their molecular necrosis. A hard, deep-seated, painful, circumscribed inflammation of the subcutaneous tissue, caused by pyogenic micro organisms infecting the skin so virulently that it looks deep red, underneath which the tissues suppurate, and discharge out drops of pus through various holes.

Sizes:—The sizes of carbuncle varies from that of a rupee to even the whole extent of the back. Sometimes it develops to a circular size with a diameter of nine or ten inches.

Season:—It is a very common trouble in India during summer.

Causes:—Excess of sugar in the blood of a Diabetic, which irritates the tissues, which are invaded by micro-organisms. This irritation does not subside in the presence of High-Blood Sugar, and is responsible for a spread of inflammation caused by the micro-organisms laden on the finger of the patient or any media of contact, which set up severe itching, with the result that the scratched skin by the nails or any acne, receives much quantity of bacteria over it, from where the infection travels deep under the skin, resulting into supuration and gangrene of the part involved. The pus underneath has either to go deep, or thus to slough the deep fascia or the muscles or the bone, or coming out of the skin by making various holes in the sloughed skin. More havoc is played if the deeper structures of organs lying below are thus affected.

Weak health, diabetes, profuse sweating complicated with dirt, irritation of the sweat glands by prickly heat, which get easily infected by staphylococci by the use of dirty mats as bed excite carbuncle. It sometimes takes the course of a typical gangrene.

*Specially contributed to Medico-Surgical Suggestions.

In India during summer people ordinarily lie out side on the floor of a verandah which may not be very clean and use the ordinary mats called "*madoor*" for their beds. Sweats, epithelial debris, dirt, moisture, all being matted together on the mat which is not very frequently washed, make a hot bed of culture for the staphylococci. On the other hand frequent sweating causes prickly heat on the back, which by constant scratching gets very easily inoculated by the staphylococci.

Sex and Age:—Men and women are equally affected by carbuncle between the ages of thirty or fifty.

Not only for Diabetes but whenever the health goes down below par for any cause *e.g.*, insufficient food or bad environments, carbuncle may develop. In hot weather there is a general disinclination for proper food resulting in general break down of ordinary health which predisposes an attack of carbuncle.

Onset:—Onset is marked by one or two prickly heats getting intensely painful and burning. They very soon become pustular and turn into regular boils. The part swells up a little, it becomes red, hot, painful and intensely tender to touch. The patient feels generally ill with high temperature, malaise and sickness. The pain and burning sensation disturb the sleep at night. In eighty per cent, or even more of the cases, the back is the seat of the lesion. From the nape of the neck to the buttock it may develop at any part of the back but they are very common at the upper part between the shoulders. Boils follow a boil, and one point follows the other at the adjoining site in a concentric way. The most external ones become pustular at first which on bursting discharge seropurulent exudate. The central zone shows signs of sloughing out, then it becomes decidedly gangrenous.

Different Zones:—There are four distinct zones:—From the centre, (1) the most central zone consists of slough with discharge of pus. No anatomical structure can be made out except a few islands of skin here and there. (2) The next zone is a ring of boils, the points of which are all discharging pus and a few giving out the hard "*cores*" consisting of the hair follicles. (3) The next or the third zone consists of a few isolated pustules only some of which are oozing out seropurulent or serous exudates. This is the most painful and burning areas. (4) Lastly the area beyond looks distinctly oedematous, red, very angry looking and swells up. Here hair follicles are distinctly prominent, the orifices of the glands are clearly visible, and there is a constant discharge simulating seborrhoea, exuding a tingy and thick oily discharge which quickly dries up at the site in bead like crust. This area extends about a couple of inches or a little more all round. This zone is the highest zone so far as the level is concerned. It slopes down all round outside. The third zone or the zone of pustules is also at the same level from where the level

slopes down in a concentrated way towards the central or the sloughy zone giving the whole area of lesion an aspect of something like a cribriform crater. During operation if the whole area is not cleaned off by thorough excision it may end in sloughing out the whole mass bit by bit leaving a deep gap, or gangrene may extend and patient may be carried away by death from absorption of septic material and blood poisoning.

Sugar in the Urine:—It may be diminished for a time, when a carbuncle is going to from. Master the Rule "*If Sugar in a Diabetic Patient's Urine is suddenly diminished, without any special treatment, same complication is going to develop or has already developed.*"

Toxaemia:—Fever is present in case the dose of toxin is absorbed into the system. Cases of long standing, who are not properly or not at all operated upon, look very anaemic, with such sort of septic fever. Evening fever with rigor, dry tongue, quick pulse, insomnia and marked anaemia are the important points to judge this symptom.

Aetiology:—The same as in boils. *Staphylococcus aureus* sometimes helped by *staphylococcus citreus* and *albus*. The condition is predisposed by constitutional or general disease such as Diabetes, Brights, Alcoholism, or malnutrition excited by unclean surroundings described above.

Morbid Anatomy:—Same as in boils from which it differs only in its extension via the cellular tissues mainly in the deep fascia, with many discharging points giving a cribriform appearance. The base of the carbuncular mass is densely hard and it creaks on cutting with a knife. This is due to infiltration of the deep fascia. On section the cut surface looks studded with grey dots representing the staphylococcus colonies. It gives an appearance of an ant-hill with hundreds of holes all connected with one another.

Clinical Varieties:—1. Carbuncle at the back is typical and it has already been described above.

2. Malignant facial carbuncle. It starts from boils or pimples on the face especially the lips and the nose. It simulates the phlegmonous erysipelas of the face. Sometimes it is very difficult to differentiate it from erysipelas unless the boils show many heads and points. The carbuncle is caused by *staphylococcus*, while the erysipelas is the result of the actions of *streptococcus pyogenes*, although both may combine to form the suppuration and cellulitis. These latter cases which are typically gangrenous are generally fatal, and the cause of death is pyaemia unless the patient is carried away before by the infection travelling through the angular vein and its connections into the skull and cause cavernous sinus thrombosis.

3. Carbuncle on the anterior aspect of the neck. The course is typical, but the termination may be fatal due to infection. Prognosis therefore is grave.

4. Carbuncle of other situations of the body such as buttock, groin, limbs are not very uncommon with people suffering from diabetes. Unless implicated with phlebitis, the prognosis is like that of the carbuncles at the back.

Diagnosis:—A carbuncle may be confused with anthrax, or an ulcerated syphilitic gumma. From the malignant pustules of anthrax it is sometimes very difficult to differentiate. *Microscopical examination is the conclusive evidence.* In the facial type of carbuncle firm extensive oedema of the lips, cheek, nose, eye-lids round a pustule with the development of fresh pustules and the appearance of slough of the cellular tissue at the bottom of the boils are the characteristic signs. The pain is severe and the fever is of asthenic type. The facial veins soon get thrombosed and can be felt as a firm cord. Pyaemic symptoms soon supervene.

In Malignant Pustules:—The Vesicles contain dark serum. The base of the pustule is formed of dry slough. There is little pain and suppuration is entirely absent. The constitutional symptoms such as rigors etc. usually show themselves two or three days after the local signs are prominently developed.

A Sloughing Gumma:—Is a painless affection, of chronic nature. Gumma sometimes occurs where it is unusual for carbuncle to develop as round the knee joint, or in muscular tissues which is never involved by carbuncle. A carbuncle is a much more acutely painful affection. History and other syphilitic signs confirm the diagnosis. Carbuncles have a distinctive odour which is quite characteristic of the lesion. Examine the urine for sugar although it is not present in all cases.

Prognosis:—The outlook of the carbuncles at the back is favourable. It generally stops progressing after a limit is reached and the slough separates in a slow and tedious process. The prognosis of carbuncle at other vital sites as the face or neck is grave and serious. If properly operated upon, and nicely treated, recovery is certain. Old patients with weak health loose lives on account of low resistance of vitality, but none should be disheartened or dishearten the patient by telling that bad prognosis is in sight. The modern treatment is unique, and some life is certainly prolonged even if there is no hope of surviving the patient from this malady. I may say that it should be a general rule to operate and treat the carbuncle of any severe type at any age. Bold Surgeons achieve success, while half hearted ones do fail.

