

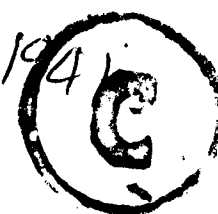
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NO. 2

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MEDICO, SURGICAL SUGGESTIONS.

FEB - 1941

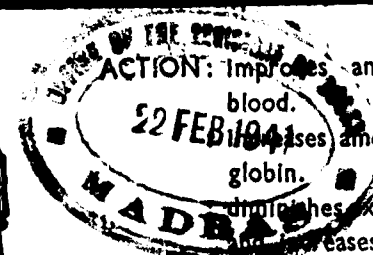
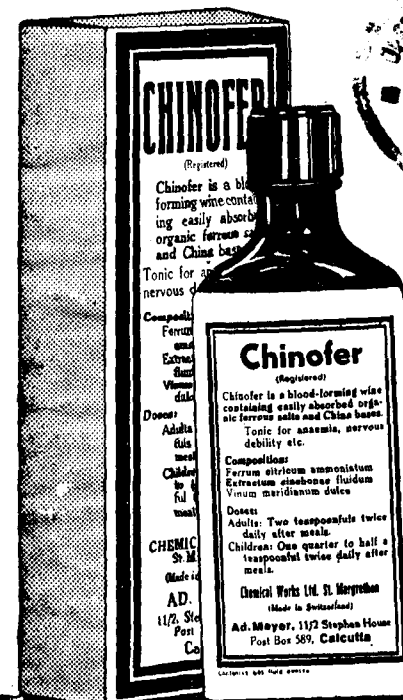


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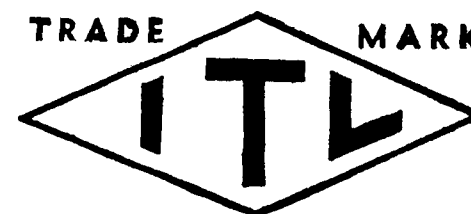
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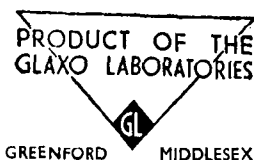
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MEDICO-SURGICAL SUGGESTIONS

(Established 1932.)

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FEBRUARY 1941

NO. 2

DENTAL CARIES*

BY DR. S. K. GUPTA, B.D.S.C., B.O.

Surgeon, Dentist and Analyst, Railway Road, Pasrur, (Dist. Sailkot.)

Definition:—Caries of the teeth is a process by means of which molecular death of a part of a tooth gradually takes place, due to the action on the dental tissues of various germs.

It is not a specific disease due to one special kind of germ, but rotting away of the tooth from the action of one or more kinds of germs which have the power either of producing acid or liquefying collagen, or of doing both.

History:—Caries or decay of the tooth is more or less a disease of civilisation. Though it existed among the ancients, for there are traces of this disease found in ancient skulls, and mention has been made in ancient Hindu Medicine, of fillings with gold and silver, presumably for the decay of teeth, yet its prevalence at that distant age was nothing compared to that at the present day. That it is a disease of civilisation is proved by the fact that even the present day primitive races are free from it, compared to the modern civilised races. It is further shown by the alarming extent of the prevalence of this disease among the American and European school children, 75—95 per cent. of whom are found to be suffering from caries.

Introduction:—The mouth cavity even in the healthiest condition is a good incubator for all sorts of germs. In it are found all the conditions viz., the temperature, moisture, medium etc., that are needed by the germs for their development. Naturally all sorts of bacteria are greater in number and more virulent in action in unclean mouths than in clean ones.

Prevalence of Caries:—To what extent dental caries is prevalent in India could not be ascertained, for there are no records available owing to the backward state of dentistry here. From what little information I have collected from my personal dental practice and

*Specially contributed to the "Medico-Surgical Suggestions", Madras.

from the experience of other colleagues, I put down the extent of this disease among Indians to between 30-55 per cent.

A. *Ancient Races*:—Statistics show that races like Romans and Egyptians which had reached a degree of civilisation were more susceptible to dental caries than the races living under more primitive conditions.

B. *Existing Primitive Races*:—Skulls of practically all the races over the world have been examined and they show clearly that races whose principal diet is meat are more free from caries than those existing only on vegetable diet.

C. *Modern Civilised Races*:—Dental caries has increased considerably not only in the human beings but also in the domestic animals.

Frequency and Distribution:—The first permanent molars (Mandibular more than Maxillary) are most prone to caries due to unhygienic condition of the mouth. The caries commences on the occluding surface. The second permanent molar comes the next, the lower more than the upper; then the lower premolars, second more than the first; the upper premolars, upper incisors and last of all lower incisors which are practically immune to caries because of cleaning action of the tongue and lips. Caries is more common in females than in males and more active during the period of growth.

MORBID ANATOMY AND PATHOLOGY

A. Naked Eye Appearances.

(i) *Enamel*:—The first sign is that, it loses its normal polish, next, a whitish spot appears which gradually becomes darker. When the process is slow, discolouration is more marked than when it is rapid. Later on the part disintegrates and a cavity is left. The destruction advances until the dentine is exposed. When caries begins on the approximal aspect of teeth, the actual point of contact is not always decalcified, but around it is a zone showing decalcification. When decay of teeth starts on the approximal surface and reaches the under surface of the enamel of the crown e.g. on the molars and premolars, then enamel on that part appears bluish-white due to decalcified tissue which has not been removed by saliva. As the caries reaches the surface, enamel grows whiter and eventually becomes so thin that mastication fractures it and the under surface is found to be soft and cheesy. This is known as "*Secondary Dental Decay*." In children in whom rickets develops after the first year i.e. after the eruption of incisors, these teeth decalcify, become brown and leathery. In these children the saliva is acidic.

(ii) *Dentine*: It becomes tough, hard and not soft and cheesy as with the case of enamel. After softening it undergoes disintegration and acidity is formed and pigmentation develops. The process may spread in any direction, with varying rapidity. In a tooth of

ordinary structure, the cavity is cone-shaped. The apex of the cavity is directed to the pulp in "*penetrating caries*." In badly developed teeth, the decay extends laterally and undermines the enamel. In hypoplastic teeth caries spread rapidly near the junction of the enamel with the dentine and causes the enamel to break away. In "*rapid caries*" the dentine may come off in big masses. This condition is also known as "*Caries Humida*." In this case the action of the acid is more than that of the germs. When the process is slow the action of the germs keeps pace with the action of acid.

(iii) *Cement*:—It is less commonly effected than the enamel or dentine. It generally starts at the neck of the tooth. The cemental substances become soft, disintegrate, leading to the formation of shallow cavities. The cavity becomes wide but seldom deep. An exception occurs at the junction of two roots of a molar tooth; the cavity here is often deep.

(iv) *Nasmyths' membrane*:—For sometime the crown of the tooth is covered with a thin layer of epithelium known as Nasmyths' membrane. It is also destroyed in caries but there is not much change in colour.