Complications:—Pyaemia, Septicaemia, Diabetic coma, Pneumonia, Phlebitis and other signs of Toxaemia and septic absorption, and typical Gangrene.

Treatment:—Treatment of Carbuncles consist of:—(1) Local measures (a) Operative and (b) Non-operative. (2) General measures.

1. **Local Measures:**—(a) **Operative:**—The only sure treatment is thorough excision. At the back the whole mass including a little of the hyperaemia area must be excised out and completed by stellate like incisions at the area indurated with infiltration.

The most important points of the operation are the following:—

1. It should always be done under general anaesthesia unless otherwise contra-indicated, but excision is impossible without it.

2. Excise the whole sloughed out tissue right down to the muscular layer, cleaning every bit of the slough at the base.

3. Take the line of a round sweeping incision just a little beyond the pustular zone.

4. Put small stellate like incisions diverging out from the line of the round sweep incisions keeping a radial arrangement all converging to the centre of the carbuncle. The length of each of these incisions should be about half an inch, more or less. The depth should be down to the deep fascia. They should be an inch apart from each other.

5. Separate the skin from the deep fascia at the area of hyperaemia and insert antiseptic gauze underneath the fringes. Simple tincture iodine in the best. Ether may be used if it is feared that iodine will stain and mask diseased foci although iodine is more preferable. In non diabetic cases strong carbolic acid may be used which should be washed away by a swab.

The above procedure will hasten the healing and reduce the area to the condition of an ordinary red healing ulcer, in seven or eight days' time. Moreover any other kind of incision such as cross incision will leave unexplored areas colonised by the bacteria.

The operative method bleed freely when the incision are made. The best course to adopt in the circumstances is to finish the sweep as quickly as possible. The excision being completed, a little firm pressure with gauze would readily stop the oozing.

(b) **Non-Operative Treatment:**—At the early stage when the lesion is of rupee size or smaller it may be treated as an ordinary boil. A small crucial incision helped by gentle section made by Klapp's bell and dressed frequently by cold boric compress gives satisfactory result.

2. **General Treatment:**—In diabetic cases if time permits before operation, the estimation of blood sugar should be determined and a suitable dose of a few units of Insulin may be injected. In any case after operation it must be administered. Put the patient on tolerable carbohydrate diet. Fasting treatment may be tried in case the patient has got some vigour to tolerate hunger and as soon as the sugar is free, find a limit of, which restricts the quantity of carbohydrate diet such as wheat floor or any carbohydrate containing diet, which when eaten, does not bring sugar in the blood or in the urine.

Iron, Quinine, salines in large doses, opiates, stimulants, sedatives should be administered according to the condition of the patient, and as indications would arise. Fresh air, sunny room, clean bed, out-door drive, good nutritious food, profuse bland drinks, are all to be attended to. Infections of *Staphylococcus aureus* vaccines help recovery quickly. If auto-vaccine could be made from culture prepared from the patient's discharge it would be better but otherwise stock vaccines of mixed *staphylococcus* should be used. It should be started from five millions cocci dose or less at the start and gradually increased to five hundred million cocci at suitable intervals watching the reactions. Vaccine therapy should never be done before operation.

Caution:—The most important questions before operation are, the methods to relieve pain, the choice of general anaesthesia in relation to the incidence of diabetes and avoidance of carbolic acid in any form in such cases. After operation one should be particularly careful about extension underneath the flaps. The first dressing may in some cases be done under gas anaesthesia.

Cases have been noted, when carbuncles at the other part of the body have appeared, and formed double carbuncle. It is ridiculous to mention that the Great Physicians used to pack goat's flesh in the Carbuncle Wound, and termed it 'Ghost Khor Phora'. The Indian Hakims imitated this bogus treatment, and I know a case where goat's flesh was being packed in the cavity of the wound. I do not recommend this hopeless method, but wonder awful imagination at the folly of the quacks.

After Treatment:—All along the course of convalescence the general conditions should be particularly watched and treated. Skin grafting should be undertaken when the case is beyond all doubtful conditions and that also from another donor.

When pus does not cease, while the whole of the cavity is cleaned, and free of slough, we must put a patient under X-Ray examination to know if the bone underneath has necrosed.

OBSERVATIONS WITH AKTISOL ALBUM (SHIONO) IN VENEREAL DISEASES.

BY W. N. WELINKAR, L.R.C.P., M.R.C.S.(ENG.),

Lecturer in Venereal Diseases, Grant Medical College.

Specialist in Venereal Diseases, Sir J. J. Hospital, BOMBAY.

Regarding sulphanilamides so much has been said in the recent medical literature that there is little point in entering into a lengthy discussion of the action of this drug. It was first used in the treatment of gonorrhoea by Dees and Colston. We have been using this drug for nearly two years now and we have found that it has certainly revolutionized the treatment of gonorrhoea in all its stages. Owing to war it was getting more and more difficult to obtain the drug. Therefore, to arm against the eventuality of suddenly being confronted with non-availability of certain products which one may have tried and have become used to, it became imperative for one to inquire into the practical utility of other Sulphanilamide group preparations. With this object in mind, we tried the drug "AKTISOL-ALBUM" (Shiono) in the venereal cases under our care.

AKTISOL (Shiono) has the composition of para-amino-phenyl-sulfanamide. We used this drug in as many cases as we could but owing to the patients not attending the hospital regularly, it was not possible to test the merits of this drug as thoroughly as one would have liked to. However, we watched the results in a few cases who received the full course and in a number of cases who did not complete the treatment. Most of the cases thought that they were cured as soon as the acute condition was over, and did not attend for final tests though in many cases one test at least was done. We also tried the drug in venereal diseases other than gonorrhoea such as soft sores and "Climatic Bubos".

In the cases of gonorrhoea we relied on microscopical examination of stained smears by Gram's method for diagnosis and tests. In the case of soft sores, the diagnosis was based upon the clinical syndrome of the disease and the intradermal tests of Ducre's *Bacillus* vaccines. In the case of Climatic Bubo, we entirely depended on the clinical signs, as Frei's antigen was not available. The following table gives a short summary of cases which were under our observation.

Summary.—The drug was tolerated remarkably well both by male and female patients, the average weight of the patients being 100 lbs. Sulphur free diet was not observed though the manufacturers recommend such. They advise no concomitant treatment with mercury and silver compounds on account of incompatibility. Yet in the case of Impetigo Contagiosa we did give Ung. Hydrag. Amm.

for local application without any bad results. In one case antisyphilitic treatment with arsenical injections was also given, apparently with no bad results; the patient is still having anti-syphilitic course of injections. Vomiting, nausea, cyanosis, were conspicuous by their absence in all our patients treated with Aktisol-Album (Shiono).

Dosage.—The average dose was given 2 tablets 3 times a day though 3 tablets 3 times a day were also given. The course should be at least for 10 days, 2 tablets 3 times a day.

Regarding Climatic Bubo cases, we could not say that these cases were really Lymphogranuloma Inguinalis or the so-called "6th Venereal disease" cases as we could not establish the diagnosis by Frei's antigen which was unavailable. The clinical findings of absence of sores, painful inguinal indolent periadenitis were the chief points we bore in mind. These cases also showed febrile reactions and were chronic in as much as one case was going on for six weeks without softening of the adenitis or disappearance of pain. These two cases did respond to Aktisol-Album (Shiono) whatever may be the cause of the Bubo. In one case aspiration of Bubo proved sterile. In the cases of soft sores, (two recorded out of ten) the response was equally good and sulphanilamides are certainly worthwhile trying in such cases. Impetigo contagiosa cases did clear sooner with this drug than ordinarily.

Conclusions:—Comparing the cases under routine treatment and routine treatment combined with Aktisol Album, one certainly is struck with the rapid results one meets with in the latter. For instance, in case of acute gonococcal urethritis, with Janet's irrigation, the discharge does not stop for 8-10 days but with sulphanilamides. I have seen the discharges lessening and completely disappearing within 24 hours. Of course it would be ridiculous to call such cases "cured" but the fact remains that it is a tremendous relief to the patient, to be without a messy discharge. I would like to sound a warning that it is also true that a patient treated with Sulphanilamide is more dangerous as a potent carrier of gonorrhoea than one who has not received this drug. Therefore, it must be brought to the patient's mind that if he or she does not remain under observation and carry out the "Test for Crue" for at least three months, after the cessation of treatment, he or she will be a danger to self and to the society in general.

My thanks are due to the Hon. Clinical Assistants of the V. D. Department for their co-operation in carrying out these investigations.

Name	Age Sex	Findings	Diagnosis	Treatment	Result	Other diseases	Adjuvant treatment.	Remarks.
(1)	40 M	Acute Anterior Urethritis. History of exposure. Two glass test.	Gram. negative. Intracellular diplo-cocci present in large numbers.	2 tablets 3 times a day for 3 days.	Discharge practically stopped. Patient felt better 1st test negative.	Syphilis W.R. +	Anterior irrigation with 1:10000 Pot. permangan. once a day signs of toxicity Alk. mix. 1 oz. 3 times a day.	The patient did not attend further for tests. No signs of toxicity were complained of by the patient.
(2)	25 M	"	"	"	"	Nil	"	"
(3)	24 M	"	"	"	"	Nil	"	"
(4)	30 M	"	"	"	"	Nil	"	"
(5)	45 M	"	"	2 tablets 3 times a day for 7 days.	"	Nil	"	"
(6)	25 F	Acute cervicitis.	"	2 tablets 2 times a day for 3 days.	Cervix clear. 1st Test Negative.	Nil	Douching and Alk. mix. 1 oz. 3 times a day.	The patient did not attend for further tests. No signs of toxicity were complained of by patient.
(7)	25 F	Acute cervicitis with Bubo. W. R. +	Gram. negative. Intracellular diplococci present in large numbers.	3 tablets 3 times a day for 3 days.	Bubo subsided. Cervix clear. 1st test negative.	Bubo, 1st syphilis.	Douching and Alk. mix. 1 oz. 3 times a day.	The patient did not complete treatment. No signs of toxicity were complained of by patient.
(8)	14 F	Acute cervicitis.	"	1 tablet T.D.S. for 7 days.	At the end of 7 days smears were examined, were negative to gonococci and pus cells.	Nil	Douching only.	After an interval of a week another course was given. Smear test negative.

Name	Age	Sex	Findings	Diagnosis.	Treatment.	Result.	Other diseases.	Adjuvant treatment.	Remarks.
(9)	18	F	"	"	2 tablets T.D.S. Discharge less. for 3 days.		Nil	Nil	The patient did not complete treatment. No signs of toxicity were complained of by patient.
(10)	18	M	"	"	"	"	Nil	Nil	"
(11)	25	F	History of exposure many years back. Heavy threads in urine.	arthralgia chronic post-erior urethritis	2 tablets 3 times a day for 3 days.	Pain in joints much less.	Nil	Posterior irrigations with 1:10000 Pot. permang. once a day. Alk. mix. 1 oz. 3 times a day.	Patient felt better, did not attend for further tests.
(12)	30	M	History of Gonorrhea some yrs. back. Urine heavy threads present mild arthralgia.	Gram negative Diplococci present	3 tablets 3 times a day for 3 days.	Joint pains much better.	Nil	Anterior irrigations with 1:10000 Pot. Per. once a day. Alk. mix. 1 oz. 3 times a day.	Patient did not attend for further tests.
(13)	22	M	Pustular eruptions on buttocks & thighs & secondary Bubo.	Impetigo contagiosa.	3 tablets 3 times a day for 4 days.	Pustules dried. Bubo subsided.	Nil	Ung. Hydrarg. Amm. locally.	Patient was very well after the treatment and impetigo markedly improved.

(14)	25	M	Inguinal Bubo (proadenitis) for 1½ months. No sore present. No Urethritis, W. R. positive.	Climatic Bubo with Syphilis.	3 tablets 3 times a day for 10 days.	Bubo subsided in size one showed softening after 2 days. It was aspirated and the pus was sterile	Syphilis	Anti-syphilitic treatment was conducted with Aktisol.	Bubos completely subsided. Pain swelling subsided. Patient is still having anti-syphilitic treatment.
(15)	30	F	Bubo. W. R. Negative.	Bubo with leucorrhoea.	3 tablets 3 times a day for 5 days.	Bubo Subsided.	Leucorrhoea,	Douching & Alk Patient improved. 1 oz. 3 times a day.	
(16)	25	M	History of Gonorrhea 6 years back. Arthralgia.	Chronic Gonorrhea.	2 tablets 3 times a day for 8 days.	Number of pus cells diminished markedly. Joint pains much better.	Nil	Irrigation with 1:1000 Pot. permang. once a day Alk. mix. 1 oz. 3 times a day.	Did not complete course.
(17)	25	M	Painless proadenitis in inguinal regions without sores on the penis	Climatic Bubo.	2 tablets T.D.S. for 3 days.	Bubos did not suppurate. Urine no threads.	Nil	Nil	Bubo subsided. Patient did not attend to complete course.
(18)	25	M	Bubo with multiple painful sores on the penis.	Soft sores, intradermal test +	2 tablets T.D.S. for 3 days.	Sores healed. Bubo softened was aspirated; subsided.	Nil	Local dressing with lotio Nigra. Glycerine Belladonna to Bubo.	Did not attend. Apparently cured.
(19)	30	M	Bubo, soft sores on penis.	"	"	Sores healed. Bubo subsided.	Nil	"	"
(20)	7	F	Vulvo-vaginitis.	Gram. Negative intra-cellular diplococci present	1 table T.D.S. for 8 days.	Discharge markedly lessened. 1st Test negative.	Nil	Douching & Alk. Mix. ½ oz. 3 times a day. Hip Baths.	Did not attend further tests.

SUMMARY TABLE OF CASES TREATED WITH
AKTISOL—ALBUM.

Clinical Diagnosis	Number of patients treated	Remarks.
Gonorrhœa acute.	10 cases	After 3-4 days, the discharge had stopped and no gonococci were found in smears; one test only.
Gonorrhœa chronic.	4 cases	"
Gonorrhœal vulvovaginitis.	1 case	"
Climatic Bubo (Proadenitis)	2 cases	The Bubos subsided. One case was complicated with a W. R. + and was given N.A.B. after 10 days Aktisol-Album.
Soft Sores.	2 cases	After 6 tablets a day for 3 days sores healed and Bubos subsided.
Impetigo.	1 case	This was a very interesting case. 4 days Aktisol-Album of 9 tablets a day, the condition cleared up remarkably well.

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ACUTE CORONARY THROMBOSIS. 143003.

E. COWLES ANDRUS, M.D., F.A.C.P.

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The clinical syndrome which is called "acute coronary thrombosis" is, in reality, the result of myocardial infarction, acute ischemic necrosis of ventricular muscle. It is an incident, perhaps an accident, in the life of a person with atherosclerosis of the coronary arteries. It is only one of the three modes of disability resulting from coronary sclerosis, the others being angina pectoris and congestive heart failure. As the diagnostic criteria of disease of the coronary arteries, so clearly presented by Dr. Marvin, are becoming more widely understood, it is plain that acute coronary thrombosis is at once more prevalent and, when properly recognized, less dangerous than was previously supposed. It is not, to my knowledge, possible as yet to intervene effectively in the chain of events which ends in coronary occlusion, but by establishing the diagnosis early after the onset of symptoms, the subsequent course may often be changed entirely in direction.