The pathological anatomy of caries consists of the destruction of Nasmyths' membrane, if present, by infiltration with germs to such an extent as to obscure all structure. A tough layer of soft tissue composed mostly of germs is often found adhering to the surface of enamel in the early stages of caries, and it is hard to say if this is altered Nasmyths' membrane or a product of germ activity. Its importance lies in the fact that it is very difficult to remove by the ordinary methods of cleansing teeth, and if it harbours acid-forming germs, by holding such acids in close contact with the teeth, it may greatly aid the process. On the other hand, it seems that at times such films may be protective.

(V) PHENOMENA ACCOMPANYING CARIES

(a) *Translucency*:—The part situated between the pulp and the carious process appears translucent. The affected area is cone shaped with its apex towards the pulp.

Cause of Translucency:—There are many theories but the most commonly accepted theory is following:—

The opacity of the normal dentine is due to the fact that the matrix and contents of the tubes have different refractive indices. If these indices approximate, the dentine becomes translucent and we know that there is some change in the contents of dentine tubes which effects its refractive index.

(b) *Pigmentation*:—The colour may vary from pale yellow to dark black. More acute (rapid) the process, the lighter is the colour. It is produced probably by the action of 'chromogenic bacteria.'

B. *Chemical Change*:—There is decrease in the amount of lime salts (1/13 of the original) with the loss of organic tissue.

C. MICROSCOPIC APPEARANCES

(i) *Enamel*:—The surface is covered with a mass of micro-organisms. The enamel is decalcified between the rods and the interstitial cement substances, the enamel globules being set free and dissolved and washed away. The enamel may be penetrated to a considerable extent before breaking down of the tissue occurs.

(ii) *Dentine*:—From within outwards: (a) An unstained part in decalcified condition. (b) The streaks (lines) of stain which are germs occupying the dentinal tubes. (c) The globular masses which are also germs occupying the matrices and tubes also scattered irregularly. These have been termed as '*Liquefaction focii*.' (d) Irregular masses occasionally form, by the fusion of liquefaction focii.

(iii) *Cement*:—It resembles caries of the dentine.

(iv) *Pulp*:—It is inflamed causing a shooting pain.

BACTERIOLOGY

A number of micro-organisms are associated with the production of Dental Caries but the proofs are not definite. The pathogenic organisms found in the mouth are:—

1. Staphylococci.
2. Pneumococci.
3. Klebs-Loeffler or Diphtheria bacilli.
4. Tubercle bacilli.
5. Oidium or Saccharomyces albicans or Thrush fungus.

The non-pathogenic organisms found in the mouth are:—

1. Streptococcus brevis.
2. Micrococcus tetragenous.
3. Leptothrix buccalis.
4. Spirochaete dentium.

Of these the Streptococcus brevis is the most constant, and is found in healthy as well as unhealthy mouths. What function these non-pathogenic organisms perform, is not definitely known.

It is interesting to note the comparative immunity of the system, against the attack of the pathogenic organisms that are mostly found in healthy or unhealthy mouths. The two reasons for it are:—

1. The bactericidal action of the saliva.
2. The resisting power of the body due to antitoxic property of the blood.

The organisms concerned with the decay of teeth are of two kinds.

Acid-producers:—They are anærobic (capable of thriving without oxygen) e.g. Streptococcus brevis, bacillus necrodentalis, staphylococcus albus and aureus, and Sarcina albus.

2. *Liquefiers* (of Dentine): They are Aerobic (requiring oxygen for their growth) e.g. Bacillus mesentericus ruber, Bacillus gingivæ pyogenes etc.

PROCESS OF CARIES AND SOURCE OF THE ACID

Caries is a chemico-parasitical process consisting of two parts:

1. Acid formation and softening of the tooth substance by means of these acids.

2. Liquefaction and digestion of the softened tooth-tissues.

For the working of this process two things are absolutely necessary viz., the bacteria, and such suitable food-stuffs (carbohydrates) which can be decomposed into acids by the action of these bacteria.

Lactic acid is the principal form of the acid which is formed from the carbohydrates which lodge on the teeth and undergo fermentation. In the case of *sugars* of the grape sugar group, galactose is directly fermentable. The cane-sugar and milk sugar are only fermentable by taking water into their molecules. The dextrose and levulose are acted upon by lactic acid ferment:

Cane-sugar + H₂O → Dextrose + Levulose → Lactic Acid ferment and gases.

In the case of starch, the ptyalin (ferment contained in the saliva) converts it into grape sugar which is directly fermentable. Experiments have proved that when fermentable albuminous materials are combined with saliva, very little acid is formed, but when such substances e. g.; meat, undergo putrefaction acids are formed. Saliva neutralises these acids and the reaction of the saliva remains alkaline. Vegetable food is less likely to ferment when raw than when it is cooked.

When once the decalcification has taken place by means of acids, then the remaining and the major portion of the tooth substance is dissolved by the action of bacteria which act upon the dentine in the same way as the pepsin of the gastric juice acts upon the albuminous materials. It has been shown that the primary decalcification of the enamel often starts around and not at the point of contact. This fact seems to indicate that the food in the fluid condition is maintained around the point of contact by capillary attraction. Again the mouths which are kept quite free from the solid food caries is often found in pits and fissures, etc., which clearly indicates that the disease must arise from the action of fluids and not solids. Solid food cannot ferment so rapidly as the liquid food, because, the particles cannot move about so freely in the solid substances as they can

in fluids. With the solid substances the acid formed by the germs remain just around them and cannot disperse; and when the acid is raised to a certain percentage, it does not allow the germs to grow. Some observers do not agree with my view. The mucus secreted by the glands of the mucous membrane often has an acid reaction due to the presence of acid phosphates, lime; and soda; and this acid reaction is in some way related to the rapid caries in cases of mouth-breathers.

SPONTANEOUS ARREST OF CARIES.

It may occur in milk or permanent teeth but only in those which has living pulp. Among the permanent teeth, it is mostly met with in under-developed 1st permanent molars. The teeth show extensive loss of tissue, enamel of the masticating surface, and of the sides disappears. The exposed dentine is dark and the probe will show that it is harder than normal. Microscopically the dentine shows the colour of caries which is more dark. Surface is irregular and the dentinal tubes end abruptly on the surface.

It is not clear how the arrest of the process is brought about. Probably it is due to vital activity of the tooth itself, because the process is not arrested in pulpless teeth. Arrest of caries is met with in pulpless teeth as well but the death of the pulp always occurs after the arrest of caries.

EXPERIMENTAL PRODUCTION OF CARIES.

Perfectly sound teeth cut in pieces are placed in a mixture of saliva, meat and bread. The mixture is changed frequently for 3 months and kept at a temperature of 98.4° F.; at the end caries of the teeth was found but there was no translucency and pigmentation. If these teeth are exposed to coffee, tea, tobacco, fruits etc., shades of different colours develop.

Sound teeth, if placed in 2 per cent. glucose broth in test tubes and streptococcus salivarius added later on, showed penetration of dentine by germs from the pulp side after 14 weeks.

Teeth were kept in cow's milk (50 to 100 c.c.), the milk was curdled and was acid in 2 or 3 hours; it remained so for 2 to 3 days and then changed to alkaline with a bad smell; the dentine became soft with white opaque patches on the enamel and the dentine tubes showed germs in them.

SUSCEPTIBILITY AND IMMUNITY TO DENTAL CARIES.

For this purpose teeth are clinically divided into three groups:-

1. *Native* :—The teeth of primitive races.
2. *Sclerotic* :—Hard teeth immune to caries.
3. *Malacotic* :—Soft teeth susceptible to caries.

For examination of certain markings on the enamel surface, the following method may be employed.

The tooth may be washed with water, dried, surface cleaned with Lysol or Ether and Alcohol; graphite may then be applied by cotton and excess may be removed. It marks the pits and pitches and in this way the surface of the enamel is seen traversed by a series of parallel grooves and ridges to which the name '*Imbrication lines*' is given. These lines are mostly marked at a point 2/3 of distance between the neck and the crown and they are more marked in the third type than in the second; and more in the second than in the first. These lines are intimately related to the striæ in the enamel and they are the result of intermittent and interrupted calcium deposit. Hence imbrication lines can be regarded as an index of the degree of calcification of the enamel. Organic contents of the enamel is 1 to 2 per cent. (the rest is calcium); and increase in the amount of organic matter lessens the resistance of the enamel and helps caries. Caries follows the lines of organic matter. The difference in the hardness of the enamel is not due to quantity but to the quality of the inorganic salts of calcium salts. Pits and depressions, cracks and fissures predispose to caries.

INFLUENCE OF LIME SALTS IN THE FOOD.

In districts where the soil and drinking water are rich in lime, the teeth are sounder than in the places where lime is deficient. If the teeth are regular and in good position, the caries is less likely. Some mouths are self-cleansing and others are not. In the former, the caries is less common than the latter.

Saliva :—A strong flow of saliva flushes the mouth and removes the particles of food from the teeth. If it becomes less and thick, caries increases. High alkaline saliva is the best means to check the progress of caries. Diet, rich in calcium increases the alkalinity and the quantity of saliva and it favours good quality of teeth. Potassium Sulphocyanide forms, 1 per cent. of the total solids in the saliva. It has got no effect in any way on caries.

Pregnancy increases susceptibility to caries because calcium is required for the formation of bones of foetus, but of that there is no proof.

Feeding in infancy influences the disease. Breast-fed children have the best teeth. Starchy foods given along with the milk to the babies is harmful.

Adenoids :—Mouth-breathing causes the food to adhere to the teeth and increases the number of germs and helps caries.

Poverty :—Poorest children have the best teeth because they take most simple food and cannot afford starchy substances such as cakes and sweets.

ETIOLOGY OF DENTAL CARIES.

There are several theories propounded by different investigators. They are:

1. Chemico-parasitic.
2. Inflammatory.
3. Electrical.
4. Chemical.

Of these the first that "Caries is a chemico-parasitic process" as already been described above is the best and most accepted by the Dental Profession.

The form of relative positions of the human teeth favour the lodgment of food-stuffs in solid form or in solution upon or between the teeth. Of the three groups of food-stuffs (protein, carbohydrates and fats), the carbohydrate group is specially concerned in bringing about caries. Of the three groups of carbohydrates (cellulose, starch and sugar), cellulose undergoes no change in the mouth. Starch is first converted into dextrose and by microbic ferments into lactic acid and other organic acids in minute quantities. Sugars are directly fermentable and are converted into acids whereas cane-sugar takes up water and is broken up by ferment *Invertase* produced by bacteria into dextrose and levulose:

Cane-sugar + Water (*Invertase*) $\rightarrow$ Dextrose + Levulose which directly ferment and produce *lactic acid* by the action of germs. These acids attack the teeth where they are produced i.e., wherever the food is solid or fluid and have lodged on or between the teeth and they lead to decalcification first of the enamel or cementum and later on of the dentine. The decalcification of the enamel results in its complete destruction; because of the decalcification the organic matter does not hold together and as it is of nitrogenous nature, it undergoes decomposition by the action of bacteria in the presence of moisture and suitable temperature. This decomposition is brought about by a ferment produced by the bacteria in the mouth. The prevalence of caries in modern races is due to the soft character of food and increased consumption of carbohydrates.

PREDISPOSING CAUSES OF CARIES.

The causes of Dental Caries may be divided into two main parts:

1. Local:
 - (a) Connected with mouth.
 - (b) External.
 - (c) General Environment.
2. General:
 - (a) Heredity.
 - (b) Pregnancy.
 - (c) General Environment.

Local Causes:—The formation of the teeth has much to do with the occurrence of caries. *Teeth with weak structure are more prone to caries than well formed teeth.* A normal arrangement of the teeth and the gums in Nature is a great safeguard against the caries. This arrangement is that "all the teeth come in contact with each other at one single point, forming the V-shaped spaces in between them; and which are filled up by the gum tissue." This arrangement will enable Nature's cleansers (the tongue and lips) and the artificial means such as brush and tooth-pick to keep the teeth clean. But any condition (e.g., abnormally placed teeth or loose and receded gums) which alters this natural arrangement, will tend, by creating spaces facilitating lodgment of food-stuffs and micro-organisms, to promote the development of Dental Caries.

External Causes:—The acid and mucoid condition of the saliva due to dyspepsia, febrile conditions and pregnancy is a potent cause of caries. The acid condition works as a direct cause, while the other works indirectly by sticking on to the teeth and so helping the food particles to stick on them.

Such food as has the tendency to lodge on teeth-surfaces and to undergo fermentation is the kind that best promotes the inception of caries. *Hence the carbohydrate food is more likely to cause caries than the protein or meat diet. It is said that meat eating individuals are more free from caries than those whose staple food is carbohydrates.* But the experience of medical practitioners and Dental Surgeons in India is otherwise. It is a common experience that Indians who practically live on carbohydrates are infinitely more free from Dental Caries than any meat eating European nation.

GENERAL CAUSES.

(a) *Heredity*:—Heredity does play some part in the causation of Dental Caries. Its action can be considerably neutralised if proper attention is paid to the food and the habit of cleansing of the mouth of the individual.

(b) *Pregnancy*:—Some women show marked tendency to caries of the teeth during this period. *This may be due to the acid condition of the saliva or to the vomiting attacks during this period.* This predisposition during this period is not constant, for there are instances of mothers of many children possessing good teeth and showing no such predisposition at all. It is believed that this predisposition to caries has its origin in the beliefs amongst mothers.

(i) that it is natural for a mother to suffer dental troubles during this period,

(ii) that she must let her teeth and mouth severely alone during this period, lest any effort at cleansing on her part would hurt or disfigure the foetus.

If only mothers can be made to keep their mouths clean during this period, this factor would cease to be a cause of dental caries and it would be in the interest of both the mother and the baby, that pregnancy should cease to be a predisposing cause of caries.