ETIOLOGY.

The prevalence of coronary thrombosis cannot be accurately determined; mortality statistics include diseases of the coronary arteries but do not distinguish coronary occlusion as a primary cause of death. By examination of random samples of death certificates in New York State it has been estimated that between 400,000 and 500,000 attacks of coronary occlusion are sustained annually in the United States, an incidence of 1 attack per 54 men and 189 women over 40 years of age, with a total mortality of nearly 120,000.

Atherosclerosis of the coronary arteries with its sequela, acute coronary thrombosis, is probably a metabolic disorder with a constitutional basis. A certain body type, or build, is inherited and in adults determines weight within limits. Arteriosclerotic heart disease in middle age and beyond is notably more common among persons who are overweight; its mortality rate is twice as great as among the underweight. With body type, a tendency to coronary disease may thus be inherited. In certain families, leaving aside all diabetics, there runs a demonstrable aberration of cholesterol metabolism, and with it, a familial record of coronary disease. Particularly among the instances of coronary occlusion in individuals under 55 years of age the blood cholesterol is elevated in a large proportion. Since it is not possible, by any reasonable form of diet, to raise the blood cholesterol in normal persons, it is to be assumed that here again an inherited peculiarity of fat metabolism may contribute to

the advance of coronary arteriosclerosis. It is probably the abnormalities in fat metabolism which provoke the high incidence of arteriosclerosis and coronary thrombosis among persons with diabetes.

But lest this apparent adoption of predestination as to coronary disease seem to altogether Presbyterian, it should be stated as a belief as yet unsupportable by figures that over nutrition due to over indulgence may also be a principal contributing factor in the etiology of coronary thrombosis.

The association of coronary thrombosis with arteriosclerosis and hypertension is striking, but concerning other contributing factors, remote and immediate, there is less agreement. In some cases in which coronary occlusion occurs during or following surgical shock it has seemed plain that the lowered blood pressure and resultant slower coronary flow have contributed to thrombus formation in the vessel. However, there is no etiologic factor which is obviously common to all or to many cases. In some, coronary occlusion follows unusual exertion; in many more it develops during sleep. This lack of conclusive agreement regarding the relation of various factors to the onset of coronary thrombosis undoubtedly rests upon the fact that the abnormality rarely develops precipitately. The onset of symptoms probably marks the most recent of a series of events. Certain it is that examination of an occluded coronary artery from a patient who has died suddenly, soon after the first symptoms, may reveal several stages of developing thrombosis.

PATHOLOGIC CONSIDERATIONS.

In anatomic terms acute coronary thrombosis is the result of a progressive lesion in the arterial wall. In this the first important stage is the rupture of capillary blood vessels supplying the intima. This usually occurs into or near an atheromatous plaque. It may narrow the arterial lumen without rupture of the intima or thrombosis. Or, more frequently, rupture of nutrient capillaries may lead to intimal necrosis and secondary thrombus formation within the lumen of the vessel at this site.

In a physiologic sense either of these events impedes the flow of blood through the artery. Whether or not death of the patient or infarction of the heart muscle supervenes is determined by other factors. Of considerable importance is the contemporary nutritional requirement of the affected section of the myocardium; infarction is the more liable to result the greater the load on the heart relative to the coronary flow. But the immediate result of the accident is principally governed by circumstances antecedent thereto, the anatomic arrangement of the coronary vessels and the adequacy of the collateral circulation. In one third of human hearts the arterial blood supply to the myocardium is evenly balanced between right

and left arteries; these suffer least from thrombosis of one. In about one fifth the left artery furnishes more blood than the right; these suffer most, and the left artery is more frequently sclerotic than the right. The extent and efficiency of collateral circulation is, paradoxically, conditioned by the previous inadequacy of circulation; inter-coronary anastomoses will have developed principally when and where such have been needed. In certain hearts examined post mortem it appears that closure of the main stem of one or even both coronary arteries has taken place without infarction of the heart muscle. Obviously this must have occurred gradually, in the presence of adequate anastomotic channels.

In the event of acute coronary thrombosis a greater or lesser portion of the myocardium is completely deprived of its intrinsic blood supply. Necrosis probably commences at once, although histologic evidence thereof is scarcely demonstrable for nearly 6 hours, and proceeds to involve the entire thickness of the ventricular wall save for a thin layer adjacent to the endocardium, where some nutriment is seemingly derived *via* the Thebesian vessels from the blood in the ventricular cavity. The necrotic area is soon invaded by polymorphonuclear leukocytes and mononuclear cells. These later penetrate and phagocytose the necrotic muscle fibers. By the end of three or four days after the occlusion new-formed capillaries grow into the infarct and with them connective tissue fibroblasts. Later intercellular collagen fibers appear, adding strength to the fibrous scar. Where the infarct extends to the endocardium, mural thrombi form within the ventricular cavity. The entire cycle from necrosis to repair is completed within 4 to 8 weeks according to the size of the infarct. There is considerable evidence indicating that healing is delayed by general impairment of the myocardial blood supply, by circulatory failure or by anemia.

SYMPTOMS AND SIGNS.

Pain or discomfort, which may vary in degree from a vague substernal misery to a terrible oppressing choking agony, is an almost invariable accompaniment of acute coronary thrombosis. The cause of such pain is still uncertain, but several characteristic features deserve particular mention: 1. In its classical form it involves the left pectoral, subclavicular and supraclavicular regions, medial surface of the upper arm and ulnar portion of the forearm. 2. It may be accompanied by a sensation of cold and numbness. 3. It is often diffuse, sometimes encircling the chest; it occasionally appears as a dull continuous ache in one or both axillæ. 4. It rarely, if ever, commences in that area of the precordium which overlies the ventricles; it commonly begins beneath the upper sternum or even manubrium. 5. Occasionally the pain may be "referred" to the epigastrium, but in my experience this is most frequently true when accompanying failure of the right ventricle

has caused acute passive congestion of the liver. Finally, two features serve to distinguish the pain of acute coronary thrombosis from that of angina pectoris, its unremitting continuity for hours or even days, and the fact that vasodilating, depressor drugs (nitrites) fail to afford relief. From the pain of pericarditis it differs in that it is not influenced by change of position.

It is evident that the area of myocardium involved by the infarct is totally incapable of performing the function of a muscle. Depending upon its size this segment may more or less seriously interfere with the capacity of the ventricles to maintain the normal cardiac output. In the event of a small infarct the systemic arterial pressure may remain unchanged or, under the influence of pain, may actually rise. But when a larger portion of ventricular muscle, particularly of the left ventricle, is involved, the systemic arterial pressure usually falls. The patient frequently complains of extreme weakness or faintness. A sudden fall of blood pressure may bring on syncope or even a convulsive seizure. Symptoms of congestive failure may appear rapidly, dyspnea and pulmonary edema or splanchnic congestion with nausea. The latter may in some cases mark the onset of symptoms and persist for hours. In certain instances intractable nausea and bradycardia suggest that the vascular occlusion has set in train a series of manifestations of intense vagal stimulation.

Certain objective features are of the greatest value in establishing the diagnosis of acute coronary thrombosis. Upon examination the heart may occasionally appear to be entirely normal. More commonly the rate of beat is increased, the heart may be enlarged, and its sounds distant. Gallop rhythm may become audible or, after the first day following the infarction, a pericardial friction rub. Various irregularities of cardiac rhythm may appear abruptly, ventricular extrasystoles, paroxysmal ventricular tachycardia, auricular fibrillation or auriculoventricular block. Although not unusual as a consequence of more generalized interference with the nutrition of the myocardium, as with diffuse coronary sclerosis, delayed intraventricular conduction does not commonly result from acute coronary thrombosis. It has been suggested that the intraventricular conduction system, bundle branches and Purkinje network, is spared because of its position immediately underlying the endocardium, a zone usually spared in the progress of necrosis following coronary occlusion. Ventricular fibrillation is presumably the proximate cause of death in the majority of fatal cases.