General Environment :—It is a fact that not only the human teeth have deteriorated and still are deteriorating in the present generation, but that the whole constitution is undergoing a similar change; and that the resisting power of the present generation is much lower than that of our ancestors. Under the circumstances, the satisfactory cause of this must be sought for in the general environments of the present generation than in any particular local condition. So it is believed that the present day cooking and the mad rush of modern civilisation are responsible for the great prevalence of this disease. The present day culinary art needs the food-stuff to be free from fibrous parts, and so prepares it that the cooked food is liable to stick on to and in between the teeth, and undergo acid fermentation in the mouth, thus starting the process of dental caries. The second factor works by lowering the vitality of the teeth and by leaving little time for the individual to carefully attend to the hygiene of his mouth and teeth.

DETECTION OF CARIES.

The detection of caries in an early stage is often difficult. *Inspection* may detect a white spot on the enamel, a small hole, an altered translucency of a part of the tooth, a darkening of an area, or the lodgment of food, and it is necessary to inspect every part of each tooth that is accessible. Then a *fine probe* may detect a widening of fissures and deepening of natural pits and holes between the teeth or at the margin of fillings, or may pierce friable enamel or softening dentine, or cause pain when passing over an exuding surface. The passing of silk or strands of wool between teeth will detect roughness.

SIGNS AND SYMPTOMS OF CARIES.

1. Lodgment of food stuff.
2. White spot in the place of decay.
3. White spot turning black with the advancement of decay.
4. Cavity formation.
5. Beginning of pain, though very slight,
6. Pain becomes severe on the cavity extending to the pulp.

As described above, the pain comes on at a later stage in the process of decay. The pain is due to irritation of the fine ends of the nerve in dentinal tubules and will vary with the situation and extent of the cavity. If it is on the occluding surface, change in the temperature or introduction of an irritant will produce pain which increase as the cavity deepens. In cavities under the gums or on the approximal surfaces, the pain is often not felt until the pulp is

involved. The pain varies also with the temperament of the individual. Some patients do not complain of any pain even if the disease is advanced. One may be better advised not to wait for the treatment till this symptom appears for then the treatment becomes more painful and more troublesome.

TREATMENT OF DENTAL CARIES.

1. Preventive or prophylactic.
2. Remedial.

Prophylactic Treatment :—

(a) *Mechanical* :—The mechanical process is brought into operation when the hard fibrous portion of the food that is usually consumed by these uncivilised people, thoroughly scrubs the teeth clean during the action of mastication. The teeth are also scrubbed clean by the strong movement of the muscles of the tongue and lips, and the coarse fibrous matter contained in the foodstuffs.

(b) *Chemico-physiological* :—This process is brought about by the antiseptic property of the saliva, and its amylolytic action on certain parts of the food-stuffs.

(c) *Hydro-dynamical* :—This process operates by the parts of the mouth being continually washed by the constant flow of saliva, and by its being forcibly pumped through the crevices between the teeth, by the muscles of the tongue and lips.

(d) *Bacterial* :—This process operates through certain bacteria, whose presence is beneficial to the health of the mouth cavity.

So, taking Nature as our guide, our preventive treatment must consist of :

(i) The adoption of firm, fibrous food stuffs that would scrub the teeth clean and call forth the play of the muscles of mastication.

(ii) Encouraging the flow of the saliva, in order to wash clean the interspaces between the teeth, and to exercise its antiseptic action. This can be best done by introducing natural organic acids, such as are found in fruits, in our meals especially as a final course.

This line of preventive treatment if scrupulously followed, would suffice; but very often it is found that some artificial means at prevention may be needed in addition to these.

(e) *General Instruction* :—The prevention of caries is the ideal of dentistry. Insist upon breast-feeding, if practicable. A diet in early years which requires efficient mastication should be given. Avoid pulpy foods; give crust or toast instead of bread soaked in milk. Better give a piece of bone instead of minced meat. Avoid the use of carbohydrates which are easily fermentable. Sugars are most harmful and so sweets should be forbidden. The last good-stuff

given in a meal should not be of sugar or starch. The meal should finish with a fruit or cheese in case of children. Fruits are specially useful as they are slightly acidic and stimulate the flow of saliva. Give meals at regular hours to children and discourage mouth breathing. Avoid tender teeth because they hinder mastication and because the natural action of mastication is interfered with. Treat them by filling or extraction. The proper cleaning of teeth after the meals is most important. It should be done after each meal. Therefore, nothing should be eaten between the meals. The cleansing of teeth is especially needful after the last meal of the day. Brushing the teeth early morning only is useless. The kind of toothbrush is not so important as the manner of using it. Whatever the type it must be kept properly cleaned. Best is to dip it in a strong solution of Sodium Chloride after use, and leave it to dry in direct sun-light. Another method equally useful is to keep the brush permanently in a glass of 3 per cent. Lysol. It will be softer than it would, if it were kept dry but it will not rot as it does if left out in the air in an ordinary way. There is no need to wash the lysol out of it before use because it is a good tooth-wash, alone or with a paste or powder. A dentifrice may be prescribed in the form of liquid, paste or powder. For emergency mouthwash, one of the following may be selected:

	Part per 10,000 (Nearly 1 per cent).
Chloramine	... 100
Chinosol	... 80
Lysol	... 50
Mercury Chloride	... 2
Formaline	... 5
Phenol (Acid Carbolie)	... 100
Thymol	... 5
Hydrogen Peroxide (H ₂ O ₂)	... 100
Boric Acid	... 300
Potassium Permanganate	... 50
Potassium Chlorate	... 200

The order is of efficiency.

Prevention of Caries by Artificial Means :—

As dental caries is a chemico-parasitical process, naturally something counteracting the chemical and parasitic processes, must form the key-note of the preventive treatment. So the treatment must consist of antiseptic to counteract the effects of parasites, and antacids to counteract the effects of acids that result from the chemical action.

Antacid Mouthwash.

R/				
	Magnesium Carbonate	...	...	3 iv.
	Aqua Rosæ	...	...	oz iv.

Prepare mixture.

Sig : One teaspoonful to be kept and sluiced about in the mouth, morning and night. Shake before using.

Acid Mouthwash.

R/				
	Acid Borici	...	...	gr. xv.
	Eucalyptol	...	...	m. ii.
	Tinct Card Co.	...	...	m. x.
	Aqua Rosæ	...	...	an 3i.
	mft, Lotio.			

Sig : to be used as mouthwash in $\frac{1}{2}$ a cupful of warm water 3 or 4 times a day.

Tooth-Powder

R/				
	Crete præcip	...	...	oz 1.
	Pulvis. Iridis	...	...	gr. xxx.
	Oil of cloves	...	...	m. xx.
	Otto Rosæ	...	...	m. iii.

Prepare powder.

Sig : To be used with tooth-brush, morning and bed time.

Brushing of Teeth —

Too vigorous brushing should be avoided as it produce grooves on the teeth especially when is it done on the same direction. Brush up and down and inside the teeth especially the lower ones.

If gingivitis, tell the patient to massage the gums towards the teeth with finger and thumb or with soft tooth-brush. Scaling and burring of teeth should be done, if necessary.

Remedial Treatment of Dental Caries :—

It is a purely dental treatment, and consists of the removal of the decayed portion. The sterilisation of the adjacent healthy tooth tissue, and filling up of the cavity with suitable filling materials. There are many materials used for the filling of carious teeth. Each has got its advantages and disadvantages. The following are the substances commonly used.

1. Gold.
2. Amalgams.
3. Osteoplastics.

4. Synthetic cements.
5. Gutta Percha.
6. Porcelain.
7. Solutions of gum resins with cotton wools as temporary filling.

Characters of an Ideal Filling:—It must be hard, capable of standing any chemical action of the mouth. It should be non-irritant. It should be non-conductor of heat and electricity and it should be easy of adaptation. It should retain its shape and its colour after insertion. The colour should resemble the natural tooth. At present, no material fulfils these all conditions.

When dental caries is extensive and the crown of the tooth is destroyed, the tooth can be contoured up by means of an artificial crown. The remedial treatment within the reach of the medical practitioners is the use of drugs such as Silver Nitrate and Absolute Alcohol, in cases of slight caries in adults or the extensive superficial caries in children. *This treatment must consist of the application of the saturated solution of pure sticks of Silver Nitrate twice or thrice at intervals of a week, and the application of Absolute Alcohol once a day during the intervals.* During the application of these drugs, saliva must be kept away from the parts treated. These drugs act by precipitating the protoplasm in the dental tubes. Beyond this treatment a medical man may do the radical treatment, e.g., the extraction of the tooth.

Conclusions:—

The medical practitioner would be better advised to stop short at palliative treatment, and leave the legitimate dental treatment to properly qualified dental surgeons.

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THERAPEUTIC APPLICATIONS OF SULFANILAMIDE AND ALLIED COMPOUNDS.

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The introduction of the sulfonamides into medicine for the successful treatment of bacterial infections is rightly regarded by many as marking a new epoch in the history of medicine. The number of new compounds belonging to this class is growing almost daily, and along with this growth there have also arisen many serious problems; the chief of these is the prevention of the promiscuous use of these new chemicals.

One of the most important aids in checking the promiscuous use of these new drugs, many of which are powerful and have dangerous toxic effects, is the new Federal Food, Drug, and Cosmetic Act, which has become operative recently. One of the provisions of this new law is that no new medicinal agent shall be introduced into general practice until the Government has released it. This provision thus makes it imperative that a new drug must be carefully studied in the laboratory, then tried clinically by physicians who have the facilities and training for doing careful research work. Only after these preliminary studies which entail an enormous amount of work does the Government release the drug for use in general medical practice.

It may be well to reiterate briefly the type of examination to which a new substance should be submitted before it is used on human beings:

1. If at all possible, the exact composition (qualitative and quantitative) should be known; or, if not obtainable, the detailed method of preparation of the product.
2. Acute toxicity studies on a sufficient number of laboratory animals of different species should be made; studies on one species alone may be very misleading.
3. Chronic toxicity experiments at varying dosage levels and with different species must be performed in order that any possible cumulative effect of the drug may be noted.
4. Careful and frequent observations of the animals are necessary, so that a composite picture of the clinical course is available. The data on many drugs are very deficient in this respect.

5. Careful pathologic examination of the tissues with appropriate stains is necessary.

6. Effects of the drug on animals with experimental lesions of various important excretory or detoxifying organs, especially of the kidneys and liver, should be studied.

7. The rate of absorption and elimination of the drug, its path and manner of excretion, and the concentration levels in the blood and tissues at varying times after administration must be determined.

8. The possible influence of the presence of certain foodstuffs or drugs should be noted. For example, magnesium sulfate should not be administered to a patient undergoing treatment with sulfanilamide.

9. Careful examinations for idiosyncrasis or untoward reactions should be made.

It is recognized that some will consider these safeguards to be too rigid and that they may simply be considered an ideal. It can correctly be charged, in fact, that some of the pharmacopoeial drugs have not been studied along such lines. Admitting this, it is nevertheless regrettably true that many human lives have been sacrificed by the failure to meet the standards of these preliminary tests and that many more lives will be sacrificed if such standards are not put into effect. Any essential compromise with these requirements will inevitably exact a toll of deaths or injuries among the public. The life and safety of the individual should not be subordinated to the competitive system of drug exploitation.

It is now well recognized that to obtain the best therapeutic results it is necessary to maintain an effective blood level of the drug. This is done by giving a large initial dose which is followed by smaller doses given at repeated intervals. Experimentally the giving of the drug mixed with the food has proved very useful in maintaining a constant or even blood level.

SULFANILAMIDE.

Sulfanilamide is excreted partly unchanged in the urine and partly as the acetyl derivative which is less efficacious therapeutically. Sulfanilamide also passes rapidly into the spinal fluid. This fact is most important in helping to explain its efficacy in meningococcus meningitis. The drug also passes into transudates and exudates.

In order to quickly attain an effective level of the drug in the blood the common practice is to give an initial large dose which is followed by repeated smaller amounts over a period of several days. The following tables 1 and 2 illustrate the point:

TABLE I

The amounts of Sulfanilamide necessary to establish effective blood levels (10 to 15 Milligrams per cent.) in quickly in patients ill with severe Hemolytic Streptococcal, Meningococcal, Pneumococcal or Welch Bacillary infections.

Wt. of Patient	Initial Dose per Os		Maintenance Dose per Os q. 4 hours (Day and Night)		Total Dose First 24 Hours		Total Daily Dose Bicarbonate of Soda	
	Kilos	Pounds	Grams	Grains	Grams per Kilo	Grains per Pound	Grams	Grains
70	150		4.8	1.2	0.15	1.2	3.6	60
60	125		4.2	0.9	0.15	1.2	3.0	50
45	100		3.6	0.9	0.18	1.3	3.0	50
35	75		3.0	0.9	0.23	1.8	3.0	50
23	50		3.0	0.6	0.26	2.0	1.8	30
11	25		1.8	0.3	0.3	2.2	0.9	15

TABLE II

The amounts of Sulfanilamide necessary to establish effective blood levels (4 to 8 Milligrams per cent.) in patients ill with mild or moderately severe tissue infections in which Sulfanilamide therapy is indicated.

Wt. of Patient	Calculated Daily Dose			Dose per Os. q. 4 Hours (Day and Night)		Total Daily Dose of Bicarbonate of Soda	
	Kilos	Pounds	Grams	Grams per Kilo	Grains per Pound	Grams	Grains
70	150		5.4	.07	0.6	0.9	3.6
60	125		5.4	.09	0.7	0.9	3.6
45	100		5.4	.12	0.9	0.9	3.6
35	75		4.2	.12	0.9	1 of 1.2*	60
23	50		3.6	.16	1.1	5 of 0.6	40
11	25		1.8	.16	1.2	0.6	30
* 1 dose of 1.2 grams followed by 5 of 0.6 grams.							
						0.3	1.2

If the infection is mild or of moderate severity, the large amounts of sulfanilamide indicated in table 1 are not needed and the doses of the drug as outlined in table 2 will generally be sufficient to control the infection.