Other manifestations are due to the development and resolution of the infarct itself. Within 24 hours of the onset of myocardial necrosis the patient's temperature rises, rarely above 101°F.; this fever persists in uncomplicated cases for 3 to 7 days. Leukocytosis appears at about the same time and persists longer, 1 to 3 weeks.

Concomitantly, sometimes later, the erythrocyte sedimentation rate increases, reaching 30 to 40 mm. in an hour and is persistently rapid well into convalescence. It may, indeed, be that this last manifestation is not to be ascribed to the presence of the infarct alone, since it persists sometimes long after the infarct has been replaced by scar tissue. Thrombi formed upon the endocardial aspect of the infarct are not infrequently dislodged with consequent embolism to lungs, brain, limbs, or viscera. Barely the embolic phenomena are the first objective signs indicating acute coronary thrombosis. Finally, in a fortunately very small proportion of instances, rupture of the ventricle may take place with sudden death. When it occurs, cardiac rupture usually happens within 2 weeks of the infarction.

I have omitted thus far any discussions of the characteristic electrocardiographic abnormalities which identify infarction of the myocardium. In the first place, it should be emphasized that records taken early or even for some time following the infarction may not deviate significantly from the normal. More commonly distinguishing abnormalities appear in the electrocardiogram within a short time after the acute vascular closure. In contrast with the abnormalities of auriculoventricular (P-R interval) and intraventricular (QRS interval) conduction which accompany diffuse myocardial damage or coronary sclerosis, the electrocardiographic manifestations of acute coronary thrombosis involve characteristically the S-T segments and T waves.

TREATMENT.

Any patient presenting any symptoms or signs conceivably due to acute coronary thrombosis should be treated as such until the possibility is clearly excluded. It must be the primary aim of the treatment to afford immediately adequate and continued rest to the patient and to his circulatory apparatus. It is frequently better to put the patient to bed in the nearest available room rather than subject him to the strain of a ride to the hospital. The patient should be fed and attended so that he is spared all effort, less on account of imminent danger to life than in order to hoard his energy. As soon as the diagnosis can be established some opiate should be given to relieve pain and anxiety and to afford quiet. Morphine is usually undesirable because of its tendency to provoke nausea; pantopon ($\frac{1}{3}$ grain hypodermically) or dilaudid ($\frac{1}{8}$ grain hypodermically) are preferable and may be repeated. Papaverin has afforded relief in some cases.

Diet should be soft, not bulky, and administered in 5 feedings instead of 3. During the acute stage and in early convalescence the diet may well furnish less rather than more than the total caloric requirement of the patient.

Constipation should be combatted and a daily bowel movement produced without resort to vigorously stimulating catharsis. Milk of magnesia and oil, glycerin suppository, or small oil or glycerin-water enemata usually suffice.

Digitalis is indicated only when evidence of cardiac dilatation or congestive failure is apparent, or when auricular fibrillation is present with a rapid ventricular rate and significant pulse deficit. When indicated it should be given in therapeutic doses with care to avoid toxic effects. The routine administration of quinidine on the assumption that it might ward off ventricular fibrillation is entirely unwarranted, in my opinion. It is of value in the occasional case of ventricular tachycardia or to suppress premature ventricular beats when these are so numerous as to embarrass the cardiac output.

The value of xanthine derivatives seems well established. Theobromine sodium salicylate (5 to 7 grains T.I.D.), with or without an added $\frac{1}{4}$ or $\frac{1}{2}$ grain of phenobarbital, or aminophyllin by mouth or hypodermically, should be given early and continued into convalescence. In some instances these drugs appear to ward off pain on effort after recovery from the acute disease. In others, the intravenous administration of aminophyllin (0.25 or 0.5 Gm.) dramatically diminishes the pain in the early stages.

It is scarcely necessary to plead the benefits of oxygen therapy, but it should be emphasized that it is of value early in the course of this disease and not alone as a measure of last resort when signs of failure have become apparent. Whatever the basic cause of the pain which attends acute coronary thrombosis it is certainly dependent in large part upon myocardial ischemia. Oxygen therapy by means of a tent or mask will often afford gratifying relief. By the first method a concentration is attainable in the atmosphere of 40 to 50 per cent and may be continued indefinitely. By means of a mask pure oxygen may be administered, although this is usually not well tolerated for long periods. In addition to its effect on pain, oxygen therapy, in so far as it increases the oxygen content of the myocardial blood supply, enhances the healing of the infarct and the function of the remainder of the ventricular muscle.

Beyond these immediate measures none is so good as time. But this must be prescribed with caution; too little may wreck the results of early treatment, too much may contribute unnecessarily to an invalid reaction. How long should rest be continued? If one may judge from the rate of healing observed post mortem in fatal cases the largest infarcts heal within 6 weeks or 2 months. No clinical signs make possible accurate evaluation of the size of the infarct, but a general impression may be gained from the concomitant degree of circulatory embarrassment. Cardiac dilatation or prolonged fall in pressure should call for rest of 2 months with very

gradual return to activity. Light massage and passive exercises should be performed before the patient sits up or stands.

Along with the increasing accuracy of early diagnosis of acute coronary thrombosis the prognosis has improved distinctly. This is due less to any improvement of therapy than to a better comprehension by the profession of the natural history and usual course of the disease. The secondary results of early diagnosis, complete rest during the first few weeks, and gradual return to activity have also contributed much toward the better prognosis. The result is that coronary thrombosis no longer always entails complete and permanent disability; half of its victims return to full or part time work within 6 months to 1 year.

The location of the infarct, anterior or posterior as distinguished by the electrocardiogram, does not significantly influence the outlook for recovery. Certain other factors do, however, appear to bear a determining relation: 1. Number of attacks; the immediate and remote prognosis become worse with each successive attack; 2. age of patient; the immediate prognosis and the outlook for complete rehabilitation are better in younger than in older patients; 3. cardiac enlargement; the probability of recovery and the likelihood of return to active life are twice as great in patients without cardiac enlargement as in those with enlarged hearts; 4. congestive heart failure and angina pectoris; the development of symptoms and signs of congestive heart failure during the acute stage of the disease, or the persistence of these, or of pain on effort, during and following convalescence, distinctly impair the prognosis in any given instance. Both are indications that the remainder of the coronary circulation is insufficient.

In four years, 1936 to 1940, 284 cases of coronary thrombosis were observed in Johns Hopkins Hospital, 205 in men and 79 in women. The incidence was highest in the sixth decade of life. One hundred and forty-four were private patients, 140 were admitted to the wards. Thirty-one private patients died in the hospital, 59 patients died in the wards. The death rate varied from 20 per cent among patients under 40 to 48 per cent among those between 60 and 70.

There were no therapeutic measures or nursing procedure available to the private patients which were not applicable on the wards. The professional care after the patients were admitted was in many instances rendered by the same physicians. The implication of these figures is that among the private patients the disease was recognized and hospitalization was advised at an earlier stage. It is in this direction that we must look for the next advance toward the more successful treatment of acute coronary thrombosis.

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THE TREATMENT OF INFECTED TRAUMATIC WOUNDS

BY DAVID J. LEWIS, M.D.

Springfield.

In discussing the subject of infected wounds I am going to limit my remarks to the care of traumatic wounds that from the start of treatment must be considered already infected or certain to be so complicated. What wounds should we classify in this manner? The mere presence of bacteria on the wound surface does not mean that they are infected. All wounds have bacteria on their surfaces, whether they are made intentionally under aseptic precautions or made accidentally without regard for asepsis. As long as the bacteria remain on the wound surface they contaminate the wound. As soon as they invade the tissues beneath the surface they infect the wound. Some general surgeons are now so conscious of this contamination of operative incisions that they are advocating soap and water cleansing of all such wounds before closure, even advocating a special soap for this procedure.