The most common *toxic reactions* following the use of sulfanilamide are listed below:

Mild

Anorexia
Nausea
Acidosis—prevented by NaHCO₃
Incidence 2-4 per cent.

Dizziness
Headache
Scotomata
Abdominal Cramps
Flatus
Diarrhea

Moderate

Simple Fever—3-9 per cent. Important warning sign. Stop use of drug.

.. Cyanosis 90-100 per cent. (Long regards it of little clinical importance.)

Leukopenia or Leucocytosis.

Skin Rash—less 1 per cent. porphyrinuria and sensitivity to light.

Peripheral Neuritis—rare

Jaundice—rare } Stop use of drug.

Severe.

Agranulocytosis—0.5 per cent.

Hemolytic Anemia 3.0 per cent.—(more common in children). Stop use of drug.

Primary Optic Atrophy - rare.

Nicotinic acid is reported to reduce reactions.

TABLE III.

THE CLINICAL TOXIC MANIFESTATIONS OF SULFANILAMIDE, SULFAPYRIDINE AND SULFATHIAZOLE WITH THEIR TIME OF APPEARANCE IN THE COURSE OF THERAPY

TOXIC MANIFESTATIONS.	SULFANILAMIDE.	SULFAPYRIDINE.	SULFATHIAZOLE.
Nausea and Vomiting	Uncommon Occurs early	Very Frequent Occurs Early	Rare
Dizziness	Common Occurs Early	Common Occurs Early	Uncommon
Psychoses*	Rare Occurs Early	Not Reported	Not Reported As Yet
Neuritis*	Rare Generally Early	Not Reported	Not Reported As Yet
Cyanosis	Very Common Occurs Early	Faint, Common Occurs Early	Uncommon Occurs Early

Acidosis	Common if Soda is not given Occurs at any Time	Not Reported	Not Reported As Yet
Fever*	Common Generally 5th to 9th Day May Occur 1st to 21st Day	Uncommon Generally 5th to 9th Day May Occur from 1st to 21st Day	Common 5th to 9th Day
Rash*	Common may take Almost Any Form Generally 5th to 9th Day May Occur 1st to 21st Day	Not Very Common Generally 5th to 9th Day May Occur 1st to 30th Day	Very Common Generally 5th to 9th Day
Hepatitis**	Rare Early or Late	Rare Early or Late	Not Reported As Yet
Hematuria*	Not Reported	Common 1st to 10th Day	Common 1st to 10th Day
Anuria with Azotemia**	Not Reported	Not Uncommon 2nd to 14th Day Blood Pressure Normal Fundi Normal	Not Uncommon
Acute Leukopenia** with Granulocytopenia	Not Common 1st to 10th day	Common Especially in Children 1st to 10th Day	Not Uncommon 3rd to 10th Day
Agranulocytie Anginia**	Uncommon Generally Between 17th to 25th Day May Occur 14 to 40th Day	Uncommon Generally Between 17th and 25th Day May Occur 14th to 40th Day	Not Reported As Yet
Hyperleukocytosis*	In Presence of Acute Hemolytic Anemia	In Presence of Acute Hemolytic Anemia	Not Reported As Yet
Mild Himolytic Anemia	Very Common Early and Late	Common Early and Late	Not Reported As Yet
Acute Hemolytic, Anemia*	Common Especially in Negroes Generally 1st to 5th Day	Uncommon Generally 1st to 5th Day	Reported as Occuring
Purpura Hemorrhagia**	Rare	Day	Not Reported As Yet
Injection of Solerae* and Conjunctivae	Not Reported	Rare Not reported	Common Especially in Conjunction with Rash and Fever 5th to 9th Day
Visual Disturbances	Rare	Rare	Not Reported As Yet
Jaundice**	With Hepatitis or Acute Hemolytic Anemia	Rare	Not Reported As Yet
Painful Joints*	Reported	Not Reported	Reported with Rash, etc.
Stomatitis*	Rare	Not Reported	Not Reported
Gastro-Intestinal Tract	Bleeding Rare Diarrhoea Uncommon	Bleeding Reported	Not Reported

*Best to Stop Drug and Force Fluids.

**Imperative to Stop Drug and Force Fluids.

Taken from a lecture by Dr. Perrin Long, delivered in Washington, D. C. May 15th 1940, before Pan American Scientific Congress.

TABLE IV.

DISEASE	1	2	3	DISEASE	1	2	3
Hem. Strept. Inf.	****			Non-Hem. Strept. Inf.	0		0
Tonsillitis	****			Anaerobic Strept. Inf.			
Pharyngitis	****			Pneumococcal Inf.			
Peritonsillar Abs.	****			Pneumonia			
Ludwigs Angina	****			Peritonitis			
Acute Sinusitis	****			Otitis Media			
Otitis Media	****			Mastoiditis			
Mastoiditis	****			Sinusitis			
Meningitis	****			Meningococcal Inf.			
Erysipelas	****			Brucella Inf.			
Scarlet Fever	****			Gonococcal Inf.			
Cellulitis	****			Male Gonorrhea			
Pneumonia	****			Female Gonorrhea			
Empyema	****			Vulvo Vaginitis			
Peritonitis	****			Arthritis			
Puerperal	****			Endocarditis			
Septicemia	****			Ophthalmia			
Osteomyelitis	****			Staphylococcal Inf.			
Ulcers	****			Sepsis			
Impetigo	****			Pneumonia			
Miscellaneous	****			Carbuncle			
Viridans Strept. Inf.	****			Meningitis			
Abscess	****			Endocarditis			
Osteomyelitis	****			Osteomyelitis			
Tooth Sockets	****			E. Coli Tissue Inf.			
Endocarditis	****			Chancroid			
Meningitis	****			Typhoid Fever	0	0	0
Septicemia	****			Paratyphoid Fever	0	0	0

1 = Sulfanilamide
2 = Sulfapyridine
3 = Sulfathiazole

**** = Preferred Drug.
** = Active.
* = Slight Activity.

Taken from a lecture by Dr. Ferrin Long, delivered in Washington, D. C. May 15th, 1940, before Pan American Scientific Congress.

SULFAPYRIDINE.

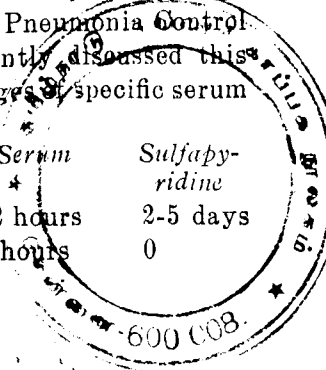
Sulfapyridine, (known abroad as M.&B. 693 and Dagenen) was introduced into medicine in 1938 by Whitley who reported success with it in the treatment of experimental hemolytic streptococcal, meningococcal and pneumococcal infections in mice. A great deal of work has already been done on this drug by numerous workers. The main clinical use and toxic reactions are listed in the accompanying tables 3 and 4 which are taken from a lecture by Long delivered in Washington on May 15, 1940, before the Pan American Scientific Congress. In these tables are also given the toxic manifestations and main clinical uses of sulfanilamide and of sulfathiazole so far as they are known. In comparison with sulfanilamide, sulfapyridine is irregularly and often poorly absorbed. Nausea and vomiting occur frequently and are serious disadvantages. The drug is conjugated to the acetylated form in blood and tissues to a higher degree than is sulfanilamide. The drug is excreted through the kidneys in the free and conjugated form. It is excreted more slowly than sulfanilamide. Gross hematuria with and without signs of renal failure have been noted in patients receiving sulfapyridine. It is thought that the hematuria is associated with the formation of acetylsulfapyridine calculi in the renal lobules and pelvis. A direct toxic effect on the kidney has not yet been ruled out. It is advisable to administer enough fluids to keep the urine output slightly above normal in patients receiving sulfapyridine, so as to lessen the possible chances of calculus formation.

Adequate dosage standards have not as yet been agreed upon by investigators who have used this drug in the treatment of pneumonic and other infections. It is suggested that adults suffering from pneumonia be given 4 grams as an initial dose, followed by 1 gram every four hours, which is continued for 24 to 48 hours after the temperature has fallen to normal. Thereafter the dose could be reduced to 0.5 gram every four hours for 24 to 48 hours longer. If the drug is stopped too soon recurrence of the pneumonic process may occur. Blood levels of 4 mgm or more of free sulfapyridine for each 100 cc of blood seem to be necessary for prompt therapeutic response.

In this connection mention should be made of the relation of special sera to sulfapyridine in the treatment of pneumonia. Dr. O. Robertson, Professor of Medicine in the University of Chicago and Chairman of the Advisory Council of the Pneumonia Control Commission for the State of Illinois, has recently discussed this problem. He pointed out the respective advantages of specific serum and sulfapyridine therapy as follows:

- | | | |
|--|------------|----------|
| 1. Termination of disease in early cases : | 6-12 hours | 2-5 days |
| 2. Time of physician required for administration | 2-4 hours | 0 |

Serum
Sulfapyridine



3. Discomfort of patient during treatment	very little	may be marked
4. Toxic reactions	slight	may be marked
5. Fatalities from treatment	O (with conc. + rabbit serum)	(though rare)
6. Serum sickness	15%	0
7. Occurrence of complications	rare in early cases	?
8. Period of hospitalization	10 days+	10 days+
9. Results of therapy on mortality	5-8%	5-8%

Dr. Robertson indicated the limitations to the treatment of pneumococcal pneumonia with:

(A) <i>Specific Antipneum. Serum</i>	(B) <i>Sulfapyridine</i>
1. Serum sensitiveness	1. Patients with damaged kidneys
2. Asthmatics	2. Patients with liver disease.
3. Very small children	3. History of previous sulfapyridine therapy with reaction.
4. Severe Cardiovascular disease	4. Development of toxic reactions during treatment
5. Type III pneumococcus infection	
6. Types for which serum not immediately available	Serum can be given in all these conditions.
7. Pneumococci which do not fall into 32 types	Sulfapyridine can be used in all these conditions.

He also gave the indications for combined Serum and Chemotherapy in Pneumococcus Pneumonia as follows:

(A) Full dosage of both serum and sulfapyridine when.

1. Treatment is begun after the 4th day.
2. There are three or more lobes involved.
3. The patient is 45 years of age or over.
4. The patient is pregnant or in the first week of puerperium.
5. The blood culture is positive.
6. The pneumonia is complicated by heart disease or other serious chronic disease.
7. In any case if there has been no response to sulfapyridine after 18 hours of therapy.
8. The pneumonia is caused by pneumococcus type III.

(B) Half dose of serum and full dose of sulfapyridine in uncomplicated cases of pneumonia in young adults with limited pulmonary involvement without bacteremia.

Thus far it would seem as if the results obtained in the treatment of pneumonia with sulfapyridine are some what better than with specific serum or with the combined drug and serum therapy. In a recent analysis of 3000 cases treated under the pneumonial

control program of the State of Illinois the mortality with serum treatment alone in 170 patients was 14.7 per cent., with sulfapyridine in 1,157 patients mortality only 3.8 per cent., while in 1,464 cases on combined drug and serum the mortality was 10.9 per cent. In 41 patients with type I pneumococcus pneumonia the mortality was 4.8 per cent., while in 117 similar cases treated with sulfapyridine the mortality was 3.3 per cent. These figures must be accepted as tentative, and should be analysed further to be sure that all the factors concerned have been properly adjudicated.

SULFATHIAZOLE.

Sulfathiazole the newest of this group of drugs with promise of therapeutic value is still in the experimental stages. A preliminary report has recently been issued by the Council on Pharmacy and Chemistry under the name of Dr. Long. While it is still too early to make any too definite statements what is said should be regarded as preliminary in nature. It produces little or no vomiting or nausea—a decided advantage. It is more readily absorbed from the gastrointestinal tract than is sulfapyridine and in this respect it approaches sulfanilamide. In the blood the ratio of free and conjugated fractions of the drug also resemble that of sulfanilamide. It is more rapidly excreted through the kidneys, and in the urine much less of the drug exists in the acetylated form than is the case with sulfapyridine. Mild hematuria has been noted in several patients. It may be expected that sulfathiazole will produce essentially similar toxic manifestations observed following the use of sulfanilamide and sulfapyridine.

From the view of rapidly curing pneumonia sulfathiazole is not as effective as sulfapyridine. The possibility of peripheral neuritis as a toxic manifestation must be considered. Sulfathiazole is definitely superior to sulfanilamide or sulfapyridine in the therapy of staphylococcal infections and of tissue infections produced by the colon bacillus. In urinary tract infections caused by these organisms, not enough information is available at the present time to say anything about it. The drug is active in gonococcal infections. It does not seem to pass readily over into the spinal fluid. In over 200 cases seen by Long and associates, 20 per cent. major toxic reactions including fever, rash conjunctivitis and scleritis hematuria anuria and also several instances of granulocytopenia have been seen. As far as is known sulfathiazole does not produce peripheral neuritis.

In conclusion, I should like to stress once again that we are only at the beginning of what promises to be a most fruitful period in chemotherapy. Many new compounds will appear in rapid succession, and it will require the close co-operation of physicians and research workers to ensure steady progress. Undue enthusiasm and abuse of these, potent drugs will do more harm than good, and may be even costly in the loss of human lives.

(Illinois Medical Journal, Nov. 1940.)

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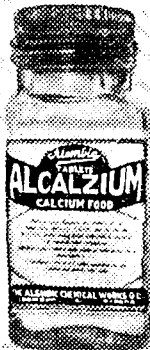
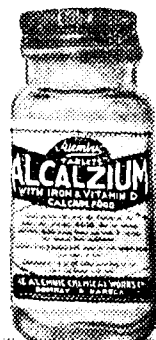
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MEDICO-SURGICAL SUGGESTIONS

VOL. X

FEBRUARY 1941

NO 2

THE HONORARY MEDICAL SERVICE.