The incubation period that separates the stage of contamination from that of infection is usually considered to be from 6 to 8 hours after the inception of the wound. This time, of course, is necessarily an average time because the invasive power and rate of different organisms vary. Usually bacteria of human origin are more rapid in their invasion and a virulent infection may develop in a few hours. (A recent British study of some of the World war wounds showed that 15 per cent. of the cases were obviously infected with the hemolytic streptococcus on admission to hospitals after an average of 12 hours from wounding. In a much greater number the infection was evident after a day or two, in some instances running to 90 per cent of organisms causing the invasion. Colebrook states that this one organism was responsible for about 70 per cent of the deaths from wound infection and caused most of the serious infection complications.) Some wounds are contaminated by these virulent bacteria at the time the wound is caused. In many instances, however, these organisms gain entrance to the wound when first aid measures are attempted or improper examination of the wound is made. It is necessary then for all of us to limit our first aid attempts to covering the wound with a sterile dressing until we can prepare ourselves, our instruments and surroundings for an aseptic approach to the wound. Some wounds are so badly soiled that from the start we know they will have bacterial invasion below the surface and it is unwise to close them even after the most careful cleansing and debridement.

We may consider a wound infected when it has not received treatment for six to eight hours after inception or is so soiled that removal of contamination is impossible.

We should approach this infected wound as carefully as though it were clean. The operator should be scrubbed, gowned and gloved to prevent the introduction of any virulent organisms from his person. The skin about the wound should be shaved, washed with soap and water and the area draped before the dressings are removed. All instruments must be sterile and should be used but once. Surface toilet should consist of removal of dead tissue, foreign bodies and blood clots. In the normal process of repair, when the space between the two wound surfaces has been bridged by healthy granulation tissue and all debris has been absorbed, bacteria can no longer grow and produce infection because of the high degree of immunity exhibited by young fibroblasts and angioblasts. If, on the other hand, the accurate apposition of the healing surfaces is prevented by the presence of blood clots, serum, or devitalized tissue, then excellent culture media is provided on which bacteria may grow unchecked and from which they may invade surrounding living structures. Hemorrhage which has continued from the injury or caused by the cleansing efforts should be stopped by pressure or fine ligature. All tissues must be handled carefully. Cannaday has said that theoretically the tissues of open infected deserve more carefully handling than clear wounds because they will be without the protective covering of the skin. The surface and pockets of the wound should be cultured to determine what organisms are present. Meleney has for several years been directing our attention to the importance of knowing accurately what organisms are on the surface of the wound and contributing to or causing the infection. During the last few years we have learned of several chemicals that have the effect of improving the patient's ability to combat certain infections. To make use of these agents and to be able to use them intelligently we must know what organism is to be combatted. The usual culture is made for the study of the aerobic bacteria. Many anaerobic or aerophilic organisms are present in pocketed, undermined or deep tissue wounds and their presence will not be found unless special cultural methods are used.

If tendons or nerves are severed it is unwise to attempt their immediate repair in a wound that is infected or that undoubtedly will become so. In this situation it is well to give care to the wound to promote rapid closure with a minimum of scar and plan a secondary tendon or nerve repair at a later time (Bunnell has introduced non-irritating removable fine steel wire for the repair of finger tendons and states that it is possible in some instances to do a primary repair of tendons as late as twenty-four hours after injury.)

If a fracture complicates the wound, the bone and periosteum should be cleansed, soiled bone being sacrificed. Any large piece should be saved even if completely detached. It is now possible to

use vitallium in various forms for the internal fixation of such fractures, if this procedure does not require much operative manipulation. The fracture should be aligned and held in position by skeletal fixation. We use plaster around large Kirschner wires, the wires being placed through normal tissues at some distance from the wound. The wound is then held open with a vaseline pack over which absorbent dressings are applied. One of the most interesting reports of treatment of a large number of infected Spanish war wounds shows efficiency of this type of treatment. Joseph Trueta of Barcelona treated over 15,000 wounds of the extremities, many being compound fractures and most of them considered infected wounds by the standards I set at the beginning of the paper. In brief, he clears away all bruised tissues being careful to preserve as much skin as possible and all large bony fragments. The cavities are left open and packed with gauze; a cast applied over the skin immobilizing the joints above and below the injury; the cast not removed until the development of intolerable odor or soaking of the cast by drainage. All cases were given gas gangrene tetanus antitoxin. The fear that such a treatment of fractures must lead to an increase in sepsis, especially gas gangrene was found to be groundless, and the method, by facilitating evacuation of the patients and dispensing with apparatus had great advantages under war conditions. In 1075 cases that he was able to follow there were 6 deaths, only 2 of which were due to gangrene. He reports good results in 91 per cent. of cases.

An infected wound should never be closed. Should it be drained? If there are pockets in the wound or dissections along fascial or muscle planes that will not be benefited by gravity drainage, it is usually wise to insert Dakin's tubes and continue Dakin's technique until the wound bacterial count has shown a satisfactory decrease and until all devitalized tissue has been removed by this chemical debridement. If dead tissue or bone has been removed and a dead space exists between the deeper layers of the wound that cannot be closed by elastic pressure on the wound edges, I have found the vaseline pack of great value. Orr popularized the use of this pack in the treatment of the open wounds following removal of the sequestræ of osteomyelitis. It is surprising how rapidly an infected wound will fill with healthy granulations if this pack is correctly applied and left alone for several weeks. This pack is not placed for the purpose of effecting drainage but is introduced to exert pressure against the walls of the wound thereby suppressing exudation and stimulating granulation tissue. If vaseline or rubber strips are inserted for the purpose of drainage they should be removed by 48 hours and not replaced. (I have noticed around the local hospitals, daily dressing of infected pocketed wounds, drains pulled out and reinsured, usually without preparation of the

surrounding skin field. I believe this procedure is unnecessary and an almost certain way of introducing additional organisms into the wound.) Koch has recently made this significant statement concerning the drainage of wounds: "If the wound opening is adequate and well placed, drainage will continue as long as exudation is present. If it is not, insertion of a drain will not compensate for the error." Even superficial wounds should be dressed with the idea of facilitating drainage. Here wet dressings changed frequently enough to prevent drying of the wound secretion will be effective. Isotonic salt solution is really better for this purpose than most any other solution, because its prolonged use does not cause tissue edema and maceration so frequently seen when boric or plain water packs are used.

The use of chemicals in an effort to prevent and cure infections in wounds has received impetus during the last few years since we have had experience with the 'magic bullets,' sulfanilamide and sulfapyridine. Numerous reports of lowered morbidity in wounds which were treated with these chemicals have appeared. The use of sulfathiazol should give still better results since it is effective against the staphylococcus and certain anaerobes as well as against all of the organisms that the other chemicals combat. The usefulness of these substances seems to be in preventing infection in wounds, the bacteriostatic action of the chemical delaying bacterial invasion and allowing a longer time for the natural body defense mechanism to get effectively into action. How effective these drugs will be locally after the wound is once infected has not been reported. Their general administration to a satisfactory blood concentration is indicated if wound culture shows the presence of an organism susceptible to their action. There is a great deal of doubt about the necessity or value of the use of antiseptics on the dressings. These drugs in the dressings may prevent outside organisms from getting into the wound from the surrounding skin but there is evidence to show that most of them are harmful to the surface cells of the wound. Recent studies show merthiolate less toxic than phenol, iodine, mercuriochrom and metaphen but not an ideal antiseptic because it is more toxic to connective tissue and epithelial cells than to bacteria. McClure reports the mercurials, bichloride, metaphen and merthiolate, as having a high toxic coefficient, with the cresols and hexylresorcinol relatively low. Meleney has proven the value of the use of zinc peroxide where contamination with anaerobic and micro-aerophilic organisms is suspected or proven by culture. This chemical is an oxidizing agent and must be applied as a creamy suspension in sterile distilled water to every part of the wound surface. When delayed closure of such a contaminated wound seems advisable, it may be packed wide open with gauze saturated with zinc peroxide. I have found this material of great value in cleaning

up dirty wounds, especially wounds about the mouth. The method has the disadvantage of requiring daily dressings and the changing of packs because the material is active for only 24 hours.

For wounds infected with *B. pyocyaneus*, acetic acid wet dressings are sometimes indicated. There are a few organisms against which we have specific bacteriophage. Moist phage dressings are indicated in these cases. Occasionally a wound has in it devitalized tissue that cannot be removed surgically because of possible damage to nearby vital tissues or because of the danger of breaking through nature's inflammatory defense wall. In these cases some of the protein digestants may be of value. It is now well established that the introduction of maggots into a suppurating and poorly healing wound definitely assists in the process of healing. Part of this beneficial effect can be attributed to the fact that the maggots remove necrotic tissue and thus clean the wound, making it poorer soil for the growth and proliferation of pyogenic organisms. Investigation has shown that allantoin is excreted by maggots, and that it greatly promotes the healing of infected wounds. Urea in a 2 per cent. solution is also efficacious in the treatment of such wounds. It is well known to biochemists that strong urea solutions are excellent solvents for protein material. Many investigators agree that concentrated solutions or even crystals of urea are superior to the dilute solutions first employed. The advantages of this treatment is its low cost of freedom from tissue irritation. It is absolutely nontoxic. Robinson has recently advised using 2 per cent. sterile solutions of ammonium bicarbonate for dressing wounds where tissue digestion is helpful. He feels that the effectiveness of maggot therapy in such wounds was their ability to form urea which was acted upon by urease to form ammonium bicarbonate. This substance is very effective in the digestion of protein. It is nonirritating to living tissue.

(Loehr reported using cod liver oil dressings for burns in 1943. Since then he had advocated this substance as a dressing for infected wounds. The substance in the oil which is responsible for the ability to improve the healing of chronic and infected wounds was first thought to be the Vitamin A. Subsequent investigations seem to indicate that the unsaturated fatty acids are the active constituents. In fact, an excess of Vitamin A applied locally seems to retard wound healing. Getz has studied the effect of various oils on the healing of chronic tuberculous ulcers and has made these interesting findings. Healing was more rapid in the animals treated with cod liver oil. Paraffin, lanum, olive and cottonseed oils were inactive. Halibut and tuna liver oils were slightly less potent than cod liver oil in spite of having a higher vitamin content. The saponifiable fraction was irritating and suppressed healing while the non-saponifiable fraction caused more rapid healing than the whole cod

liver oil. This fraction also seemed to have a systemic effect in combating the tuberculosis. The active principles were shown not to be either Vitamins A or D.)

In closing my comments on the use of antiseptics in wounds, I would remind you of a recent timely statement of Koch. "Sterilization of the wound area must take place before healing can proceed with any degree of rapidity. The most important single factor in hastening sterilization is simple cleanliness. To permit macerated epithelium, encrusted wound secretion and coagulated blood to cover skin edges and the area about a wound is to provide favourable conditions for bacterial growth. Too often the surgeon satisfies his conscience by pouring an antiseptic solution over the wound area day by day, and convinces himself that he is 'treating' the infection."

The primary dressing should be applied in such a manner that we make use of all of the procedures known to improve tissue metabolism and to prevent spread of infection. Our first object is to get the wound surface covered with healthy granulation tissue. In making this dressing I use a fine meshed gauze or sheeting that has been impregnated with vaseline. This is applied over the raw surface and fluffed gauze is packed over it in sufficient quantity so that when anelastic bandage is applied there will be slight even pressure over the entire wound. The portion of the body which has been wounded should be immobilized sufficiently to give absolute rest to the injured area while active infection is present. This immobilization is effected so that movable parts of the body are in the so-called positions of function. It is well to allow gravity to aid venous circulation by slightly elevating the wounded area. This is especially important in extremity wounds. It is well to keep the temperature of the body about the wound at a constant high level by a heat cradle. Tissue resistance is higher and metabolism is better at temperature between 98 and 100 Fahrenheit.

After the wound has been cleaned and dressed, it should be left alone as long as possible. The odor of the exudate or the discomfort occasioned by its presence will usually force the surgeon to redress the wound every three or four days. The wound should be left alone but not forgotten because during the first few weeks, at least, we must be careful not to delay in our detection of such complications as gas bacillus infection or cellulitis. An elevation of pulse or fever should call for an inspection of the wound and surrounding tissue. When a wound is Dakinized more frequent dressings are necessary. If wet dressings are applied to a wound surface with the idea of facilitating drainage, they must be changed often enough to accomplish that purpose.

There is much need for care in preventing outside infection entering the wound when it is being redressed as there was at the time of primary care. David has recently written an editorial on the

need for careful dressing and points out that "if the skin surrounding an infected wound is treated as if it were potentially a sterile field, the wound itself is spared contamination by shifting dressings. The granulation tissue contains most of the fighting forces of the body against infection. Left to its own devices and protected from constant recontamination, it soon demonstrates its ability to combat the infection upon it. The result as a wound covered with a red, healthy bed of granulation tissue, with all evidences of healing.

The granulation tissue covering the wound should be covered with skin as soon as active infection has subsided and dead tissue has been eliminated. This can be accomplished by pinch grafts in the presence of slight infection or a large perforated split thickness graft if the wound is relatively clean. Early skin grafting on a suitable surface prevents tissue scarring and greatly shortens the time of convalescence. When the wound has been covered by skin, our job is only partly over. If the wound has been on the extremities, the immobilization and inactivity that the treatment has caused has resulted in weakening of muscles and stiffening of joints. As soon as active infection has subsided the joints and muscles should have periods of active exercise and as soon as skin covers the wound, warm baths and light massage should be given until there has been a satisfactory return of function. Neglect of this part of the treatment may result in joint or muscle stiffness that may remain as permanent disability.

In our concern for the care of the wound we must not lose sight of the fact that the local resistance of tissue to infection and the rate of wound healing depend a great deal on the general condition of the patient. To increase the body's resistance to the pathogenic anaerobes we routinely use gas gangrene tetanus antitoxin, 3,000 units intramuscularly at the time of first dressing and a similar dose on the third day. A diet high in protein elements raises the ability of the body to resist infection and increases the rate of wound tissue growth. This type of feeding is given routinely to our cases with open wounds. Dehydration definitely delays wound healing and the fluid balance should therefore be accurately controlled at all times. Vitamin A in adequate amount is necessary for proper epithelial metabolism. Vitamin C is apparently necessary for the laying down of the intercellular substance in wound healing. Anemia or a low blood protein is liable to develop in a patient ill with an infected wound. The onset of either condition seriously reduces the patient's ability to resist the infection or heal the wound. Blood and serum transfusions are so easy to give that there is no excuse for letting a patient remain under this handicap. We routinely give our fracture patients a quart of milk a day to insure adequate calcium and phosphorus intake. We also give Vitamin concentrate to patients who have a poor appetite or who are going to be ill for a few weeks or longer.

Summary: 1. The wounds when first seen must be relieved of all foreign material such as foreign bodies, blood clots or dead tissue.

2. The wound should be left open and treated by Dakin's dressings or some modification of Orr's technique.

3. The injured area should be immobilized, dressed with slight pressure and elevated if possible.

4. The wound should be cultured for aerobic and anaerobic organisms. If a specific form of chemical therapy is available for the organism found it should be used.