The honorary medical system as worked in the Madras Presidency is a topsy turvy caricature of the system obtaining in England. In England the chief men are honorary, with an extensive private practice, and they work in the hospitals in an honorary capacity. The juniors, such as the assistant medical officers, house surgeons and clinical assistants are paid a decent living wage and get a thorough training. In India it is quite the other way. The chief physician or surgeon is a highly paid individual, with all the facilities of a Government hospital at his command with almost unlimited opportunities. To further augment his high pay by indulging in private practice, even at the sacrifice of his official duties. The junior men, consisting of recent products of the medical colleges, have to work as clinical assistants and house surgeons, in an honorary capacity. In some instances the house surgeons are made to work almost the whole day for a niggardly pittance of twenty or twenty five rupees per month, and they are definitely prohibited from having any private practice. Look at this picture and that!

The honorary system was ushered in several years ago as a very great advance in imparting medical relief in this country. It was claimed that it increases the number of medical men available to look after the suffering public, that it saves the government a lot of money, and that at the same time it gives opportunities for young medical men to gain more experience in practising their profession by associating them in state hospitals. But in actual practice these honorary men have been acting as fifth wheels and supernumeraries, and have been looked upon by the service men as intruders. No perceptible saving of money has resulted. On the other hand new jobs on fatter salaries have been created.

Many young medical men have been lured into the honorary system. They have probably spent the best part of their life imbibing the costly medical education, and after graduation what do they

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get? Honorary service. After a strenuous career in college and after the expenditure of an enormous sum of money he is expected to work for nothing. We have not heard of honorary work in any other profession, but a medical man is expected to do honorary work. We have not heard of an honorary judge delivering judgments in the high court. We have not heard of an honorary police officer looking after law and order in any country. We have not heard of honorary soldiers fighting their adversaries. We have not heard of honorary professors of chemistry or history lecturing to college students. We have not heard of honorary work in any other profession, but the medical man is exploited and asked to work honorarily. Other professions are able to eat the fruits of their labour but the medical man has only to see them eating those fruits and be satisfied. The medical man has to submit to exploitation, get more experience, do honorary work, and—go hungry. What a prospect in this twentieth century for the noblest of professions!

There is a group of individuals who vociferously and publicly proclaim the virtues of the honorary system, and quietly get into any paid job that comes along. That shows how the natural wind blows. The call of the stomach is no less important and imperative than the call to service. It is an incongruity to have a highly paid service and an honorary service working side by side and competing with each other on unequal terms. The state looks after and finances several beneficial services for the good of the people, and it is but right that it should look after the medical requirements of the public through the medium of an efficient and contented band of medical men rendering the best service and getting their due recompense for such service.

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A NEW METHOD OF TREATING LEUCODERMA.*

BY D. PANJA, M.B., (CAL.) AND P. A. MAPLESTONE, D.S.O. D.SC.,
M.B., B.S., D.T.M.

(From the Medical Mycology Inquiry, under the Indian Research Fund Association, School of Tropical Medicine, Calcutta.)

The successful treatment of leucoderma is, for two reasons at least, an important problem for dermatologists and particularly for those who have to treat dark-skinned races, first because the disfigurement is much more apparent than in those with fair skins, and second because of two widespread but erroneous belief that is a variety of leprosy and that a victim of the disease accordingly must be shunned by his fellows for fear of infection.

A great deal of work has been done on skin pigmentation, but the full understanding of the complicated chemical changes, which are dependent on endocrine functions, is far from being fully established, so treatment of leucoderma is mainly empirical and is rarely attended by satisfactory results.

Many years of experience of treating hundreds of cases at the Calcutta School of Tropical Medicine has led us to place our faith in external application of the oil extracted from the seeds of *Psoralea corylifolia* and known as *bavchi* oil, together with internal administration of intestinal antiseptics, but although persistence in this form of treatment is occasionally rewarded with success, partial or total failure has much more often been the final result. Recently, we have carried out a number of experimental treatments in which the oil has been injected intradermally instead of being rubbed on the skin, and our results have been so good that this short note is being published to draw the attention of other workers to this method. It is felt, however, that there is still much room for improvement in our technique, and this is only in the nature of a preliminary report.

The oil so far employed for injection is exactly the same as that used for external application.

Method of treatment.—The patch to be injected is carefully cleaned with alcohol, and the oil is injected intradermally using an ordinary hypodermic syringe fitted with a fine needle. The amount of oil in each individual injection is a single drop (between 0.05 and 0.1 c. cm.) The number of injections varies with the size of the depigmental patch, small spots of 1 m. or so in diameter only needing a single injection in the centre. In larger patches the injections are spaced about 1 cm. or a little more apart, until the whole area is covered. In two to three weeks, formation of pigment can be noticed, beginning at the

*Reproduced from I. M. G. February, 1940.

site of the needle puncture from which point it spreads centrifugally. If when deposition of pigment ceases, the contiguous areas of new pigmentation have failed to coalesce, a second and if necessary a third course of injections can be given in the intermediate white patches, untill the whole area is normally pigmented once more. If the patch to be treated is several inches in diameter, and in consequence needs numerous injections to cover it completely, it has been found better to give a few injections fairly wide apart in the first instance and to follow with intermediate injections a few days later, than to try and cover the whole area at one sitting. The reasons for this modification are that injections are followed by a good deal of pain and if many are given at one time the oil is sometimes followed by a small focal ulcer which remains small if the injections are wide apart, but, if several of these ulcers were to occur in adjoining injection sites, they might coalesce and form a relatively large lesion. It should be noted, that apart from the extra discomfort at the time and the subsequent scarring, ulceration has no ill effects on the final result as the scar always becomes pigmented.

The precautions outlined above in the treatment of large depigmented patches indicate the disadvantages of this form of treatment in its present form, and we are now engaged upon trying to lessen or eliminate the pain and the tendency to ulceration, by variations in the method of injection and the addition of different substances to the oil.

TREATMENT OF SCORPION STING

BY P. KRISHNA RAO, B. SC., M.B., B.S., D.T.M., & H. (Eng.)

*Pathologist, Victoria Hospital, and Lecturer in Pathology,
University Medical School, Bangalore.*

The usual signs and symptoms are intense burning sensation at the time and the place of sting and gradual spread of pain along the course of the vein, or lymphatics. In some cases, the shock is so great that the patient perspires immensely, shows all signs of collapse and may even die.

Scorpion poison was found to be acid in reaction and it was possible to render it neutral by the addition of an alkali in proper proportion. The poison from the stings of various types of scorpions was collected in a sterilized watch glass and diluted with a small quantity of distilled water. Half the quantity of the poison was then poured out, into another watch glass and mixed with a 2 per cent. sodium bicarbonate solution to render the mixture neutral to

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From the above and other experiments it was inferred that the poisonous effect of scorpion sting is due, in all probability, to some material which is acid in reaction and could be rendered neutral and innocuous by the addition of an alkali. An alkaline substance which is not harmful to the human body while injected, is all that is required, to cure the toxic effects of the scorpion sting. Sodium bicarbonate has been selected on account of its being very handy and not injurious to the human body in any way.

Eighty-four persons stung by scorpions were treated by the author by this method with great success.

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