5. There is no need for the routine application of antiseptic solutions to the wound itself.

6. The patient's general condition should be carefully watched and kept near normal conditions.

(*Illinois Medical Journal*, Dec. 1940.)

Medical News & Notes.

TATA MEMORIAL CANCER HOSPITAL, BOMBAY, was opened by the Governor of Bombay on February 28, 1941. This is the first and foremost fully equipped hospital in India for the treatment of cancer. Sri S. Saklatwala welcomed the Governor and said that the hospital was dedicated to the treatment and study of cancer and allied diseases. It is intended for the treatment of patients with tumours, both benign and malignant and for the diagnosis of tumours. The clinic for new patients will be open every morning from 9 to 11 A.M. where the patients will be seen, the diagnosis made, and the necessary laboratory investigations carried out. Only when the diagnosis is established the type of treatment will be discussed at the staff meeting and agreed upon. If the tumour requires surgery, he will be admitted to the hospital. If x-ray or radium treatments are to be started, this will be done as an out-patient, unless the patient is extremely ill and needs pre-operative preparation before surgery or x-ray treatment. The use of the hospital beds is reserved for those most seriously ill and the staff of the hospital will be best judges regarding this question. Accommodation will be provided gradually to a total of 100 beds and treatment will be free to the poor. In this hospital, bedside study of the patients, treatment and research would be intimately co-ordinated. The clinician will be a research worker and the laboratory investigator will have the opportunity of extending his experience to the hospital patient. Most of the clinicians and the laboratory staff will devote their whole time to the work of the institution. It is proposed to have frequent conferences of the

staff which will formulate a unified programme of work. The institution hopes to develop into a centre of teaching for medical men all over the country in the diagnosis and treatment and control of malignant disease. The institution has received advice from and is modelled after similar institutions in England and America. The staff of the hospital consists of Lt. Col. Sir J. N. Duggan, Superintendent; Dr. J. L. Pool and Dr. R. B. Cohn, Surgeons; Dr. V. R. Khanolkar, Pathologist; Dr. K. P. Mody, Radiologist; Dr. B. N. Sircar, Anaesthetist; Dr. D. R. Meherhomji, Resident Surgical Officer; Dr. R. Naidu, Physicist; Dr. L. H. Athle, Assistant Radiologist; Dr. V. V. Gharpure, Assistant Pathologist. This hospital is the first and foremost of its kind in the east.

KAMALA NEHRU MEMORIAL HOSPITAL, ALLAHABAD, was opened by Mahatma Gandhi on February 28, 1941. He opened the main gate of the hospital with a silver key and said that the hospital is for all alike without distinction of caste or creed or community, because "disease and death are with us all without any such distinction." In a statement, Dr. Jivaraj Mehta, M.D., Secretary of the Hospital Committee, says that the construction work being over the hospital starts on its mission of mercy. Amidst large open spaces, the hospital is a modest but elegant piece of masonry. From the central block housing the offices of the hospital, the wards start off at various angles in three directions. Adjoining the offices are the two operation theatres and four labour rooms. The building is all brick work with a flooring of reinforced brick slabs. All the floors are tiled and the operation theatres have marble flooring. The wards are open, airy, spacious and well lighted, and special attention has been paid to sanitary requirements. In the wings to the right and left are situated two general wards with 12 beds each; these beds are free. There are 7 semipaying beds and 6 full-paying ones with separate private accommodation for each. An antenatal clinic and dispensary have been provided in the hospital building, where women in pregnancy or those suffering from diseases peculiar to women will be attended to. Quarters have been provided not only for the medical and nursing staff but also for the labour staff. The hospital has been able to secure the services of a highly qualified surgeon, Dr. M. S. Satyapriya Majumdar, F.R.C.S. (Eng.) as its superintendent. The hospital is intended to be utilised for labour cases and for women suffering from special gynaecological troubles.

THE PREMIER RADIOLOGICAL INSTITUTE, Mylapore, Madras. (Director Dr. K. R. Doraiswamy) The deep x-ray therapy installation in this Institute was switched on by Major-General N. M. Wilson, I.M.S. Surgeon-General with the Government of Madras on 6th January 1941. A large gathering of medical men and women was present. After tea, the visitors went round the Institute and inspected the installation. The Institute has facilities for x-ray

diagnosis (Radiography and fluoroscopy) x-ray treatment with a stock-proof oil-cooled tube with a working range from 80 K.V. to 200 K. V, diathermy, electro-cardiography, and ultra-violet rays, and other electro-medical equipment.

THE INDIAN MEDICAL SERVICE.

According to the Associated Press. The Indian Medical Service, which at the outbreak of the war, was about two thirds European and one-third Indian has so rapidly expanded that now there are twice as many Indian as European officers.

The officers undergo a course of fourteen days intensive military training before being posted to military hospitals for further hospital and technical training. The course of training includes elementary drill, discipline and lectures and demonstrations on message writing, anti-gas training, military hygiene, military law, military medical organisation in peace and war, revolver training, care and maintenance of M. T. route march, collection and loading of casualties etc. This training extends over a total of seventy-five working hours. On completion of their course, officers are posted to various hospitals in the command.

Training centres have been established at Rawalpindi, Quetta Lucknow and Poona and selected candidates are first posted to one of these stations for fourteen days' intensive training of purely military nature. A certain number of trained officers are detailed for overseas service after they have completed their three months' probationary period.

It is explained that the expansion of the army in India, which has been in progress since the outbreak of the war has necessitated a corresponding increase in the number of medical officers required. The I. M. S. war reserve, which is employed in civil departments in peace time, is intended to provide the necessary trained nucleus around which an expanded medical organisation of the army can be built in war time. This reserve has been called up in regular monthly batches and the time is not far distant when the recall will be complete.

This reserve, however, was never intended to be more than a nucleus of the expansion required. Moreover it could only be mobilised gradually if the civil departments were not to be dislocated. A large number of doctors have therefore been recruited from among the general public and very many more will be required as expansion continues. The system of recruitment, which obtained before the war, has accordingly been replaced by appointment to emergency commissions in the Indian Medical Service, on the same lines as the policy adopted for the Indian army. All those who are appointed to such emergency commissions will be eligible for permanent commissions after the war.

Two hundred and twenty-one Sub-Assistant Surgeons (emergency branch) have been trained since June 1940 at Lucknow alone while there are others at Rawalpindi, Quetta and Poona. The training covers a period of one month.

As in the case of the Indian Medical Service, recruitment to the permanent and reserve cadres of the Indian Medical Department (Sub-Assistant Surgeons branch) was suspended at the outbreak of the war and replaced by appointment of an emergency type. These appointments are open to medical men possessing licentiate qualifications. Candidates now appointed to the emergency cadre will be eligible for permanent appointments after the war to the regular cadre of the Sub-Assistant Surgeons branch of the Indian Medical Department as well as to the provincial subordinate medical department.

The Provincial Governments have played considerable part in the supply of doctors for the army. A large proportion of the Sub-Assistant Surgeons now employed with Indian troops have been obtained from provincial medical departments in accordance with the terms of the bonds executed by these Sub-Assistant Surgeons undertaking liability for military service at the time of their appointment to the provincial cadres.

Obituary.

The death in Bombay of Mr. H. Walker, Managing Director, Messrs. May & Baker (India) Limited, Calcutta, came as a great shock to his many friends and business associates.

A member of the Pharmaceutical Society of Great Britain, the late Mr. Walker worked in West Africa and in Burma prior to joining Messrs. May & Baker (India) Limited in 1933. He was subsequently appointed Managing Director and will always be remembered by the staff for his ability and rigid sense of fairness as well as for his consideration and kindness.

The late Mr. Walker became ill in October last and his death finally resulted from cardiac failure.

The large number of letters and telegrams received, and the wreaths received from Bombay and Calcutta were a tribute to one who was a popular employer and a truth friend.

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